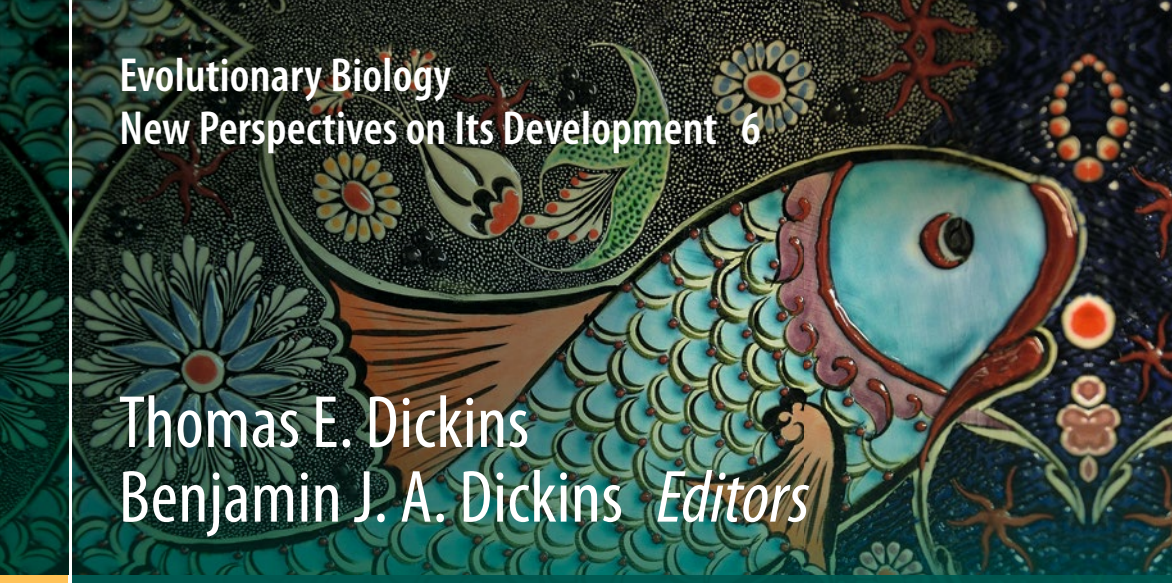

Evolutionary Biology
New Perspectives on Its Development 6

Thomas E. Dickins
Benjamin J. A. Dickins *Editors*

Evolutionary Biology: Contemporary and Historical Reflections Upon Core Theory

Springer

Evolutionary Biology – New Perspectives on Its Development

Volume 6

Evolutionary biology has been a remarkably dynamic area since its foundation. Its true complexity, however, has been concealed in the last 50 years under an assumed opposition between the "Extended Evolutionary Synthesis" and an "Alternative to the Evolutionary Synthesis". This multidisciplinary book series aims to move beyond the notion that the development of evolutionary biology is structured around a lasting tension between a Darwinian tradition and a non-Darwinian tradition, once dominated by categories like Darwinian Revolution, Eclipse of Darwinism, Evolutionary Synthesis, and Post-Synthetic Developments.

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* * *

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To Anne and David for their endless parental investment.
To Nicola, Jack, and Isabella for continued kinship.

Preface

The current volume is a contribution to the Springer series *Evolutionary Biology – New Perspectives on its Development*, edited by Richard Delisle. The aim of this series is to reflect the dynamic nature of evolutionary biology from its foundation to the present day, while analyzing it with fresh eyes. This dynamism is a product of theoretical development and empirical discovery, and it is not always the case that both have moved in step with one another across the years. Moreover, evolutionary biology is no stranger to forthright disagreement, prolonged debate, and resolution.

Perhaps the most pervasive historical narrative in evolutionary biology concerns the *Modern Synthesis*. The Modern Synthesis is variously treated as a period of significant empirical development, as a deliberate attempt to unify much of biology, and as a specific set of theoretical commitments. Over the last twenty years, the term, *the Modern Synthesis*, has been deployed to represent a static and conceptually inadequate theory in want of updating considering new findings within areas such as evolutionary developmental biology. Most commonly, this position is associated with those who seek to create an *Extended Evolutionary Synthesis*, by either drawing in new findings or through more radical re-engineering of the conceptual architecture of evolutionary theory.

The Extended Evolutionary Synthesis amounts to a set of theoretical claims, grounded in a version of the history of evolutionary biology. In this volume, our ambition was to draw together both theoretical and historical analyses of these claims to ground them in their past but also to understand their potential reach. The book contains chapters making direct comment on the uses of history in this recent debate and chapters that question previous historical assumptions and categorizations. There are also chapters that philosophically analyze the central arguments between those advocating for an extension and those who see no requirement for a new theory. And there is one chapter that presents a case study of post-Modern Synthesis discovery and theoretical development. Our authors are historians, philosophers, and scientists. Many biological phenomena are discussed, and the dynamism of evolutionary biology is well represented across thirteen central chapters, commentaries, and replies. You will find here scholarship across a broad

intellectual landscape and our hope is that this volume presents a balanced contribution that will prove useful to those interested by or engaged in current debates in evolutionary biology.

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Benjamin J. A. Dickins

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Introduction

1

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Abstract

In this chapter, we make some general comments about the nature of scientific explanation and use them to contextualize recent debates within evolutionary biology about the adequacy of what is sometimes termed standard evolutionary theory. These comments serve to introduce the aims of the book and we then summarize the chapters to follow, relating them to the opening themes.

Keywords

Modern synthesis · Extended evolutionary synthesis · Idealization · Abstraction

1.1 Introduction

This book is about the debate between advocates of what has been called an Extended Evolutionary Synthesis and the defenders of the Modern Synthesis, sometimes also referred to as the standard theory of evolution. We will come to discuss the broad parameters of this debate shortly, but first, we wish to develop a context within which to place this debate.

The history of evolutionary theory is one of argument. Gradualist biometricians were locked in a dispute with saltationists about Darwinian gradualism in the late

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nineteenth and early twentieth century (Pence 2022). Weismann's distinction between somatic and germ lines conflicted with those Darwinians who saw a role, following Darwin, for Lamarckian process in evolution (Romanes 1888). The twentieth-century success of population genetics was at times challenged for its apparently simplistic assumptions about genetic effects upon the phenotype (Rao and Nanjundiah 2011). In the mid-twentieth century, the perceived emphasis upon natural selection, gradualism, and adaptationism in contemporary evolutionary theory led to robust opposition (Eldredge and Gould 1972; Gould and Lewontin 1979).

The preceding list is a gloss and undoubtedly misses many other discussions and disagreements through the 170 years of evolutionary thought. It is not our intention to survey them in this Introduction. Instead, we wish to make some general comments about disagreement before moving on to outline the content of this book and what the reader should expect.

Recent scholarship in philosophy of science has focused upon understanding as an epistemic aim that is distinct from explanation (Elgin 2007; de Regt 2017; Potochnik 2020). Following de Regt, we can claim to understand a phenomenon when we are in receipt of an explanation, however for that explanation to work we also need to understand the theory from which it is derived. In this case, understanding is a pragmatic skill evidenced by the correct deployment of theory (de Regt 2015). Under this schema, to understand a particular phenomenon is a macro-level ambition, to be contrasted with lower-level theoretical understanding. As a result of this relational architecture, theory can be seen to be applied in a variety of circumstances with different explanatory aims, and different understandings. Potentially each macro-level activity yields new variation in the pragmatic deployment of theory.

For phenomena to be understood theories need to be intelligible, which means they need to fit with human cognitive capacities and intelligibility should not be regarded as an intrinsic property of the theory but rather as a relational property (see also Potochnik 2017). De Regt (2017) defines intelligibility as "the value that scientists attribute to the cluster of qualities of a theory (in one or more of its representations) that facilitate the use of the theory" (p. 40). De Regt gives a brief history of radical behaviorism as an example of developing intelligibility. The initial aim of radical behaviorists was prediction and control, a positivist ambition that sought to uncover nomic, mathematically described relations between stimuli and behavior. The quest for such laws failed, according to de Regt, because the mathematics were largely unintelligible in the absence of a theoretical structure. This problem was directly addressed by a new strategy of hypothesizing unobservable, intervening constructs to explain stimulus-response relations. This was dubbed mediational neo-behaviorism (Moore 2013). These mediating variables provided theoretical intelligibility through a functional explanatory framework which in turn enabled successful prediction. In de Regt's terms, the mediational move was the development of a micro-level theory that could be deployed to understand specific phenomena. That understanding was of a functional and predictive kind.

From the preceding views, de Regt draws out the necessary and sufficient conditions for scientific understanding. First is the *Criterion for Understanding*

Phenomena, which states that a phenomenon is understood if and only if it has an adequate explanation based on an intelligible theory. Second, an intelligible theory must "conform to the basic epistemic values of empirical adequacy and internal consistency" (2015: 92).

The second clause is important. De Regt notes that astrologers, for example, may well feel that between them they have a series of intelligible explanations for the phenomena they are interested in. However, it is widely agreed beyond astrology that their theories fail on grounds of both empirical adequacy and internal consistency. To this end, astrology cannot claim to have delivered a scientific explanation.

Potochnik has recently discussed the role of idealization in science, claiming it to be a method for intelligibility in line with de Regt's view (Potochnik 2020). She is focused upon causal explanations, and how to deal with the huge complexity imposed by the world in an intelligible manner.

> (F)aced with the need to grapple with this complex world, scientists face cognitive, and other, limitations. These limitations make it difficult to secure causal knowledge, to make accurate predictions, and to pursue science's other aims in this causally complex world of ours. Or, perhaps better, this point can be phrased positively: simple patterns are cognitively valuable. Simple patterns support human influence on and understanding of our world. There is thus a basic mismatch between the cognitive value of simple patterns and the world's complexity... So, in the face of this mismatch, we often resort to lying a little bit: we artificially simplify the parts of accounts that we are not interested in to improve our access in a variety of ways to the parts we are interested in. This is one service that idealizations provide. (Potochnik 2020: 934–935)

Idealizations are false assumptions, or assumptions made without regard to their veracity. This is a deliberate strategy and is not to be confused with the empirical refutation of a model. In this case, the model is tested and found to be false, but it was not deliberately rendered false. An example of an idealization would be the assumption of a frictionless plane when measuring the velocity of an object sliding down a ramp, or that of an infinite, panmictic population in population genetics. Both idealizations make mathematics more straightforward, more applicable, and thus more intelligible. Potochnik discusses idealizations deployed to represent causal patterns:

> Causal patterns are patterns insofar as they are regularities that are limited in scope and that may permit exceptions. The ideal gas law characterizes the approximate behavior of most gases, although its predicted relationships break down at low temperatures and at high pressures. It also ignores molecular size and intermolecular forces. Recall the idealization of an ideal gas composed of noninteracting point particles; this idealization achieves that neglect. Accordingly, even within its scope of application, the ideal gas law has exceptions... (T)o represent a causal pattern is to show how changes to a system would, over some range of circumstances, precipitate changes in other feature(s) of the system. The ideal gas law shows, for example, how temperature increasing in a sealed container of gas with a fixed volume increases the pressure. Mastery of causal patterns is exactly the kind of thing that beings who prize simplicity need in order to operate in and grapple with a causally complex world like ours. (Potochnik 2020: 935)

When idealizing, truth can be sacrificed to increase intelligibility and to gain understanding of causality. Idealizing is not a factive pursuit, it is a macro-level aid to understanding. But, as de Regt makes clear, any underlying theory needs to be empirically adequate and internally consistent. There is an expectation that empirical facts will be gathered to support the theory, and in turn to justify the intelligibility strategy of idealization.

There is a subtlety to Potochnik's view. Her claim is that the causal patterns invoked by an idealization must be embodied by the system in question. But this does not preclude many other causal patterns being embodied within the same system. The introduction of falsehoods to create an idealization assists the scientist in pragmatically grasping the causal pattern, enabling its use as a tool for understanding. Not only must the tool map to embodied phenomena, but, in line with de Regt's view, it must also map to the cognitive agent, the scientist. This makes scientific understanding a human product, and one that can be directed to many different aims.

> Suppose the temperature increase in a sealed container of fixed volume was in fact a can of aerosol hair spray left in a car on a hot day. This phenomenon embodies the pattern described in the ideal gas law. It also embodies the pattern of the greenhouse effect: the short wavelengths of visible light can enter through the glass of the closed windows, but the longer wavelengths of infrared light radiated by the objects in the car that absorbed the light cannot exit through the glass as easily. These patterns relate to different aspects of the phenomenon, and which is of interest depends on which aspects we are focused on. As these simple examples show, different patterns embodied by some phenomenon may be closely related to one another or wholly unrelated (or anywhere in between). (Potochnik 2020: 936)

Potochnik's view, that there are many different projects within science each of which may require its own idealizations, its own methods of rendering intelligibility, has led her to develop a view on disagreements in science (Potochnik 2013). She notes that some disagreements in biology have been or are treated as ideological debates. Among her examples is the clash between the anti-adaptationists (Gould and Lewontin 1979) and those defending adaptationism as an optimization approach. She notes how Gould and Lewontin's original paper invoked religious language to characterize adaptationists, clearly labeling this approach as an ideology. Furthermore, Potochnik points to defenders of optimization who openly reference it as a *world view* or *leap of faith*.

> Either you subscribe to the Optimization Research Program as your worldview, or you reject it... These positions are presented as ideological in the sense that they involve adherence to a systematic set of ideas, a comprehensive way of looking at things. The set of ideas in question is viewed as fundamental to the domain under investigation, and adherence to one side or the other is taken to be a total commitment. This ideological tenor therefore suggests that there is a rift in theory, that there is dispute regarding the basic understanding of these types of phenomena. (Potochnik 2013: 119)

Potochnik argues that we can fruitfully move away from much ideological grandstanding by taking what she refers to as a methodological approach. By this,

she means that we ought to characterize science as model based, and those models as idealizations in the manner discussed above. To this end, models adopted by groups of scientists will contain deliberate falsehoods to facilitate intelligibility under specific task demands. Where interests, or focal phenomena diverge, we perhaps should expect to find different idealizations in play, and it is very easy to present differences in idealization as distinctions in ideology. One reason for this, Potochnik claims, is that scientists often commit to "simple causal processes with broad domains of application" (Potochnik 2013: 121). Yet, we should really agree that phenomena are the result of multiple, complex causal pathways and that a focus on a particular route through such a tangle is a commitment to producing an intelligible explanation. In this sense, *a* causal model is privileged above others, but that privilege should only be seen as a pragmatic expedient, not an ontological commitment. It is therefore to be seen as methodological because it is a method for providing a workable explanation within a specific domain of enquiry.

1.2 Extending the Modern Synthesis

In 2014 *Nature* carried a debate between two groups of scholars (Laland et al. 2014). The question under discussion was "does evolutionary theory need a rethink?" One group argued that yes it did, and urgently. The second that no it did not, and that all was well.

The advocates made a core claim that mainstream evolutionary theory focused almost entirely on gene-level explanations, a criticism aligned with the term *gene-centrism* which captures the concept of privileging the gene in evolutionary explanations. Their counter was that developmental processes should be recognized as contributory factors in evolution. This idea is at the heart of the Extended Evolutionary Synthesis (EES) movement and was most clearly expressed by Pigliucci when he called for the unification of theories of genes with those of form (Pigliucci 2007). In this way, the extended synthesis effectively promotes the introduction of mechanistic theories of form into evolutionary theory.

> We hold that organisms are constructed in development, not simply "programmed" to develop by genes. Living things do not evolve to fit into pre-existing environments, but co-construct and coevolve with their environments, in the process changing the structure of ecosystems.
>
> The number of biologists calling for change in how evolution is conceptualized is growing rapidly. Strong support comes from allied disciplines, particularly developmental biology, but also genomics, epigenetics, ecology, and social science. We contend that evolutionary biology needs revision if it is to benefit fully from these other disciplines. The data supporting our position gets stronger every day.
>
> Yet the mere mention of the EES often evokes an emotional, even hostile, reaction among evolutionary biologists. Too often, vital discussions descend into acrimony, with accusations of muddle or misrepresentation. Perhaps haunted by the spectre of intelligent design, evolutionary biologists wish to show a united front to those hostile to science. Some might fear that they will receive less funding and recognition if outsiders—such as physiologists or developmental biologists—flood into their field.

> However, another factor is more important: many conventional evolutionary biologists study the processes that we claim are neglected, but they comprehend them very differently. This is no storm in an academic tearoom, it is a struggle for the very soul of the discipline. (Laland et al. 2014: 162)

While the above opening statement references a specific scientific idea, that developmental processes should be considered within evolutionary theory, the language deployed is distinctly ideological (Potochnik 2013). Some of those opposed to the EES are allocated emotional responses, those advocating for the EES are battling for the fundamental essence of the discipline. By implication there can only be one winner, there is no room for coexistence.

The advocates for a rethink establish a distinction between the Modern Synthesis, which they see as the period in which population genetics emerged, and Standard Evolutionary Theory (SET). It is not entirely clear what the status of SET is, but the authors claim that it incorporates much of the Modern Synthesis. Thus, SET sees new random variation established by genetic mutation, and natural selection as the sole source of adaptation. In making these claims the advocates side neatly with Gould's view of the hardening of the Modern Synthesis into a panadaptationist, gene-level theory (Gould 2002; Dickins 2021).

> In our view, this "gene-centric" focus fails to capture the full gamut of processes that direct evolution. Missing pieces include how physical development influences the generation of variation (developmental bias); how the environment directly shapes organisms' traits (plasticity); how organisms modify environments (niche construction); and how organisms transmit more than genes across generations (extra-genetic inheritance). For SET, these phenomena are just outcomes of evolution. For the EES, they are also causes. (Laland et al. 2014, 162)

Here we see an explicit claim about the introduction of other causes beyond mutation and selection. By implication, the idea is that those pursuing SET and those pursuing the EES have the same broad target in mind—evolutionary explanation—but the EES is seeking to proliferate causes under a development conception of evolution in which those processes, while possibly the outcome of evolution can also affect subsequent evolution. Put another way, there is an implicit assumption that both groups of scholars share the same explanatory task. The EES claim is that SET is causally inadequate to that task because they miss detail. Thus, causal explanation is the focus in this debate.

The advocates move to an example:

> (C)ichlid fishes in Lake Malawi are more closely related to other cichlids in Lake Malawi than to those in Lake Tanganyika, but species in both lakes have strikingly similar body shapes. In each case, some fish have large fleshy lips, others protruding foreheads, and still others short, robust lower jaws.
>
> SET explains such parallels as convergent evolution: similar environmental conditions select for random genetic variation with equivalent results. This account requires extraordinary coincidence to explain the multiple parallel forms that evolved independently in each lake. A more succinct hypothesis is that developmental bias and natural selection work together. Rather than selection being free to traverse across any physical possibility, it is

> guided along specific routes opened up by the processes of development. (Laland et al. 2014: 162)

In this quotation we see a particular causal strategy in play. The authors claim that a SET account of morphological similarity across cichlid species challenges credulity, as it appears to rely on multiple instances of the same mutation occurring and being selected. Introducing developmental path dependency as a further cause is argued to reduce search space for natural selection and render the model credible. Crucially the EES claim is not that there is no role for selection, but rather that selection is assisted by a narrowing of the parameters over which it must search. Theories of this kind often rely on modular models of development (Brakefield 2006, 2011; Kirschner and Gerhart 2010; Newman 2010) which reduce lethality effects associated with mutation by reducing the number of genetic mutations required to enact a change in morphology and enhance evolvability. Where selection does operate, in these models, is over regulatory genes that might change where in time a developmental module is activated, or might enhance the outcome of developmental modular processes etc. (Dickins 2021).

The cichlid example is instructive. The first thing we might note, in keeping with Potochnik's clear view that our causal world is complex (Potochnik 2020), is that it is very unlikely that either the SET or the EES approach will tell us the whole story about the evolution of cichlid morphology and its ecological distribution. We should accept that both accounts are idealizing. For example, a SET account might adopt the idealization of single locus selection to mount an optimization model of cichlid adaptations. In doing this no account of development processes would be made, but development would be assumed. A part of that assumption would be that to all intents and purposes the variation resulting from development was insignificant to the adaptationist generalizations sought. Meanwhile, the EES account would highlight developmental variation and make a case for the mechanisms of development enabling more effective selection. But that selection may be modeled in single locus terms again, with a focus upon regulatory genes. Moreover, the precise mechanistic account of development is unlikely to capture all developmental causes, and further idealizations will be introduced at some point. For example, the idea of developmental modules is most likely an idealization designed to neatly capture some dependencies in development. How encapsulated and domain specific such dependencies are becomes a matter of empirical interest in each and every case.

The intuition we are seeking to prime is that whilst both the SET and EES approaches, in this case, deploy idealizations it is not clear that the idealizations are in contradiction to one another. Indeed, we might claim that the SET approach simply assumes development, while the EES incorporates a version of it, and in that way SET is more abstracted than EES, where abstraction is a process of reducing detail to gain generality (Levy 2021). Moreover, the less abstracted EES account is rendered this way simply because its project is different from that of SET. We might say that the EES project is to reduce abstraction in evolutionary biology by introducing proximate developmental mechanisms to show their effect upon evolutionary dynamics and trajectories.

Table 1.1 Brown's (2022) three types of adaptationism and structuralism summarized. In both positions, the commitment becomes softer from empirical to methodological versions. The former contains an ontological claim while the latter amounts to a set of guiding principles to do science and generate findings

	Adaptationism	Structuralism
Empirical	Selection is powerful, has causal primacy, and can be used to explain and predict evolutionary outcomes	Developmental constraint has causal primacy and can be used to explain and predict evolutionary outcomes
Explanatory	Selection has unique explanatory importance as it can address apparent design	Developmental constraint can answer the important questions of diversity, disparity, and complexity
Methodological	Adaptation is a good initial hypothesis for scientists enabling subsequent work	Scientists should look at disparity and diversity and developmental constraint is a good initial hypothesis

A sticking point for this intuition is that the advocates have suggested that the SET account of cichlids requires an extraordinary coincidence. In doing this they are suggesting it is a false account. Initially, this might appear devastating to our argument about abstractness. If the SET account is wrong, then its abstractions are false and unrelated to any true account. However, the EES invocation of development to reduce the search space for selection does not in fact do away with this problem. It is still present in that account also. In the version we have presented a mutation (presumably in a regulatory gene) is posited, and it will be selected because it is attached to a highly conserved developmental program that is well insulated against internal disruption by mutation. That regulatory mutation must have happened more than once. What the advocates are really arguing against is the idea that the entire developmental suite was re-engineered by mutation and selection on two separate occasions, which would be a non-parsimonious claim, if made. As the SET account holds development constant this is in fact not a necessary commitment for that position. The advocates thus fill in the detail of how that constancy might be delivered and in so doing render a less abstract explanation, but no less idealized.

Recently, a related argument has been made. The claim is that much of the debate between SET and EES turns on a distinction between structuralism and adaptationism, which are seen as two separable scientific projects in their own right (Brown 2022). Brown claims that both structuralism and adaptationism are attempts at understanding "phenotypic diversification and the mechanisms that generate it" (2022: 2). Structuralism is concerned with developmental constraints, bias, and innovation as causes, while adaptationism is concerned with adaptation and natural selection, approaching this in diverse ways from population genetics to behavioral ecology. Brown's innovation is to separate three different kinds of adaptationism and structuralism: empirical, explanatory, and methodological (Table 1.1).

Empirical commitments to adaptationism or structuralism amount to an ontological claim for causal primacy in evolutionary explanations. This is to be contrasted with the softer explanatory approaches which begin to make explicit claims about

the explanatory targets and then point to specific frameworks as more useful. To this end, adaptationism is focused upon apparent design while structuralism focuses upon the diversity, disparity, and complexity of phenotypes. Finally, methodological approaches are more pragmatic affairs, in which explanatory targets differ but the framework adopted is merely seen as the best starting point for hypothesis generation and test. However, Brown notes that while methodological adaptationism seems to be a straightforward commitment for most adaptationists, due simply to the great success of this approach, EES advocates claim that in recent years the amount of new developmental data calls this assumption into question. Brown suggests that the argument is that adaptationism as a method is now eclipsed by the availability of structuralist data. She further notes that structuralists can still see adaptation as an explanatory target, but that their shift is away from the pure externalism of selection toward a more interactive, or constructivist model of building those traits. In this way, developmental causes at a minimum have explanatory parity with selection.

We think Brown has the EES claim right here, but we would note that this still amounts to a shift of explanatory focus as described above. If the overall aim is to explain phenotypic diversification and the mechanisms that produce it, then we can see these two approaches as an effort toward that. However, the devil is in the detail. For an adaptationist, the question of phenotypic diversification is one of what is selected, what is retained in the population, and why. Selection is the mechanism, and as inheritance is required, genetic variation is the source of novelty under constancy assumptions about development (as above). For the structuralist, the question of phenotypic diversity is a question of the multiple causes of variation in the phenotype prior to any selection. This does not mean that they must deny selection, selection is simply not a principal focus. As suggested the structuralist is less abstracted than the adaptationist. But Brown might claim that the developmental constancy of adaptationism is an idealization. We think this is both an idealization and an abstraction because the variation introduced by development is in fact already captured as an idea in SET through the concept of reaction norms. Reaction norms are understood as the available range of phenotypic expression for a genotype, and it is assumed this impacts upon evolutionary dynamics downstream (Stearns 1989). Reaction norms are therefore an available explanation for SET, but they are idealized out of the main account under constancy assumptions (in part because it is assumed selection has operated to design conditional response in phenotypic expression (Nettle and Bateson 2015). If EES advocates were to argue for unbounded phenotypic variation, with no connection to genetic variation, then they would be in want of a new mechanism for inheritance to continue their evolutionary accounts. Of course, some theorists have latched onto epigenetic inheritance as a possible second mechanism but the causal dependency of epigenetic effects upon genes is strong and this claim unsubstantiated (Dickins and Rahman 2012; Futuyma 2017).

We will stop here. Our aim is not to launch into a full analytic argument about key claims on either side of this debate. Instead, we want to show how a range of engagements is possible with this literature. At one pole, there are clear analytic disagreements to be had and this tends to happen as explanatory aims overlap or are entirely in common. At the other, there is the complete separation of explanatory

targets and the emergence of specific strategies to gain understanding. With this view in mind, we turn now to discuss what is to come in this volume.

1.3 This Book

This book aims to survey various aspects of the debate between EES and SET advocates. There is no ambition to be partisan, but rather to inspect key claims and to place them in contemporary and historical contexts. Our authors are drawn from science, and the history and philosophy of science, offering various perspectives on elements of the argument as it has been played out over the last two decades. We have also introduced a discursive element to the book, producing commentaries on chapters, from within the author list, and allowing response. Our hope is to convey the liveliness of debate through this method, but also to expose further lines of thinking beyond the original chapters. In this section, we will survey the main chapters of the book. Each main chapter is grouped with its commentary and reply into an individual part.

In Part 1, Chap. 2, Betty Smocovitis addresses the uses of history in the debates surrounding the Extended Evolutionary Synthesis. She opens by urging caution, there are many pitfalls to deploying historical narrative in support of particular views about present theories. An early example of this is her analysis of the term *Modern Synthesis*. This is what historians label a *trace* because it indicates something about the actor who deployed it. In this instance, that actor was Julian Huxley, and Smocovitis discusses the mid-twentieth century mood to which he was addressed in labeling the emergence of evolutionary biology *the Modern Synthesis*. What this term is not is something derived from secondary analyses by historians. A modern synthesis has not been unearthed as a movement. Rather Huxley's clever coinage, used in a book designed for a wide, if intellectually curious audience, has stuck. This does not mean that our early twenty-first-century mood will unambiguously and correctly interpret the term as it was intended. Smocovitis also discusses the later adoption of *evolutionary synthesis*, by Mayr and others, to firmly focus on the discipline-building activities of evolutionary biologists while controlling the historical narrative. This habit of leading scientists to write their own histories of evolutionary biology was common, and Smocovitis surveys several key contributions revealing different sub-disciplinary emphases, alternative lists of the core architects, and even disagreement about the duration of any synthesis. These observations draw Smocovitis to a critical analysis of how certain key terms are casually deployed in the extended synthesis debate, without care for their origins and conceptual place in the past, sowing confusion and much crosstalk. Her plea is for histories of science to be produced that try to place the work of scientists in their appropriate moment, not just through the interpretation of their scientific publications but also by understanding the zeitgeist of the moment in which the work was done; partial histories are clumsy rhetorical tools (see also Chap. 11). She ends by ably demonstrating the inadequacy of some partisan histories deployed in the recent debates about extension, and relates them to prior arguments in the 1980s, when standard evolutionary

theory was also declared to be in decline. A clear message from Smocovitis, and one important for readers of this book, is that there never has been a single, monolithic theory of evolution. But there has always been disagreement and diversity within evolutionary biology.

Chapter 5 (Part 2), by John Welch, parses the many arguments about creativity and natural selection that have arisen in the literature. As a result, the chapter spans a considerable section of the history of evolutionary thought in order to position key claims about creativity. Welch focuses the chapter on the idea that the theory of natural selection was specifically developed to deal with apparent design in organisms, with the concept of adaptation. This is one source of creativity in biology and does not rule out others, but Welch makes a case for how these different forms of creativity can be related to and separated from one another, in part dependent upon the scientific focus brought to bear. While he does not directly discuss abstraction and idealization, the contemporary views of modeling discussed above are evident within his analysis. He also demonstrates how a tendency to lose ascription in the literature has led to ambiguity and, at times, hyperbole with an inevitable loss of explanatory traction. Ultimately Welch's chapter provides a conceptual structure that supports a form of pluralism, that of need. Under this account, natural selection, and thus the standard evolutionary theory derived during the Modern Synthesis, has a central and organizing role but is not to be considered the sole causal source of trait variation and nor is selection isolated from the dynamics of organismic agency. The explanatory needs of researchers should determine which aspect of the conceptual architecture is most relevant to them, while understanding its place relative to the whole.

In Chap. 8 (Part 3) Jan Baedke and Alejandro Fábregas-Tejeda present a historical and philosophical analysis of the role of the organism in evolutionary biology. This chapter complements that of Welch in that it analyzes the history of the claims about the role of organismic agency in evolution. They outline the emergence of organicism, the non-reductionist, non-vitalist third way position that fell out of favor in the twentieth century. Reasons for this decline are given and include a lack of institutional support as well as specific, reductionist moves in the philosophy of biology developed by Mayr and others during the Modern Synthesis. Baedke and Fábregas-Tejeda then make the case that the Extended Evolutionary Synthesis is advancing a new version of organicism, albeit without explicit reference to their third-way forebears. The EES movement, by granting the organism a role in evolution, looking to reciprocal interactions between organism and environment, and by showing organisms to be agents actively shaping their environments, is introducing the main themes of organicism as outlined by Baedke and Fábregas-Tejeda. But Baedke and Fábregas-Tejeda conclude their chapter with a note of philosophical caution. While the organicists and advocates for an EES have clearly laid out what they see as missing elements in evolutionary biology, those elements need to be drawn into a theory that displays key explanatory virtues which include the proper deployment of abstractions and idealizations. The implication is not that this is an impossible task, but rather that is an essential one, if any form of pluralism

is to be defended. For the authors this is an effort that cuts across debating sides—all need to decide upon their explanatory standards and give reason for them.

In Chap. 11 (Part 4) Erik Svensson, in keeping with the preceding chapters, looks to the historical antecedents of the Extended Evolutionary Synthesis and criticisms of the ideas emerging from the Modern Synthesis. A key point from Svensson is that these criticisms often ignore the developments in evolutionary theory since the historical synthesis period (for example, neutral theory). Central to Svensson's argument is the idea that the Modern Synthesis was a period of intentional synthesis between the subdisciplines of biology, under a shift from a natural historical, organism-focused discipline to a process-based one. While he notes it may not have included all elements of biology, he claims this effort toward synthesis should not be understated. It is this synthetic ambition that continues to this day in evolutionary biology and allows neutralism and other developments to become incorporated. Evolutionary biology is a dynamic discipline and not one in crisis as some critics have suggested. To this end, Svensson sees the Modern Synthesis as the formation of a framework for doing science, not a formal theory, and he makes a case for two broad schools of thought emerging during the synthesis to support this claim: one in the UK and the other in the USA. This leads Svensson to make the stronger claim that the criticisms of the Modern Synthesis are often focused upon an inaccurate presentation of contemporary evolutionary theory based in partial accounts of the past that neglect the inherent pluralism of the synthesis period. Svensson sees evolutionary biology as encompassing several theories that are determined by the focus of the researchers in question, and this again is in keeping with modern views of scientific explanation outlined at the beginning of this chapter. But he also recognizes that empirical evolutionary biology has raced ahead of theory in recent years as many novel findings have accumulated. There is a job to be done here, but his view is that it will not be achieved by misrepresenting the past and calling for reform—instead, the history of evolutionary biology shows a flexible discipline that has consistently integrated and re-engineered itself.

David Haig draws our focus to teleological and teleonomic explanations in Chap. 14 (Part 5). He begins by discussing the odd nature of evolutionary theory when addressing cause and effect. Where we normally hold that an effect cannot precede its cause, evolutionary theory renders this more complex showing how the outcomes of genetic variation (which are effects) can then cause the perpetuation of those genetic variants and the associated traits due to selection. This is the distinction between type and token causation. It is the treatment of cause and effect in biology that is of concern to Haig, but before he directly addresses this issue, he argues for differences in interpretation based on need. While his comments are founded in a manner consistent with Derrida (an unusual move within theoretical biology) his argument is in keeping with the idea that scientific interpretations of facts are derived with a specific explanatory purpose in mind. Following Svensson's comments, Haig sees a tendency for establishing strawmen but on both sides of the debate about an Extended Evolutionary Synthesis, in order to support specific interpretations at the expense of others. Given this, Haig sets to analyzing, and re-interpreting the work of Laland and colleagues on reciprocal causation and related matters, with a mind to

discerning differences in purpose. That difference is about how design is explained, much as Welch suggested in Chap. 5. Where Haig sees natural selection as an adequate account, Laland and colleagues do not. They downplay the role of selection in building purpose, while at the same time play up the role of individual organisms as agents in their own design. Haig analyzes a variety of related claims and concludes that this pluralism of views is to be expected not least because the science of purposive life is hard. But he diagnoses a reluctance to address teleology head-on as a cause of many differences. If we were honest in seeing this as a clear explanatory target, Haig implies, all sides might cooperate to positive end. In the meantime, he advocates treating each argument kindly, born of its own, possibly slightly different purpose.

In Chap. 17 (Part 6), T. N. C. Vidya, Sutirth Dey, N. G. Prasad, and Amitabh Joshi provide a comparative analysis of the Darwinian core of evolutionary theory and the Extended Evolutionary Synthesis. The authors also lay out what they consider to be the six core questions for evolutionary biology: (1) the origin of new life forms; (2) the prevalence of alternative life forms; (3) the origin of new species; (4) the presence of alternative species within and among life forms; (5) the origin of new trait variants, and (6) the prevalence of alternative trait variants within and among species. All six are subsumed under the overarching explanatory aim of accounting for the relatedness, diversity, and adaptedness of species. These six categories have been addressed to differing degrees since the emergence of Darwinian evolution. For example, the authors claim (1) as the domain of evolutionary developmental biology, or evo-devo, which has only recently gathered pace and status within biology. Vidya et al. then move to an analysis of the Darwinian core and make a number of claims. Notable among these is the idea that natural selection, as a concept, was prefigured in Ancient Greek philosophy, and found in many writings since then. Rather than the conjuring of this idea from empirical work, Darwin's great contribution is instead the atomization of the individual into traits and changing the concept of heredity to contain the notion of trait generation and transmission. Vidya et al. place the removal of developmental concerns at this Darwinian point in history, thanks to this new concept of heredity. A detailed historical analysis of Darwin's contribution follows, outlining how the Darwinian core dealt with the six questions above. This core has proved resilient with only uniformitarianism and gradualism receiving serious challenge and removal. The Modern Synthesis is described as factually broader than the preceding Darwinism, but conceptually narrower, and again Vidya et al. address the responses of the Modern Synthesis to the six questions. Much of the core was retained by the MS, including the views on development, but what was lost was Lamarckism, group selection, and non-genetic inheritance. What was gained was a mechanism of genetic inheritance via Mendel. But Vidya et al. are not uncritical of the Modern Synthesis, taking aim at misconceptions in quantitative genetics, the use of fitness at the microevolutionary level, all of which feed into a direct analysis of the Extended Evolutionary Synthesis in comparison to the Darwinian core and the Modern Synthesis. Here Vidya et al. are broadly sympathetic to the aims of the Extended Synthesis, seeing roles for development and non-genetic mechanisms, while also

finding the commitment to gradualism within the Modern Synthesis troublesome. But the authors also disagree with the use of the term *synthesis* in both the Modern and the Extended cases. They prefer the notion of a Standard Evolutionary Theory, which emerged during the period referred to as the Modern Synthesis, because this theory subsumed new empirical findings but did not engage in a dialectical interaction with opposing ideas, leading to a true synthesis. The Extended advocates make the same error, but Vidya et al. note earlier writings from this group discussed Standard theory, rather than the Modern Synthesis, hinting at rhetorical purpose rather than a true commitment. The theoretical additions favored by the Extended lobby and themselves are, they note, additions made when considering less well-researched categories from within the six core questions of evolutionary biology. In this way, Vidya et al. argue for a form of explanatory pluralism in the context of a subtler reading of the history of evolutionary biology which demonstrates that much of the pluralism was in place within the Darwinian core, with some notable exceptions. This core runs through the Modern Synthesis (or standard theory) and the Extended Synthesis.

Chapter 20 (Part 7), by António Rodrigues and Andy Gardner, makes the case for inclusive fitness theory as a scientific revolution, one overlooked by advocates of the Extended Synthesis and yet, the authors claim, containing many of the aspects of evolutionary theory that are requested in an extension. Critically, Rodrigues and Gardner position inclusive fitness theory as a post-Modern Synthesis development. However, they also position it as a theoretical development in the study of adaptations, a fundamentally Darwinian project. The chapter ably moves from Darwin to Fisher's fundamental theorem, to Hamilton's rule, which is given clear exposition. It is here that Rodrigues and Gardner point to the revolution. Hamilton not only formalized fitness, but in developing inclusive fitness he removed it from being a purely individual-level concern. Individual organisms are not expected to maximize their own fitness, but rather inclusive fitness—this changed the purpose of adaptation within evolutionary theory. When discussing the relation of Hamilton to Price's famously substrate-neutral depiction of selection, the authors highlight a lesser-known aspect of Hamilton, which is that inclusive fitness theory can also be expressed in non-genetic terms. Indeed, the authors show how inclusive fitness theory can operate at several levels of abstraction to derive useful explanations. So, while originally conceived as a theory of individual-level biology, inclusive fitness models can also be applied at the level of the gene (e.g., imprinting and intragenomic conflict), or the group following the application of Price to multi-level selection. With the theory outlined Rodrigues and Gardner turn to a list of six central requirements for extension, originally given by Laland and colleagues (Laland et al. 2015). For each one, they offer an inclusive fitness theory account. For example, they argue that reciprocal causation is at the heart of inclusive fitness theory because it attempts to model an evolving population as a part of its own selective environment. They reinforce this point with an account of dispersal where organisms will disperse, reducing their personal fitness to zero, to increase the fitness of their remaining relatives. Where dispersal is not lethal, kin competition has been shown to reciprocally balance selection for altruistic dispersal, while this dispersal in turn

modulates kin competition. For Rodrigues and Gardner, this is a textbook case of reciprocal causation that emerged during the 1970s. They continue with such analyses to conclude that core Extended Synthesis requirements for reciprocal causation and a focal role for organisms were the very concepts that motivated the development of inclusive fitness theory, while others were subsequently incorporated as the theory developed. Given all of this, Rodrigues and Gardner wonder why inclusive fitness theory has been ignored in the debates around the Extended Evolutionary Synthesis. Their suggestion is that advocates of extension have primarily focused upon increasing the realism of models, by adding parameters. In doing this scholars are seeking to reduce the abstractions within models, while inclusive fitness theory has been aimed at producing an abstract generalizable explanation that cuts across many phenomena. To this end, the Extended Evolutionary Synthesis is less a scientific endeavor and more a set of quibbles, to borrow a phrase from Rodrigues and Gardner.

In Chap. 23 (Part 8) Stephen Abedon provides a case study in post-Modern Synthesis theory development in phage biology. Owing to their relatively late discovery and starring role in the development of molecular biology, phage life cycles have been characterized in painstaking mechanistic detail. Abedon shows how concepts from ecology, and optimal foraging theory, can be brought to bear on phage life cycles. This first entails re-parsing the life cycle so that variations can be considered that modulate reproductive output, measured in whole organisms. Focussing primarily on lytic cycles, Abedon shows that the intracellular development of phages imposes an opportunity cost on phages that is traded off against the number of offspring produced upon cell lysis (the burst size). Ecological parameters are then considered that influence this trade-off in addition to organism-level constraints that limit optimization. Abedon thereby reveals an array of intrinsic and extrinsic features to be considered in formal and informal models. This exemplifies how applying resources from evolutionary theory is both productive and attention-directing: focussing the researcher on features of the life cycle that may, at first, appear trivial such as the kinetics of phage adsorption by host cells. Two features that are challenging to incorporate in formal models are reproductive variance and relatedness. Considering the first, Abedon describes the concept of effective burst size showing that a single parameter can be devised that incorporates multiple details (including host cell density). Returning to the broad framework (viz., weighing time spent within hosts in the form of a virocell against time spent extracellularly as "free phage") Abedon shows that this can be brought to bear on the lysis/lysogeny switch exhibited by temperate phages. Also at the comparative level, Abedon separates lytic and lysogenic life cycles from chronic-productive infections by mapping these to semelparous versus iteroparous reproductive strategies, respectively. We also see examples of reciprocal causation in this chapter with phages influencing their own biotic environment by depleting host cells. Overall, like all good science, the application of theory by Abedon is seen to be at least as generative of questions as it is of answers.

In Chap. 26 (Part 9) Tom Dickins focuses upon the topic of plasticity. The chapter begins with a discussion of Pigliucci's argument that a mechanistic theory of form is

required within evolutionary biology (Pigliucci 2007). Phenotypic plasticity is central to such claims and has been used to counter the purported gene-centrism of the Modern Synthesis: form, and variation in form is caused by more than genetic information. Dickins embeds the concept of plasticity within a brief history of the introduction of the genotype-phenotype distinction and the reaction norm. The reaction norm has been interpreted in several ways since Woltereck, but Dickins follows contemporary views that it was Dobzhansky who thoroughly incorporated it into the Modern Synthesis. The idea of plasticity emerging from this is one of robustness, an available array of responses to environmental variability that can preserve the genotype. Where this is patterned and positively affects fitness we can look to adaptation. It is here that gene-centrism emerges as a complaint because the robustness view appears to privilege the gene at the expense of other explanatory resources: the genotype delivers a responsive phenotype. This criticism has its roots in developmental systems theory that emerged from criticisms of Lorenz's instinct concept in ethology during the 1950s. Dickins explores this criticism by deploying a distinction between instructional and cybernetic information. The former is a more colloquial usage, but Dickins argues that it is both an abstraction and an idealization that deliberately ignores the nature of data + context interactions found in the cybernetic treatment. The instructional idealization works within population-level evolutionary models, where developmental processes are assumed to have happened, as genes are seen as a necessary condition for development but also for inheritance and thus evolution across time. By definition, this idealization does not apply to developmental concerns. However, the cybernetic view of information should be seen as the basis from which the abstraction to the instructional view is made. Dickins argues that a cybernetic position was always at the core of the Modern Synthesis, but researchers switched into colloquial, instructional idealizations to make population-level evolutionary accounts intelligible. The key point is that this is not incompatible with the more detailed view. To this end, the plasticity of development is to be understood as a complex interaction between genetic data and the context of all developmental resources, much as developmental system theory states. That interaction is visible to selection, just as Dobzhansky argued. Dickins finishes by unpicking various claims made on behalf of West Eberhard (West-Eberhard 2003) pertaining to the role of developmental plasticity in evolution. He shows how her view is entirely consilient both with a cybernetically grounded account and thus with the standard theory emerging from the Modern Synthesis.

David Shuker discusses sex allocation in Chap. 29 (Part 10). In common with several authors in this book, Shuker sees evolutionary theory as a dynamic discipline that has changed since the formal period of the synthesis, including major theoretical developments such as neutral theory and inclusive fitness theory. But these developments did not require a paradigm shift away from the framework developed during the Modern Synthesis. Instead, these are explanatory efforts that have emerged within the framework as new empirical findings have come to light. Given this, Shuker is perplexed by the calls to Extend the Modern Synthesis, not least because he interprets extension as a desire to replace. In order to explore this, he takes sex allocation—an empirically and theoretically rich area of biology emerging

early in the history of the discipline, gaining refinement through the synthesis and later from inclusive fitness theory—and asks what the Extended Synthesis would change in order to explain this phenotype. Shuker begins with a comprehensive summary of the major theoretical transitions in sex allocation from Darwin onward. He then summarizes the core claims of the Extended Evolutionary Synthesis as a focus upon organismic agency and an emphasis upon non-genetic inheritance. These claims are then related to sex allocation. Unsurprisingly, Shuker sees organismic agency as central to sex allocation, with adjustments made given sensitivities to crucial environmental contingencies. Shuker does not make a claim about the kinds of mechanisms one should allocate to agency but merely commits to their being mechanisms that are facultative and thus make decisions. This is a minimal view of agency, but one seen in the writings of the Extended Evolutionary Synthesis also. Shuker firmly places this kind of decision architecture within the domain of plasticity, another topic central to advocacy for extension, and makes a strong case for sex allocation as a canonical form of plasticity in enabling multiple outcomes from a genotype. Shuker continues to relate sex allocation to non-genetic transgenerational effects, development, niche construction and many of the key topics of discussion within extended evolutionary circles. As with Rodrigues and Gardner, Shuker asks why the Extended Synthesis has ignored this active domain of evolutionary biology that appears to tick all their boxes. In addressing this Shuker refers to recent philosophical discussion about explanatory virtues and varieties of explanatory purpose. For Shuker, this is something already enshrined within the field, thanks to Tinbergen's four questions. The expectation was not that each biologist addresses all four, but rather that the collective effort of all biologists across these domains would lead to a complete understanding. Shuker's strong conclusion is that the extension sought is less extension and more detachment. He argues that advocates of the Extended Evolutionary Synthesis have a laser-like focus upon the construction of form and in so doing are simply not addressing evolutionary questions in the first place.

Douglas Futuyma discusses the evolving evolutionary synthesis as an interaction between theory and empirical findings (Chap. 32, Part 11). Early in his chapter Futuyma discusses the generality of population genetics, which was central to what he refers to as the *Evolutionary Synthesis.* [1] Population genetics tells us how evolution works, but not about specific features or taxa, it can predict short-term outcomes such as allele fixation, but it cannot explore macroevolutionary trends, such as diversification. Here, Futuyma shares some of the concerns of the Extended Evolutionary Synthesis, as the biology of actual organisms is absent from population genetics. But he continues, claiming that much of the synthetic effort was to then accrue evidence of genetic variation from multiple biological disciplines and to look to the evolution of morphological traits, etc. in terms compatible with population genetics. Not all disciplines were included, and he lists physiology, development,

[1] Some historians of science prefer to use the term Evolutionary Synthesis to denote the process of theoretical synthesis and to separate it from claims to modernity (Smocovitis 1996).

and ecology as missing parties, noting disagreement about some aspects of exclusion in the historical literature. Futuyma then moves to outline controversies within evolutionary biology: neutralism, levels of selection, sympatric speciation, punctuated equilibria, and adaptation and constraint. The lesson he draws from analyzing these controversies is that, while some of the initial suppositions may have proved incorrect, they each helped to improve knowledge and understanding. Futuyma does not use this observation to defend the role of controversy but rather to hint at the robustness of the original theory, developed during the synthesis. Futuyma then discusses the Extended Evolutionary Synthesis, as a controversy, and with particular focus on the 2014 exchange in *Nature*, that he was a part of (Laland et al. 2014). He carefully goes through each of the major areas discussed in that exchange—niche construction, evolutionary developmental biology, plasticity, and inclusive inheritance—demonstrating what has been studied within these areas prior to the advent of calls for an Extended Evolutionary Synthesis. Futuyma pulls no punches and makes clear where claims for extension are overwrought, in particular choosing to see niche construction theory as a simple rebranding of community ecology. But he is also charitable and argues that while controversy may not be the best mechanism for moving science forward (he claims it is impossible to counterfactually know whether it is), advocacy for extension may provide a useful service in highlighting important work that requires further integration into evolutionary biology. Thus, the thrust of Futuyma's argument is that this is business as usual, as we saw during the Evolutionary Synthesis, and not a wholesale demolition of core theory but rather integration of empirical findings leading to improved understanding in a manner he aligns with the Kuhnian concept of normal science. Futuyma's argument about the generality of the population genetic approach to evolution amounts to a claim for high-level abstraction, and his view of normal science to the reduction of abstraction through the addition of empirical detail. He is quite clear that empirical details can challenge and overturn theory, but at this point in the history of evolutionary biology he sees no evidence of this having happened.

Central to the Extended Evolutionary Synthesis claim is the criticism that Standard Evolutionary Theory, emerging from Modern Synthesis, is gene centric at the expense of the organism (Chap. 8). In Chap. 35 (Part 12), J. Arvid Ågren discusses the changing fortunes of both gene and organism concepts in evolutionary biology. He surveys various views from within and without the Modern Synthesis, demonstrating how the gene's eye view of evolution has divided biologists and philosophers since it first emerged in the work of Williams and then Dawkins (Dawkins 1976; Williams 1996). Both authors made cogent arguments regarding the problem of design as the central problem for evolutionary biology and for shifting from the Darwinian focus upon individuals and groups to the level of the gene to address this problem. Ågren argues that the project of understanding adaptations requires solving a beneficiary problem—what are adaptations good for? Williams' and Dawkins' answer is genes, understood as having the properties of longevity, fecundity, and copy-fidelity. It is this understanding that shifted the concept of the gene from a purely molecular or particulate one to that of a *replicator*, in Dawkins' terminology. In determining the replicator concept,

organisms were assigned to the category of *vehicle*, lacking the essential properties for evolutionary process, but enabling genes in their purpose through their differential survival and reproduction. The focus upon adaptation is related to Paley's natural theological legacy by Ågren, but he also discusses the role of Fisher's early population genetics. Unlike Wright, who believed he was modeling organismic evolution, Fisher was focused upon adaptation and genes and Ågren shows how Fisher introduced a simple segregation between genetic and environmental variation (variance) that resulted in the genome being seen as an environment, in the same way as more standard ecological factors were. The final move that cemented the gene's eye view was the rejection of group selection and specifically naïve *good of the species* versions of this argument. It is this decomposition of evolution to a gene-level process, and the relegation of organisms to the vehicle category, that has led to concerns that gene-centrism is detrimental to biology. Those advocating for an extension to the Modern Synthesis are keen to prioritize the phenotype in evolutionary explanations, and therefore they seek a role for the organism. In this way, they are reverting to a Darwinian core (Chap. 17). But as Ågren shows, this tension existed within the Modern Synthesis also. We have already referenced Wright, but Mayr too clashed with the gene-centric Haldane in the infamous bean-bag genetics dispute. Moreover, inclusive fitness theory marks an organism-focused post-synthesis development that is neutral about genes even though it can be cashed out in gene's eye terms (Chap. 20). This aspect of inclusive fitness theory has frustrated Dawkins and Maynard Smith, as Ågren makes clear, because of the difficulties of calculating a value at the individual level, which has led Dawkins to re-emphasize the utility of the gene's eye view for gaining explanatory traction. Ågren discusses genetic conflicts and shows how this challenges the inclusive fitness whole organism view that there is a unity of purpose in the organism. This leads to some inclusive fitness theorists idealizing such conflicts as absent to deliver models for specific phenotypes. It is here that Ågren concludes that inclusive fitness theory works for specific explanatory targets and as a result this supports the ambition to keep organisms at the center of evolutionary accounts. But this is also a clear statement that this ambition existed before the calls for an extension of the Modern Synthesis, as well as a pragmatic point about the differing utilities of the gene's eye and organism-level approaches. Under Ågren's interpretation, inclusive fitness theory is an idealization, claiming unity of purpose at the level of the organism. It is perhaps a thin idealization as the major transitions in evolution are regarded as cooperative shifts to such unity, but nonetheless it is a deliberate falsehood (Maynard-Smith and Szathmary 1995). This does not imply, however, that the gene's eye view is without idealizing assumptions.

Mitchell Distin focuses upon evolvability in Chap. 38 (Part 13). He opens by claiming that evolvability was effectively overlooked during the development of modern evolutionary biology. This was in part due to the abstract nature of evolutionary theory (Chap. 32) and a lack of connection to nature. This obscured complex evolutionary dynamics emerging beyond the individual, and Distin claims a division between theory and empiricism emerged that was enhanced by a commitment to the logical positivism of the early twentieth century. The ambition was to produce

mathematically expressed nomic statements about biology, something that did not suit evolvability. By implication, Distin also relates this to a neglect of ecology, and states that the discipline of evolutionary ecology is now coming of age and enabling a true focus upon evolvability, for example within discussions of evolutionary rescue. He also points to other developments around genetic evolvability mechanisms such as stress-induced mutagenesis, which he argues point to high-level selection at the species or lineage level, something perhaps anathema to earlier critics of naïve multi-level selection accounts (Chap. 35) and Distin aligns this move with specific pluralist and multi-causal explanatory approaches within the philosophy of science. The phenomenon of evolvability demands a different kind of causal model than commonly deployed by Standard Evolutionary Theory. From here Distin moves to discuss evolvability as the cornerstone of the Extended Evolutionary Synthesis, where evolvability is conceptually embedded within evolutionary developmental biology, or evo-devo. However, Distin does not wish to support the view that evo-devo is the focus of evolvability, instead preferring to see evo-devo as a method of thinking about evolvability. Indeed, Distin is clear that evolvability as a generic concept has been present in evolutionary thinking for many decades, as he carefully documents, but as above, its traction has been impeded by the abstract nature of the evolutionary biology emerging during the Modern Synthesis. This has led to a high degree of conceptual fuzziness in contemporary discussions of evolvability and Distin offers to untangle this first with an analysis of the evo-devo view of non-genetic evolvability. He surveys the work on modular developmental programs (see Kirschner 2013 for an overview) that reduce lethality effects associated with novel mutation by reducing the number of mutations required to introduce novelty. While praising the innovation and progress made in this field, Distin sees it as hindering understanding of genetic evolvability, and obscuring distinctions between short and long-term evolvability. The kind of genetic causality Distin favors is that which leads to differences in mutation rates between species, something that Distin notes natural selection can operate over. For Distin, the focus upon evo-devo makes the causal account limited and perhaps too abstract. He wants evolvability to encompass both developmental and genetic causes and for the causal model of evolvability to clearly explain the different roles of each component. For Distin, then, evolvability is a unitary phenomenon with broad application thanks to a complex, causal mechanism. Indeed, early on he labels evolvability as an emergent dispositional property, causally relevant at higher levels of organization over long stretches of time. Thus, evolvability is multiply caused, and in turn, causes evolutionary change. Distin uses this discussion to draw out a philosophical point about pluralism. The fuzziness of the evolvability concept in the literature ought not to encourage an unrestricted pluralism, allowing multiple different causal idealizations to emerge, each operating within its own explanatory bailiwick. Instead, we need a restricted pluralism, in which we aim to minimize, but not necessarily eradicate, plural approaches, in order to sharpen our focus upon the phenomena. The subtext of Distin's chapter is that the initial abstractions of the Modern Synthesis led to a formal neglect of various phenomena, including evolvability, but also gave licence to an anything-goes culture among those addressing evolvability. To bring evolvability

into evolutionary biology a clear set of phenomena associated with that label need to be decided, and a part of that decision will be made with reference to Standard Evolutionary Theory. In this way, any extension of the Modern Synthesis is quite simply an extension of its phenomenal reach.

1.4 Summary and Conclusion

Our summaries of the chapters are intended as a rough guide to what follows. They do not do full justice to the rich, detailed work that the authors have undertaken and presented. While we have drawn out a theme about idealization in this chapter this is by no means the only emerging theme, nor do we expect our readers to necessarily agree with it. Instead, our hope is that this volume will stimulate continued discussion and engagement within the broader community of scholars who think about evolution and how to account for it.

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Part I

Every Evolutionist Their Own Historian: The Importance of History, Context, and the *Extended* Evolutionary Synthesis

2

V. Betty Smocovitis

History, if used as a repository for more than anecdote or chronology, could produce a decisive transformation in the image of science by which we are now possessed.
Thomas Kuhn The Structure of Scientific Revolutions*, 1963.*

Neither the value nor the dignity of history need suffer by regarding it as a foreshortened and incomplete representation of the reality that once was, an unstable pattern of remembered things redesigned and newly colored to suit the convenience of those who make use of it.
Carl Becker, "Everyman His Own Historian," 1931.

Abstract

This chapter opens with a discussion of scholarly practices within the history of science, noting a distinct difference between professional historians and scientists-turned-historians. History is important and how it is done has implications not only for our understanding of the emergence of a discipline but also for contemporary debates within it. This theme is followed with a detailed analysis of the often-partisan uses of history to define disciplinary boundaries, to found disciplines, and to criticize them. Parallels are drawn between the anti-adaptationist debates of the 1980s and recent calls for an

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extended evolutionary synthesis in their uses of history. This chapter questions the core idea that there is a monolithic evolutionary theory, or that there ever has been. Instead, detailed historical analyses, relying on more than interpretation of academic publications, show a dynamic and often conflicted field of scientists.

Keywords

Historicism · Presentism · Modern synthesis · Evolutionary synthesis · Neo-Darwinism · Standard evolutionary theory · Extended evolutionary synthesis · Context

2.1 Introduction: Every Evolutionist Their Own Historian

Entering the conversation about the so-called *extended* evolutionary synthesis, and its use of history reminds me of a classic 1932 essay by Carl Becker, one of the most reflective of US-based historians long associated with Cornell University in Ithaca, New York. Titled "everyman his own historian," Becker drew the analogy between everyday historical reckonings that human beings perform in their daily tasks and the work of professional historians, scholars trained in the use of historical evidence, methods, and publications (Becker 1932). His intention was to make the case for the importance of history in everyday life, and to diminish if not eliminate its professional pretentions with respect to scientific history, the newfangled application of scientific methods to understanding the past in objective terms. But while he supposedly received a standing ovation when he first delivered it as his 1931 Presidential address to the American Historical Association, the essay, and its analogy, eventually backfired because it introduced a kind of historical relativism that Becker himself had not intended. It also highlighted the kinds of interpretive ambiguities few historians were comfortable embracing, and indeed, had been working hard to eliminate from their work. People do have the tendency to recall the past in light of the present, and to selectively ignore or forget some things, while remembering others; the gallons of ink spilled on discussions of selective memories, for example, speaks directly to this (Pohl 2004), so why should historians be any different, or so Becker's logic goes. Everyone is going to craft their own history, and erase it when they see fit, and though there may be a price, and a big one at that, to be paid if others do not like it, everyone is entitled, in a sense, to do this, especially when matters of self-existence and identity come to play. This latter point needs underscoring because boundaries and identities are determined, and bloody wars are fought, over matters of history. It does matter, hugely.

Becker was of course addressing history, *writ large*, but much of what he relayed in his essay also speaks to the history of science, at least as I see it. To be sure, historians of science do have their own issues; we frequently refer to "scientist's histories," oftentimes deriding them because they lapse into the kinds of naïve mythologizing that celebrate great discoveries, ideas, and historically white men (Forman 1991). Such histories, or narratives, can simultaneously marginalize or

exclude alternatives, while at the same time serving to sanitize the messiness and conceal the arduous work, or the sociopolitical dimensions, involved in the practice of doing science, especially when something like consensus-building comes to play. Such histories can also be blinkered, or skewed by "Whig," presentist, or even self-serving perspectives, and these can then shape entire disciplines once they are embodied in textbooks as the received wisdom of the field that enroll novices to the craft (Ashplant and Wilson 1988a, b draw these distinctions at length). Thomas Kuhn drew attention to most of this in his celebrated *The Structure of Scientific Revolutions* in 1962 (Kuhn 1962), of course, but this is why historians of science are wary when history is evoked by scientists-participants, especially in the context of disputes and arguments over who or what is right, and who or what is wrong, or when deployed in an arsenal when conflicts break out over the direction of an entire field.

Apart from pointing all this out, however, historians of science are pretty well powerless to do much of anything about it, especially if, following Becker's logic, we are subject to the same kinds of interpretive ambiguities ourselves. Scientists can and will write their own histories as they see fit especially in a science like evolutionary biology which is a historical science that has included more than its share of evolutionists-turned-historians like Ernst Mayr and Stephen J. Gould (Nitecki and Nitecki 1992). As I have argued elsewhere, their use of history as well as philosophy has accompanied their crafting of evolutionary theory, both for scientific and wider, more popular audiences (Smocovitis 1992, 1996). And it is not part of some devious, underhanded, or even deliberate process that serves self-interest narrowly defined; it is part of the active, and ongoing process of constructing disciplinary narratives that unify and lend coherence to the group and define the identities of its members, while at the same time unwittingly serving to exclude and marginalize others; in short, they do what sociologists of science call "boundary work," they determine the contours of the discipline and who or what will be inside and who and what will be outside its boundaries (Gieryn 1983, 1999; Shapin 1992). And when the disciplinary narrative of an area of study like evolutionary biology can tap into, or become part of, a wider narrative of say, the Enlightenment, or of the history of science in western cultural contexts, and when it can encompass an originary narrative of humanity, or of life as a whole, it can become part of a *grand unifying narrative* (another technical term) that undergirds understanding of the world, structuring a worldview or *Weltanschauung* that may tell us something about the "human's place in nature" (Greene 1981; Smocovitis 1992, 1996).

I begin my chapter with these historical reflections because I think that they are at play in many of the contemporary discussions and debates over evolutionary theory especially pertaining to the *extended* synthesis. My task, as I see it, is not to weigh in on scientific matters, but instead to draw on the historical record as revealed by the traces left behind by the historical actors, and to engage and understand them *on their own terms,* avoiding presentism to the extent that I can, and then to examine the uses of history by contemporary evolutionary biologists, especially as understood and deployed in some contemporary discussions and disputes. In this way, I hope to offer some historical observations, try to reduce, if not eliminate some

misunderstandings, and to offer some clarification in the way of moving the discussion forward in a more productive direction. My firm belief is that despite the frequent use and referencing of history, many of these disputes and discussions are based on several historical misunderstandings, rely on a problematic, confused, and underexamined terminology, and at times adopt a wholly *ahistorical* view of evolution, especially of evolutionary theory. Indeed, they are too much from the "every evolutionist their own historian" perspective and thus lead to unnecessary confusion and discussions that are often at cross-purposes.

2.2 Laying the Groundwork: Historicism, Contextualism, Presentism, and the Language of *Synthesis*

In the way of laying some groundwork, I would like to start with the word "historicize", or "historicism." To some, it might appear like academic jargon, the kind of thing common in the humanities, associated for example with literary theorists like Frederic Jameson, and his well-known slogan "always historicize" (Jameson 1981). But I would argue vigorously that there is nothing jargon-esque, or fashionable about it to those of us studying history as well as a historical science like evolutionary biology. It simply means to embed in history, to render a thing as an object of history, and to give it meaning and signification within a historical web of beliefs and practices; it means to think diachronically, processually, or indeed, in evolutionary terms. Let me offer a concrete example: does a fossil have much meaning without a sense of where it is found, not just in what kind of environment, but in what kind of temporal sequence, or context, of what came before, what is found near it, and what comes afterward? The answer is not really, because the meaning of fossils is understood in historical sequences as well as environmental contexts. The same holds true for ideas, beliefs, practices, and even scientific theories. But realize the twist in accepting this; it means that ideas, beliefs, practices, and even scientific theories are embedded, or situated in particular contexts and understood diachronically; they change, evolve, and come to be. They do not transcend history and culture but take on a more local or historical specificity, and themselves evolve, though the process is very different from that operating in organic evolution. The view that theories evolve, moreover, is not much of a novel or even radical idea to reflective evolutionary biologists; Douglas J. Futuyma has made this point more than once and argues for it indirectly in his contribution in this collection (Chap. 32), for example (and see CALLEBAUT 2010).

This gets me to my next word, "context" or "contextualize," which adds the cultural, or social, and political to the mix, especially when referring to ideas, beliefs, practices, and even theories, which do not arise *de novo*, but draw on raw materials from the past and prevailing currents of thoughts, habits, or what in the humanities are called "circulating discourses." To the historian, this is a kind of reframing of the older concept of *zeitgeist*, though this current version may also incorporate material practices as well as the prevailing thoughts or *spirit of an age* along with a closer attention to language because thoughts—ideas—are embodied in words. Ideas

themselves are not viewed as *unit particles* that arise from the brain of a Zeus, a "great man of science," or a "genius" to be transmitted and bounced off others in a billiard-ball-like causal process, but instead exist in the forms of words comprising languages that circulate, moving around in a cultural system not unlike materials that cycle in ecosystem ecology, but in that process may alter in meaning. So, to contextualize means to embed words within other words in texts that enable us to access or understand cultures that are different from ours (Smocovitis 1996). To the evolutionary biologist, such a view of context, also provocatively opens the door to a plurality of legitimate theoretical perspectives, each a kind of culture, emphasizing one or other aspect of evolution, based on particularities and specificities of methods, training, generation, field, or organismic system. It is provocative because it challenges the assumption that there is one singular, unifying, or grand theory, and instead opens the door to the possibility of multiple overlapping theories emerging from context and history.

We move then to another important word that I have already used in the introduction and that is "presentism," the tendency to project the present into the past or to interpret the past in light of the present. Lapsing into presentism gives us not just a distorted view of the past, but turns history into a kind of extractive industry, full of moral and ethical quandaries since it often comes at the expense of the historical actors who are rendered voiceless in the process. What is it we actually want to know, anyways? Do we want to know something about *them*, or do we want some affirmation from the past about *us* today? If we want to know something about them, we have to make every effort to understand them as they were, especially if we are trying to trace our historical roots. L. P. Hartley's famous opening line to his novel *The Go-Between* that the "past is a foreign country" was not just a pithy statement about the past (Hartley 1953); we enter the past much as foreigners encountering another culture, needing to learn another language. Suffice it to say that presentism is a violation of basic historical methodology and something to be avoided; paying careful attention to the words and phrases as we trace their occurrence helps us to avoid doing this. So, I end this section of my chapter underscoring the importance of language as a way of mediating between history and context and stating explicitly that, along with Kuhn and subsequent students of the history and philosophy of science, science is best viewed as a historically rooted and culturally embedded practice. Such an approach enables us not just to mediate between historicism and context, but to also draw on a mix of approaches from history, philosophy, sociology as well as anthropology, in a kind of multidisciplinary approach that will help us gain a greater understanding of the past on its own terms. That in turn, I believe, may allow us to avoid miscommunication and some of the more contentious conversations in evolutionary biology today (Smocovitis 2021).

2.3 The Modern Synthesis, The Evolutionary Synthesis, and Neo-Darwinism: Drawing Distinctions

Next, I would like to sort through some of the actual language of the synthesis focused on naming and draw some useful distinctions in terminology. In the process, I hope to also cover some ground in the history of evolutionary biology. We have for example frequent reference to the "modern synthesis" of evolution. This term comes straight out of the title of Julian Huxley's well known and oft-cited comprehensive treatment of evolution originating in the late 1930s and published in 1942 as *Evolution: The Modern Synthesis*. It was a widely read book aimed at a more general, semi-popular audience, the kind of synthetic book offering a scientific worldview that Huxley was very good at writing. Heir to the Huxley family legacy, Julian was a gifted writer and popularizer of science, becoming a kind of celebrity in his own time. Putting his skills to use, Huxley wrote the 1942 book drawing on an earlier essay of 1936 titled "Natural Selection and Evolutionary Progress" that reflected his belief in a progressive view of evolution, that would also help ground a secular, liberal worldview that was increasingly divided by extreme ideologies (Smocovitis 1996, 2016). The publication of the 1942 book has traditionally heralded that a new and *modernized* evolution had emerged, one that synthesized Darwinian selection theory with Mendelian genetics (Mayr and Provine 1980). But perhaps equally important, Huxley framed this "modern synthesis" with his introduction that offered a kind of early disciplinary narrative of what became a new field of study, namely evolutionary biology. As a disciplinary term, "evolutionary biology," was only just gaining currency, and Huxley was just starting to use it interchangeably with the broadly conceived but generic "evolutionary studies" (see Smocovitis 1996, p. 162 for more on the etymology of "evolutionary biology" and the history of the discipline). Setting up this "modern synthesis of evolution" Huxley stressed the preceding two decades of work that turned a mostly descriptive natural history-oriented study of evolution into a rigorous science grounded in observation and experiment that had drawn novel insights from population genetics and mathematical modeling. This, according to Huxley, made possible the fusion of genetics and Darwinian selection and served to reanimate Darwinism following a period he named "the eclipse of Darwin" that had seen several theories that had either built on, amended, rivaled or even challenged Darwinian selection theory, especially after the year 1900 when Mendelian genetics began to gain currency. Like a "mutated phoenix risen from the ashes of the pyre," he wrote with dramatic flair, this "reborn" Darwinism made natural selection a "fact of nature capable of verification" and made natural selection one of the fundamental principles of biology. Biology itself, according to Huxley, was undergoing its own "phase of synthesis" bringing together a set of newer sub-disciplines previously isolated and often "contradictory," and was in the process of becoming a "more unified science," rivaling the "unity" of sciences like physics and chemistry (Huxley 1942 pp. 13–28).

Without getting too heavily into the details of Huxley's intentions, and his life and work, subjects that have been extensively explored (Keynes and Harrison 1989; Greene 1990; Waters and Van Helden 1992; Smocovitis 1992, 1996, 2016; Bashford

2022) we can think of his "modern synthesis" as an *actor's phrase* or even as a kind of actor's category with a discrete periodization (interwar and wartime) because it is associated with the title of Huxley's book. The phrase "modern synthesis" thus has direct relevance not just to understanding Huxley but also speaks to the context of the 1930s and very early 1940s and possibly the two decades preceding that, since Huxley was trying to trace the tortuous history, in a kind of "rise and fall narrative" of Darwinism in setting up his argument for a modern synthesis of evolution; what I am getting at here is that for our purposes, the term "modern synthesis" comes from a *primary* source. In historical parlance it is a trace, one that directly reveals something about the past and the historical actor who used it and gave it signification. This is in contrast to *secondary* sources, works by subsequent commentators, usually historians or people trying to reconstruct history, offering interpretations of the past, based primarily on following traces and using primary sources; that, and the scholarly understanding that has accumulated in the historiography, a term which refers to an understanding gained from a given body of historical literature that has been written or produced by scholars working in the subject area.

Consideration of secondary sources takes us to yet another term used frequently by some evolutionary biologists, usually those who consult historical scholarship more frequently, namely the "evolutionary synthesis." The term is most closely associated with Ernst Mayr and William B. Provine's edited collection of 1980, a now classic, foundational, and entry-level work for anyone interested in the history of evolution. The collection grew out of two workshops held in 1974 devoted to the subject and organized by Mayr and Provine. It included a number of original participants, or "architects," of the evolutionary synthesis (a self-designated or identifying label used by Mayr and others who were still alive to reflect on their work and its outcomes; see the index in Mayr and Provine 1980 for the list of conference participants) but it also included a number of historians and philosophers, many of whom, like Provine were junior scholars and keen to understand the history and philosophy of evolutionary biology, especially at time when the physical sciences had long held too much dominance; an examination of evolutionary biology had the potential to yield novel insights, especially because it was a historical science, unlike physics or chemistry.

As such, the workshop, the abundant transcripts, biographical sketches, and correspondence (all safeguarded and deposited in archival collections) along with the published edited collection is an important and fascinating mix of both secondary as well as primary sources, sometimes indistinguishable, that anyone interested in the history of evolution may consult. Indeed, one of the most interesting aspects of the "evolutionary synthesis" is the length to which Mayr and Provine (but especially Mayr) went to ensure that all the materials would be available for future reference. I do not believe this was accidental but view it as part of Mayr's strategy of drawing attention to "the evolutionary synthesis" and to actively fashion it as a major historical event in the history of science. His choice of terminological change from "modern synthesis," which had gained some currency after Huxley, was in response to a title that appeared too generic and too vague and was getting lost to other syntheses. Retitling it to "evolutionary synthesis" left little doubt that this was a

synthesis that took place pertaining to evolution. Coinciding (or close enough) with this, too was the publication of his *magnum opus* in the history and philosophy of biology, *The Growth of Biological Thought*, in 1982, which placed evolution as the centerpiece of both the history and a new philosophy of science (Mayr 1982). Along with his earlier efforts in supporting a *Journal of the History of Biology*, and subsequent publications in these areas, Mayr unfolded a long-term project at cojoining the science of evolutionary biology, and the evolutionary synthesis, with the growing field of the history and philosophy of biology, in so doing, of course both inventing, and reinventing as well as trying to control the narrative of the synthesis and the narrative of evolutionary biology (Smocovitis 1992, 1996).

But he was not alone, at least with respect to the evolutionary synthesis. Examination of the transcripts and the published collection shows that the architects of the evolutionary synthesis all engaged in similar attempts to control the narrative which was usually centered on: (1) their disciplines or sub-disciplines; (2) their preferred methods that included emphases on experimental work, or quantitative work or the site where the work was done, for example the laboratory, or field, or desk, etc. (this is another way of framing that old experimentalist-naturalist tension that dominated an earlier period in the history of biology; see Allen 1979), (3) their institutional and national contexts, often largely taken for granted, especially if they were in dominant Anglophone nations, (4) their own achievements. In short, though many of the accounts offered invaluable perspectives in the way of serving as primary sources for historians, the historical accounts by the architects had to be read especially critically, since they centered on themselves, often complaining that they, or their fields, or their predecessors, frequently evoked in hagiographic terms, had not gained sufficient historical recognition for their contributions (Mayr and Provine 1980). An exercise in selective memories and historical reconstruction, many of the architects of the evolutionary synthesis demonstrated a classic textbook version "confirmation bias," especially in the transcripts which did not rely much on material evidence, but mostly memory.

As expected, furthermore, these architects frequently disagreed with historical interpretations that did not line up with their first-hand experiences or memories, or even with the invitation list which excluded some areas and individuals while placing too much emphasis on others. G. Ledyard Stebbins, the chief botanical architect and author of the 1950 *Variation and Evolution of Plants*, the last and longest of the works that ushered in the field of plant evolutionary biology (Stebbins 1950; Raven 1974; Smocovitis 2006), for example, attended the meeting but complained of a pronounced zoo-centric cast to the workshop. G. G. Simpson did not attend, claiming he had poor health but also because he did not think it the proper place to discuss his contributions. My sense reading his correspondence was that it was not so much ill health that prevented his participation, but that he did not want to compete with others or share in the glory; he had his own story to tell, which he eventually did share in his autobiography published in 1978 especially after reading the transcripts and feeling that the other architects entirely misunderstood his role in the evolutionary synthesis and the importance of his work. Similarly, Conrad Waddington, the noted embryologist who was an early critic of the "modern

synthesis," heeded the call for a historical account and gave his own version of the story in a semi-autobiographical work he immediately completed and published in 1975 (Waddington 1975). But no one was justified at being more aggrieved than theorist Sewall Wright who was not invited to the workshop at all; Mayr had thought him too old, too verbose, and with some 22 other geneticists invited, would have just drawn too much attention to genetics to the detriment of other fields like systematics, Mayr's own area. Mayr was also in a dispute with Wright over evolutionary theory (Mayr 1992). Wright, it should be noted, did get to tell his side of the story second hand, by working closely with Provine who eventually published an enormous biography that assessed Wright's role in the whole of evolutionary biology titled *Sewall Wright and Evolutionary Biology* (Provine 1986; see also Provine 1971). Mayr incidentally, was always aggrieved about the attention given to Wright and even to Stebbins and worked hard at cultivating historians, as well as leaving behind abundant oral histories and other historical materials for our use.

The disagreements were also not just about larger interpretive points, about chronologies, periods, fields, or which individuals were important. It also included basics like the years demarcating the evolutionary synthesis. Ernst Mayr, for example, gave the dates 1936–1947, which included Theodosius Dobzhansky's *Genetics and the Origin of Species* but left out much of the major work of theoretical population geneticists R. A. Fisher, J. B. S. Haldane, and Sewall Wright (Mayr 1980). It also marked the endpoint with the celebratory Princeton meetings that brought an international group of evolutionary biologists together for the first time after the war and led to the publication of an important collection for the occasion that Mayr himself co-edited (Jepsen et al. 1949; Mayr 1980). These choices enabled him to downplay theoretical population genetics while raising the importance of systematics, his preferred area of study while also excluding the work of botanists since Stebbins published his book bringing "botany to synthesis" in 1950. As Kim Kleinman has shown, furthermore, Mayr likely alienated Edgar Anderson who had given the botanical side of the Jesup Lectures shared with Mayr, so the "viewpoint of the botanist" did not accompany Mayr's "viewpoint of the zoologist" (Kleinman 2013). Subsequently, Stebbins had based his book on his Jesup Lectures at Columbia University in 1946, but problems with copy editing and Columbia University Press caused a delay in publication; that, and the fact that Stebbins brought together an astonishing range of fields and bodies of literature that had accumulated in plant evolution. Because it drew on such a disparate set of literature about the plant world, it became the longest of the Jesup Lecture series, Columbia University Press books, and arguably the most complicated in organization (Smocovitis 2006). To Mayr, this "delay" was not due to publication issues or the difficulty of the task, or of his own relationship with Anderson, but to the failure of botanists to keep in step with zoologists, and indeed one of the historiographic subthemes he introduced in the collection was to explore reasons for this "delay." A consummate zoologist, moreover, Mayr did not include a comparable discussion of zoology and the synthesis, because it was obvious to him that it needed no explanation; it was all animal-centric, in other words. For this reason, I have called "zoology and the synthesis" the "invisible subject" (Smocovitis 2003).

Yet more basics that were in dispute included who would count as an architect, and who was but a mere minor player, what fields dominated the evolutionary synthesis, which fields lagged, and which fields were not actually included. Even the list of important publications varied. Few disagreed with the inclusion of the books that came out of Jesup Lecture Series at Columbia such as Dobzhansky's *Genetics and the Origin of Species*, Mayr's 1940 *Systematics and the Origin of Species*, and Simpson's 1944 *Tempo and Mode in Evolution*, as well as of course Stebbins's "tardy" 1950 *Variation and Evolution of Plants*. But what about R. A. Fisher's 1930 *Genetical Theory of Natural Selection*, J. B. S. Haldane's 1932 *Causes of Evolution*, or even Julian Huxley's 1940 edited collection *The New Systematics*? Huxley's 1942 book was of course to be included, but even then, architects like Mayr never thought it reached the status of an original focused scientific work such as his or Dobzhansky's; he thought it incoherent and inferior and rarely gave it the credit he gave the other Jesup Lecture, Columbia University Press publications, as did some of the other architects like Stebbins. And what about major figures who did not write books but instead concentrated on articles like Sewall Wright, or books that were written in other languages, not yet available in English and not as widely known, such as Bernhard Rensch's 1947 *Neure Problems der Abstammungslehre*, only later translated to *Evolution Above the Species Level* in 1959? In short, even determining the list of major works was subject to discussion, along with the inclusion of the authors, both of which were made especially complicated by the lack of agreement over the actual timeline.

Provine himself, the junior partner in the collaboration, largely skirted Mayr's more restricted timeline. He instead employed the wider period 1920–1950, which he felt was more inclusive of theoretical population genetics, his own area of interest (Provine 1971), as well as the publication of what he viewed as the last work that brought in botany to the evolutionary synthesis, namely Stebbins's *Variation and Evolution in Plants* (Provine 1980). But he also recognized the difficulty of determining exactly what happened, and who to include and what the major works should be. In his Epilogue to the collection, written under some pressure by Mayr as senior editor, Provine indirectly admitted this, but stated all participants seemed to agree that a consensus emerged about the mechanism of natural selection during the interval of time between 1920 and 1950. He further concluded that it was "an event of first-rank importance in the history of biology" (Provine 1980; p. 399). But reflecting on this later in the decade, and no longer under the influence of his senior collaborator, Provine changed his mind, pointing to a kind of eliminative historical moment ejecting alternatives to natural selection and termed the event as the "evolutionary constriction" (Provine 1989, 1992). In a series of subsequent publications, he softened even on that, as he assessed Mayr's contributions, and his role in evolutionary biology (Provine 2004, 2005). By 2001, his view had changed so much that he wasn't even sure much of anything happened during the evolutionary synthesis, questioning the very fact of it even being an important historical event (Provine 2001, 2014).

Thus, if the 1974 workshops and the 1980 co-edited collection revealed much, it was that there was little agreement about the historical event of the evolutionary

synthesis, including eventually later in Provine's case, that it had happened at all. There is therefore no "standard historical narrative" to be found, so anyone upholding this has either not examined historical sources available, replicated one of the many participant's narratives, more than likely Mayr's, or is setting up a "straw story," a convenient narrative, that they can then knock down. What we do have available is a fascinating array of multiple narratives some of which overlap with each other, but some of which also depart from each other in other ways. We have Huxley's narrative, Simpson's narrative, along with Stebbins's and Provine's narratives as well as his co-organizer Mayr's, who likely exerted the greatest influence because he retold it in his many publications over an unusually long career. Ironically, the lack of agreement is embodied in the actual title of the collection *The Evolutionary Synthesis: Perspectives on the Unification of Biology*, though few people pay attention to the second half of it, and the fact that the collection offers "perspectives," many of which happen to be very disparate. Despite the fact too, that the title makes the connecting link between evolution and the unification of biology, few of the actual participants made this claim. As I have argued elsewhere, this emphasis on unification belongs very much to Ernst Mayr acting in his capacity as an avian systematist, evolutionary biologist, as well as historian and especially philosopher keen to preserve the unity as well as the autonomy of biology. He also happened to be one of the organizers and founders of the *Society for the Study of Evolution,* as well as the first editor of the journal *Evolution,* functioning as a discipline builder and gatekeeper of evolutionary biology (Smocovitis 1992, 1994a, b, 1996). Mayr and Provine's evolutionary synthesis thus did not result in some grand historical consensus about the event, though all participants did agree that the evolutionary synthesis warranted much more serious historical attention. Some scholars continued to delve into the area, and the publication of the volume, which was widely read and discussed by both scientists and historians, drew in another generation of historians who began to turn their attention full time to evolutionary synthesis. As a term, the evolutionary synthesis thus entered the historian's vocabulary, a subject for serious scholarly inquiry precisely because it embodied a wide range of interpretive issues, twists, and turns ripe for exploration by anyone interested in historiography. It is also full of the kinds of ambiguities alluded to by Becker, and indeed a nice demonstration of the "everyman his own historian" theme.

Yet another term used often in conjunction with modern synthesis and evolutionary synthesis is "Neo-Darwinism," sometimes used interchangeably with "twentieth century Darwinism." It has thus come to have a more generic meaning used often indiscriminately, and at a times in a kind of "quick and dirty" fashion, equating it to the twentieth-century version of nineteenth-century Darwinism. It has also been an especially contentious term in evoking the many meanings associated with "Darwinism," ranging from the role played by natural selection, the primary but not sole means by which Darwin thought species change took place or in his theory of descent with modification (recall that there was no reference to evolution in his first, 1859 edition of *On the Origin of Species*) to the interpretive twists added by the many "Darwinian" commentators as well as critics. For good reason, Morse

Peckham wrote a now-classic essay on this phenomenon titled "Darwinism and Darwinisticism" on the occasion of the Darwin Centennial of 1959 (Peckham 1959). Then too there is the association with "Social Darwinism," with its distasteful Spencerian connotations, made all the worse by it taking the form of an "ism" so that a negative ideological valence is often assigned to it. The term has been used in such a variety of ways in part because of the wide-ranging impact of Darwin's work, but also because it has been in use for such a long time. As Ernst Mayr pointed out in an insightful essay on the topic titled "What is Darwinism Today" published in 1984, the term "Neo-Darwinism" was first coined by George John Romanes, one of Darwin's loyal advocates, who applied it to August Weismann's celebrated revisionist interpretation of Darwinian evolution without the inheritance of acquired characters in 1895 (Mayr 1984a, b; for an excellent discussion of the several means by which organic change happened in Darwin's *Origin* of 1859 see Provine 1985). Since then, it has been widely used and misused often in pejorative terms usually. Conrad Waddington, one of the earliest critics of the synthesis, for example, used it in a negative sense (Wilkins 2015), while Stephen J. Gould was especially fond of using terms like "ultra-Darwinism" hurled as a kind of invective against the "hyper-selectionism" of opponents like Richard Dawkins (see Prindle 2009 for more on Gould's mix of politics and science). Mayr was especially wary of the use of the term "Neo-Darwinism," because it was so often thrown around, but also because it was associated with a view of evolutionary theory that was crude, simplistic, and overly reductive. He wrote his 1984 essay in a highly critical response to a book titled *Beyond Neo-Darwinism* by Mae-Wan Ho and Peter Saunders whose naïve and crude formulation of evolutionary theory he described as a "caricature" (Mayr 1984a, b). Similarly, Erik Svensson (Chap. 11) warns against using "Neo-Darwinism" in conjunction with "modern synthesis" and notes the confusion and destruction the conflation of these two terms has engendered. Examining the use of "Neo-Darwinism" for over 100 years now, I would simply say it carries too much baggage of all kinds to be useful and should only be explicitly used as a historical actor's term, the actor being Romanes.

Some other important terms that I think should be examined and used with greater care in the way of drawing distinctions include:

1. The synthetic theory of evolution, which refers to the theory of evolution as it emerged during the period of the evolutionary synthesis that is also carried over to contemporary use, and too often used interchangeably with the modern synthesis and Neo-Darwinism (see also Beatty 1986)
2. Evolutionary biology, which refers to a scientific discipline that emerged during the period of the evolutionary synthesis, where discipline refers to *both* a community or a collective of scientists interacting through organizations and societies and journals identifying themselves as members, as well as epistemic beliefs, practices, scientific methods, textbooks of instructions, rituals, and commemorations. Discipline, it should be noted here, must never be confused with only the social aspects of science; by definition, it embodies science. It is, in short, a way of getting at a collective view of what will count as the science of

evolution, in both its theoretical and practical formulations. Contextualism here can help us out because it collapses the internalist view of science and the externalist view of science (Smocovitis 1996)

3. *The* synthesis, which is a convenient, if vague shorthand, an abbreviation to refer to a constellation of some mixture of the above, and at times even all of the above or used to imply non-committal neutrality.

Sadly, all the terms I have examined are frequently used not just casually, but interchangeably, almost as a rule. This has led to an inordinate amount of confusion and miscommunication (Chap. 11). If we situate these in history, and if we properly *historicize* them (e.g., "Modern Synthesis," "Neo-Darwinism"), we might be free to move forward with more appropriate language for dealing productively with not just the past, but also the present.

In this section, I have tried to do three things. First, I have tried to draw distinctions between important words and phrases or terms that are used interchangeably, but in fact refer to different things. Second, I have offered something of the historical backdrop surrounding some of these terms in the way of filling in the historical picture for the reader, and third, I have drawn on a number of historical publications including my own, because they attempt to write history *on its own terms,* using the full set of resources available to the historian. By that, I mean the reflective, methodologically precise means of working with historical discourse drawn on archival publications and correspondence, and not just published work. This latter point needs underscoring because working only with scientific publications gives us only one aspect of the historical development of evolutionary biology and a distorted one at that. Publications may be the preferred source for much of the old-fashioned history of ideas, and useful if read as contextual, circulating discourses, but are nonetheless often inadequate when used alone. In this, Peter Medawar's famous question about the "fraudulence" of the modern scientific paper (Medawar 1963) has more than a kernel of truth to it. It was an exaggerated claim, of course, but he was right in that science is messier than is generally acknowledged, and this is rarely if ever revealed in the sanitized formatting of the modern scientific publication; to get at a more faithful, and inclusive understanding takes digging into sources commonly located in historical archives, such as correspondence, diaries, field and lab notebooks, videos, oral history interviews, or at times even dusty attics, in addition to work with publications.

2.4 Moving Targets, Déjà Vu Moments, and the Importance of History to the EES

Ironically, just as the 1980 volume was enrolling scholars into the study of the evolutionary synthesis, just as the term itself was entering the historian's vocabulary and making it into a historical event subject to scholarly scrutiny, a body of literature began to appear that began to challenge its meaning, its significance, and its status in evolutionary theory, which itself was thought to need amendment, completion,

Table 2.1 Challenges to the synthesis I: challenged, unfinished, or dys-synthesized. From Smocovitis, V.B. *Oxford Bibliography*, "Modern Synthesis," 2019

Publication	Summary
Antonovics, Janis. 1987. The evolutionary dys-synthesis: Which bottles for which wine? American Naturalist 129:321–331	Offers historical perspectives on what the evolutionary synthesis did not achieve and calls for a dismantling of the methodological and conceptual difficulties that continue to plague evolutionary biology
Eldredge, Niles. 1985. Unfinished synthesis. Biological hierarchies and modern evolutionary thought. New York: Oxford University Press. [ISBN: 0-19-503633-6]	Book-length treatment arguing for a rethinking of the modern synthesis in terms of palaeobiological reforms that stress different evolutionary processes operating at different levels
Gould, S. J. 1980. Is a new and general theory of evolution emerging? Paleobiology 6:119–130	Building on theoretical developments from paleobiology such as punctuated equilibrium, S. J. Gould's paper called for a new synthesis of evolution
Gould, S. J. 1983. Darwinism and the expansion of evolutionary theory. Science 216: 380–387	Major article calling for a revision and expansion of the modern synthesis of evolution
Futuyma, Douglas J. 1988. Sturm and drang and the evolutionary synthesis. Evolution 42: 217–226	Tempered review of controversies over the modern synthesis and whether or not it required revision on the occasion of the 40th anniversary of the journal Evolution, founded in the wake of the modern synthesis
Mayr, Ernst. 1984. The triumph of the evolutionary synthesis. Times Literary Supplement, 2 November, pp. 1261–1262	Spirited defense of the modern synthesis for the general reader
Ho, M.-W., and P. T. Saunders, eds. 1984. Beyond Neo-Darwinism: An introduction to the new evolutionary paradigm. New York: Academic Press. [ISBN: 9780123500809]	An edited collection of papers that attempted to argue that Neo-Darwinian theory was over and required a new paradigm to explain new phenomena
Stebbins, G. Ledyard, and F. J. Ayala. 1981. Is a new evolutionary synthesis necessary? Science 213:967–971	Defense of synthetic theory by one of the chief architects and his collaborator in evolutionary genetics in response to Gould (1980) and others

overhaul, or dissolution. Indeed, Mayr's 1984 essay, titled "What is Darwinism Today?" was part of a cluster of publications he produced in response to work that began to appear in the late 1970s challenging the synthesis. The body of literature drawing attention to it was huge, and the titles alone point to the stunningly diverse and confused configurations that demonstrate the interchangeable use of the terms modern synthesis, evolutionary synthesis, Neo-Darwinism, and synthetic theory. Table 2.1 presents a sampler that I compiled for the *Oxford Bibliography* on the Modern Synthesis published in 2019.

Additional titles convey the overall arc of the debate: *The Evolutionary Synthesis is Only Partly Wright, But Not Wright Enough* (a reply to the former), *Beyond Darwinism? The Challenge of Macroevolution to the Synthetic Theory of Evolution,*

Evolutionary Theory at a Crossroads: The New Biology and Philosophy of Science, Challenges to the Synthesis, The Synthetic Theory Strikes Back, and *Darwinism Stays Unpunctured.* These essays, articles books, and peer-reviewed publications were echoed in the popular press, which, in the early 1980s had become especially attuned to science. Editorials, columns, and feature articles in *Science* and *Nature* painted a picture of the evolutionary community in turmoil, at times even in crisis, with leading evolutionists feuding with each other in print, at conferences, or even on the television set (see Smocovitis 1996 chapter two for details on the extreme rancor and silliness engendered by some of these disputes that included a lot of political posturing over areas like sociobiology and its grounding in the synthesis). To survey it today, one would think that an epic battle were being waged over the heart and soul of modern evolutionary biology, instead of a sometimes lively but also contentious and sometimes confused set of conversations between evolutionary biologists about the synthesis in the 1980s. Needless to say, the feuds did little to resolve long-standing issues of methods, fields, mechanisms, level of evolution, organismic models, or other particularities of evolutionary biology though they did accompany the emergence of a discipline, or subdiscipline known as paleobiology (Smocovitis 1992, 1996; Sepkoski and Ruse 2009; Sepkoski 2012) and garner attention, if not fame for some of the participants. It also attracted a number of philosophers to evolution interested in the structure of evolutionary theory, which they quickly discovered defied any model of a scientific theory; they variously described as a "supratheoretical framework," a "hypertheory," and a "metatheory" (Burian 1988; Wasserman 1981; Tuomi 1981; Caplan 1978). Other philosophical literature disputed that the synthesis happened (Rosenberg 1979; van Balen 1988) while yet others focused on the integrative features of the synthesis to understand integration within scientific fields of study (Bechtel 1986). Added to the mix of historical perspectives from the 1980 workshop and the feuds between evolutionists reaching the popular press, philosophical inquiry thus led to even more confusion over terminology, and basic historical understanding. In short, the 1980s were full of conversations about "the synthesis," with the word evoking such an interpretive mess that philosophers surveying the history described it as a "moving target" (Burian 1988; and see Gayon 1990 for a useful review of the 1980s).

Interestingly, the disputes abated by the late 1980s to early 1990s but enough of the synthesis, or modern synthesis, or synthetic theory as well as Neo-Darwinism, in all their varied configurations appear to have remained sufficiently intact for the next generation of critics who began to appear sometime early in the next century, following a stunning range of developments in evolutionary science starting with developmental biology, long thought to have been excluded from the evolutionary synthesis (Waddington 1953; Amundson 2005 and see Fábregas-Tejeda and Vergara-Silva 2018 for a more recent treatment). First came calls for an "expansion" of the synthesis (Kutschera and Niklas 2004), and then calls for an "extension" in 2007 (Pigliucci 2007), which drew renewed but critical attention to the "modern synthesis" after a well-publicized meeting in Vienna that resulted in an edited collection (Pigliucci and Müller 2010a, b) that precipitated a new round of conversations and disputes. A rash of literature began to appear accompanying

what was perceived as new ideas or new phenomena in evolution being studied or entirely new areas of inquiry emerging that include niche construction and ecology, epigenetic inheritance, plasticity and accommodation, modularity and evolvability, and genomics and network theory (see Scheiner and Mindell 2020 for a more detailed explication of these topics; and see Lewens 2019 for a survey). It all culminated with the appearance of two back-to-back articles in 2014 in the pages of *Nature* by Laland et al. (2014), and the response by Wray et al. (2014) that resulted in an explosive series of conversations and debates enabled by social media, which at times energized the community of evolutionists, and at other times led to outright hostility as well as more silliness in some of the more extreme reactions. My sampler of 2019 only highlights some of this literature (Table 2.2):

Reading it one experiences a déjà vu moment because of the parallels with the 1980s, especially in the increasingly rancorous style of argumentation often at cross purposes. Indeed, as Erik Svensson (Chap. 11) has noted, some of this literature joining the fray has already proclaimed that evolutionary biology is experiencing another "crisis" (Dupré 2012). The extent to which it actually is in such a "crisis," and parallels the disputes of the 1980s merits further exploration, though in my mind at least, the level of emotion engendered by criticism of existing evolutionary theory bears a striking resemblance to the 1980s. But apart from sharing the sense that evolutionary theory needs amendment, either in the way of an expansion, extension, or overhaul, are there any commonalities in these works which seem to continue to snowball? From the perspective of a historian, the answer is yes, there do appear to be some commonalities, at least in my reading from most of the accounts traveling under the banner of the "extended evolutionary synthesis," commonly abbreviated to the "EES."

First, as with the criticisms of the synthesis in the 1980s, terminological and conceptual confusion abounds. Starting with the naming of EES, we have descriptive references usually made to the extension of not something called the "evolutionary synthesis," as we would expect, but instead for the "Modern Synthesis", capitalized, or "MS" sometimes used interchangeably with the historical event of the evolutionary synthesis, as well as with the "standard evolutionary theory" or the "SET." Given the frequent reference to the MS one would have thought that "extended modern synthesis" would have made a more precise name instead of "extended evolutionary synthesis." And then, too little of a substantive historical nature is stated about Huxley's own version of the modern synthesis and little or no attempt is made to historicize or contextualize it, treating it instead as some transcendent, essentialized entity. Indeed, the MS which should ideally refer to Julian Huxley's synthesis is used to refer to the synthetic theory, and the historical event of the evolutionary synthesis, as though the synthetic theory emerged intact in the period of the 1930s–1940s and then became frozen in time, immutable as it were, and often working to the detriment of pluralism in the field. Adding to the confusion, the more problematic term of Neo-Darwinism is thrown in often as well. Second, the question is often raised as to whether the SET served to exert a constraining, restrictive or at times even hegemonic role in the development of evolutionary biology. In other words, often there is an argument made that x, or y, or z got left

Table 2.2 Challenges to the synthesis II: extended, expanded or overhauled? From Smocovitis, Oxford Bibliography, "Modern Synthesis," 2019

Publication	Summary
Carroll, Sean. 2008. Evo-devo and an expanding evolutionary synthesis: A genetic theory of morphological evolution. Cell 134: 25–36	Lucid article explaining why developments in evo-devo pose problems for the modern synthesis
Dickins, T. E., and Q. Rahman. 2012. The extended evolutionary synthesis and the role of soft inheritance in evolution. Proceedings of the Royal Society B: Biological Sciences 279: 2913–2921	Useful overview for understanding why the turn to epigenesis creates a need for an extended synthesis
Jablonka, Eva, and Marion J. Lamb. 2008. Soft inheritance: Challenging the synthesis. Genetics and Molecular Biology 31:389–395	Argues soft inheritance and the mechanisms underlying epigenetic inheritance challenge the modern synthesis of evolution, whose theoretical framework is inadequate for modern evolutionary biology
Kutschera, U., and Karl J. Niklas. 2004. The modern theory of biological evolution. An expanded synthesis. Naturwissentschaften 91: 255–276	Early paper describing inadequacies of the modern synthesis and presaging the appearance of the extended synthesis
Laland, K., Tobias Uller, and Marcus Feldman, et al. 2014. Does evolutionary theory need a rethink? Yes. Urgently. Nature 514:161–162	Polemic multi-authored piece drawing attention to the need for a revision of the modern synthesis
Müller, G. B. 2007. Evo-devo: Extending the evolutionary synthesis. Nature Reviews Genetics 8:943–949	Brief argument summarizing the need for an extended synthesis
Pigliucci, M., and G. Muller, eds. 2010. Evolution: the extended synthesis. Cambridge, UK: Cambridge Univ. Press. [ISBN: 9780262513678]	Edited collection with a range of papers by scientists and philosophers in support of an extended synthesis and against the modern synthesis
Pigliucci, M. 2007. Do we need an extended evolutionary synthesis? Evolution 61:2743–2749	Brief argument laying out the rationale for an extended synthesis
Wray, G. A., Futuyma, D. A., Lenski, R. E., MacKay, T. F. C., Schluter, D., Strassman, J. E., Hoekstra, H. E. Does evolutionary theory need a rethink? No, all is well. Nature 514: 161–164	Defense of the modern synthesis in response to Laland et al. (2014)

out, or was marginalized in some way, where x, y, or z is usually an entire field, an area of inquiry dominated by an individual or a group, or even a phenomenon, so that the progress of evolutionary science has been hampered or stalled by this absence. Yet little in the way of historical evidence is presented for this, and this is certainly the case for the belief that some deliberate act or an individual excluded, say embryology which was about to be transformed to developmental biology (see Horder 2010 for one history of developmental biology; and see Fábregas-Tejeda and Vergara-Silva 2018 for a substantive discussion of the issues). Third, and finally, starting especially with Pigliucci (2007), the edited collection by Pigliucci and

Müller (2010a, b), and continuing to Laland et al. (2014) we see at least one more very important thing in the EES literature: a grounding for these arguments in *history,* either explicitly or tacitly, especially in reference to the MS not just used interchangeably with the SET, but often accompanying the belief in some kind of standard historical narrative, though as I have noted above, no such thing exists, unless of course one wants to buy into Mayr's narrative, a naïve and uncritical, or even possibly even a dishonest proposition. Whatever the case, in the EES, history matters and serves as a foundational starting point in making the case for the inadequacy of the MS or the SET today. As a recent philosophical survey of the same literature in EES notes: "[t]he debate over the EES is in part a historical one" (Lewens 2019, p. 711). I entirely agree.

Ironically, however, the published work on the EES rarely references works of history in any significant, or meaningful way, let alone engage it, including even referring to the Mayr and Provine (1980) volume, which, as I noted above, is the scholarly entry point for a historical understanding of the evolutionary synthesis. Laland et al. (2014) for example make a point of noting that the core of current evolutionary theory was forged in the 1930s and 1940s and then proceed to relay the "story" of the SET along the lines of a standard historical narrative of some sort, yet the paper does not reference any historical work, nothing in the way of a secondary source nor even a proper primary source in support of its historical claims. Even Pigliucci and Müller in their too-brief "conceptual history" justifying the need for the EES that introduced their 2010 co-edited volume engages only a handful of well-known historical publications lightly, and make serious errors of omission and downplay, if not dismiss entirely the work of Simpson and Stebbins and even Mayr, though they do elevate Huxley, as I will show in the next section (Pigliucci and Müller 2010a, b). Reading this body of literature, we are left to think that not only is the history of the evolutionary synthesis a settled past, but that the entire history of evolutionary biology has been written, agreed-upon, and cast in stone, but strangely enough irrelevant to actually reference carefully, or to explore further. One also gets the sense that a well-defined body of evolutionary theory crystallized and emerged in the 1930s and 1940s and continued mostly unchanged as the SET from the period of the 1940s onward, at times serving to choke off dissenting opinion, the latter point being a kind of subtheme in much of the EES literature as a whole. Tellingly, few or no historical details charting most of the twentieth century is ever provided, and historical works are not properly consulted, nor are historians included in the abundant conferences, collections or in the many collaborative interdisciplinary efforts; yet Laland et al. (2014) keenly note the interdisciplinary nature of the EES effort, though interdisciplinary here appears to mean the inclusion of a small group of biologists and one philosopher, as with the earlier Vienna meeting by Pigliucci and Müller, which included some 16 participants, 14 of whom were scientists, and two of whom were philosophers (Pigliucci and Müller 2010a, b). My concern here, it should be noted, is not so much with the exclusion of historians, but with the treatment of history because the historical record itself, is almost never consulted, and if primary sources are included, they are selected to bolster a presentist view, devoid of context or historicist thinking (see also Dickins 2021 for

further treatment of this history). The synthetic theory, or the MS used interchangeably emerges, intact, as though it were assembled in one piece, without much labor or work, and consensus between a large and disparate group of evolutionary scientists appears to have been reached rapidly, staying that way largely until the beginning of the next century when calls are made to reform it. Curiously, little mention is made of the tumult of the 1980s and what happened afterward. Pigliucci and Müller do tell us in their introduction that scientists do not have the time to be reading the original published works, though of course that does not stop them from coming up with their own historical narrative. To be sure, practicing scientists might not have the time to be digging into archives and correspondence and engaging in critical historical methods, recalling events of the 1980s, or even in reading every single published work of the period, but why then ignore the works of historians who do, and who may have come up with some helpful insights? Where, furthermore, is all this history that is being deployed as the background narrative justifying the call for reform or amendment coming from, precisely? Does it emerge from textbooks, or is it mere mythological lore retold by practitioners as part of some process of enculturation? Astonishingly, Pigliucci and Müller, do in fact use a textbook definition of the MS in their brief history, quoting a paragraph from *Evolutionary Biology* (Futuyma 1986), as proof of the existence of their understanding of the MS or SET which supposedly emerged in the 1940s and continued unchanged to the late 1980s (Pigliucci and Müller 2010a, b; see also Dickins 2021 for more detailed analysis of Futuyma's textbook). To sum up: although history is being evoked and used to justify the need for the EES, it is incomplete, and so rudimentary, that it resembles a caricature, not just of evolutionary theory itself, but of its historical emergence as well as its subsequent evolution, or lack thereof. At best, it is a kind of "scientist's history" because it is deployed against a kind of convenient caricature, literally a textbook account, to make the case for EES; at worst, it is no real history at all; indeed, at times it is weirdly *ahistorical*, especially for an argument that rests on a foundation of history.

2.5 History and the EES

What is missing in the EES history, in short, is the actual history, which happens to be well worked especially in tracing the overall arc of the development of evolutionary theory after Darwin (see Provine 1986; Ruse 1996; Gayon 1998; Hodge 2008, 2009; Bowler 2009). The historical record does not support the view that a monolithic, or a standard evolutionary theory (SET) appeared during the period of the evolutionary synthesis, or even immediately afterward during the years around the 1959 Darwin Centennial when some, like the late Stephen J. Gould, looked back, and argued for the rise of a "new orthodoxy" and the "hardening of the synthetic theory around a selectionist core" (Gould 1983a, b). Gould's own historical turn, as we recall, was part and parcel of the first round of challenges to the synthesis that he was leading to bolster his critique of the adaptationist program (Gould and Lewontin 1979). That is why Gould wrote the introduction and was behind the reissue of

Goldschmidt's 1940 text, *The Material Basis for Evolution* with Yale University Press which did not just resurrect Goldschmidt but reinvented him as a "heretic" and anti-hero (Goldschmidt 1940; new introduction by S. J. Gould 1982). But when it came to understanding the history and structure of evolutionary theory, however, Gould struggled with finding a conceptual core and tracing its history; it took him some 1500 pages to attempt that (Gould 2002).

Interestingly, a similar resurrection and reinvention was attempted, this time of Julian Huxley by Pigliucci and Müller with the reissue of his 1942 book as the "companion" volume to their collection of 2010. In the foreword, Huxley is described as an "intellectual giant", and a "first-rate scholar," whose book introduced the Modern Synthesis, which laid down the "conceptual structure underlying all of evolutionary biology for the twentieth century." Huxley is also praised for his pluralism and treatment of complex topics like species definitions and speciation, but also for providing a "breathtaking panoramic view" of evolution that showed "academic rigor and scholarship" (Pigliucci and Müller 2010b). Curiously, they describe the book as controversial, and while they take pains to show that it was an unusual hybrid of a semi-popular work, they do not say much more about its reception or impact within the evolutionary community, only that it sold many copies and went through multiple editions. That it may have sold copies because it was intended for a wider audience, and did not necessarily have much impact on the evolutionary community itself is not sufficiently noted; and although Huxley was indeed a major public intellectual whose influence in promoting evolution, "modernized" as it were, cannot be understated, he also engendered controversy, not so much for his actual science, but because of his overbearing personality, his reformist zeal, as well as his many political engagements. Huxley ruffled more than a few feathers and disrupted more than one institution with a unifying evolutionary vision that he often impatiently and unilaterally tried to implement. And while some viewed him as polymathic, he also stretched himself thin in his many organizational, administrative, and public duties, coming across more as an intellectual gadfly than a focused scientific researcher. Combined with his mental health struggles, these attributes, all contributed to an erratic and turbulent scientific career (Armytage 1989; Waters and Van Helden 1992; Smocovitis 1999, 2016; Cain 2010). Thus, the attempt to reinvent him as a daring, original, and controversial scientist pushing the envelope in evolutionary theory and laying down the "conceptual structure" of the science for the twentieth century is not entirely accurate, though of course it might suit Pigliucci's and Müller's historical justification for the EES.

A focus on Huxley's role in the synthesis is warranted, moreover, and there is much historical work that remains to be done on the impact of his 1942 book, but if the attempt is to argue that the conceptual structure introduced in his book was the basis for all of evolutionary theory embodied in the MS or SET, and that it remained unaltered in the twentieth century because one can see it, or echoes of it in Futuyma's textbook of 1986, then that would be doing a serious injustice to the historical record. For one thing, it misses what happened concurrently, namely the publication of Mayr's 1942 book, the systematist's response to Dobzhansky's book of 1937, and what happened afterward including the publication of Simpson's 1944 book which

addressed rate and mode of evolution drawing on observable evidence in the fossil record, as well as Stebbins's 1950 attempt to reconcile variation and evolution in the plant world with that of animals; this is in addition to ignoring the abundant work by individuals like the Carnegie team of Jens Clausen, David Keck and William Hiesey and others (Núñez-Farfán and Schlichting 2001). It would entirely miss the many rounds of negotiations, the give and take, the back and forth, the moments of agreement and disagreement that were all part of a process of exchange and consensus-building between the many individuals involved in the synthesis, their disciplines, their methods, and their organismic systems, and the extent to which consensus was actually reached, if at all. Indeed, the fine-grained level required to understand consensus-building, discipline-building, or theory construction, would be entirely lost, as would the intricacy and importance of the collaborations leading to "trading zones," the crucial moments of exchange between theorists and practitioners, one of the more fascinating aspects of the history of evolution during this period of time (Galison 1999). And it misses the fact that architects like Mayr and Stebbins thought Huxley's book was incoherent and superficial because it was an unoriginal semi-popular book; they were of course writing their own. This is what the advocates of the EES commonly miss and is the case with Pigliucci and Müller's introductory essay in their co-edited book as well as their foreword to Huxley (1942) (Pigliucci and Müller 2010a; Pigliucci and Müller 2010b).

Yet another aspect that is rendered invisible in EES histories is the extensive international sociopolitical infrastructure enabling, and indeed shaping the emerging new science of evolutionary biology: the many conferences, workshops, and other important organizational efforts that enabled the communication between individuals from wide-ranging areas who increasingly looked to each other and to their understandings and methods in solving common problems of evolution (Smocovitis 1992, 1996). Starting in the mid-1930s groups such as the Biosystematists (originally the Linnaean Club) on the west coast of the USA brought together individuals who shared concerns in systematics and evolution and sought new methods from genetics and cytology for systematic purposes and to understand mechanisms of speciation and build workable phylogenies. Huxley himself had been active in organizing events and symposia in the UK that led to books like his edited *The New Systematics,* and Ernst Mayr and A. E. Emerson were instrumental in forming the Society for the Study of Speciation, which gave way to the National Research Council-backed Committee on Common Problems of Genetics and Paleontology later changed to Committee on Common Problems of Genetics, Paleontology and Systematics at the request of Mayr—all a series of organizations that eventually led to the founding of the first international organization after the war in 1946, The Society for the Study of Evolution, and the launching of the first international journal *Evolution.* Such a journal served as the forum for the exchange of ideas in the growing community but that also inadvertently served to determine what would be included, and what would not. During the war years, furthermore, when travel to conferences was difficult, a series of long, substantive, and formal epistolary exchanges between evolutionists were mimeographed and distributed to interested members. These bulletins, comprised of 115 pages in four volumes edited

and distributed by Ernst Mayr remain the best existing source to show the active process of negotiation taking place between evolutionists, the precise positions being held and what mattered the most to participants, in addition of course, to the more private correspondence deposited in archives. Importantly, the final bulletin included a formal notice by G. G. Simpson, just returned from war service, that a field common to the disciplines of genetics, paleontology, and systematics had emerged which pointed to a growing consensus, though the details remained to be worked out (Simpson 1944b).

Sources such as these matter. They tell us something about the points of agreement, but also the points of disagreement, which, despite the growing consensus that a new unifying discipline named evolutionary biology was emerging, remained considerable. This was apparent, when, in 1946 a large international group of evolutionists under the auspices of the Committee on Common Problems of Genetics, Paleontology and Systematics came together at Princeton University on the occasion of Princeton's Bicentennial for a conference on common problems in genetics, paleontology and systematics (Smocovitis 1992; Cain 1993). The co-edited collection and accompanying materials that appeared in 1949 serve as a crucial historical source, in revealing what, if any consensus had emerged (see also Jepsen and Cooper 1946 for a preliminary summary, the program, the participants, and a photograph). The foreword by Glenn L. Jepsen explicitly notes that the conference and volume did not reflect a "single synthesis of its three titular subjects, i.e., genetics, paleontology and systematics", but instead comprised a "compound of data, of ideas, and of conclusions" and that researchers were given "aggressive encouragement to stray beyond the conventional limits of their subjects." Indeed, according to Jepsen, the authors included in the collection were "subjected to close association with practitioners in other fields" at the conference and "many critical, penetrating, and sometimes embarrassing questions and comments were exchanged in an attempt at mutual education" (Jepsen et al. 1949, p. viii).

Reading the historical sources, one does not get the impression that some crystallizing moment took place, or that any hard consensus was reached; the final paper in the Jepsen volume, the summation on the meeting as a whole by geneticist H. J. Muller, spoke directly to a "common ground of theory" that made possible a "convergence" of evolutionary disciplines like genetics, systematics and paleontology and the emergence of a new and "synthetic" type of evolutionist (Muller 1949). It is important because it remains one of the few clear declarations of what constituted the core elements of the synthetic theory, and the points of agreement, here paraphrased as: (1) that natural selection was the primary mechanism for evolutionary change; (2) that it operated at the level of small, individual differences making evolution a slow, gradual process; (3) and that the same evolutionary process that operated at lower levels, for example, beneath the species, also accounted for higher-order phenomena (this is frequently framed as the continuum between microevolution and macroevolution which Dobzhansky had promoted (Adams 1968, 1980)). Muller's tone was celebratory, but also reflective, and pointed to the way to work that remained to be done, as well as the dangers facing humanity in the aftermath of a bloody world conflict.

Taken as a whole, furthermore, the summation that Muller provided, indicated that no new, revolutionary or conceptually profound ideas had emerged, and that the points of agreement were not necessarily emphasized to the same extent by all the participants or all individuals keen to align themselves with the new science of evolutionary biology. Let us recall that Richard Goldschmidt became one of the best-known dissidents right from the start (Smocovitis 1992; Dietrich 1995), and that Ivan Schmaulhausen (1949) and Conrad Waddington (1953) pointed to the inadequacies of the synthesis because its emphasis on mathematical population genetics left out embryology and other fields (Gilbert 1994). Even at the 1959 Darwin Centennial which was intended to be a great celebration—and a public demonstration of unity—a voice of discontent was heard from paleontologist Everett Olson who called for greater attention to phenomena in paleontology (Olson 1960; Smocovitis 1999). The same consensus, furthermore, could easily fall apart on closer examination. As Mark Adams recently showed, some participants showed appreciable resistance to the microevolution and macroevolution continuum (Adams 2021), and even good friends like Dobzhansky and Stebbins, whose 1950 book attempted to align plant evolution with the general processes of evolution in Dobzhansky's 1937 book disagreed, and hugely, over the relative importance of phenomena like hybridization when they actually tried to collaborate for a textbook on general evolution (Smocovitis 2006). Muller's brief summation is thus consistent with Provine's sense that what happened to evolutionary theory in the interval of time between 1930 and 1950 involved not so much building on any one new idea or development, as slowly reworking, and eliminating rival or alternative theories of evolution. There is little historical support for a bold new paradigm of evolution, any one crystallizing moment, or even a monolithic standard evolutionary theory emerging at that time. What was notable in the late 1940s instead, was a spirit of consensus and agreement, especially centering on the efficacy of natural selection as a primary mechanism of evolution, and a growing community of practitioners who redefined themselves as evolutionary biologists, and who began to function as the unifiers of biology.

Nor was the loose consensus and agreement an end in itself. Calls for greater synthesis, unification, and integration continued to be made, building on those of Schmalhausen, Waddington, and others, including J.B.S. Haldane, one of the mathematical modelers who himself saw the synthesis only as a kind of stage, a "developmental instar" in a path toward a maturing evolutionary theory. Thus, whatever, the deficits and criticisms, the loose consensus continued to become more inclusive, as a process of synthesis and integration brought disciplines like anthropology into alignment with evolutionary biology. Elsewhere, I have traced some of the major developments following the evolutionary synthesis of the 1940s leading to the watershed moment of the Darwin Centennial of 1959, because it demarcated the long struggle to establish Darwin's theory of natural selection, and Darwin himself as a kind of "founding father." It also brought together a stunning assortment of workers who agreed that evolution could serve as the unifying principle of modern biology. But while the centennial served to draw attention to what the core disciplines would be, it also brought attention to what remained to be included, a

process that continued well into the 1960s following first an apparent clash with the newer and more reductive science of molecular biology, and then with the integration of molecular methods that led to amendments and modifications in evolutionary theory. That history has been charted, albeit briefly (Smocovitis 1999, 2020). Here, I need only note that a process of integration continued as areas like development were slowly integrated with evolution, and at a much earlier time than the 1990s as stated by some advocates of EES. That process began as early as the late 1950s, when architects like Ledyard Stebbins explicitly recognized the importance of the gene to character transformation and reoriented research to developmental genetics (Stebbins 1965). A greater emphasis on history, and one more inclusive of plants and other organismic systems as well as animals, would have revealed that development was not being actively excluded by evolutionary biologists, but that by the 1960s, Stebbins, Dobzhansky and others had begun to speak more explicitly about its importance as well as the importance of integrating wider areas of the biological sciences. Yet another emphasis, this time on language historically traced, would also show that the language of "synthesis" and "unification" was being slowly replaced by the language of "integration," culminating with the emergence of yet another academic discipline traveling under the banner of "integrative biology" (Wake 2001, 2003, 2008). The synthetic theory of evolution did appear to be challenged from within, by evolutionary biologists during the period of discord in 1980s, and while some made considerable noises that the synthetic theory was inadequate, and in need of reform or disassembly or overhaul, others continued to work quietly at amendment, revision or reintegration. At present, what counts as the synthetic theory, the MS, or even the evolutionary synthesis, its central tenets and first principles appear to vary (e.g., Kutschera and Niklas 2004; Scheiner and Mindell 2020), depending on the particularities of the field, the experimental system, the methodology, and interestingly enough individual preference and generational factors. It nonetheless has abundant instantiations, provides rich resources for investigation for a wider and more inclusive community of workers, and retains its explanatory power. Those unifying properties articulated by people like Huxley and others, are still upheld by a wide community of workers but especially by integrative biologists (Wake 2001, 2003, 2008), who I view as the successors to the synthesis, and can be seen to be especially productive in extending its reach to areas like medicine, agriculture, computer science, and even historical and cultural modeling. For good reason, some speak of theoretical and applied evolutionary biology, and as Vidya et al. note that is because "evolution is far bigger than all of us" (Chap. 7). So, to close this section of my chapter on history and the EES, it might be more useful to think in terms of multiple evolutionary theories that work productively for biological investigation, instead of one monolithic or standard evolutionary theory, as the EES advocates seem keen to promote (Scheiner and Mindell 2020; Dickins 2021). Although subject to interpretive bias, as Becker and others have noted, the historical record based on primary sources, on critical historical and linguistic methods, that is inclusive of social and political dynamics, and not just philosophy, gives us a far more complex and nuanced view of the history of evolution, evolutionary theory, evolutionary biology, and even the so-called "MS."

2.6 "Every Evolutionist Their Own Historian": Summation and Closing Thoughts

In this chapter, I have drawn on Carl Becker's famous essay to make a point about history and the evolutionary synthesis and arguments for the extended synthesis: history matters, and hugely. But historical reckoning is everywhere in the history of evolutionary biology starting from Julian Huxley's disciplinary narrative setting up his view of the "modern synthesis" of evolution to the most recent arguments for an extended synthesis. It figures in the work of evolutionary biologists like Stephen J. Gould but especially Ernst Mayr who played important roles in writing it and rewriting it to bolster claims about evolutionary theory, demarcate the boundaries of evolutionary biology, and marshal support for making the historical event of the evolutionary synthesis, as an important historical event. I have pointed to reasons for confusion and miscommunication mostly based on imprecise terminology and asked for greater sensitivity to context and the historicity of ideas, beliefs, practices, including theories and to a more critical use of a diverse body of sources beyond published works. I have drawn attention to the absence of any one dominant historical narrative on the synthesis coming out of the workshops organized by Mayr and Provine and subsequently by other scholars; and I have drawn on my own past historical work and that of other historians to show that there was no one, monolithic evolutionary theory to emerge out of the 1940s, and to continue unchanged until the EES. Indeed, I have argued that there was a loose consensus, and what did emerge was minimal in nature, something involving more of a reworking, or refinement, involving the elimination of existing understanding that is much more consistent with a kind of pluralism that I believe continued—and continues—to characterize evolutionary theory and evolutionary biology today.

As I have also noted, the challenges to the synthesis today seem to bear a noteworthy resemblance to the challenges of the 1980s, which did not lead to major upsets or to some revolution in evolutionary understanding of the natural world. Indeed, little actually unraveled then, so that there is reason to think that little is unraveling now, at least so it seems to this historian. A more careful emphasis on language, an acceptance of the historicity of ideas, beliefs, practices, and even theories, as well as greater attention paid to history, as subject as it is to interpretive bias, indeed recognizing the very fact that it is going to be biased, may help lead to greater understanding, communication, and indeed to what I would like to think is better science. Finally, let me state that the "synthesis" has actually moved on, morphing into "integration" in biological circles, and although it remains a topic of scholarly interest in historical and sociological circles, it is best left out of active, productive scientific conversation, unless of course it is accompanied by critical historical inquiry. Ironically enough, what has kept it alive and relevant in scientific circles are some of the endless conversations and unnecessary disputes surrounding the EES.

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Yes Indeed, Evolutionary Biologists Should Pay More Attention to History: A Commentary on Smocovitis

3

Erik I. Svensson

Abstract

Vassiliki "Betty" Smocovitis has done a thorough and admirable job in highlighting the complex history the Modern Synthesis (MS) or the Evolutionary Synthesis (ES) as she prefers to call it. Her interesting contribution should hopefully increase awareness that the history of evolutionary biology is important even for researchers today who are mainly interested in solving practical questions of more immediate importance. Indeed, evolutionary biology has a rich history, and that in itself should motivate more dialogues and communication between biologists and historians to avoid perpetuating strongly biased views in ongoing conceptual debates about the future of our field.

Keywords

Evolutionary synthesis · Extended evolutionary synthesis · History of evolutionary biology · Modern synthesis · Population genetics

Evolutionary biology is a historical science. By "historical" I do not mean only that the field is focused on the history and evolution of life itself, but also that the field has a rich and diverse intellectual history that we need to pay careful attention to, if we want to make progress, avoid repeating past mistakes and avoid re-iterating what has already been said in countless and old debates without adding substantially new arguments to move the discussion forward. While this might seem obvious to most of us—and even trivial to some—I believe a neglect of history and sometimes even a

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distortion of history has unfortunately shaped many of today's passionate debates about the state of evolutionary biology, its past, and the possible need for extension, including calls for a so-called "Extended Evolutionary Synthesis" or EES (Pigliucci 2007; Laland et al. 2014, 2015; Müller 2017).

Few academics are more qualified than Vassiliki "Betty" Smocovitis to critically evaluate the history of evolutionary biology and clarify the meaning and frequent use of terms like *"The Modern Synthesis," "The Evolutionary Synthesis,"* and *"Neo-Darwinism"* which are frequently used interchangeably and often conflated. Her chapter in this volume should be essential reading for anybody with a strong opinion about what the Modern Synthesis (MS) was, whether one is critical of it and wants to extend it (Pigliucci 2007) or whether one sees it as a positive historical scientific event or process, like I tend to do and many others (Charlesworth et al. 2017; Futuyma 2017; Svensson 2018). Smocovitis early work in her masterly historical overview *"Unifying Biology"* (Smocovitis 1996) and the arguments she presents in the chapter in this volume clearly show the heterogeneity and conflicting views among the "founding fathers" (Dobzhansky, Mayr, Huxley, Rensch, Simpson, and Stebbins, to mention only a few names) of the MS (or ES, for those who prefer that term). These scientists differed fundamentally in their views about the evolutionary process, what was important to study, the relationship between micro- and macro-evolution, the role of natural selection vs. drift and the role of population genetics vs. other fields like systematics, natural history, and paleontology. Contrary to what is commonly claimed by recent critics of the contemporary evolutionary framework (Pigliucci 2007; Laland et al. 2014, 2015) there exists no such thing as a *"Standard Evolutionary Theory"* (SET), nor did anything ever exist. Neither does there exist anything such as *"The Modern Synthesis Theory"* as claimed by other critics (Walsh 2015; Müller 2017).

Similarly, claims that the MS largely *is* population genetics (Pigliucci 2007) are historically false and have no empirical support, at least not from those who have actually studied the original sources, documents and correspondence between the scientists involved in the establishment of the MS, as Smocovitis repeatedly emphasizes in her chapter. One could instead point to considerable disagreements and public debates between influential figures in the MS, including the famous debate between Ernst Mayr and J.B.S. Haldane about the utility and limitations of so-called "bean bag genetics" (Crow 2008; Dronamraju 2011). In the more recent decades, we have also seen much research and heated debates about the relative importance of stochastic factors vs. deterministic factors like selection in evolution (Gould 1981; Orzack 1981; Coyne et al. 1997; Wade and Goodnight 1998; Blount et al. 2018). These scientific debates, which are still ongoing (Blount et al. 2018), challenge simplistic historical narratives about a monolithic MS and a dogmatic contemporary evolutionary framework that have coalesced around some rigid structure like SET or *"Modern Synthesis evolutionary biology,"* as has frequently been claimed by some critics (Pigliucci 2007; Dupré 2012; Laland et al. 2014; Walsh 2015; Dupré 2021).

Indeed, I believe that the main take-home message of Smocovitis' chapter should be this: evolutionary biologists (including myself) should pay more respect to history

and collaborate with historians, rather than trying to write their own "Whiggish History," whether to play up their own importance like Ernst Mayr frequently did (Mayr 1959, 1993) or whether to attack strawman historical narratives about the rigid and monolithic MS and sweepingly describe a conflict-free history of evolutionary biology (Pigliucci 2007; Noble 2013, 2015, 2021; Walsh 2015). Instead, the way forward in this debate is for biologists to humbly realize what Smocovitis is trying to convey: there does not exist a single "true" historical narrative of our complex field. There will most likely never exist any such a narrative. As I underscored in my own contribution (Svensson, Chap. 11, this volume), rather than having a single theory, we have several co-existing and partly overlapping conceptual and empirical frameworks in evolutionary biology, which include currents like adaptationism, selectionism, mutationism, neutralism, and various structuralist schools inspired by evolutionary developmental biology research ("Evo Devo"). To understand our history we also need to look beyond the scientific publications and pay attention to the intellectual and social infrastructure of the field, including the formation of scientific societies, organization of meetings, and the launching of scientific journals, including *Evolution* (Smocovitis 1996). We, therefore, need multiple and complementary *perspectives* on the history of our field (Mayr and Provine 1998), rather than trying to establish *the* historical narrative. Too often, debates in evolutionary biology and calls for *"reform," "extension,"* or even *"replacement"* have been initiated by critics who have explicated highly biased historical narratives about a monolithic and dogmatic research field that has not changed as part of their reform agenda (Gould 1980; Pigliucci 2007; Noble 2013, 2015; Laland et al. 2014; Walsh 2015). We can do better, I think. Rather than criticizing or defending the MS (or our own favorite narrative about it!), we should instead strive to genuinely understand the complexity of our field's rich history, as admirably clarified by Smocovitis in her nuanced chapter.

Paying more attention to the history of our field, and collaborating with historians, could help to avoid the spreading of myths, misunderstandings, and erroneous claims about evolutionary biology. For instance, it has been stated that physiology has been neglected in evolutionary biology and that consideration of physiology will *"rock its foundations"* (Noble 2013), which is a strange claim, given decades of past and still ongoing exciting research in evolutionary physiology (Bogert 1949; Garland and Adolph 1991; Huey et al. 2003). Hopefully, future debates in the coming decades about the current state and history of evolutionary biology will use Smocovitis' chapter as a starting point and hopefully the intellectual connections between biologists and historians will also become strengthened. Biologists and non-biologists alike should pay due attention to her chapter and its take-home message.

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History, Evolution, and the "Rashomon Effect": A Reply to Svensson

4

V. Betty Smocovitis

Abstract

This paper uses the "Rashomon Effect" to understand the inherent challenges in a pluralistic theory of science with an equally complex narrative history. It is a reply to Erik I. Svensson's, "The Structure of Evolutionary Theory."

Keywords

Rashomon effect · Pluralism · Perspectivism · Evolutionary theory · History

Erik Svensson has done an excellent job of summarizing the major points of my paper. As a historian, I cannot offer any real opinion or commentary about the need for, or legitimacy of, the "extended evolutionary synthesis" (EES), in its many incarnations, or engage in meaningful conversations about the future direction of the science of evolutionary biology and what it should include, reject, or amend. Instead, what I have tried to do is to draw attention to the use of history in evolutionary biology as a whole, and the fact that a historical process—or narrative-making about the past—has always been operating, and is still ongoing, shaping the contours of the field, or serving as a tool for unification, as well as in helping to determine the identities of its members, and its relations to related fields. I have drawn on this more general history to convince the reader that it is always part of the way we think about evolutionary science, though we may not always be aware of that, and more specifically to examine the relationship between history and the EES. Despite the fact that its advocates evoke history and use it as an important

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justification for extending the modern synthesis (MS), substantive engagement with the past is too often lacking, or often comes across as a convenient caricature instead of nuanced engagement with a complicated past. It is a tall order to ask practicing scientists to do that, of course, but formal or informal collaborations would be a productive start. In today's collaborative, team-oriented approach to science the inclusion of a historian does not seem that far-fetched. Barring that, an enhanced awareness of the critical methods of history, the importance of sources used, and even engaging the existing scholarship would help lead to better insights about evolutionary science. That Svensson seems keen on doing that, is no surprise, because his co-edited collection titled *The Adaptive Landscape in Evolutionary Biology*, included historians and philosophers of science (Svensson and Calsbeek 2012). Dedicated to exploring the meaning, significance, and use of the "adaptive landscape," one of the most powerful metaphors in evolutionary theory first evoked by mathematical population geneticist Sewall Wright (Provine 1986), the collection traced its history from 1932 to the present. It may serve as an example of the kinds of collaborations that would help historicize large and ambitious projects and offer critical perspectives that might lead to a better understanding of science, or at least help diminish some persisting misunderstandings. I do believe that combined with a greater sensitivity to language, and a more precise terminology, many of the more rancorous debates surrounding the EES might be avoided.

I am also very happy to note that Svensson appreciates the importance of sociopolitical aspects of science. By that, I do not mean what is external to science, or about how politics "gets in" but that the very process of doing science is itself a sociopolitical process. Svensson seems comfortable too with the view that science is historically rooted and culturally embedded practice, a view of science shared with people like the late Stephen J. Gould and the late Richard C. Lewontin. The point of the paper was to call for the historicity of ideas, beliefs, practices, and even theories, and to stress the processual aspects of both science and history, not always an easy way to think, though it should be, given that evolutionary is a historical science; Svensson has agreed with that too.

Finally, I am happy—relieved actually—that Svensson seems comfortable with the "every evolutionist his own historian" theme and with the perspectivism my paper brings out. It is not easy to live in a pluralistic universe and not easy to accept that there may be multiple evolutionary theories, or multiple narratives, each plausible or legitimate in some way but at times with contradictory elements. Questions framed by uncertainty that evoke positionality or perspective such as "what counts as the synthetic theory, and to whom?" or "whose historical narrative will we draw on to understand the past?" challenge us as epistemic beings, and can lead to a paralysis of sorts, if not blow more than a few fuses in our own thinking. We would all prefer a more simple "straight story" and a clearly structured and well-defined theory, a grand theory (Skinner 1985), if you will, that reliably helps us explain the world both including history and science. There rarely is just one simple story, or one singular monolithic theory, however, and we must find ways to accommodate to that. I do believe Vidya et al.'s (Chap. 17) claim that "evolution is bigger than us" is very helpful here, and with them, believe that whatever its configuration, evolutionary

theory still provides us with abundant instantiations, rich resources for future investigation, and can still retain in its explanatory power (Smocovitis 2020; Scheiner and Mindell 2020). We are also not alone in trying to come to terms with the mind-bending qualities associated with perspectivism, narratives, and historical truth. Let me close with a reference to a classic film from 1950 titled *Rashomon,* a word which, in Japanese means "dispute." Directed by Japanese director Akira Kurosawa, the film takes us through four eyewitness accounts of a violent rape and murder, each participant taking turns, but telling a very different story emphasizing varying circumstances and placing blame on different individuals. The film was so effective in drawing attention to perspectivism, that it gave rise to the term the "Rashomon effect" (Davis et al. 2015). It is maddening to the viewer to come to terms with this, especially with an ending that never tells us what actually happened and who is actually to blame, but that is the point of Kurosawa's direction; if we focus on the contradictions and the dispute, we miss entirely the points of agreement, and that it turns out might be the most important part—the fact that all agree that a violent crime was committed. No one disputes that. In the same way, a historical event like the evolutionary synthesis may be subject to varying interpretive claims, some of which are contradictory, but focusing only on that takes away from what really does matter, and that is that it continues to count as a historical event.

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Part II

5 The Creativity of Natural Selection and the Creativity of Organisms: Their Roles in Traditional Evolutionary Theory and Some Proposed Extensions

John J. Welch

Abstract

Biological adaptations appear designed for a purpose, and so they result from a "creative process" almost by definition. Traditional evolutionary theory assigns a special role in this process to natural selection, with theorists invoking selection both to explain the appearance of purpose, and to predict what the purpose of adaptations will be. At the same time, traditional theory recognizes that many other factors might influence the evolution of adaptations. These factors might, for example, increase evolvability and accelerate adaptation, or bias evolution towards a subset of the possible adaptive outcomes. Such factors are also creative in a sense, but not in the same sense as natural selection. Challenges to traditional theory have sometimes championed organisms as a neglected source of creativity in evolution. This could be interpreted as the radical claim that non-human organisms—like people—are novel sources of purpose in nature, generating apparently designed outcomes that are not directed at reproductive success. But it might also be interpreted as the uncontroversial claim that organisms—like many other things—sometimes act in a way that accelerates adaptation or makes some adaptive outcomes more probable than others. Ambiguity about their claims has led to theories attracting unwarranted enthusiasm and unwarranted scepticism, and distracts us from the criteria by which the theories should be judged.

Keywords

Adaptation · Evolvability · Genetic assimilation · Niche construction · *Astyanax mexicanus* · *Mycobacterium tuberculosis*

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"This is an art
Which does mend Nature—change it rather; but
The art itself is Nature."
– The Winter's Tale: IV.iv

5.1 Introduction

A major claim of the EES is that factors other than natural selection play a creative role in evolution, and that traditional evolutionary theory has either denied or neglected this fact.

> In contrast to how evolution has traditionally been conceived, in the EES the burden of creativity in evolution does not rest on natural selection alone. (Laland 2018; see also Laland et al. 2015)

This chapter discusses the claim above, and its conclusion will, in short, be partial agreement. I argue that an important strand of traditional thinking does view natural selection as, in some sense, uniquely creative. And this traditional claim is, in principle, open to empirical challenge. However, I note that other, equally traditional strands have always recognized a very wide range of creative factors. This is because the different strands focus on different questions, and so they use the word "creative" to mean different things (e.g., Fisher 1934: 116; 1950; Huxley 1942: 28; Muller 1949: 461; Dobzhansky 1974: 329–32). Unless we can distinguish clearly between these different meanings of creativity, challenges to traditional evolutionary theory become difficult to interpret.

In expanding the argument above, the chapter considers creativity in evolution only in the context of explaining *adaptations*. While adaptations are not the only interesting things in biology (Dawkins 2004: 380), our claims about traditional thinking are not even plausible in any other context. Adaptations were, recall, used as evidence of a creator (Paley 1802; Maynard Smith 1969: 82; Ospovat 1978, 1980) and so they result from a "creative process" almost by definition. Adaptations were, moreover, what the theory of natural selection was proposed to explain (Fisher 1930; Simpson 1947; Williams 1966). If traditional thinking does grant natural selection a uniquely creative role, it must be its role in the evolution of adaptations.[1]

And the role, I argue in §5.2, is to explain why adaptations appear designed for a purpose. Darwin's theory differs from most of its rivals because it predicts what the purpose of adaptations will be: reproductive success (e.g., Maynard Smith 1969;

[1] The main alternative topic would be speciation, such that "creative factors" lead to the origin of new species. But this cannot be natural selection's uniquely creative role, because biologists have long recognized that speciation can occur in lots of different ways (see, e.g., Mayr 2001). With speciation, as with evolvability (§5.3.1.1), a major challenge is to determine the relative importance of the different factors; and as with evolvability too, another major challenge may be to explain why speciation is so slow (e.g., Felsenstein 1981; Barton 2020; §5.3.1.2).

Gardner 2017; Haig 2020: 338–45). This makes Darwin's theory testable, and scientifically useful. For a factor to be creative in the same way that natural selection is creative, it must be an alternative source of apparent purpose. Human creators are an obvious example, because we create complex apparently purposive artefacts that have little or nothing to do with reproductive success (Dawkins 2004: 377). §5.2 introduces this account of selection's creativity, and contrasts it with alternative accounts, which emphasize selection's ability to *initiate* or *direct* evolutionary change (Beatty 2016, 2019; see also Allen 1980; Stoltzfus and Cable 2014). I argue that these other accounts, though widespread, and grounded in scientific practice, can be ambiguous or even misleading if our aim is to understand adaptations.

Next, §5.3 considers factors in evolution that can justly be called creative in quite different senses, even though these senses also concern adaptations. While events of differential survival or reproduction have a direct connection to apparent purpose, many other factors might play important roles in the evolution of adaptations. These factors might, for example, increase a population's capacity for adaptive evolution (perhaps explaining why adaptations could evolve at all), or they might bias evolution towards a particular subset of the possible adaptive outcomes (explaining why these *particular* adaptations evolved). Although their role is quite different from natural selection's, factors of both types might be described as "causes of adaptations" in some sense (e.g., Endler and McLellan 1988: 408); and as a result, they have sometimes been called creative too (e.g., Fisher 1950; Gould 1982: xxx–xxxii; Goodnight and Wade 2000: 318). Creative factors in *these* senses are very common indeed. In fact, nothing is excluded from the list in principle, since absolutely everything can influence the course of adaptive evolution. The real challenge, therefore, is to understand the relative importance of the different factors. §5.3 tries to show how difficult this can be.

These different meanings of creativity underpin two different approaches to studying adaptations. One approach is most concerned with the defining feature of adaptations—their function—and so it naturally focusses on natural selection. The other approach considers the evolution of adaptations as a dynamical process, and so it needs to consider a wide variety of causal factors. Researchers focussed on these different approaches have often shown mutual incomprehension or hostility (e.g., Williams 1985; Grafen 1988; Ross 2002; Frank 2013; Queller 2020), but both types of research have been major parts of evolutionary biology since the very beginning, and so both must qualify as traditional[2].

Finally, §5.4 considers some self-described challenges or extensions to traditional evolutionary theory. Each proposal concerns adaptations (e.g., Waddington 1960: 386; Popper and Eccles 1977: 13; Lewontin 1985: 95), and each proposes *organisms*

[2]This is why "traditional thinking" will not be identified with any single historical period. "The Modern Synthesis" is, moreover, very variously and often unhelpfully characterized, sometimes as a quasi-mythical event, like the Dissociation of Sensibility, and sometimes as a shorthand for a strict set of tenets, difficult to identify with any actual scientists. These are historical idealizations as bold as any found in population genetics.

as additional sources of creativity in evolution. It follows from §5.2 and §5.3 that these claims could be interpreted in different ways. They might, for example, be claims about the purpose of adaptations—claims that non-human organisms, like people, can generate complex purposive outcomes that are *not* directed at reproductive success. Alternatively, they might be claims that organisms (or their actions) belong on the long list of factors that sometimes accelerate adaptation, and sometimes make certain adaptive outcomes more probable than others. I suggest that confusion has been caused by a failure to distinguish clearly between these very different claims—and that only the first would challenge traditional Darwinism. Such ambiguities have led to theories attracting unwarranted enthusiasm, and unwarranted scepticism; and this distracts us from the criteria by which the theories should be judged. If non-human organisms are creative in the same sense as, say, random genetic drift or recombination, their importance to evolution should be gauged in the same way.

5.2 How Natural Selection Is a Creative Process: Speleology and Teleology

This section considers two sets of ideas that are often invoked when discussing the creativity of natural selection. The first set of ideas is quite hard to characterize, but it involves natural selection both *going first* in bouts of evolutionary change; and ensuring that subsequent changes act in the same direction. Beatty (2016, 2019), for example, summarized a diverse array of literature, and concluded that "the creativity of natural selection is best understood in terms of [. . .] selection *initiating* [. . . and] *directing* evolutionary change" (2019: 705). The second set of ideas involves the appearance of purpose. Nature is full of apparent design, and natural selection explains this fact, without invoking a creator (Darwin 1859; Simpson 1947; Ospovat 1978).

Below we will see that both directionality and purpose are part of ongoing scientific practice, with both used to distinguish natural selection from other processes. However, we will also see that the two ideas are very weakly coupled—so that directionality need not imply purpose, and vice versa. Only the appearance of purpose could underwrite claims that natural selection is somehow *uniquely* creative. These points are illustrated with an extended example.

5.2.1 Blind Cave Fish: Creativity as a Source of Directionality

Animals inhabiting subterranean caves descend from surface-dwelling ancestors. The descent often brings modifications, including elongated sensory appendages, reduced pigmentation, and loss of functional eyes. Such phenotypes have evolved convergently in distantly related taxa, from planthoppers to platyhelminths, and caudata to crustacea (Poulson and White 1969; Culver 1982; Juan et al. 2010). In the nineteenth century, this convergent evolution was the subject of debate between

Darwinians and Lamarckians (Lankester 1893; Culver and Pipan 2015)—with Darwin famously defending the Lamarckian position (Darwin 1859, Ch. 5; Jeffery 2009). More recently, researchers have debated whether the convergent regressive evolution is adaptive, or a non-adaptive by-product of relaxed negative selection combined with shared mutational biases (Protas et al. 2007; Jeffery 2009; Rétaux and Casane 2013; Culver and Pipan 2015; Wilkens 2020).

To test these hypotheses, Protas et al. (2007) compared the surface and cave forms of the Mexican tetra, *Astyanax mexicanus*. As is typical, cave forms of this fish are colourless and blind, with eyes that are fully formed at birth, but which are reabsorbed within a few weeks. From a second-generation cross, Protas et al. mapped quantitative trait loci (QTL) for pigmentation (melanophore number), and adult eye size. Results showed that pigmentation QTL varied in direction, such that cave forms carried some alleles that increased pigmentation and other alleles that decreased pigmentation. By contrast, the directions of the eye QTL were consistent, with all cave alleles acting to reduce the eyes. Following arguments of Orr (1998), Protas et al. concluded that the two cases of phenotypic change had different causes; the loss of pigmentation was due to drift and a net mutational bias, while the directionality of the eye QTL implied that eye loss was adaptive and driven by selection.

This inference of selection from rapid directional change is common to many standard tests (Spitze 1993; Orr 1998; Walsh and Lynch 2018, Ch. 12; Schneemann et al. 2020; Fraser 2020) but—as fully acknowledged by Protas et al. (2007)—it does have serious caveats (Rétaux and Casane 2013; Walsh and Lynch 2018, Ch. 12; Aardema et al. 2020; Wilkens 2020). Directional QTL can also result from strongly biased mutation (Wilkens 2020), or from genetic correlations with other traits under selection (Simpson 1949: 149–50; Grafen 1988: 459–60; Protas et al. 2007; Yamamoto et al. 2009). Moreover, the results do not imply that the loss of eyes was *initiated* by selection. As once stressed by Lamarckians (MacBride 1925), some reduction in eyes occurs when surface forms undergo early development in the dark (Bilandžija et al. 2020).

Taken together, this work has two clear implications for creativity in evolution. The first is that rapid convergent evolution can occur in many ways. Many processes can initiate and direct evolutionary change—and so be creative in this sense. The second implication is that directional evolution, even when driven by natural selection, need not lead to paradigmatic adaptations. As an example of adaptive evolution, the loss of cavefish eyes is entirely representative of both empirical work (Endler 1986; Weber 1996, 2004; Sanjak et al. 2018) and theory (e.g., Charlesworth 1993; Orr 2000; Welch and Waxman 2003; Hansen et al. 2006; Pavlicev et al. 2011). But the absence of eyes is, at best, a marginal case of an adaptation[3]. Tests for

[3] Darwin, for example, found it "difficult to imagine that eyes, although useless, could be in any way injurious to animals living in darkness" (1859: 137; see also Weismann 1889: 86), and the fitness benefits of eye loss remain hypothetical. Anderson (1893) speculated that eyes might be "exposed to injury, destructive inflammation, and the attacks of parasites", while Protas et al. (2007) called attention to the metabolic and energetic cost of eye maintenance (Young 1971; Linsenmeier and

adaptation, like that of Protas et al. (2007), will naturally focus on these marginal cases. But they are not the cases that the theory of natural selection was proposed to explain. If we want a theory of form (Pigliucci 2007), we need to see beyond the cave.

5.2.2 Blind Watchmakers: Creativity as a Source of Purpose

It is worth recalling why the vertebrate eye is a paradigmatic case of an adaptation. The reason is not its complexity nor its orderliness (both complexity and order are widespread in the inorganic world). The reason is its apparent design, with the "design [. . .] inferred from the relation which the parts bear to one another in the prosecution of a common purpose" (Paley 1802: 157; see also Fisher 1930: 38; Simpson 1947; Williams 1966; Ali and Klyne 1985; Leigh 2001). Even though the eye contains many imperfections, and features that cannot be understood as purposive (Paley 1802: 43; Ali and Klyne 1985: 8–13; Williams 1992: 152–3), the inference of function is difficult to deny. It's staring us in the face.

This definition of an adaptation makes no reference to natural selection, and that is why Darwin's theory is testable (Maynard Smith 1969; Lewontin 1978: 222; Williams 1992: 40; Reeve and Sherman 1993; Rosenberg and Bouchard 2005; Neander and Rosenberg 2012; Gardner 2017). When we find new, clear cases of apparent design in nature, Darwin's theory predicts what their ultimate purpose will be: reproductive success. This basic point was emphasized in the strongest terms by Darwin and by his critics.

> If it could be proved that any part of the structure of any one species had been formed for the exclusive good of another species, it would annihilate my theory. (Darwin 1859: 201, see also 199, 211, 239 and 242)[4]

> 'Natural Selection' acknowledges that if ornament or beauty, in itself, should be a purpose in creation, it would be absolutely fatal to it as a hypothesis. (Owen 1868: 808)

Of course, reproductive success is achievable in many ways (Foley 2004), and the theory does not deny mutualism, altruism, ornament, or beauty. Kin selection and sexual selection are sometimes distinguished from natural selection (in the narrow sense), to recognize three different means of achieving reproductive success, each yielding purposive phenotypes of characteristically different kinds. The theory of group selection (in the old sense) predicted a fourth class of adaptation

Braun 1992); these costs *increase* in perpetual darkness (Kimble et al. 1980; Wangsa-Wirawan and Linsenmeier 2003), and may be at a particular premium for teleost fish (Damsgaard et al. 2020), and in caves (Simon et al. 2017).

[4] Of course, much scientific effort has been devoted to refining and formalizing Darwin's claim (e.g., Hamilton 1964), and the version quoted is potentially misleading (oak galls, for example, are part of oaks, but form for the exclusive good of wasps).

(Wynne-Edwards 1962; Williams 1966; Maynard Smith 2001; West et al. 2007)—showing that mistakes about design criteria can lead to failed predictions.

Darwin's theory, then, cannot accommodate all observations. Complex, apparently purposive phenotypes that do not function to increase reproductive success, would suggest "that nature might not be so dogmatic about the reproductive imperative" (Eldredge 1995: 219), and be genuinely challenging to Darwin's core claims.

Most challenges of this kind have not succeeded, either because reproductive success was too narrowly defined (Popper 1978: 345–6; Eldredge 1995: 39–40; Foley 2004; Garson 2019), or because researchers were too quick to *assume* a lack of adaptive function (Cain 1964). For example, box jellyfish (class Cubozoa) have camera-type eyes, very like those of fish; yet unlike vertebrates, jellyfish lack optic nerves and brains to process the visual information, and their lenses are too close to the retina to clearly focus an image (Gerhart and Kirschner 1997). We should not conclude, however, that jellyfish eyes represent "form without function" (Coates 2003; Land and Nilsson 2012; Garm et al. 2016). Box jellyfish show high levels of specialization among their 24 eyes, and a nervous system capable of peripheral processing (Garm et al. 2006; O'Connor et al. 2010); lenses close to the retina improve light capture and might also provide mechanical support and protection against photo-damage (Nilsson et al. 2005; Land and Nilsson 2012: 76–9). Moreover, box jellyfish do use their camera eyes successfully, to navigate in near-shore habitats, such as mangrove swamps (Coates 2003; Garm et al. 2007, 2011), and to actively hunt, including via bioluminescence (Garm et al. 2016). It is also notable that the eyes are present only in the motile medusa stage—when they are useful—and not in the sessile polyp; and are lost upon regression to that stage (Valley and Martin 2011). Another indirect argument for functionality is the persistence of these traits for very long periods of evolutionary time. There are many examples of mutational pressure degrading functional structures quite rapidly in the absence of stabilizing selection (as with the cave-dwellers discussed in §5.2.1; Romero et al. 2003); yet cubozoan eyes have withstood this pressure for around half a billion years (Cartwright et al. 2007).

Research on eyes illustrates how Darwin's notion of function is *useful* for biologists. Attributing purpose to adaptations can lead to explanatory and predictive science (Williams 1966, 1985; Mayr 1983). While the usefulness sometimes applies to the simplest, most marginal cases of adaptation (results in §5.2.1, for example, drew attention to the cost of maintaining eyes; fn. 3), the usefulness is much clearer for adaptations that are complex (Rudwick 1961; Mayr 1983; Nilsson 2013; Neander 2017; Levin and Grafen 2019). This is partly because observations of some aspects of a phenotype can be used to predict or understand others (Maynard Smith 1952; Cf. Lewens 2005); and partly because biologists often rely on their understanding of normal proper function to make generalizations about variable populations and complex systems (Neander 2017). For both reasons, Darwin's concept of purpose plays an under-acknowledged role in functional biology (Dawkins 1982, Ch. 3; Mayr 1983; Neander 2017; Garson 2019, Ch. 9). The usefulness of Darwin's idea tells us that recognizing purpose in the products of natural selection is neither *ad hoc* nor whimsy. There is no equivalent for complex natural structures that form without

natural selection—such as caves, formed by the speleogenetic dissolution of calcium carbonate in limestone (Audra and Palmer 2011; Cf. Fodor 1990: 79; Lewens 2005).

These points bear repeating because many find them embarrassing. Even if it has proven useful to attribute purpose to the products of selection, isn't this, at best, a stopgap for some future, more grown-up science, restricted to causes and effects (Simpson 1947: 488; Mayr 1961; Williams 1966; Ghiselin 1983: 363; Dennett 1987, Ch. 10; Ross 2002; Charlesworth 2006; Neander 2017; Haig 2020, Chs. 1 and 9)? The worry about this purist position is that, without *some* notion of purpose, we struggle to describe the defining feature of biological adaptations—the thing that makes eyes importantly different from caves—and so we cannot even state the problem to which natural selection was the solution. This makes the importance of natural selection to traditional evolutionary theory extremely difficult to express (Krimbas 1984; Rosenberg 2000, Ch. 5; Grafen 2003; Rosenberg and Bouchard 2005); it becomes difficult to state what traditional theory affirms, and what it denies.

5.2.3 Complex Adaptations and the Lack of Creative Direction

Sections 5.2.1 and 5.2.2 contrasted two accounts of selection's creativity, stressing directionality and purpose, respectively; but in one important respect, this is a false dichotomy. For example, Fisher's Fundamental Theorem (Fisher 1930, Ch. 2) is best understood as an attempt to show how natural selection explains biological adaptations (Fisher 1930: 37-38, 1936: 59; Grafen 1988, 2003, 2018), and it does so by isolating selection's strictly directional "improving" tendency (Fisher 1930: 37; Bennett 1956; Turner 1985; Queller 2020; Kokko 2021).

Note, however, that the tendency is directional only in a very specific and limited sense, not least because selection-induced changes to the environment (broadly conceived) will often reduce reproductive success (Dawkins 1976: 74; Felsenstein 2000: 609–11; Frank 2013; Kokko 2021). More broadly, results such as Fisher's tell us very little about the evolutionary dynamics (Bennett 1983: 222; Grafen 1988: 455). There is no reason to believe that complex adaptations will evolve via the sort of straightforwardly directional process discussed in §5.2.1. Complex adaptations, by definition, involve many related parts, and so their evolution must involve phenotypic changes of very different kinds. It follows that many of the selection pressures will be dynamically and internally generated, as parts of a complex phenotype adapt to one another (Darwin 1872: 216). It also follows that changes of function are very likely (changes, that is, in the way that reproductive success is achieved). Darwin's achievement was precisely to separate the idea of design from advance planning or foresight; and so it is natural that exaptation, preadaptation, potentiation, and co-option were all baked into his original conception of *adaptation* (Darwin 1872: 183–4; Fisher and Stock 1915: 50–1; Simpson 1947: 495; Haig 2020: 223, 273–5); all were certainly involved in the evolution of eyes (Fernald 2006; Piatigorsky 2008; Nilsson 2013; Picciani et al. 2018). Evolution is a recursive process, and adaptation is always recognized in retrospect (Mayr 1992: 131; Haig 2020, Ch. 11). All of this implies, incidentally, that the *origin* of a complex

adaptation, along with the factor that *initiated* its evolution, will often be difficult to define in any non-arbitrary way (Wund 2012: 5).

Even at the level of alleles and their fitness effects, there is no requirement for the evolution of adaptations to be strictly directional. An adaptation would be no less canonical if its evolution had required the drift-driven fixation of weakly deleterious alleles, as essential intermediate steps. Sewall Wright, the co-founder of population genetics, was explicit that such "backward steps" might often be necessary (Wright 1932; Simpson 1947: 494–5; Provine 1986; see below); and the point is also acknowledged, if sometimes implicitly, in classical quantitative genetics (Frank 2013: 55; Barton 2017: 98, 102–4)[5], and the study of molecular evolution (Maynard Smith 1970: 564; Lenormand et al. 2009).

So, while selection may be a directional, improving process, the evolution of adaptations need not be directional in any stronger sense. Historically, debates about orthogenesis saw discussions of creativity in evolution, and the adequacy of selection, become strongly focussed on observations of long-term directional trends (Bergson 1907/1998; Osborn 1921; Simpson 1947: 486–7)[6], but this topic needs to be distinguished from the core problem of adaptation. The hallmark of a biological adaptation is not that all the necessary changes went in a common direction, but that many aspects of the eventual phenotype can be viewed, in retrospect, as serving a common purpose—and that is what natural selection explains.

5.3 Processes That Are Creative in Other Ways: Balance and Bias

Whether they are described as directional selection or stabilizing selection, events of differential reproductive success play a special role in explaining evolutionary outcomes that appear designed for reproductive success. Nevertheless, as implied by §5.2.3, these events do not constitute a complete account of the evolution of adaptations. Factors can do different explanatory work from natural selection, and yet still be, in some sense, causes of adaptation. These factors are sometimes called creative too.

[5] The role of drift in quantitative genetics is easy to miss because the modelling tracks phenotypic distributions instead of allele frequencies (e.g., Walsh and Lynch 2018), but when the genotype-to-phenotype map is sufficiently complex, selection on single alleles will often be weak, so that many allelic substitutions are driven by drift (Frank 2013: 55; Barton 2017: 98, 104). Quantitative genetic theory allows us to model evolution when the map is arbitrarily complex (Fisher 1918; Barton 2017: 96; Barton et al. 2017), although this point is also obscured when the theory is identified with its first-order approximations like the Breeder's Equation.

[6] Bergson, for example, proposed his theory of "Creative Evolution" because "adaptation explains the sinuosities of the movement of evolution, but not its general directions" (Bergson 1907/1998: 102). Simpson (1949, Ch. 11) argued that most of the non-illusory trends were adaptive; although for Simpson this implied that "orientation in evolution is not determined solely by some characteristic within the evolving organisms or solely by external factors in their environments, but by both and by interplay between the two" (1949: 142; see also 149–50).

§5.3.1 and §5.3.2 develop the claims above. Examples of creative factors are taken from two different bodies of research. The first involves Wright's Shifting Balance process (Wright 1932; Provine 1986; Coyne et al. 1997; Wade and Goodnight 1998); the second involves mutational biases, i.e., the fact that some mutations are more common than others (e.g., Muller 1947; Maynard Smith 1960, 1970: 564, 1981; Maynard Smith et al. 1985; Santiago et al. 1992; Lynch 2007, Ch. 6; Barton 2017: 99). The same two processes were discussed together by Simpson in 1947, as two sorts of "preadaptation" consistent with "the synthetic understanding of evolution" (Simpson 1947: 494–5).

5.3.1 Creativity as Increasing Evolvability

Natural selection only generates adaptations under certain conditions, and a complete account of adaptation must explain when and why those conditions hold. One of Wright's central beliefs was that selection and mutation alone, would not provide sufficient conditions for the impressive adaptations observed in nature. Populations evolving "under strict control of natural selection", Wright argued (1932: 359), would become trapped at suboptimal fitness peaks (i.e., combinations of alleles that are relatively unfit, but still fitter than the alternative combinations reachable via mutation). Wright's solution to this problem was his Shifting Balance theory, and invoked random genetic drift. Drift's role was to act as a "trial and error mechanism", increasing the variation available to selection, so that "the species may continually find its way from lower to higher peaks" (Wright 1932: 358; Provine 1986: 207; see also Frank 2013: 55; Barton 2017: 98, 102–4). In Wright's Shifting Balance, then, genetic drift plays an important role in the evolution of adaptations, helping to direct evolution "from lower to higher". For some authors, this is enough to make drift a creative factor.

> This explicitly allows a more creative role for stochastic processes in adaptive evolution. (Goodnight and Wade 2000: 318)

(See also Crow 1991: 973; Wade 1996; Wade and Goodnight 1998: 1547; Lenormand et al. 2009: 163.)[7]

Wright's theory also shows how the idea of creativity can become associated with the idea of *evolvability*, which following Uller et al. (2018: 952), I define as "the capacity for a population to undergo adaptive evolution" (see also Barton 1995). With this definition, the Shifting Balance, like much of classical population genetics, is shown to be a theory of evolvability. Wright's view was that, without drift, populations would lack the capacity for adaptive evolution; drift's creative role, therefore, is to raise evolvability to adequate levels.

[7] Note that Wright himself, unlike the authors cited, did not call drift creative (e.g., Wright 1980), confirming that the word is used in different ways.

As a creative factor in this sense, drift is far from unique. Ongoing adaptive evolution would be impossible, for example, unless some of the possible mutations were either beneficial or sufficiently weakly deleterious (§5.2.3). Several authors have identified properties of developmental systems that make such fixable mutations less rare than they would otherwise be (Goldschmidt 1940; Waddington 1953b; Maynard Smith 1958: 317–8; Lewontin 1978; Gerhart and Kirschner 1997; West-Eberhard 2003; Kirschner and Gerhart 2005; Uller et al. 2018: 952). Just as drift might increase the quantity of variation, these biases might increase its *quality* (Simpson 1947: 494–5; see also Huxley 1942: 449–51; Stebbins 1985: 515–6). If drift can be called creative, these developmental biases seem to qualify too (e.g., Gould 1982: xxx–xxxii).

5.3.1.1 Creative But Unimportant?

A consequence of the argument above is that more-or-less anything might play a creative role in evolution. While sources of apparent purpose in nature seem rare, factors that might increase evolvability are very common indeed. A population's capacity to undergo adaptive evolution will depend, at least, on the quantity, quality, and heritability of its variation, and on the selection pressures it faces—and almost any feature of the world might affect one or more of these things.

One challenge, then, is to identify factors that are *important* determinants of evolvability. To show what this means, it is easiest to give an example. Consider therefore the shuffling of genetic material, via sex and recombination (Weismann 1889; Williams 1975; Barton 1995). We know that sex and recombination are important for evolvability because their rates vary widely in nature (Bell 1982; Eckert 2002; Vos and Didelot 2009) and their absence is consistently associated with conspicuous failures of adaptation (Maynard Smith 1978; Normark et al. 2003; Vrijenhoek and Parker 2009). Absent recombination explains a large class of genuinely puzzling observations: from the low diversification rates of clonal lineages that we might expect to have a huge competitive advantage (Williams 1975; Schwander and Crespi 2009), to the degeneration of Y or W sex chromosomes (Bachtrog 2008), and the maladaptive frequencies of plumage variation in the ruff (*Philomachus pugnax*; Küpper et al. 2016; McNamara and Leimar 2020: 19). There is, moreover, direct experimental evidence that losing sex and recombination reduces a population's capacity to adapt (e.g., Rice 2002; Cooper 2007; Becks and Agrawal 2012; Kosheleva and Desai 2018).

Sex and recombination do not *always* increase evolvability (e.g., Charlesworth and Barton 1996; Tilquin and Kokko 2016)—and this is to be expected. Adaptation is not simply continued change in a given direction (§5.2.3), and so factors that accelerate or facilitate adaptation under some conditions, almost always retard or prevent adaptation under other conditions (i.e., some timescales, or patterns of environmental change; Ancel 2000; Huey et al. 2003; Hendry 2016: 34; Barton 2017). Nevertheless, taking the evidence as a whole, it seems likely that a world without sex or recombination would contain much less adaptation than the world that we live in.

For other possible determinants of evolvability, the case for importance is far more difficult to make. Many clades, for example, have undergone both spectacular increases and spectacular decreases in their adaptive diversity (e.g., the Miocene hyracoids; Schwartz et al. 1995), but it has been difficult to show convincingly that any of these events was caused by changes in evolvability (Haig 2020: 228), let alone by changes of a particular kind, such as changes in the level of drift in their subpopulations, or changes to developmental systems making beneficial mutations more or less likely. And neither theory, nor data, make a compelling case for either factor[8].

5.3.1.2 What Needs to be Explained?

While debates about relative importance are thorny and intractable, the subject of evolvability is marked by more fundamental disagreements. These are disagreements about what needs to be explained.

Again, sex and recombination are a telling special case. While many other features of genetic systems appear designed for accurate transmission (Ganai and Johansson 2016), sex and recombination appear designed for exactly the opposite (Weismann 1889; Bell 1982; Burt 2000; Leigh 2001). Sex, especially, seems to be an adaptation for increasing future evolvability at great cost to current fitness—and is therefore a counterexample to the standard Darwinian account of the purpose of adaptations (§5.2.2; see, e.g., Fisher 1930: 119; Eldredge 1995, Ch. 12). As is well known, sex and recombination present a paradox for traditional Darwinism (Turner 1967; Williams 1975; Maynard Smith 1978; Bell 1982; Feldman and Liberman

[8] A variety of results have questioned whether drift is required for peak shifts—especially when selection pressures vary in space and time (Wright 1931: 167; Fisher and Ford 1950; Weatherhead 1986; Williams 1992, Ch. 4; Price et al. 1993; Whitlock 1997; Weinreich and Chao 2005; Whibley et al. 2006; Bell 2010). And while many populations are spatially subdivided (Provine 1986: 270; Harrison and Taylor 1997; Yang et al. 2019), there is no evidence that levels of drift match the "sweet spot" required to maximize evolvability (Coyne et al. 1997; Barton 2017), or have any tendency to evolve in that direction (e.g., Peck 1992). Nor is there evidence that large well-mixed populations are conspicuously maladapted. Regarding mutational biases, a range of different results suggest that adaptation might not be limited by the rate of beneficial mutation (e.g. because frequent beneficial substitutions can interfere with one another; Weissman and Barton 2012; but see also Wright 1932; Maynard Smith 1976; Maynard Smith et al. 1991; Arnold 1996; Schluter 2000; Welch and Jiggins 2014; Rousselle et al. 2020; Barton 2020). Other work has shown that decreasing the severity of deleterious mutations might lead to extinction, because weakly deleterious mutations persist for longer (Gabriel et al. 1993; see also Kondrashov 1988). Biases that make beneficial mutations more likely, or deleterious mutations less severe, need not, therefore, lead to substantial increases in evolvability. It is important to note that none of these arguments is conclusive. The decisive measurements—on real-world fitness landscapes, or levels of maladaptation relative to some hypothetical optimal kind—remain very difficult (Maynard Smith 1978; Williams 1992, Chs. 4, 9 and 10; Crespi 2000; Hereford et al. 2004; Kaznatcheev 2019); and there is still no consensus about the relative contributions to adaptation of large- and small-effect mutations (Simpson 1947: 494-5; Bell 2010; Rockman 2012; Boyle et al. 2017; Barton 2017: 105-6; Barghi et al. 2020). In addition, some developmental biases are difficult to quantify, while others, like "key innovations", evolved only once (Williams 1992: 35); so unlike with sex and recombination, we cannot use natural or induced variation to perform tests.

1986; Otto 2009; Barton 2010; Lehtonen et al. 2012; Altenberg et al. 2017). They are not only consequential for adaptation in nature, but surprising in themselves; they call out for some special explanation.

For other determinants of evolvability, the situation is once again more ambiguous. For some authors, explaining the possibility of beneficial mutations remains a central unsolved problem, comparable to the paradox of sex (see e.g., Lewontin 1978; Kirschner and Gerhart 2005, e.g.: 13, 29, 166; Wagner 2014). For others, the appearance of a problem is an illusion of perspective. Researchers who trace extant lineages back in time (Calcott 2009) see only the "the unbroken string of triumphs" (Dennett 1987: 317; Orr 2013), and not the countless billions of mutations that failed to persist[9]. The generation of adaptive variation will always seem remarkable if the maladaptive variation (which everybody agrees is the larger part) is shielded from view[10]. When should we conclude that the rate of beneficial mutation is surprisingly high? Wouldn't it be far more puzzling if *all* properties of developmental systems acted to make mutations more deleterious?

Intuitions are equally divergent about yet another, and still more basic question: Is the overall rate of evolvability itself surprisingly high? This debate began with the most searching objections of Darwin's earliest critics, about the time required for complex adaptations to evolve (e.g., Mivart 1871, Ch. 2; Darwin 1872, Ch. 7; see also Hull 1973, Ridley 1982, Crow 2008). While disagreeing with their conclusions, Wright agreed with these critics that *something else* was needed to explain the rapidity of adaptive evolution (Wright 1932: 358), and many subsequent authors have agreed with Wright (see Williams 1992: 127-8; Charlesworth et al. 2017: 8-9). However, decades of work now suggest that adaptive phenotypic evolution is often surprisingly slow. The slowness is apparent over short periods of time (where realized phenotypic change is often less than would be predicted from estimated selection gradients: Merilä et al. 2001; see also Hereford et al. 2004; Morrissey and Hadfield 2012; Kokko 2021), and over long periods of time (where long-term rates are much slower than short-term rates: Gingerich 1983, 2009; Williams 1992, Ch. 9; Hansen 2013; Kokko 2021). An inability of populations to adapt rapidly is also implied by the time lags between the apparent origin of new niches and their occupation (Stromberg 2005; Labandeira 2007; Hansen 2013; Erwin 2015), and from the absence of traits that "should have evolved" (Williams 1992, Chs. 9–10; Ruxton and Humphries 2008). Why, for example, has *Streptococcus pyogenes* (group A strep) remained susceptible to penicillin for so many years (Horn et al. 1998; Hayes et al. 2020; though sadly, see, Olsen et al. 2020)? Theories that aim to

[9] Analogously, Wright's views about evolvability were heavily influenced by his focus on artificial breeding in populations much smaller than most of those in nature (Jones et al. 1968; Provine 1986: 239; Caballero et al. 1991; Weber 1996, 2004; Weinreich and Chao 2005).

[10] Overall mutation rates, like recombination rates, are the subject of a generalised reduction principle (e.g., Altenberg et al. 2017), but unlike recombination rates, there is no evidence that mutation rates *can* be reduced to zero, especially in stressful conditions, when all sorts of biological functions are poorly performed.

explain high levels of evolvability must seem much less compelling to those who conclude that evolvability is not, in fact, surprisingly high.

5.3.2 Creativity as Choice Between Peaks

Wright's Shifting Balance theory, while very influential, also faced severe criticisms. The most influential criticisms were not aimed at the posited role for drift (§5.3.1); instead, they questioned Wright's claim that the eventual *outcomes* of the Shifting Balance would be determined by fitness differences alone (Haldane 1959; Coyne et al. 1997; Crow 2008). Wright had argued that the very fittest peaks, once reached with the aid of drift, would tend to spread via differential migration. Wright's critics argued that peaks might spread for reasons that were either unpredictable (Gavrilets 1996; Coyne et al. 1997; see also Dobzhansky 1974: 331–2; Lewontin 1978: 228; Williams 1992: 31), or predictable, but unconnected to fitness. For example, genotypes might spread preferentially if they contained alleles that are dominant rather than recessive (Mallet and Barton 1989; Barton 1992; Coyne et al. 1997)—an argument that also underlies *Haldane's sieve* (Haldane 1924; Turner 1981: 112). The particular adaptive outcomes that evolve, therefore, might depend on factors such as dominance relations, not only on the fitness differences of the phenotypes.

In a multi-peaked fitness landscape, where several adaptive outcomes are possible, a huge variety of factors might direct or bias evolution towards a subset of those outcomes. An excellent recent example involves the transition-transversion bias in single-nucleotide mutations[11] (Gojobori et al. 1982; Yampolsky and Stoltzfus 2001), and adaptive evolution in *Mycobacterium tuberculosis* (Payne et al. 2019). Like many pathogenic bacteria, *M. tuberculosis* is treated with antimicrobial drugs—an intelligently-designed attempt to cause its local extinction—and it has evolved resistance repeatedly (Osório et al. 2013; Eldholm and Balloux 2016). Payne et al. (2019) showed that the mutations which conferred resistance in *M. tuberculosis* were surprisingly often transitions, and that this could not be explained by selection. Their evidence for the latter claim was twofold; first, they showed that transitions were overrepresented not only in resistance mutations, but also at putatively neutral sites elsewhere in the genome; second, they showed that several of the amino acids that conferred resistance could have evolved via either transitions or transversions, due to redundancy in the triplet genetic code. This last result implies strongly that the mutational bias had no substantial effect on evolvability in this case. The bias was not, therefore, a creative factor in the sense of §5.3.1, and so could not itself have evolved to facilitate adaptation. Nevertheless, the bias *had* influenced the adaptive

[11] Of the four bases in DNA, C and T are pyrimidines with a single ring, while A and G are purines with two rings. Transition substitutions are pyrimidine-to-pyrimidine or purine-to-purine, and so conserve the number of rings, while transversions change the number of rings. There are twice as many possible transversions as transitions, allowing us to define a "surprising" overrepresentation of transitions.

outcomes. Without considering the transition-transversion bias, we could not explain why these particular adaptive mutations evolved.

Many similar results have been securely established, both for genomes and phenotypes (for some beautiful examples see Alberch and Gale 1985; Houle et al. 2017; Lange and Müller 2017; Hayden et al. 2020[12]), and so debates have tended to concern their implications, or how exactly they should be described. Most relevant to this chapter are claims that the biasing factors need to be recognized as *creative*, because they direct evolution toward particular adaptive outcomes (see, e.g., Gould 1982: xxx–xxxii; West-Eberhard 2003: 10; Arthur 2004: 38). Making this claim requires some rhetorical care. It is important that the population not be directed towards the fittest possible outcome—for otherwise, a naive pan-selectionist account, ignoring the biasing factor, would have made identical predictions. However, to merit the label *creative,* it seems equally important that the biasing factor not be viewed as a merely negative influence, or a constraint on selection (e.g., Uller et al. 2018).

How do the results of Payne et al. (2019) speak to this debate? On the one hand, it is plausible that the *M. tuberculosis* data do reflect a type of evolutionary constraint. Its low mutation rate, near clonality and strong population subdivision (Achtman 2008; Eldholm and Balloux 2016) would all have reduced the variation available to selection (Maynard Smith et al. 1985: 266; Stoltzfus 2019), and so would the selection pressure itself, with its intensity, recency, transience, and patchiness. Limited variation might have prevented *M. tuberculosis* from reaching a single globally optimal fitness peak, even if one mechanism of resistance were appreciably superior to the others.

On the other hand, results must also reflect what Lenormand et al. (2009) called "evolutionary freedom". There are many effective mechanisms of antimicrobial resistance, most achievable via many mutational routes (Blair et al. 2015). It was precisely this wealth of options that allowed mutational bias to choose between them. If evolutionary freedom is sufficiently high, biases can affect outcomes even when standing variation is plentiful. For example, the sexual displays of many male animals have a strong arbitrary component, and this may explain why mutational biases (including as a source of genetic correlations in the standing variation) play such an important role in classical models for the evolution of such traits (Fisher 1930: 135–9; Lande 1981; Kirkpatrick 1987; Pomiankowski et al. 1991; MacNamara et al. 2003; Fuller et al. 2005; Henshaw and Jones 2020; Veller et al. 2020).

If the role of biases is not, therefore, exclusively negative, should we conclude that they do the same sort of explanatory work as natural selection—that selection and biases are creative in the same sense? From one perspective, this is quite true. In *M. tuberculosis*, both selection and bias led to predictably non-random sets of

[12]Note, however, that the controls used by Payne et al. (2019), involving neutral sites and the redundancy of the genetic code, are rarely available for other types of mutational or developmental bias.

mutations becoming common. However, from the perspective of §5.2.2, the differences are more striking than the similarities. The theory of natural selection solved the problem of apparent design in nature, by underwriting a scientifically useful concept of function or purpose. The transition-transversion bias cannot do this. This would be true even if the bias acted consistently across all life (which it doesn't: Keller et al. 2007; Seplyarskiy et al. 2012; Long et al. 2016), but it is even more true for mutational or developmental biases that act only for certain traits in certain lineages at certain times.

5.3.3 Summary So Far

We have seen that many different factors might—in one sense or another—play a creative role in evolution. All such factors can—in one sense or another—direct evolutionary change—and all can—in one sense or another — be causes of adaptations. While there are longstanding and intractable debates about their relative importance, a detailed dynamical description of the evolution of adaptations might need to include many such factors.

Of course, researchers might still want to focus on a small subset of the factors. For example, from fitness trade-offs alone, we can make useful and general predictions about pathogen virulence (Cressler et al. 2016). Such methods suggest, for example, that the deadly nature of *M. tuberculosis* is linked to its persistence outside of its host (Walther and Ewald 2004; Martinez et al. 2019); and that some regimes of antibiotic treatment will work better than others (McLeod and Gandon 2021). Such methods provide good enough results on the conditions that virulence is connected directly to reproductive success, that evolvability is sufficiently high, and that biases are either weak or transient (e.g., Hammerstein 1996) or involve aspects of the adaptive phenotype that are not of direct interest (e.g., exactly how the host is harmed, or which particular amino acid changes confer resistance).

This is a pragmatic case for focussing on selection alone, but as we have seen, it is not the only case available. Natural selection is not just one creative factor among many but is creative in a way that the other factors are not. The part of the evolutionary dynamics that approximates optimization or rational choice, may be of intrinsic interest, even if a fuller dynamical description is also available (Grafen 1988, 2003; Ross 2002; Frank 2013). Methods that focus solely on natural selection (e.g. McNamara and Leimar 2020, Chs. 1, 4), even if woefully inadequate for many tasks (Lewontin 1978: 216; Williams 1992: 31; Eshel 2005: 16), are essential for understanding the purpose of adaptations.

5.4 The Organism as a Source of Creativity: Agency Work and Make-Work

This final section will examine some proposals to extend evolutionary theory by recognizing *organisms* as additional sources of creativity in evolution. As we have seen (§5.2 and §5.3), this could mean different things, and I argue below that the proposals can all be interpreted in different ways. First, though, we need to explain why such proposals are both plausible and potentially challenging to traditional theory.

5.4.1 The Art of Misdirection: Human Creativity as a Novel Source of Purpose

The major reason that natural selection was first called creative is the analogy between its products and those of human creators (Paley 1802; Osborn 1921: 134; Dobzhansky 1974: 335). The creativity of natural selection is therefore modelled on the creativity of (some) organisms. But the idea of creativity had to be transformed as it was naturalized (Rosenberg 2016). Natural selection makes its "choices" at the population level, and so its goal must always be the same: reproductive success. By contrast, human creativity is characterized by a sort of individual agency[13], and can be employed towards a wide variety of ends. This explains why it seems so natural to link "the marvel of creativity [. . .] the freedom to create; [and] the freedom of choosing our own ends and our own purposes" (Popper 1978: 343; Dennett 2003). Goals that are generated, reflected upon, and endorsed by the agents themselves, will often *conflict* with reproductive success; "there is no more sombre enemy of good art than the pram in the hall" (Connolly 1938: 116). And yet such goals are often achieved, with results that demand to be understood as purposive, but which have little or nothing to do with reproductive success. For Dawkins, this is the evidence that "we, alone on earth, can rebel against the tyranny of the selfish replicators" (1976: 201). What distinguishes human creative endeavours from the products of natural selection is the variety of purposes manifest in their outcomes (Fisher 1950; Dawkins 2004: 377; Ross 2007; Sterelny 2020).

Of course, humans are very far from alone in manifesting goal-directed, flexible, and agential behaviour. Such behaviours are very common products of natural selection and are standard Darwinian adaptations—tools for achieving reproductive success in the face of environmental uncertainty (Dennett 1975; Haig 2007; Lenormand et al. 2009). Standard approaches to studying such adaptations often attribute agency to organisms (Grafen 1999; Levin and Grafen 2019; McNamara and Leimar 2020), and they do so as a scientific tool—i.e., because it is useful (Gardner 2019). In such research, reaction norms are treated as "strategies" of the adaptive

[13] This view is culturally specific (e.g., Niu and Sternberg 2006), but it does seem to be the relevant one for debates about evolutionary theory.

agents, and predictions can be sensitive to the ability of agents to assess their own internal state and modify their phenotype accordingly (e.g., Dawkins 1980), or to process information more generally. This explains why *learning* was part of this tradition from its earliest days (Müller 1879) and continues to be at its cutting edge (McNamara and Leimar 2020, Chs. 5 and 8).

But while these methods do attribute agency to organisms, they do deny them one important, and even defining attribute of human creativity: plurality of purpose. This is why the agency can often be reassigned from the organism to hereditary replicators or even to "Mother Nature", without greatly altering the predictions (Dawkins 1976, 1982; Dennett 1995). Moreover, the organisms don't even need to do anything for the methods to be useful (Dennett 2019: 356). Viewed as adaptive agents (Grafen 1999), cavefish are wilfully blind; this is not because they actively absorb the eyes they are born with, but because their adult phenotype has an adaptive rationale.

To summarize: humans prove that organisms can be creative in the way that natural selection is creative, but for (most?) non-human organisms, this type of creativity is either neglected or denied by standard evolutionary theory.

5.4.2 Theories of Adaptation Without Purpose

Let us now turn to the proposed extensions to evolutionary theory. Each has been heralded as a major contribution to our understanding of adaptations, and each involves organisms being creative in at least one of the senses discussed above (§5.2.2, §5.3.1, §5.3.2 and §5.4.1). We will note an ambiguity about which of these senses is meant. As a result, it is often difficult to know what each theory seeks to explain, how its importance should be judged, and how, if at all, it differs from traditional theory.

5.4.2.1 Popper: The Genetic Fallacy

Though his writings on biology were sparse, Karl Popper proposed some improvements to evolutionary theory (Hull 1999; see Popper 1972, 1974, 1984), often anticipating later critics (Platnick and Rosen 1987; Hull 1999; Vecchi and Baravalle 2014). Popper contrasted his own views with a statement of Darwin's (1859: 141-2). Darwin began by noting that the evolution of complex adaptations might involve changes in both behaviour ("habit") and morphology ("structure"), and concluded that it was

> difficult to decide, and immaterial for us, whether habits generally change first and structure afterwards; or whether slight modifications of structure lead to changed habits; both probably often occurring almost simultaneously. (Darwin 1859: 141–2; see also Dobzhansky 1974: 323)

Popper replied:

> I agree [with Darwin] that both cases occur, and that in both it is natural selection which works on the genetic structure. Still I think that in many cases, and in some of the most interesting cases, habits change first. [. . .] I disagree with Darwin, however, when he says that the question is 'immaterial for us'. I think it matters a lot. Evolutionary changes that start with new behaviour patterns—with new preferences, new purposes of the animal—not only make many adaptations better understandable, but they re-invest the animal's subjective aims and purposes with an evolutionary significance. (Popper and Eccles 1977: 13)

The fact that behavioural changes sometimes *go first*—Popper argued—both helps to explain "many adaptations" *and* gives "evolutionary significance" to "the animal's subjective aims and purposes". What exactly did he mean?

Popper's writings elaborate on his basic proposal in several different ways. He sometimes argued, for example, that behavioural changes are more likely to be adaptive, or fortuitously pre-adaptive, than are genetic mutations (Popper 1972: 239-42, 280-3), or that "the mechanism of natural selection becomes more efficient when there is greater behavioural repertoire available" (Popper and Eccles 1977: 13). So, by improving the quality or quantity of phenotypic variation, behaviour might increase evolvability—just like drift, biased mutation or recombination (§5.3.1). This claim is plausible, but it was not novel. Neither Darwin, nor subsequent Darwinians, doubted that animal behaviour is often adaptive, nor that it can alter the course of evolution—just like everything else can (e.g., Huxley 1942: 113-5; Fisher 1950; Maynard Smith 1958: 319). Many biologists had also made the stronger claim: that behaviour will tend to *accelerate* adaptive evolution (e.g., Schmaulhausen 1949; Mayr 1960, 1963: 604; Wyles et al. 1983; Huey et al. 2003; Merrill et al. 2015: 1428). Doubts about the stronger claim are also familiar; just like drift, biased mutation and recombination, behaviour can retard adaptation, as well as speed it up (e.g., Elton 1927; Huey et al. 2003; Uller et al. 2018; §5.3.1.1); and it isn't clear that high evolvability is what we need to explain (§5.3.1.2).

Alternatively, we could read Popper as suggesting that characteristic patterns of behaviour (motivated, if you like, by "subjective aims and purposes") might bias evolution towards a subset of the possible adaptive outcomes—just like the transition-transversion bias (§5.3.2). Again, this is part of traditional theory. Small, non-adaptive biases in mate preference can influence the evolution of extravagant secondary sexual traits (Fisher 1930: 135-9; Kirkpatrick 1987; Fuller et al. 2005; Henshaw and Jones 2020; §5.3.2). Evolutionary outcomes in a multi-peaked fitness landscape will often depend on the ancestral state (Williams 1985: 12–13; Eshel 2005: 16)—and this includes behaviours as much as anything else.

Finally, Popper sometimes argued that complex adaptations have a wider variety of purposes than Darwin had acknowledged (1978: 345; §5.2.2). Unfortunately, Popper's thoughts on *this* subject were unclear. His most famous foray into evolutionary biology was his early claim that natural selection is an untestable tautology (Popper 1974, 1978: 344; Maynard Smith 1969; Hull 1999; Nowak et al. 2017)—a sure sign that his focus was not on the purpose of adaptations (§5.2.2); and his discussions of adaptation at this time were mixed up with notions of directionality and orthogenetic trends (Popper 1972: 280; 1976: 173; §5.2.3). Later, he claimed that Darwin's theory, understood as a claim about adaptations, was "not only

refutable, but actually refuted" (1978: 345). But Popper's view of reproductive success was overly narrow, apparently excluding success in attracting mates (1978: 345-6; §5.2.2).

The result is that Popper's claims are hard to summarize. Are they plausible but unoriginal and largely uncontroversial ("behaviour—like many other things—sometimes accelerates adaptive evolution, and sometimes biases outcomes non-adaptively"); or are they radical and rich in implication, but largely unsupported ("complex adaptations need not function to increase reproductive success, but reflect new and subjective purposes")?

5.4.2.2 Waddington: Plasticity First and Last

Similar ambiguities are evident in the evolutionary theories of Conrad Hal Waddington. Unlike Popper, Waddington was an active experimental scientist, but like Popper, he criticized the evolutionary theory of his day[14], and proposed some extensions of his own (e.g., Waddington 1957, 1969/2008). The best known is *Genetic Assimilation*: the idea that environmentally induced phenotypes might become less sensitive to environmental conditions following allele frequency change (Waddington 1942, 1953a). Closely related ideas have been proposed many times (Spalding 1873; Weismann 1894; Baldwin 1896; Lloyd Morgan 1896; Schmaulhausen 1949; Simpson 1953; Ridley 1982; West-Eberhard 2003; see also Pigliucci et al. 2006; Crispo 2007; Lande 2015), but what do they aim to explain?

As with Popper's proposal, Waddington's basic idea can be elaborated in different ways (Maynard Smith 1958: 322–3; Eshel and Matessi 1998). In one version of the theory, the change in environment yields a plastic response that is straightforwardly adaptive, because the capacity to generate the phenotype under appropriate conditions was the target of past stabilizing selection. This version of Waddington's theory connects to large bodies of research on the evolution of adaptive plasticity (e.g., Cohen 1966; Philippi and Seger 1989; Grafen 1999; Chevin and Lande 2011), and especially its role in colonization (e.g., Sol et al. 2008, 2012; Wright et al. 2010; Davidson et al. 2011; Hendry 2016: 32-3; Bock et al. 2018; Liu et al. 2020). Nevertheless, in this form, Waddington's claims are almost trivial: populations can flourish in conditions to which they are already adapted; and plasticity—like eyes—can be lost when no longer selectively maintained (Williams 1966: 71-83; see also Hughes 2012).

In a second, distinct version of the theory, the change in environment yields a novel phenotype that is fortuitously pre-adaptive (Eshel and Matessi 1998; Pigliucci et al. 2006). This is plausibly true of the reduced eyes of Mexican tetra that are reared

[14] Waddington's major complaint about population genetics seems to have been its *failure to mention things explicitly* (so there is "no explicit mention of the phenotype", "no hint that phenotypes can be affected by environments", and "no mention of the fact that the effect of a given gene is influenced by the rest of the genotype" 1969/2008: 259). Of course, mentioning things explicitly is not always a theoretical virtue (Gilbert 1994: 153; Strevens 2008) and in any case, all these things are mentioned explicitly in standard quantitative genetics (Fisher 1918; Hill and Kirkpatrick 2010; Walsh and Lynch 2018).

the dark (Bilandžija et al. 2020; §5.2.1). The capacity for reduction is very unlikely to have been the target of past selection, but it might nonetheless have increased the fitness of the early cave colonists. It is not news that lucky events (like beneficial mutations) will sometimes happen, nor that the lineages we observe are more likely to have experienced good luck. A stronger claim would be that induced responses are *more likely than expected* to be fortuitously pre-adaptive. In this version, Waddington's theory strongly resembles the theories of biased mutation discussed in §5.3.1; and again, a challenge is to quantify these expectations, and generate a meaningful null (Robertson 1977: 601; Huey et al. 2003; §5.3.1.2).

In a third version of the theory, the change of environment *increases phenotypic variation* (Rendel 1967; Eshel and Matessi 1998; West-Eberhard 2003; Hendry 2016: 34), just like drift in the Shifting Balance (§5.3.1; Levin 1970; Pfennig et al. 2010; Wund 2012: 7). While new habitats might sometimes decrease variation (heritable variation in visual conspicuousness, for example, will be lower in caves than on the surface), Waddington argued that environmental novelty *per se*, might preferentially increase variation, if stress leads to failures of canalization (Lloyd Morgan 1896: 738; Eshel and Matessi 1998; Rutherford and Lindquist 1998; Badyaev 2005).

In all the non-trivial versions of Waddington's theory, environmental induction belongs on the long list of creative factors that might increase evolvability (§5.3.1)—essentially as a source of fit intermediates (Weismann 1894; Baldwin 1896; Waddington 1953b, 1960: 389-90; Simpson 1953: 111, 114; Maynard Smith 1958: 319; Ridley 1982; Ancel 2000; Avital and Jablonka 2000; West-Eberhard 2003; Papineau 2005; Wund 2012: 7; Hendry 2016; Godfrey-Smith 2017: 3). In all cases, however, it remains unclear whether Genetic Assimilation is an *important* determinant of evolvability, comparable to, say, recombination (§5.3.1.1)[15]. It is quite difficult, for example, to point to any puzzling fact about nature that Genetic Assimilation has explained.

Consider in this light a later version of the theory. This version involves *positive frequency-dependent selection*, where traits decrease fitness when rare in the population, but increase fitness when common (West-Eberhard 2003; Godfrey-Smith 2017: 3). Such traits do seem surprising at first blush; and induction could help to explain their evolution, because induced traits—unlike recurrent mutations—might appear in many individuals simultaneously, avoiding the maladaptive stage of low frequency. However, traits under positive frequency dependence can evolve in several different ways, including changes in selection pressures, kin aggregation, or localized drift as in the Shifting Balance (Fisher 1930: 159, 162; Harvey et al. 1982; Hedrick and Levin 1984; Schilthuizen and Davison 2005; Mallet 2010). And induction seems to have played no role in the best-studied example of positive

[15] Assessing the importance of plasticity to evolvability is difficult for some unique reasons (e.g. Lewontin 1985). How should the benefits of plasticity in a given trait be weighed against the benefits arising from most other traits being stably expressed? And how should we deal with the fact that much adaptive plasticity aims precisely at stabilizing other aspects of the phenotype?

frequency dependence: warning colouration in *Heliconius* butterflies (Mallet 2010; Merrill et al. 2015: 1422; Chouteau et al. 2016); nor in other impressive and recurring examples, such as cytoplasmic incompatibility in *Wolbachia* (Meany et al. 2019), and karyotypic change in animals (Hedrick and Levin 1984; de Vos et al. 2020).

But whether we consider it important or not, there seems no reason to doubt that Genetic Assimilation will often happen; and presentations of traditional Darwinism have always recognized Genetic Assimilation as a possible mode of evolution (e.g., Huxley 1942: 114; Mayr 1951; Simpson 1953: 115; Haldane 1954; Maynard Smith 1958: 322-3; Williams 1966: 71-83). How, then, did Waddington's theory acquire its enduring reputation as being challenging to traditional Darwinism (e.g., Bateson 1958; Fodor and Piattelli-Palmarini 2010: 58-60; see also Gilbert 1994)?

One possible explanation is that theories like Waddington's *had* been proposed as alternative, and genuinely anti-Darwinian answers to a different question: Why is there apparent purpose in nature? (Simpson 1953: 111, 114). Waddington himself made no such claims explicitly. Like Popper, he was not clearly focussed on the problem of purpose, believing that "natural selection [. . .] turns out on closer inspection to be a tautology", because its acting on "random mutation of Mendelian genes [. . .] could, in fact, explain anything" (Waddington 1960: 385-6; see also 1969/2008: 260-1). And yet, like Popper too, he sometimes pointed to apparently designed phenotypes that, he felt, could not possibly be functioning to increase reproductive success (§5.2.2); compare, e.g., Waddington's discussion of reptile-mimicry in insects (Waddington 1969/2008: 261) to those of Pearson (1989), Cloudsley-Thompson (1995) and Novelo Galicia et al. (2019)[16]. And Waddington also left himself open to misconstrual when he claimed to have discovered a "more convincing explanation of how the appearance of design comes about" (1960: 386).

It is also telling that Waddington's theory—like Popper's—places great emphasis on what *goes first*, which, though "immaterial" for Darwin, for Popper, was somehow related to the animal realizing its subjective purpose. Statements about priority in time, combined with causal influence, are easy to misread as statements about purposive control. Consider, for example, the claim that:

> (G)enes are followers, not leaders, in adaptive evolution. (West-Eberhard 2003: 157-8)

This is easy to (mis)read as a challenge to Dawkins (1976), as a claim that the stratagems of the "selfish genes" are somehow thwarted, and their agendas overruled. But that would only be true if induced phenotypes tend to be assimilated even when they are maladaptive—i.e., if gene frequencies dutifully shift to canalize new phenotypes, whatever their fitness consequences. If, by contrast, induction increases evolvability then—by definition—the genes would follow only where

[16] Waddington also claimed that his theory—like sexual selection before and kin selection after—explained a whole new *class* of adaptations; but these were "pseudo-exogenous adaptations"—which look like physiological adaptations but aren't—and so are not distinguished by a characteristic type of function (Waddington 1953b: 134; Simpson 1953: 113).

they were quite happy to have been led. Moreover, *neither* of these readings would imply plurality of apparent purpose, and so neither would challenge Dawkins' central claim: that adaptations function for replicator success.

When discussions of adaptation avoid the subject of apparent purpose, ambiguities often follow. Even Simpson—who was usually so clear on the subject (Simpson 1947, 1953: 111, 114)—sometimes made arguments that are easily misconstrued. For example, in his discussion of the Baldwin Effect—a precursor of Waddington's theory—Simpson not only mentioned "the *directive force* in adaptation" (1953: 116), which has no single meaning (§5.2.3, §5.3), but also made the following ambiguous claim:

> If the Baldwin effect occurs, either there is or is not a causal connection between an individual accommodation [defined as a non-heritable phenotypic change] and subsequent genetic change in a population. If there is no such connection, then the truly genetic change must occur wholly by mutation, reproduction, and natural selection, and the accommodation may be irrelevant. If there is a causal connection, the neo-Lamarckian argument is as much supported as supplanted (Simpson 1953: 115).

Simpson may have meant only that induced phenotypic changes do not cause subsequent allele frequency changes in any simple and direct sense. If so, the claim is very plausible. But if he meant to deny there ever being *any* "causal connection" between the two, then this is both highly implausible, and far more than traditional Darwinism demands (e.g., Huxley 1942: 114; Maynard Smith 1958: 322-3).

5.4.2.3 Lewontin: Changing the Subject

Popper sometimes described his improvements to evolutionary theory in yet another way.

> By adopting a new form of behaviour the individual organism may change its environment [. . .]. [I]n this way, individual preferences and skills may lead to the selection, and perhaps even to the construction, of a new ecological niche by the organism. By this individual action, the organism may "choose," as it were, its environment. (Popper and Eccles 1977: 12; see also Popper 1972: 149, 1984: viii)

Such phenomena are certainly widespread. *M. tuberculosis*, for example, constructs its niche in the human lung by upregulating the human protein PPM1A, suppressing macrophage apoptosis, and prolonging the infection (Lee et al. 2009; Schaaf et al. 2017). Many organisms also choose their environments—with no scare quotes required—when they move about (Elton 1930; Huxley 1942: 113-5). But why, for Popper, does this challenge traditional Darwinism?

> Darwinism teaches that organisms become adapted to the environment through natural selection. And it teaches that they are passive throughout this process. But it seems to me far more important to stress that the organisms find, invent and reorganize new environments in the course of their search for a better world (Popper 1984: viii)

But Darwinism has never taught that organisms remain passive. Among professional biologists, there is a broad consensus that birds sometimes search for nesting sites, build nests, and migrate. What Darwinism teaches is that complex, apparently purposive phenotypes will aim at reproductive success.

To deny that animal choice is necessary for the appearance of design, is not to deny that choice ever occurs or has consequences; but Popper is not alone in eliding this distinction. Few have doubted, for example, that habitat choice is both common and consequential. But from the cavefish debates of the 1890s until the present day, researchers have disagreed about whether this undisputed fact is an integral part, an extension, or a refutation of traditional Darwinism (Lankester 1893; Cunningham 1893; Davenport 1903; Cuénot 1914; Fisher and Stock 1915; Elton 1930; Huxley 1942: 113–5, 524; Thorpe 1945, 1965: 15–16; Simpson 1953: 111; Waddington 1960: 399, 1969/2008: 264; Odling-Smee et al. 2003; Bateson 2004; Lewens 2005; Radick 2017; Edelaar and Bolnick 2019).

A challenge only subtly different to Popper's was advanced by the great evolutionary geneticist Richard Lewontin (1978, 1985)[17]. Lewontin's target was "the classical Darwinian theory of adaptation", whose flaw was that it "depends absolutely on the problem pre-existing the solution" (1985: 95). For while "the breaking of snail shells is a problem to which the stone anvil is the thrush's solution [. . . it] is a problem created by thrushes, not a transcendental problem that existed before the evolution of the Turdidae" (1985: 99). Similarly, and more straightforwardly, the problem of evading antimicrobial drugs (§5.3.2) is a problem that pathogenic bacteria helped to bring upon themselves, not a transcendental problem that existed before they had evolved[18]. In both cases, the sole "transcendental problem" is increasing reproductive success; subproblems arise by a complicated dynamical process, in which organisms no doubt played their part (§5.2.3). Fisher had made the same point in 1950, linking it explicitly to creativity.

> Just where does the theory of natural selection place the creative causes which shape evolutionary change? In the actual life of living things; in their contacts and conflicts with their environments, with the outer world as it is to them; in their unconscious efforts to grow, or their more conscious efforts to move. Especially, in the vital drama of the success or failure of each of their enterprises. [. . .] The theory of Selection seems to me also holistic, [. . .] in the mutual reaction of each organism with the whole ecological situation in which it lives—the creative action of one species on another. (Fisher 1950: 17-19; see also Fisher 1934: 111; Dobzhansky 1974: 336; Kokko 2021)

The major difference is that Fisher took himself to be describing Darwin's theory of adaptation, not presenting an alternative to that theory.

[17] Lewontin's presentation is best known from The Dialectical Biologist (Levins and Lewontin 1985), suggesting inspiration from Hegel via Marx (Maynard Smith 2001). Popper made no such attribution.

[18] Although both problem and solution seem to predate human-designed antibiotics (D'Costa et al. 2011; Larsen et al. 2022).

While the accounts of Fisher, Popper and Lewontin are all quite similar to one another, they are, once again, open to different interpretations (e.g., Laland and Sterelny 2006: 1760). We might, for example, read the theories as ignoring the problem of adaptation altogether, and simply reminding us that everything can affect everything else. A problem with this minimalist claim is that, while obviously true, it is not obviously useful (Kitcher 2001: 413 fn.8). Standard theory acknowledges that selection pressures can change, and for all sorts of reasons (e.g., Laland and Sterelny 2006: 1760), but for any practical purpose, some features of the world must be treated as static. The important choices have always been made on a case-by-case basis (Houston and McNamara 2005; McNamara and Leimar 2020, Ch. 1), and the new theories offer little further guidance.

Two remaining interpretations are minor variants of those met above. The first stresses that organisms not only change their environments, but *improve* them, thanks to behaviour that is either adaptive or fortuitously pre-adaptive (as, e.g., with the "soil improvement" activities of earthworms, discussed by Williams 1966: 18-19; see also Allee 1940; Laland and Sterelny 2006). If "improvement" is defined in terms of Darwinian fitness, then organisms are *creative* in the sense of §5.3.1. The boldest interpretation is that organisms are not simply one cause among many, nor even one determinant of evolvability among many, but set the agenda for evolution in a more fundamental sense; again, the evidence would be instances of apparent design that reflect their new and subjective purposes. These more radical suggestions are strongest, as we have seen, in Popper's writings, but they are not entirely absent elsewhere. It is notable, for example, that Lewontin framed his theory not only against "the classical Darwinian theory of adaptation" (1985: 95), but also against Dawkins (1976, 1982; Lewontin 1985: 88-9). This is puzzling because Dawkins, in The Extended Phenotype (1982), had presented a theory which was no less interactionist than Lewontin's (e.g., organisms modify their environments with consequences for the organism's reproductive success in that environment), and where organisms are no less active (birds, e.g., building nests). Dawkins stronger claim, of course, was that some of the environmental modifications should be recognized and studied as adaptations, and as adaptations that function for replicator success (e.g., Weber et al. 2013).

One question, therefore, is whether anything in Lewontin's treatment really does challenge anything in Dawkins'. How could a gene-centred theory of the purpose of adaptations be undermined by a pair of coupled differential equations (Lewontin 1985: 104-5; Laland and Sterelny 2006)? The impression that these two approaches are true alternatives—different ways of addressing the same question—might arise from a defunct theory of purpose that was popular in the heyday of cybernetics. In this theory, agency is more-or-less identified with feedbacks, and goals with stable states (Rosenblueth et al. 1943; Thorpe 1965: 15–16; Nagel 1977; Neander 1991: 455). Waddington had also invoked these ideas, combining feedback loops—a little paradoxically—with an *active organism going first* (Waddington 1959, 1960: 401). From the cybernetic perspective, the language of agency might be equally applied to organisms, and to the earth as a whole (Lovelock and Margulis 1974; Saunders 1994); and there is little difference between co-evolution and niche construction

(Lewontin 1985: 105; Godfrey-Smith 2017: 4), because each may involve similar patterns of feedback. From the perspective discussed in §5.4.1, these situations are fundamentally different, because the responses of inorganic nature cannot be reliably predicted with the assumption of rational agency (Dawkins 1982, Ch. 14; 2004: 378-9; see also Williams 1966: 75). Feedbacks do play important roles in some traditional theories of adaptation—as, for example, with Fisher's runaway model (§5.3.2)—but, like plasticity (§5.4.1), feedbacks are not essential to the traditional accounts. The main trouble with cybernetic theories of purpose is that feedbacks and equilibria are ubiquitous, and so the theories recognize purpose where there is none to be found—i.e., where the assumption of purpose gives us no predictive or explanatory power (Bedau 1992; Mayr 1992: 130). But this very failure can be a source of appeal. An over-liberal theory of purpose can give the false impression of a true plurality.

It is important to note that Lewontin never endorsed a cybernetic account of purpose. He saw clearly that a description of a dynamical system would not do the same explanatory work as "the adaptationist program"; and that each was an essential part of biology, incapable of replacing the other (Lewontin 1978: 220-2; 1985: 97; see also Rosenberg 2000, Ch. 5). But he remained overwhelmingly hostile to actually existing adaptationism (Lewontin 1977, 2002: 9-10; Gould and Lewontin 1979; Maynard Smith 2001), and despite his own warnings, sometimes seemed willing to "throw the baby out with the bathwater" (Lewontin 1978: 228-30) and abandon attempts to explain apparent purpose altogether (Lewontin 2002, 2010).

5.5 Conclusion: Creative Ambiguity

This chapter has argued that traditional evolutionary theory does recognize natural selection as uniquely creative (Laland 2018). But it does so in one sense and one sense only. Natural selection explains the appearance of purpose in nature; it can be distinguished from alternative sources of purpose because it generates (extended) phenotypes that function to increase reproductive success.

In other senses, traditional theory recognizes many sources of creativity. A very large number of factors might either (1) increase evolvability and accelerate adaptation, perhaps, thereby, explaining why adaptations could evolve at all, or (2) influence the evolution of adaptations in ways that are interesting or predictable, but not in themselves adaptive, such as biasing evolution toward a subset of the possible adaptive outcomes (Wright 1932; Simpson 1947; Fisher 1950). Natural selection might be creative in these senses too, but not uniquely so.

The chapter has further argued that debates about evolutionary theory have often been hampered by a failure to distinguish between these senses of creativity. Symptomatic of this failure is the tendency—well documented by Beatty (2016, 2019)—to argue about whether some factor might *direct* or *initiate* evolutionary change. Neither of these terms has an obvious meaning when applied to evolution, not least because they hover between "respectable-sounding" descriptions of dynamical systems, and "important-sounding" indicators of purposive action and

agential causation (in something like the sense of Chisholm 1964). As a result, they can be applied to creative factors of qualitatively different kinds (e.g., Fisher 1930, Ch. 1; Endler and McLellan 1988: 417).

This ambiguity sometimes appears as a confusion of explanatory goals. For example, is the "ultimate explanation" of an adaptive trait (Mayr 1961) something like its complete evolutionary history, or something like its adaptive rationale (Ariew 2003; Laland et al. 2011; Gardner 2013)? To describe the former, we might need to invoke many different creative factors (and many that are not creative). To describe the latter, we need to focus on the *uniquely* creative factor: natural selection (Gardner 2013).

These different modes of creativity can also be attributed to organisms; this means that organisms might play a creative role in evolution in many different senses. Failure to distinguish between these senses may explain the assumption—found in Popper, Waddington, and many others—that traditional Darwinism *denies them all*. One of Darwin's central claims is that the apparent purpose of adaptations is not to be explained by invoking purposes that were represented or endorsed by anyone, very much including the organisms that manifest the adaptations. This implies that most non-human organisms are not creative in this very limited and specific sense. But this claim seems to have been confused with the absurd denial that organisms ever make choices with consequences, so that they cannot be creative in *any* sense. Organismal choices might be consequential in lots of different ways (e.g., Fisher 1950; Rosenberg 2000, fn8: 55-6), and only a very few of these would challenge traditional Darwinism. For example, evidence that agential behaviour, plasticity, or niche construction increases evolvability (§5.4.2), would have no bearing on gene-centred theories about the purpose of adaptations (Williams 1966; Dawkins 1976).

Blurring these distinctions is the fact that we—people—*are* novel sources of purpose in nature; we can shape the world with results that are undeniably purposive and undeniably decoupled from reproductive success (§5.4.1). Agential behaviour, plasticity and niche construction were all plausible prerequisites for our own freedom of purpose (e.g., Hobhouse 1901; Dennett 1975; Dobzhansky 1974: 326; Dawkins 1976; Popper 1978; Ross 2007; Sterelny 2020), and each has many homologues and analogues throughout nature. But none of these factors, on its own, implies a novel source of purpose. One of the real pleasures of studying evolution is noting the inauspicious origins of traits that would become hugely consequential in some lineage or other (Godfrey-Smith 2017). But we should not confuse potential and achievement. It is one thing to show that organisms are creative in the same way that we are, and quite another to show that they are creative in the same way as is genetic drift, or the transition-transversion bias: with actions that sometimes accelerate adaptation, and sometimes bias evolution towards a non-random subset of the possible adaptive peaks.

The danger of these ambiguities is that theories attract enthusiasm and suspicion for all the wrong reasons. Waddington's claim to have discovered a "more convincing explanation of how the appearance of design comes about" (1960: 386), set a very high bar for his theory, serving only to highlight its limited explanatory

achievements over the last 75 years. But this should not prevent us from acknowledging the explanatory work that it might still do in the future. Genetic Assimilation is, for example, a possible explanation for the lengthy lag phase that often separates the introduction of an alien species from its successful invasion (Pigliucci et al. 2006: 2364; Lande 2015). The importance of the process could then be judged after a systematic comparison with the many other possible explanations of this pattern (Davis 2009: 94). If it does explain the lag, the theory would have done valuable work—even if that work has nothing to do with "creativity" or with adaptations (Lewontin 1978: 216; Williams 1992: 31)—and even if it challenges nothing in traditional evolutionary theory.

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Let there Be Light: A Commentary on Welch

6

David Haig

Abstract

Any important word means many things. What is at issue in arguments about the source of creativity in evolutionary biology is opaque because the contending parties use a common vocabulary to mean different things. Welch begins with Laland's (2018) statement that "the burden of creativity in evolution does not rest on natural selection alone." Welch then specifies a sense in which he sees natural selection as *uniquely* creative, namely the production of adaptations. Other things may be created by other processes, but natural selection is the only process that produces adaptation. This specification of his intended meaning brings greater precision to a point of possible contention. Laland probably disagrees because he understands something different by the word "adaptation." Is anything more at stake than the use of different senses of "creative," "evolution," "adaptation," "produce," or where a "burden" rests? We are more likely to find common ground if we understand each other's meaning, but winning arguments is more attractive than seeking consensus. Charity of interpretation frequently succumbs to scoring of points. Life is cyclic but we tell linear stories. The linear form predisposes to asking the question what comes first and what comes after, but in a cycle one kind of thing can come both before and after another kind of thing. In the Jewish creation myth, the world is created in a spontaneous act of will. This has predisposed scientists in the Western tradition to seek the source of creativity in a single creative event. In the Hindu tradition, creation is cyclical with an important role played by the maintainer (Vishnu) and the destroyer (Shiva). This may be a more useful model of the creativeness of natural selection.

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Keywords

Creativity · Teleology · Recursion · Narrative · Vishnu · Shiva

> Artists have an interest in others' believing in sudden ideas, so-called inspirations; as if the idea of a work of art, of poetry, the fundamental thought of a philosophy shines down like a merciful light from heaven. In truth, the good artist's or thinker's imagination is continually producing things good, mediocre, and bad, but his power of judgment, highly sharpened and practiced, rejects, selects, joins together. (Nietzsche 1984: 107)

Any important word means many things. What is at issue in arguments about the source of creativity in evolutionary biology is opaque because the contending parties use a common vocabulary to mean different things. Welch begins with Laland's (2018) statement that "the burden of creativity in evolution does not rest on natural selection alone." Welch then specifies a sense in which he sees natural selection as *uniquely* creative, namely the production of adaptations. Other things may be created by other processes, but natural selection is the only process that produces adaptation. This specification of his intended meaning brings greater precision to a point of possible contention. Laland probably disagrees because he understands something different by the word "adaptation." Is anything more at stake than the use of different senses of "creative," "evolution," "adaptation," "produce," or where a "burden" rests? We are more likely to find common ground if we understand each other's meaning, but winning arguments is more attractive than seeking consensus. Charity of interpretation frequently succumbs to scoring of points.

Scratch the surface and many impassioned debates in evolutionary theory reveal different philosophical commitments toward teleological reasoning. Welch *explains* biological characters by their functions, indeed thinks this indispensable. He is happy to explain the existence of a behavior by what it achieves. But Laland et al. (2013) disagree: "functions are not causes ... the outcome of a behavior cannot determine *its* occurrence." These authors here misunderstand the recursive nature of natural selection and thus mistake a narrative convention for a fact about the world. The outcome of a behavior in one generation can be a cause of the performance of the same behavior in subsequent generations. [Laland would probably respond that I misrepresent his intended interpretation of *its*. Are behaviors of the same kind, the same behavior? It depends on whether "same behavior" refers to a single behavioral event or to events of this kind.]

An adaptationist narrative in which natural selection is the sole source of creativity is one way of telling a story. Phenotypic plasticity, learning, and other biological processes play important roles in this story but their purposefulness is portrayed as arising from some form of natural selection, where the latter is broadly defined as feedback from phenotypic consequences to differential replication of heritable material causes. In this telling of the tale, the purposiveness of learned behavior is grounded in natural selection in two senses. First, learning itself is akin to natural selection. Variation in performance informs progressive improvement at successive

attempts by feedback from consequences. Second, the ability to learn is a product of natural selection. Natural selection is presented as *cause* and learning as *effect* of that cause.

Adaptationist narratives frequently ignore genetic drift because neutral variation does not make a difference for fitness (by definition of neutrality) and therefore cannot be a source of adaptation. Adaptationists do not deny that genetic drift has important evolutionary consequences, including the finding of non-neutral sequences by random walks, and they accept that what was once neutral may cease to be neutral as circumstances change, but these are other stories. We may disagree whether drift is creative but this is a dispute about semantics not fundamental phenomena.

West-Eberhard (2003: 3) tells an enchanting tale of developmental plasticity leading genes through the perils of the evolutionary woods. In her telling, a mutation has often been seen "as the *originator* of a new phenotypic trait" but development is really "the *originator* of all adaptive change." Putting "the flexible phenotype *first*" necessitates "changes in thinking about virtually every major question of evolutionary biology" and resolves "some of its most persistent controversies" (emphases added). The creative retelling of stories enriches the understanding of a reader who accepts there is more than one way the story can be told.

Once upon a time . . . Narratives concern unique events. Judaeo-Christian creation myths emphasize spontaneous acts of creative will. Things come into being and progress toward an end. A linear conception of time has subtly shaped the way creativity is conceptualized in evolutionary biology. It brings to the fore arguments about what comes first, about the initial creative act, about original adaptations rather than uncreated exaptations. Do developmental mechanisms or genetic mutations initiate change? Does phenotypic plasticity precede adaptation by natural selection or is it an adaptive product of natural selection? Do organisms create the selective forces to which they respond or are they created by these selective forces? Causes come before their effects. Isn't that obvious?

Linear time proceeds to a goal. Cyclic time repeats with variation. In the Hindu tradition, the world goes through cycles of dissolution and new creation. Trimurti is three gods in one: Brahma (creator), Vishnu (preserver), and Shiva (destroyer). In feminine guise, these are Tridevi: Saraswati, Lakshmi, and Kali. All deserve reverence. Veneration of destruction becomes intuitive once Shiva and Kali are conceived as destroyers of evil, enemies, and the less perfect. The destructive can be generative and transformative. In modern Hinduism, Brahma, the ostensible creator, plays third fiddle to Vishnu and Shiva but Saraswati has maintained prominence through her association with art and learning and Kali's fearsome nature has been tamed in the gentle form of Parvati.

The Hindu conception of continual transformation provides a different model for thinking about evolution by natural selection, with Brahma conceived as mutation, a source of new difference, and Vishnu and Shiva conceived as the two faces of selective acts that erase difference by preserving one side of a difference and eliminating the other. The triune cycle of birth, life, and death, repeats endlessly. Cause and effect become intertwined. Mutations are subject to selection, but the

mutable material has already undergone countless prior rounds of mutation and selection. Brahma randomly modifies materials forwarded by Vishnu, then Vishnu preserves, and Shiva destroys some of these newly modified materials. Transmutation occurs before and after selective judgments, endlessly. The question who acts *first* loses sensible meaning. Trimurti and Tridevi work as a team in the cyclic creativity of natural selection.

The narrative form is linear. Life is cyclic. Reproductive recursion challenges the dogma that one must not confuse cause and effect. Chickens produce eggs that develop into chickens who produce eggs. The phenotypes of former genes determine which future genes express phenotypes. Is phenotype a cause or an effect of genotype? Does it precede mutation or arise by mutation? Does evolution shape development or is it the other way round? Many stories can be told. Many disputes in evolutionary theory reflect narrative choices of where to begin a story rather than substantive points of disagreement. It is the "loopiness" of natural selection sifting products of mutation that creates the purposiveness of living things (see Chap. 6).

A long-standing criticism of neo-Darwinism is that it assigns organisms a passive role in the evolutionary process rather than sees them as active *initiators* of adaptive change. Champions of the organism see this as an impoverished view. But is this a central claim of Darwinism? Questions of origin frequently misconstrue the nature of reproductive recursion. Organisms are parts of each other's selective environments, and their purposive behaviors are important "selective pressures" that have shaped those behaviors. This should be completely uncontroversial. Similarly, but less obviously, bodies and genomes are parts of the "environment" that selects among genetic variants that reside in those bodies and genomes. The differential abilities of organisms to make apt choices are differences on which selection can act. Fisher (1950; quoted by Welch) wrote that the creative causes which shape evolutionary change reside in "the actual life of things … in the vital drama of the success or failure of each of their enterprises." If Fisher is a heretic then no one is orthodox!

Welch foregrounds Popper's, Waddington's, and Lewontin's advocacy for a more creative role for organisms. He could have added the apostles of autopoiesis who accept teleology but reject the Darwinian explanation (Weber and Varela 2002). Living things appear purposive and creative. Some reject this as an illusion. Others accept it as real but consider natural selection an inadequate explanation and therefore must invoke other factors to explain the manifest purpose. Both mechanists, who see natural selection as a dubious metaphor, and anti-mechanists who reject natural selection as soulless mechanism wish to cleanse biology of the taint of adaptationism.

Two issues have converged in debates over teleology and creativity in biology. The first are different attitudes toward teleological reasoning. Some deny that purposes (final causes) are causal and explanatory. For many of these deniers, the language of purpose is appropriate only for human endeavors. The second is whether natural selection by itself is adequate to explain the adaptedness of living things. I believe that there is an important sense in which natural selection is ultimately responsible for all purposiveness in the world, including the purposiveness of human beings in all of their cultural complexity, and of the machines and artistic

works humans create. Feedback from consequences, including feedback from simulated consequences, is the only process I know that is able to match ends to means. This is "Darwin's dangerous idea" (Dennett 1995; Haig 2020).

On this view, as argued by Welch, natural selection of random mutations has been the source of all biological adaptations, including adaptive phenotypic plasticity, mechanisms of epigenetic inheritance, systems of adaptive mutation, and the ability to learn by ourselves and from others. (I intend "random" in the sense of not directed to an end.) Adaptationists believe that the question "what is it for?" is an essential question in biology. Finding the answer to this question is often difficult. Many biologists would prefer to avoid the question. The study of adaptation is a hard (in the sense of difficult) not a soft science. It is easy to do poorly and difficult to do well. The most productive approach is not to deride the whole enterprise by demolishing carefully chosen examples, *selected* to ridicule, but by showing how it can be done better (but perhaps I neglect the creative role of Shiva in the critique of the adaptationist program). Fisher (1950: 19) can have the last word:

> For my own part I confess to feeling heartily relieved that it is not necessary to regard the life and death drama of the myriads of individual existences as a play, a make-believe, a shadow-show, having, for all the intensity and effort squandered in them, no real effects or consequences . . . living things themselves are the chief architects of the Creative activity . . . on the Darwinian view by doing or dying. It is not the mere will but its actual sequel in the real world, its success or failure, that is alone effective.

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Creative Destruction: A Reply to Haig

7

John J. Welch

Abstract

I am not sure that David Haig and I disagree on very much, partly because my ideas are often copied from him. I strongly agree that discussions about evolutionary theory should try to generate light rather than heat, and I feel that my tone has not always been helpful for this.

Keywords

Adaptation · Creativity

> I remember, the players have often mentioned it as an honour to Shakespeare that in his writing (whatsoever he penned) he never blotted out line. My answer hath been, would he had blotted a thousand. Which they thought a malevolent speech. (Jonson 1641: 97)

I am not sure that David Haig and I disagree on very much, partly because my ideas are often copied from him. I strongly agree that discussions about evolutionary theory should try to generate light rather than heat, and I feel that my tone has not always been helpful for this.

The irritating archness of my chapter, like its excessive length, stems from my real uncertainty as to what debates about "creativity in evolution" are really about. Are they largely empty—just products of simple misunderstandings, fuelled by the shared conviction that *our* work is unjustly neglected? Or are they, by contrast, deep disagreements about causality, which I am not qualified to understand?

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At least some of the debates do seem largely semantic. As Haig's epigraph nicely illustrates, some writers will associate creativity solely with the moment of origination and initiation, while others are happy to acknowledge the role of selecting and rejecting (Eliot 1932: 12–24; Dehaene 1997: 170–2), and might agree with Stanley Kubrick—undeniably a source of direction—when he remarked "I never know what I want, but I know exactly what I don't want" (Kirkpatrick 2015: 65).

A central question, as Haig also notes, is what should count as a cause of adaptation. In my chapter, I tried to steer a middle course, arguing that, in one sense, lots and lots of things might cause adaptations (including as necessary causes—explaining why adaptations exist at all), while still insisting that, in another important sense "natural selection is the only process that produces adaptation". Unless it is understood in a particular sense, the second claim seems too far-fetched to have been believed by anybody. G. C. Williams, for example, discussed positive niche construction in the first few pages of his first classic book (1966: 18–19, see also 12–13, 31), so when he later wrote that

> Adaptation is always asymmetrical; organisms adapt to their environment, never vice versa. (Williams 1992: 484)

I don't think he *could* have meant that environments never improve, i.e., that new resources never appear, and predators and parasites never go extinct, or that animals never move to pastures new, and never build shelters that work. And I don't think Williams meant that these things rarely happen, or that evolution would have taken much the same course whether they happened or not (Fromhage and Houston 2022). Analogously I don't think that "natural selection is the only process that produces adaptation" *could* mean that mutations are never beneficial, or that plasticity is never adaptive, or that drift-driven changes are never essential intermediate steps (Cf. Gould and Lewontin 1979: 592; Endler and McLellan 1988: 408; Stoltzfus 2012). All these topics are interesting—and traditional—areas of study, but by adding items to the list, we can lose sight of what makes natural selection special. For some purposes, more does not mean better.

Evolutionary theorists can share the broad goal of explaining adaptation, and yet interpret this goal in subtly different ways. I am grateful to David Haig for showing me how we can negotiate these differences without the need for conflicts.

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Part III

The Organism in Evolutionary Explanation: From Early Twentieth Century to the Extended Evolutionary Synthesis

8

Jan Baedke and Alejandro Fábregas-Tejeda

Abstract

In recent years there have been a number of calls for integrating developmental and organismal phenomena into evolutionary theory. This so-called Extended Evolutionary Synthesis (EES) argues that evolutionary theory should not primarily explain certain evolutionary phenomena by highlighting genes and populations but organisms instead, in particular how their development and behavior biases and drives evolutionary change. Here, we offer a new historiography that focuses less on the differences between the EES and the Modern Synthesis but seeks to provide a better understanding about which theoretical and explanatory traditions the organism-centered framework of the EES draws on. This concerns especially three currently resurfacing explanatory roles granted to organisms in evolution: organisms should allow (1) contextualizing parts in development, especially genes, (2) focusing on reciprocal organism-environment relations (in contrast to, e.g., gene-environment interactions), and (3) understand the role of agency in evolution. Through this analysis, we show that the EES advances a revival of older explanatory roles granted to the organism in evolutionary research, which became marginalized in the second half of the twentieth century. This new perspective helps to re-center contemporary theoretical debates towards relevant questions of explanatory standards in evolutionary biology.

Keywords

Organism · Modern Synthesis · Organicism · EES · Explanation · Explanatory standards

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8.1 Introduction

What does the proposal to extend the evolutionary synthesis mean? *Prima facie*, the answer to this question seems to be straightforward. "Extending" suggests that there exist older or accepted consensus practices and standards of theorization in evolutionary biology that should be augmented or widened.[1] The label of *Extended Synthesis,* introduced by some evolutionary biologists (Pigliucci 2007; Müller 2007, 2017d; Pigliucci and Müller 2010b; Laland et al. 2014, 2015), seems to clarify this historical reference point even more. It suggests a relation to the *Modern Synthesis* (MS). In short, if the name is taken at face value, the Extended Synthesis tries to broaden some features of the MS. In this chapter, we argue that this historiography is limited and currently hinders fruitful theoretical debates about what is epistemologically entailed by (and explanatorily relevant within) the framework of the Extended Evolutionary Synthesis (EES; *sensu* Laland et al. 2015).

Discussion about the historical status of the EES usually adopts a contrastive approach that separates it from the MS. This holds for both its advocates (Laland et al. 2014, 2015; Pigliucci 2017; Müller 2017d; Jablonka and Lamb 2020) as well as its critics (Wray et al. 2014; Futuyma 2017; Gupta et al. 2017; Lu and Bourrat 2018; Charlesworth et al. 2017; Svensson Chap. 11, this volume). In this common contrastive framework, the innovative potential and novelty of the EES for evolutionary biology is emphasized or downgraded, respectively, depending on how liberal the MS was considered to be with respect to integrating developmental phenomena, such as developmental bias, plasticity-led evolution, and niche construction. The results of these historical assessments are then taken to be sufficient by most authors to embrace or reject the project of an EES altogether. However, this bundle of historiographic problems (e.g., how plural, or gene-centered the MS was with respect to developmental causes of evolution or channels of inheritance, how novel the ideas stemming from the EES really are, etc.) should not be conflated with the theoretical and philosophical problem of what EES-type explanations (if anything) could bring to evolutionary biology (see, for instance, Baedke et al. 2020).

In other words, the EES debate has so far been overly fixated on the labels that surround it. This focus on what is suggested by the *name* prevents drawing sufficient attention to the ideas and explanatory roles central concepts play inside the framework elaborated by Laland et al. (2015). Here, we contend that two central concepts reintroduced by the EES are that of the *developing organism* as a causally efficacious unit in evolution and the *organism-environment relationship* as a fundamental frame to study reciprocal, protracted evolutionary interactions. The so-called organism-centered perspective of the EES (Laland et al. 2015) captures the idea that organism-centered—rather than gene- or population-centered—explanations of evolution provide a perspective, often neglected since the mid-twentieth century,

[1] Such widening could be understood, for example, in terms of its models, the domain of application of theories, or what experimental practices can be derived from it (see Grisemer 2019).

that would broaden our understanding of evolution (see also Uller et al. 2020; Uller and Laland 2019). Adding explanations of developmental and organismal causes, studied in fields and research areas such as evolutionary developmental biology (Evo-Devo), epigenetics, and niche construction theory, to the causal picture of evolutionary theory should lead to "more complete explanations" (Laland et al. 2015) and a "significantly expanded explanatory capacity" (Pigliucci and Müller 2010a: 12). Interestingly, while there is often agreement in evolutionary biology over the existence of these phenomena, at the same time, their explanatory relevance is questioned (Wray et al. 2014; Futuyma 2017; Dickins 2020; Svensson 2020).

Against the background of the stalemate the EES debate has seemingly reached at this point with many evolutionary biologists talking past each other, in this chapter, we seek to shift the focus of the discussion away from debating names towards more thorough and theoretically more fruitful historical analyses. Therefore, we concentrate on the explanatory roles the organism plays in the EES, and on how these roles have been defended and criticized in the history of evolutionary biology. We take a historical approach that asks: Where does the idea of the organism as a central explanatory unit in evolutionary theory come from and what is the focus and structure of organism-centered evolutionary explanations in the EES? What roles should we grant to the organism and to organism-environment relations in evolutionary explanations? Should it matter to emphasize the organism to build a richer evolutionary science?

We will show that, when directing the attention to organism-centered explanations, it becomes possible to see that the EES unwittingly reintroduced certain roles granted to the organism in early twentieth-century organicist biology, but which later became lost or marginalized in evolutionary biology. We identify three currently resurfacing explanatory roles organisms are thought to play in evolutionary biology: organisms should allow (1) contextualizing parts, especially genes, in development; (2) focusing on reciprocal organism-environment relations (in contrast to, e.g., gene-environment interactions); and (3) understanding the role of agency in evolution.

In what follows, we, first, discuss the conceptualization of organisms in the early twentieth-century biological debates (in organicism, dialectical materialism and holism; Sect. 8.2), and identify the three above epistemic roles ascribed to organisms in evolutionary explanations (Sect. 8.3). Second, we show how each of these roles was marginalized by evolutionary biologists in the second half of the twentieth century (Sect. 8.4), before being recently reintroduced by research conducted under the umbrella term of the EES (Sect. 8.5). We close with an outlook on how this history could stir fruitful debates about the conceptual and theoretical framework underlying the EES and about the explanatory standards evolutionary biologists want their evolutionary explanations to hold (Sect. 8.6).

8.2 The Organism Before the Modern Synthesis

At the dawn of the twentieth century, intense discussions revolving around the conceptual, epistemic, and ontological foundations of biology arose in several Anglo-Saxon and German-speaking scientific communities. According to Laubichler (2017: 95–96), this debate focused on (a) the divergence between a rapidly increasing number of new empirical findings and experimental results on the one side, and a lack of conceptual and theoretical frameworks on the other, which resulted in a data crisis around 1900; (b) the attempt to establish the foundations of biology based on these new findings; and (c) the evaluation of the epistemological and methodological preconditions of biological research.

Embryologist Julius Schaxel, for instance, mulled over the state of biology at his time and asserted that "[a] general biology, a science of life as such, exists in name only" (Schaxel 1919: 2; German original). In response to this widespread sense of crisis (see also Baedke 2019), scholars reflected upon the basic concepts that underpin biology. One of such was (and still is) the *organism concept*.[2]

In the first decades of the twentieth century, especially in the interwar period, manifold biological perspectives centered on the organism emerged in different local contexts of the globe (Haraway 2004 [1976]; Esposito 2017; Baedke 2019; Baedke and Brandt 2022). In recent years, historians have begun to study them in depth mainly in three geographical and geopolitical contexts: in Great Britain (Nicholson and Gawne 2014, 2015; Peterson 2016), the United States of America (Esposito 2016), and in Germany and Austria (the tradition of German holism; Amidon 2008; Rieppel 2016; Müller 2017c; Baedke 2019; Fábregas-Tejeda et al. 2021). Different stances within the organicist movement (on this notion, see Nicholson and Gawne 2015) represented a break in the dichotomous opposition between mechanism and vitalism (see Allen 2005), and, by integrating elements from both positions, were presented as alternatives that allowed to settle this fierce, long-lasting debate in history of biology (Beyler 1996: 252; Haraway 2004 [1976]: 2; see also Schaxel 1917).[3] For example, in embryological investigations, the organicist movement tried to reconcile ontological materialism with observations of biological emergence, and, in that sense, it paved the way between unassailable vitalism and reductionist mechanism (Gilbert and Sarkar 2000: 3).

As historian Herbert J. Muller asserted, scholars from the organicist movement, in contrast to abstract vitalists and staunch mechanists, wanted to re-center biological explanations on the living organism: "The vitalists insisted that some altogether new principle—an entelechy, an *élan vital*—was necessary to explain life; the mechanists insisted that the principles of physics were not only adequate but essential. Both tended to lose sight of the living organism in their logical dispute over explanation"

[2] For a history of the organism concept from the seventeenth to the nineteenth century, see Cheung (2006, 2014).

[3] On whether the vitalism-mechanism was ever fully settled, see Peterson and Hall (2020); see also Hein (1972).

(Muller 1943: 106). For organicists, "(...) the fundamental fact in biology, *the necessary point of departure is the organism*. (...) Although parts and processes may be isolated for analytic purposes, they cannot be understood without reference to the dynamic, unified whole that is more than their sum" (Muller 1943: 107; emphasis added).

Similar to today's organism-centered perspective of the EES, members of the organicist movement defined "the living individual [as] the fundamental unity of biology" (Russell 1930: 166), and "organism" as a special way of thinking or a *proto concept* ("Urbegriff"; Bertalanffy 1928: 74).[4] The physiologist John Scott Haldane (1917: 3) christened this new biology *organicism*, zoologist William Emerson Ritter (1919: I 28) *organismalism*, and theoretical biologist Ludwig von Bertalanffy (1932: 80) "organismische Biologie" (*organismic biology*). evolutionary theory in recentSimilarly, the embryologist and theoretical biologist Julius Schaxel called for an *organismic basic conception* ("organismische Grundauffassung"; Schaxel 1919: 125) of biology.

At least three different theoretical strands can be identified inside the organicist movement (see Baedke 2019): *organicism* (e.g., Ludwig von Bertalanffy, Lawrence J. Henderson, William Emerson Ritter, Edward Stuart Russell, Conrad Hal Waddington, Paul Alfred Weiss, Joseph Henry Woodger), *dialectical materialism* (e.g., John Desmond Bernal, Joseph Needham, Marcel Prenant, Julius Schaxel), and different versions of *holistic biology* (including German "Ganzheitsbiologie"; e.g., Friedrich Alverdes, Bernhard Dürken, Kurt Goldstein, Adolf Meyer-Abich, Hans Böker, John Scott Haldane, Jakob von Uexküll, Emil Ungerer, Jan C. Smuts, William Morton Wheeler).

Members of this heterogeneous movement agreed on the following two viewpoints (Nicholson and Gawne 2015; see also Beckner 1969), albeit with different nuances: First, the organism is the most central ontological unit in biology. It transcends the properties of its parts (e.g., genes, cells), influences the part's organization in coordination with environmental cues, and actively constructs its environment. Second, the organism should be fundamental to frame scientific explanations in diverse biological subdisciplines. In particular, organismic organization, emerging in development and in constant interaction with the environment, should be the explanatory and methodological starting point of biology. Accordingly, many (if not all) biological processes—including evolutionary ones—can only be investigated effectively when the unit of the organism is considered.

Members of the organicist movement argued that the right theoretical framework of biology is neither reductionist mechanism nor vitalism, but a *third way* that builds on the above two premises. Against this background, in the next section, we focus on three central explanatory roles granted by organicist movement scholars to the

[4]For historians such as Peterson (2016: 249), British organicism was a "meta-theoretical commitment" about how to conceive organisms and living phenomena. This idea can also be found in Hein (1969), who acknowledged that what distinguished the organicist movement from mechanism or vitalism were different meta-theoretical commitments that lead to disparate standards for understanding (and evaluating) biological evidence and provided different heuristic frameworks.

organism in biological explanations. As we will see below, these discussions are thematically similar to those held within the EES debate (see Sect. 8.5).

8.3 Three Explanatory Roles of the Organism in the Organicist Movement

Thinkers in the organicist movement built their explanations of biological phenomena by ascribing chief epistemic roles to organisms. Here, we outline three central ones: (1) contextualizing parts (e.g., genes, cells) in development, (2) framing organism-environment causal reciprocity, and (3) understanding the role of agential processes in evolution.

8.3.1 Contextualizing Genes and Cells in Development

Organicists thought that the parts of organisms are molded and constituted in a dynamic interaction that involves the entire organism and its environment (Esposito 2017). Organisms, as dynamic wholes, have to be conceived as active entities that build themselves, capable of adapting and changing their forms and behaviors according to external circumstances. In a representative example of organicist rationale, E.S. Russell (1930: 149) asserted: "The life of an organism is essentially a unitary functional or dynamical process, in which *whole* and *parts* are inextricably interconnected. Both whole and parts are together the expression of the life of the individual."

The discussion about the relationship between organismic wholes and parts was important for the early twentieth-century biologists, especially in how they should conceptualize the phenomena of development and heredity. For instance, in his 1930 book *The Interpretation of Development and Heredity*, E.S. Russell asked:

> Is development to be treated as essentially an activity of the organism as a whole, or can its full explanation be found by analysing the process into its constituent elements? Is heredity essentially the reappearance and realization of the functional potentialities of the whole, or are the separate characters of the organism transmitted piecemeal, being represented separately in material form in the germ? Are development and heredity functions of the organism as a whole, or functions of its cells, or of still smaller constituent units? In general, is the organism a real unity or individual, not completely reducible to its constituents, or is it a mere composite, built up as a hierarchy of independent units? Can the whole be fully explained in terms of its parts, or must the parts ultimately be explained in terms of the whole? (Russell 1930: 2–3)

For some authors of the organicist movement, the organismic whole should always be explanatorily salient. In that same vein, Russell (1930: 240) maintained that "the organism is from the beginning a whole, from which by self-differentiation the parts are derived"; taking an idea from an epistolary exchange with philosopher of history R. G. Collingwood, he claimed that "(t)he parts are the way in which the

whole organizes itself" (Russell 1930: 240 fn1). Developmental biologist Bernhard Dürken contended that "[i]t should not be said that the organism as a whole is built up of parts, but that the organism, which is characterized through a consistent wholeness, develops parts and then, subsequently, has parts" (Dürken 1936: 17; German original). In other words, the whole temporally precedes the differentiation of the parts or, even more, for some authors, it is ontologically prior compared to the parts (Meyer 1935: 88). Thus, scholars of the organicist movement argued that organismic wholes always have to be investigated first in the study of development (see also Ungerer 1965: 80–82).

Leveraging this organicist framework of wholeness, organicist authors argued that heredity should be framed as the re-constitution of ontogenetic resources and causal interactions that bring about the constancy (or deviations) of form from one generation to the next.[5] Against the burgeoning views of geneticists of his time, Russell (1930: 16) argued that "the real cause of resemblance is the same factor that creates this organic architecture. *Hereditary resemblance is* [...] *a byproduct of development*, and will be explained only when we succeed in explaining development" (emphasis added). Heredity and development, Russell believed, should be jointly studied as they constitute two-sides of the same organic phenomenon.

8.3.2 Organism-Environment Reciprocity

The organicist movement not only rested on philosophical reflections and scientific theorizations, but was driven by multiple experiments that were carried out on the plasticity, robustness, and inextricable embeddedness of plants and animals in their environments (see Müller 2017a, c; Nickelsen 2017; Nicoglou 2018; Baedke 2019). Also noteworthy were studies on the environmental responsiveness of developing organisms, including their transgenerational effects, which were undertaken in the first half of the twentieth century (for example, the work conducted in Vienna at the *Biologische Versuchsanstalt*, directed by the Austrian zoologist Hans Leo Przibram; see, e.g., Müller 2017a, b; Nickelsen 2017: 170–175; Nicoglou 2018: 107–111).

In particular, the idea of reciprocity between organism and environment was a fundamental principle for multiple organicist positions. For example, J.S. Haldane (1884: 32–33) highlighted: "The organism is thus no more determined by the surrounding than it at the same time determines them. The two stand to one another, not in the relation of cause and effect, but in that of reciprocity." Organism-environment reciprocity was mainly construed as a relationship of *ontological co-constitution* or one of *reciprocal causation* (for a detailed analysis, see Baedke et al. 2021). According to the view of ontological co-constitution, organism and

[5] Russell (1930) strongly disagreed with the adjudication of a special causal status to lower level hereditary units such as genes or Weismann's biophors. He even argued that it is misguided "to ascribe to these units the powers and capabilities which we know only as belonging to the organism as a whole" (Russell 1930: 49).

environment are commingled and form a single interacting system that cannot be disentangled. In turn, reciprocal causation is usually defined as a feedback loop between two interacting, yet separate entities or processes (in this case, an organism and its particular environment). The notion of organism-environment reciprocity had different origins in organicism, dialectical materialism, and (German) holistic biology. For holistic thinkers, such as Haldane or the theoretical biologist Jakob von Uexküll, this idea was inspired by readings of Immanuel Kant (see Brentari 2015; see also, e.g., Ungerer 1919; Haldane 1931). In his third critique (*Kritik der Urteilskraft*), Kant described reciprocity as the distinctive organizational pattern of organisms. The organism is generated and maintained as a whole by the reciprocal interaction of its parts. This Kantian view of reciprocity between the organismal whole and its parts was expanded and applied to different levels, such as the relation between organisms and their environment (see Canguilhem 2008 [1965]).

For British organicists like embryologist Conrad Hal Waddington and theoretical biologist Joseph Henry Woodger, the perspective of A.N. Whitehead on the organism-environment relationship was highly influential.[6] Whitehead argues that there are "two sides to the machinery" of evolution (Whitehead 1925: 163). One side includes natural selection in which the externalist "givenness of the environment dominates everything." But there is another side which scholars had paid less attention to: "The other side of the evolutionary machinery, the neglected side, is expressed by the word creativeness. The organisms can *create their own environment*" (emphasis added).

Waddington picked up Whitehead's proto-niche construction idea (see Waddington 1929: 66, 1953, 1957: 104–108; Waddington et al. 1954). He argued that evolution involves not only changes in the genetic system, the epigenetic system, and the system of natural selection, but also in the "exploitive system." The last system refers to the influence exerted by the organism on its environment, which creates a feedback loop between organismal activities and environmental selection pressures: "Animals [. . .] live in a highly heterogeneous 'ambience', from which they themselves select the particular habitat in which their life will be passed. Thus the animal by its behaviour contributes in a most important way to determining the nature and intensity of the selective pressures which will be exerted on it" (Waddington 1959: 1635–1636). Waddington highlighted that, in order to develop a theory of evolution that includes the exploitive system, biologists should replace views of unidirectional causality with reciprocal causation: "we have to think in terms of circular and not merely unidirectional causal sequences" (Waddington 1960: 400; see also Baedke et al. 2021).

Another group of the organicist movement, dialectical materialists, came to similar views on reciprocity, although, compared to organicists and holists, through

[6] In general, British organicists were deeply inspired by Alfred North Whitehead's (1925) "philosophy of organism"—a systemic and processual view of the organism that emphasizes the complex interrelatedness of its developing parts with each other and the environment (see Peterson 2011, 2016; Nicholson and Gawne 2014).

quite different philosophical sources. They were influenced by Hegel's and Schelling's romantic philosophies of nature and by the writings of Karl Marx and especially Friedrich Engels' *Dialectics of Nature*. Dialectical materialists like Julius Schaxel (1931), biochemist Joseph Needham (1937) or zoologist Marcel Prenant (1935) argued that all processes in nature comprise reciprocal influences between antagonists that lead to qualitatively different and novel forms (or levels) of organization (see, e.g., Hopwood 1997). They argued that these formations (from quantitative reciprocal interactions to qualitative novel forms) could only be captured by a *dialectical biology* (Schaxel 1931: 492), a conceptual framework focusing exclusively on the mutual interactions between organisms or the organism and its environment. This dialectical framework formed the theoretical background of Levins and Lewontin (1985)'s book, although these influential evolutionary biologists did not acknowledge this older and rich theoretical tradition.

8.3.3 Organismal Agency

The observations that organisms have the ability to actively react to environmental changes, autonomously construct and maintain their organization and identity despite changes in material composition and form, regenerate, self-reproduce, etc., have long puzzled philosophers and scientists. How do we explain the apparent purposiveness of organismal development and actions? Are organisms agents of their own development and evolution? To put it simply, we could say, following philosopher of biology Robert Wilson (2005: 6–7), that "an agent is an individual entity that is a locus of causation or action. It is a source of differential action, a thing from which and through which causes operate."

Many authors that belonged to the organicist movement defended the general view that organisms "differ from machines [...] by virtue of the fact that their purposiveness is internal or immanent, and also because their form and activities are regulable" (Russell 1924a: 267; see also Nicholson 2013). The intrinsic purposiveness of organisms (in contrast to the extrinsic purposiveness of machines, always set by an external designer) means that organisms, through their activities (that are usually responsive to environmental inputs and contingencies), pursue goals of their own, such as surviving, reproducing, overcoming challenges throughout life cycles, or simply maintaining their organization in manifold developmental contexts.[7] For example, Russell devoted entire books to review the empirical manifestations of

[7]This should not be confused with the idea that there is an underlying teleology in all of Nature (what Okasha 2018 would call "agential thinking type 1"). Authors in the organicist movement embraced type 2 of "agential thinking" (*sensu* Okasha 2018), i.e., they conceptualized organisms as evolved agents, as difference-makers in the world, but that did not lead them to embrace speculative views about the underlying purposes of Nature (for example, seeing natural selection as picking out phenotypes in accordance to a preordained goal). The teleological explanations of the organicist movement were mainly concerned with the agentic character of organisms and not with a guiding *telos* in life or evolution.

organismal agency in developmental phenomena like regeneration and in animal behavior (Russell 1934, 1945; see also Rignano 1930 for a similar case).

In general, theories of organismic agency and/or the constructive potential of organisms were widely discussed by members of the organicist movement. These authors tried to develop a middle position between, on the one hand, attempts to outlaw the concept of purposiveness from the study of organisms (or to restrict it to intentional behaviors; see, e.g., Roux et al. 1912: 460) and replace them altogether with mechanistic explanations that rest on physiochemical reduction, and, on the other, vitalist endeavors to frame organismic purposiveness in terms of non-physical influences, such as Driesch's (1908) postulation of the entelechy. By drawing on rich philosophical sources, like older Aristotelian and neo-Kantian debates, as well as on phenomena such as self-organization and the explorative processes of development and behavior, they tried to better understand the goal-directedness of organisms (e.g., Haldane 1917; Schaxel 1919; Russell 1924b; Bertalanffy 1928). According to their views, the organism molds itself and its environment in development and evolution, like "clay modeling itself" (Russell 1924b: 61). In particular, the organism was conceived as an "active environment-related subject" (Meyer-Abich 1948: 39; German original).

The active role that organisms play in evolution was pointed out in myriad scientific works of the organicist movement (for an analysis, see Esposito 2017; see also Nicoglou 2018: 111–116). John Scott Haldane, for example, emphatically rejected conceptions of organisms as passive subjects in evolution (see Haldane 1935). The German botanist Emil Ungerer parted ways with the regulative ideal of the Kantian view of teleology (assumed only as a heuristic principle to guide scientific research) and, instead, investigated agency as a constitutive property of organisms in his studies of plant regulation (see Ungerer 1919). In the same line, the Dutch eco-morphologist Cornelis van der Klaauw argued that the heuristic view of agency was not enough for biology, and that organisms exhibited constitutive purposiveness during development. In his eco-morphological approach, van der Klaauw (1948) developed frameworks to study animal morphology that took several elements into account, such as the ecological setting of the organism and its environment, the functions performed, as well as the relationships between the organism and its conspecifics, and those established with other species.

The theory of *Umkonstruktion* by the German eco-morphologist Hans Böker, fueled through various field excursions and empirical research in several vertebrate species, can be interpreted as another example of investigation around some facets of organismal agency and evolution. According to Böker (1935), the organism should be understood as a historical whole that is in harmony with its parts and with the environment in which it thrives; whenever this bio-morphological equilibrium is disturbed by changes originating in its surroundings, the organism must strive to regain it, otherwise it is at risk of dying. The morphological perturbations prompted by environmental change can subsequently bias variation in the interrelated parts of the organism in a long series of changes that dovetail to restore the bio-morphological equilibrium. For Böker, this did not happen through a *bona fide* Lamarckian process, but rather, by a multigenerational selection process similar to

what would later be called *genetic assimilation* (for an analysis of Böker's stance, see Fábregas-Tejeda et al. 2021).

Even Conrad Hal Waddington would assign some important evolutionary role to organismal agency in his later works. His basic idea was that "before an organism's environment can exert natural selection on it, the organism must select the environment to live in" (Waddington 1961a: 89). For instance, he subverted the textbook exemplar of adaptation and directional selection of the peppered moth in the industrially darkened forests of Great Britain through his view of the exploitive system. Waddington (1961a) highlighted that, in the industrial melanism case, before natural selection can sort out variants according to their fitness differential, organisms first have to select the environment in which they will live. And this act that occurs during the ontogeny of peppered moths is not devoid of evolutionary significance: "The effective environment in which they are subjected to natural selection is, in fact, the darkened bark which *they themselves choose*; it is not something completely external, but is a combination of the outside world and the moth's own behavior" (Waddington 1961a: 90; emphasis added; for an overview of Waddington's evolutionary and ecological views, see Fábregas-Tejeda and Vergara-Silva 2022).

As a final example of agential thinking in the organicist movement and its heirs (of many more that could be cited), British ethologist William Homan Thorpe argued that the behaviors of organisms not only affect their development, but have downstream causal effects in the speciation patterns of populations. For example, he studied how genetic changes could follow and make an acquired behavioral preference hereditary (e.g., Thorpe and Jones 1937; Thorpe 1940), a phenomenon akin to what was later called the *Baldwin effect* by Julian Huxley and George Gaylord Simpson. In fact, historian of science Gregory Radick (2017) contended that Thorpe's thinking actually infiltrated into the Modern Synthesis. Thorpe's empirical work, as publicized by Julian Huxley in his 1942 book *Evolution: The Modern Synthesis*, served to popularize and revitalize "the fortunes of what became one of the mainstays of agential science [in evolutionary research], the Baldwin effect" (Radick 2017: 35). In that sense, Radick claims, the Modern Synthesis was not completely inimical to animal agency, a point that we will revisit in the next section.

As we saw, authors in the organicist movement granted to organisms three central explanatory roles: (1) contextualizing genes and cells in development, (2) underscoring organism-environment reciprocity, and (3) incorporating the role of agency in evolutionary processes. But what happened to these organism-centered frameworks after their heyday during the interwar period? Why do only few evolutionary biologists know about them today? To that we turn our attention now.

8.4 Streamlining the Organism After the Modern Synthesis

Recently, historians of biology have investigated some of the reasons that explain the (almost complete) disappearance of organicism from discourses, theorizations about the phenomena of the living and biological practices in the second half of the twentieth century. Donna Haraway (2004 [1976]) argued that, in the case of the Theoretical Biology Club, the organicist stance was diluted because it did not have the correct institutional (and disciplinary) support to keep it afloat (see also Peterson 2016):

> Needham tried to construct an institute around the new paradigm commitments but was unable to obtain needed financing. Beginning in 1934 he corresponded with Dr. Tisdale of the Rockefeller Foundation, which was then interested in fostering study on the borderlines of traditional disciplines. [...] Needham submitted a long memorandum outlining a plan for an Institute for Physico-chemical Morphology [...]. By 1938 the idea was dead [...]. The reasons are controversial and complex, but the success of Needham's institute certainly would have altered the course of biological investigation in England after the 1930s. Instead, factors combined to break up the collaboration of members of the paradigm community, and World War II finally sealed the issue (Haraway 2004: 134).

For example, in his original plan for an organicist research institute (see also Abir-Am 1987), Needham had nominated Waddington to lead the area of experimental embryology, Joseph Henry Woodger would head the section of theoretical embryology, and, as head of the division of genetic embryology, Needham designated Theodosius Dobzhansky (Peterson 2016: 118), who later joined the buoyant Californian group of Thomas Hunt Morgan and eventually became one of the chief architects of the Modern Synthesis.

As Abir-Am (1982: 341) argues, in the history of the twentieth-century biology, decisions in funding policies determined the course of nascent disciplines (or, at least, were central in their directions). Just as the Rockefeller Foundation turned its back on Needham, they began to push for research in what would later be called "molecular biology" (see Kay 1993). Post-war life sciences funding policies would favor research in reductionist fields such as molecular biology (see de Chadarevian 2002), rather than holistic research like that pursued by organicists. "The molecular view of life," as historian Lily Kay (1993) would call it, prevailed over what we would call the "organicist view of life" (see also Nicholson 2014). Brooks (2019: 24) says on this point: "It was, as the story goes, the politics of research funding that seemed to doom organicism: With the molecular revolution just around the corner, it seemed simply the wrong place and time for the movement to take root."

The German holists, in a similar case to the British organicists, also planned the establishment of a center to anchor organism-centered research: in 1942 Adolf Meyer-Abich and the particle physicist Pascual Jordan founded the journal *Physis: Beiträge zur naturwissenschaftlichen Synthese* in which they announced the creation of a research institute to explore organicist themes from multiple scientific disciplines (Beyler 1996: 268–269). However, the idea would not come to fruition and the journal *Physis* would not get very far (see Dahn 2019). The project to

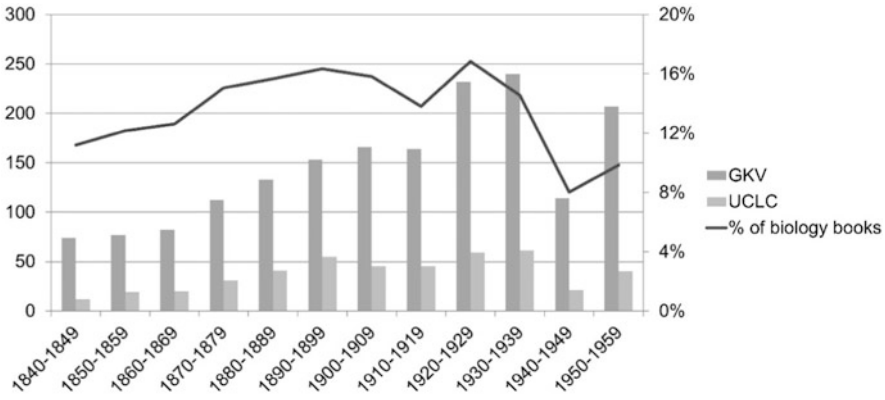

Fig. 8.1 The use of the concept of the organism between 1840 and 1959. Depicted is the number of monographs containing *organism*, *organisms*, *Organismus*, or *Organismen* in their titles. Entries are taken from two bibliographic databases: University of Cambridge Libraries Collection, UCLC (light grey bars) and German Union Catalog, GVK (dark grey bars). Only biological books are considered. Single monographs may appear more than one time in each database. The black line shows the percentage of all "organism books" (in GVK and UCLC) compared with all biological books published per year (i.e., entries in both databases matching keyword or substance for "biology" or "Biologie") (see Baedke 2019)

discipline German holism (with research institutes, journals, and specialized communities) would also not take root (see Beyler 1996). In general, German holistic biology would be discredited after the events of World War II: because of its affiliation with Nazi ideology, biologists from other latitudes would judge these theorizations as anathema and deliberately avoided citing German-speaking organicist authors (Wise 1994: 244; for analyses, see Harrington 1996; Rieppel 2016).

Moreover, around those years many older members of the international organicist movement had died (J. S. Haldane in 1936; Wheeler in 1937; Henderson in 1942; Schaxel in 1943; Ritter, Dürken, and Uexküll in 1944; Alverdes in 1952).[8] Others turned their research interests to new topics—e.g., Woodger to logic, Needham to the history of China, Bertalanffy to systems in general rather than organisms (see Nicholson and Gawe 2015; Peterson 2016), or to politics and/or popular science writing (Schaxel and Bernal). For many of the German-speaking advocates of holistic ideas, the end of the Second World War was a *caesura,* a break with the past. In the 1940s and 1950s, the (relative) number of monographs discussing organisms significantly dropped (see Fig. 8.1).

The Theoretical Biology Club disbanded and received strong criticisms from scholars like the immunologist Peter Medawar, who, although he was originally

[8] What is more, during the Nazi upsurge in Austria, organicist scientists based at the *Biologische Versuchsanstalt* were expelled from their workplace, barred from entering and some, such as its director Hans Przibram, were transported to (and later executed in) concentration camps (Taschwer 2014; Müller 2017c).

trained under the guidance of Woodger and Waddington, would not hesitate to publicly criticize organicism as a form of antiquated, speculative and useless way to do biology once he landed positions of power (Peterson 2016: 156–158). In addition to that, Ernst Mayr on several occasions discredited the work of organicists by wrongly cataloging it as "Lamarckian" (and, in the case of Conrad Hal Waddington, even as "Lysenkoist"; see Robinson 2018: 179, 184, 187, 190),[9] a label of mistrust that, however, served to dismiss the organicist *corpus* in the second half of the twentieth century and to justify that evolutionary biologists overlooked it (see Peterson 2016: ch. 11).

In this post-war scenario, the organicist movement left the international arena to dwell in obliviousness; in parallel, the Modern Synthesis, which should be understood as both a movement that sought theoretical unification and a discipline-building effort (see Smocovitis 1994), held sway. Furthermore, molecular biology and evolutionary biology (which would become increasingly gene-centered with the passing of decades) would dominate the landscape of the second half of the twentieth century. These two movements had a significant impact on the explanatory standards of evolutionary research, especially on those three epistemic roles granted to the organism by the organicist movement that subsequently were underestimated, ignored, or merely abstracted away.

First, genes became strongly decoupled from their organismic context and came to be regarded as the primary determinants of phenotypic characters. As Gawne et al. (2018) have showed, the vast majority of evolutionary biologists from the second half of the twentieth century onwards construed a simplified view of the genotype-phenotype map and lost sight of the fact that the origin of phenotypes can only be properly understood by integrating findings from all levels of organization of an organism (see Ågren, Chap. 35, in this volume). While molecular approaches offered a (to some extent successful) reductionist research program that abstracted from the context of the whole organism, population geneticists focused on the transmission of alleles and their dynamics in populations,[10] rather than on the developing organism (which was later held to be merely an epiphenomenon or a vessel of genetic programs) with all its material, concrete interactions with the environment (see Walsh 2019). Although there were some good scientific reasons for taking this

[9] Waddington always considered himself a *bona fide* Darwinian and contended that genetic assimilation was not an alternative to explanations that appealed to gradual, random genetic mutations and natural selection, but supplementary to them (see, e.g., Waddington 1961b). Through his views on developmental canalization, Waddington considered genetic assimilation to be a genuine Darwinian mechanism underpinning the inheritance of acquired characters, not related whatsoever to Lamarckian soft inheritance but depending upon the genetic capability of organisms to respond plastically to environmental changes via non-directed, preexisting cryptic genetic variation (for a detailed analysis, see Baedke 2018: 27–29; Loison 2019).

[10] One might even qualify this statement further. Medawar (1981) claimed that "[t]he most important single innovation in the modern synthesis was [...] the new conception that a population that was deemed to undergo evolution could be best thought of as a population of fundamental replicating units—of genes—rather than as a population of individual animals [or organisms in general]."

approach (Ågren 2021) and even some architects of the Modern Synthesis did not entirely forgo an ontogenetic perspective (see Depew 2017), an important consequence was that the organism was no longer understood as a major ontological and theoretical challenge that had to be addressed by evolutionary theory (see also Walsh 2015).[11]

Second, the explanatory roles of organism-environment reciprocity in the organicist movement changed significantly after the institutionalization of the Modern Synthesis and especially through later developments in evolutionary biology. In the course of the second half of the twentieth century, views of organism-environment reciprocity were increasingly marginalized (for a detailed discussion, see Baedke et al. 2021). This trend was driven by the attempt to establish clear boundaries between organisms and environments as a methodological stipulation for fruitful research—as Haldane (1936: 349) put it, this separation is "a practically and theoretically valuable abstraction" for population genetics. In this view, the environment is seen as an external causal factor, that, apart from generating selection pressures on organisms, is a "source of error that reduces precision in genetically studies," and thus one has "to reduce it as much as possible" (Falconer 1960: 140). Waddington (1957: 189) denounced this shortcoming: "Any further influence which the environment might have was degraded to the status of mere 'noise' in the system of genetic determination."

As another example of an impoverished view of the causal roles that the environment plays in evolution, Mayr (1970: 2) claimed: "the *true role* of the environment in evolution could not be understood until the nature of small mutations and of selection was fully comprehended" (emphasis added). Moreover, the persuasive split between proximate and ultimate causes advanced by Mayr (1961) resulted in a move away from the study of organism-environment reciprocity: through the lens of this dichotomy, the organism is only a developmental unit, wherein proximate causes are instantiated through the decoding of a genetic program; in contrast, the environment becomes evolutionary relevant as the reservoir and source of selective pressures, which are ultimate causes of evolution that shape the make-up of genetic programs. In line with these views, mainstream evolutionary biology increasingly adopted an asymmetric, unidirectional view of the organism-environment relationship (e.g., Williams 1992: 484).

In addition, evolutionary studies on reciprocity focused on other *relata*. Instead of organism-environment reciprocity, gene-environment reciprocity was increasingly studied in population genetics and other disciplines of evolutionary biology (Haldane 1946; Lerner 1950; Falconer 1952), for example, through path analysis (Wright 1960). New models of reciprocal relations between genes and populations as well as genes and environments (Fisher 1930; Kirkpatrick 1982) addressed population regulation by genetic feedbacks (e.g., Pimentel 1968), positive and negative

[11] An important exception, although not very influential in the last years of his career, was Sewall Wright, who still vouched for the importance of the organism in evolution against far-reaching gene-centered currents (see Wright 1980; see also Steffes 2007; Ågren 2021).

frequency-dependent selection (Fisher 1930; Wright 1969; Charlesworth 1971), and eco-evolutionary dynamics (Thompson 1998). The importance of these evolutionary models notwithstanding, and despite what some scientists claim (Brodie III 2005; Svensson 2018), the vast majority of these models did not encompass organism-environment reciprocal causation but focused on other *relata*. Because of these developments, the organism lost its previous explanatory function as a causal agent that constructs its environment, and thus its own development and evolution.

Third, and as an expansion of the previous marginalization, the view of the organism as an agent of development, and especially evolution, disappeared within the dominant framework of evolutionary biology. There, genes were construed as the sole agents of evolution, most of the times in ways that turned out to be empirically and conceptually unwarranted (see Okasha 2018: ch. 2; for criticisms of the cognate idea that genes are the main agents of development, see, e.g., Moss 2003; Griffiths and Stotz 2013). According to the gene-centric rationale, "[t]he production of whole organisms, and their differential survival and reproduction, are causally necessary consequences of the activities of [genetic] replicators" (Walsh 2017: 243). For instance, in a highly influential book, Monod (1971) contended that organismic purposiveness could be completely reduced and accounted for by citing invariant molecular mechanisms that get transmitted intergenerationally (for an analysis, see Walsh 2017).

While it is true that Modern Synthesis-inspired work integrated the import of some organismal factors into their evolutionary theorizations, such as the evolutionary role of behavior in particular cases of the Baldwin effect (although considering it rather marginal and not challenging central tenets of the synthetic theory, see Simpson 1953; see also Depew and Weber 2003 and chapters therein), most of the phenomena associated with organismal agency discussed inside the organicist movement became unheeded. One of the reasons for this development was that teleology "transmogrified" into teleonomy, as philosopher Krieger (1998) would say.

Colin Pittendrigh (1958) proposed the term "teleonomy" to encompass the study of purported end-directed processes (such as adaptation) in the hope of ridding biology from the encumbrances of the loaded term of teleology. In the hands of Mayr (1961: 1504), however, teleonomy became restricted to "systems operating on the basis of a program, a code of information." For Mayr (1985: 140), this meant that a "*teleonomic process or behavior is one which owes its goal-directedness to the operation of a program*" (emphasis in original). In particular, this implied that all the seemingly goal-directed processes unfolding in ontogeny (including agential behaviors) are under the controlling action of a genetic program inscribed in the sequence of DNA (for a distinction between closed and open programs, see also Mayr 1964).[12] Mayr's understanding of teleonomy was backed up by a widespread adoption among biologists of concepts from cybernetics and information theory (see Mayr 1985: 134, 142, 144), which collected criticisms from some authors that used

[12] For a different reading of the concept of genetic program in Mayr's work, see Dickins (2021).

to belong to the organicist movement (see Bertalanffy 1951), but ultimately to no avail.

Moreover, other influential evolutionary biologists, such as Simpson (1958: 520–521) and Williams (1966: 258–269), advocated for the epistemic legitimacy of shoehorning all agential processes under the heading of *teleonomy*. Although a scientific and philosophical debate ensued on the proper status of teleological (and teleonomic) explanations in the late twentieth-century evolutionary biology, the genetic program understanding of intrinsic purposiveness (shaped by bouts of natural selection) prevailed in the field (for an analysis, see Krieger 1998). If organisms *seem* to be agents to us, it is merely because genetic programs that encode purposive-like traits were selected for in evolutionary time: "Each particular program is the result of natural selection, constantly adjusted by the selective value of the achieved endpoint" (Mayr 1985: 141). If not vitalism, the only conceivable alternative for many evolutionary biologists was "to regard internal [organismic] teleology as a product of evolution by natural selection" (Dobzhansky et al. 1977: 96). Organismal agency, then, was rendered a mere evolutionary *product*, but not a *cause* that has some bearing on the process of evolution.

An additional problem was that many evolutionary biologists collapsed discussions concerning finality in evolution (e.g., divine design, orthogenetic trends) with the problem of organismal purposiveness and specifically of agential, goal-directed processes. This contributed to making most evolutionists think that, using Okasha's (2018) useful terminology, "agential thinking 1" (the problem of teleology in nature) and "agential thinking 2" (the treatment of evolved entities, such as organisms, *qua* agents that pursue intrinsic goals) is one and the same problem. Uncareful conceptual treatments of the subject of teleology made any discussion of organismal agency equivalent to teleology in its broadest sense.[13] Accordingly, it is no surprise that a fair number of evolutionary biologists are still today reticent to entertain the idea that organisms are causal difference-makers in the world and that some evolutionary consequences must obtain from this fact, a view which is usually despised.

In sum, many trends in evolutionary biology led to an explanatory framework that is focused on the transmission of genes and its effect on populations, rather than on the developing organism and its reciprocal interactions with the environment and agential activities. Evolutionary thinkers established a narrower conception of the organism, both internally (organisms are primarily the product of genetic programs) and externally (organisms are not agents that co-construct their environment and thus modulate their selection pressures). However, this "eclipse of the organism" in evolutionary theory (see Walsh 2015) has been increasingly challenged since the 1980s and especially in recent years.

[13] Mayr (1985) did draw a distinction between different senses of teleology in biology: (a) Unidirectional evolutionary sequences; (b) goal-directed processes; and (c) teleological systems. As organismal agency mostly pertains to (b), it is unfortunate that most biologists did not follow Mayr's pedagogical taxonomy.

8.5 Rediscovering Explanatory Roles of the Organism in the EES

Since the 1980s, many of the calls to extend, expand, or replace the Modern Synthesis (see Depew and Weber 2013 for an overview) channeled discourses that called for the re-constitution of the organism as a central unit in evolutionary biology. Stephen J. Gould, for example, judged the decline of the concept of organism as a setback to be remedied by the emergence of a reformed theory of evolution that, among other things, would return "to biology a concept of organism" (Gould 1980: 129). David Rollo, in the preface of his book *Phenotypes: Their Epigenetics, Ecology and Evolution*, stated that the purpose of his work was to return the organism to its rightful place as the center of selection and evolution (Rollo 1994: xi). Susan Oyama, putting forward her vision of the place of developmental systems theory (DST) in evolutionism, explained that her goal was to "put organisms back" into evolution or, in other words, to "restore the organism" (Oyama 2000: 30–31).[14] In a similar vein; Brian Goodwin stated in 1999:

> Organisms have *disappeared as fundamental entities*, as basic unities, from contemporary biology because they have no real status as *centres of causal agency*. Organisms are now considered to be generated by the genes they contain. [. . .] Thus organisms are arbitrary aggregates of characters, generated by genes, which collectively pass the survival test in a particular environment. [. . .] [T]here is *no causally efficacious unit* that transcends the properties of the interacting parts. This is the sense in which organisms have disappeared from biology (Goodwin 1999: 230; emphases added).

This situation, however, has changed in recent years. Especially advocates of the EES try to reestablish the organism as a central explanatory unit in evolutionary biology. This new "organism-centered perspective" (Laland et al. 2015) stresses the idea that organisms are the central explanatory units to not only understand evolutionary relevant dynamics in (gene-)regulatory processes during embryo- and morphogenesis, but also to study developmental plasticity, non-genetic channels of inheritance, and constructive behaviors that shape organisms' niches and selection pressures (West-Eberhard 2003; Jablonka and Lamb 2005; Jablonka 2017; Laland et al. 2014, 2015; Walsh 2015; Sultan 2015; Müller 2017a; Uller et al. 2018, 2020). This new framework of the EES unwittingly ties in with the central three cornerstones of organism-centered evolution once defended by the organicist movement in the early twentieth century. This includes (1) the conceptualization of genes as parts in larger extracellular, organismal and developmental contexts, and the consideration of these contextual wholes in shaping evolutionary trajectories; (2) the idea that evolution is the result of organism-environment reciprocal interaction (rather than of external environmental factors causing changes in allele frequencies and population dynamics); and (3) that organismal agency is a key

[14] For a different reading of the role of the organism in DST, or the lack thereof, see Pradeu (2010).

explanatory component for understanding how organisms co-construct their evolution. Let us briefly discuss these three revived dimensions of the organism.

First, the organism is granted an explanatory role inside the EES and related Evo-Devo views of evolution that allows properly contextualizing parts and their causal contributions in development. For Laland et al. (2015: 6), lower levels (e.g., genes) do not prevail over higher levels of organismal organization as causation runs reciprocally between them: "causation not only flows from the lower levels of biological organization, such as DNA, 'upwards' to cells, tissues and organisms, but also from the higher level 'downwards', such as through environmental- or tissue-induced gene regulation" (Müller 2017a). In particular, the view of constructive development defended inside the EES "does not a assume a bijective function (i.e., a one-to-one correspondence) between genotype and phenotype, nor grants causal privilege and programmatic jurisdiction to genes driving individual development; instead, the developmental system is viewed as responding flexibly and creatively to internal and external inputs, through condition-dependent gene expression, and through physical properties of cells and tissues and 'exploratory behaviors' of several systems" (Fábregas-Tejeda and Vergara-Silva 2018a: 179; see also Gawne et al. 2018 on the de-idealization of the genotype-phenotype map).

A recurring theme in Laland et al. (2015) is the fact that phenotypic variation can be biased by the processes and organizational dynamics of development, which channel the evolution (i.e., increasing the probability of occurrence) of certain functional phenotypes and restricting the possible space of realized forms. Developmental bias, an Evo-Devo notion, has been mobilized as an important epistemic cornerstone of the discussion of the structure and assumptions of the EES. Another important key theme for Evo-Devo, facilitated variation (*sensu* Kirschner and Gerhart 2005), is used as a conceptual scaffold by EES proponents to explain the presence of developmental biases: the core processes of development concurrently exhibit high robustness and exploratory behaviors that allow them to stabilize and select certain states over others (Laland et al. 2015; see also Uller et al. 2018).[15] Additional key themes of Evo-Devo (e.g., evolvability, modularity) are also deployed in EES explanations (for an analysis, see Fábregas-Tejeda and Vergara-Silva 2018a). Moreover, EES defenders embrace a view of development in which the organism co-constructs its own developmental trajectories by means of plastically responding to, integrating and shaping environmental cues.

Second, in recent years, evolutionary biology in general and advocates of the EES in particular tied in with the older idea of organism-environment reciprocity (see Baedke et al. 2021). This especially concerns research on phenotypic plasticity and niche construction (Laland et al. 2013, 2015; Mesoudi et al. 2013; for discussion, see Fábregas-Tejeda and Vergara-Silva 2018a, b; Svensson 2018; Buskell 2019; Baedke 2019). Here, organisms' plasticity and niche construction behaviors are studied as feedback circles that modify the natural selection pressures working on the

[15] For a counterposition on facilitated variation and its compatability with traditional evolutionary thinking, see Dickins (2021: 142–144).

constructor and other organisms (Lewontin 1983; Sterelny 2001; Odling-Smee et al. 2003; Chiu and Gilbert 2020).[16] This process is said to have a co-directive effect on adaptive evolution "by imposing a consistent statistical bias on selection" (Laland et al. 2017). Examples include the building of artifacts by animals, like nests, burrows, and mounds, the creation of shade and change of nutrient cycling by plants, and the modification of manifold physical and chemical conditions. By focusing on such examples, for instance, Clark et al. (2020) collected evidence that niche construction affects the variability and strength of natural selection in a way that is possible to distinguish between constructed and non-constructed environmental sources of selection.

In order to explain the feedback between constructing organisms and environments as well as the developmental effects on evolutionary trajectories (and vice versa), advocates of the EES argue that the traditional dichotomy between ultimate and proximate causes (Mayr 1961) should be replaced by a concept of *reciprocal causation* (Mesoudi et al. 2013; Laland et al. 2011, 2013, 2015, 2017).[17] This view holds that developing organisms are not only products but also causes of evolution and starting points of evolutionary trajectories. Thus, the proximate causes of developmental processes should not be strictly isolated from ultimate causes of evolutionary processes. Instead, proximate causes feedback to affect the direction and rate of adaptive evolution. As a consequence, investigating developmental mechanisms, from mechanisms of gene expression or cell and tissue development to organisms' constructive actions in their local environments, offer explanatory relevant information on how organisms evolve.

Some authors involved in the EES debate trace back this idea of causal reciprocity between organism and environment to Levins and Lewontin's (1985) book *The Dialectical Biologist* (see Svensson 2018) or cite Waddington (1969) to highlight proto-niche construction views (see Laland et al. 2011; see also Odling-Smee et al. 2003)—albeit without being aware of the fact that these authors are actually late examples of an older and much richer movement that took organism-environment reciprocity as a theoretical starting point to reason about evolution, independent of (and not as a reaction to) the MS.[18]

Third and finally, this renewed interest in organisms' constructive roles in shaping their selective environment in the EES reintroduced the concept of organismal agency to evolutionary theory in recent years (see Baedke 2021). EES advocates commonly use agential terms like "active phenotypes" (Watson and Thies 2019),

[16] For conceptual frameworks that distinguish diverse kinds of feedback processes in niche construction, see Aaby and Ramsey (2020) and Chiu (2019).

[17] For an overview of criticisms against Laland et al.'s concept of reciprocal causation, see Baedke and Gilbert (2020). For conceptual challenges that go along with adopting views of organism-environment reciprocity, see Buskell (2019) and Baedke et al. (2021).

[18] There are scarce references to organicist authors within the EES literature and not a single comprehensive discussion on the historical pedigree of EES-type reasoning concerning the active role of organisms in evolution. Some exceptions which rely on succint mentions are Müller (2017d: 8) and Jablonka and Lamb (2020: 1, 71).

"active agents" and "purposive organisms" (Laland et al. 2019; see also Sultan 2015; Sultan et al. 2022). Rather than embracing a spurious vitalist notion of agency and non-material purposiveness, these authors seek to highlight behavioral drivers of evolution or the general idea that organisms (and their phenotypes) are leaders in evolution (West-Eberhard 2003, 2005). Genes merely follow agential changes in evolution. In other words, organisms introduce (in a biased manner) new phenotypes into populations, which are subsequently stabilized by genes.

While, so far, no consensus has been reached on which kind of theory of agential causation should be adopted to strengthen especially the status of niche construction as a theory, several frameworks have been put forward (for general analyses on organismal agency and intrinsic teleology, see also, in this volume, Fábregas-Tejeda and Baedke Chap. 10; Fábregas-Tejeda and Baedke Chap. 15). Laland et al. (2019) draw on classical understandings of the purposiveness of organisms through thermodynamics and self-organization (see Schrödinger 1944; see also Nicholson 2018; Baedke 2019). Others have highlighted that any theory of evolutionary relevant purposive behavior of organisms should include the experiential side of niche construction (Sultan 2015; Chiu 2019).[19] It should be able to distinguish, but also integrate, the different causal and explanatory roles the organismal agent is performing by changing its environment (i.e., modifying its physical properties) and by changing its relation to it (i.e., by experiencing it differently), as both cases can have very different evolutionary effects (see Baedke et al. 2021). Another distinction has been made between agential and contributional forms of niche construction (Aaby and Desmond 2021). In the first case, organisms act as agents if niche constructing effects result from goal-directed behavior under the control of the organism (e.g., plants alter leaf-morphology to optimize light exposure). In the second case, organisms act as contributors if the effects of niche construction do not arise from a goal to perform the constructive activity (e.g., bacteria create novel niches through energy-rich detritus that different strains can metabolize).

Yet other approaches of organismal agency draw on the concept of affordances (i.e., the opportunities of action or what an organism can do based on its traits and its environment together). For example, Denis Walsh argues that organisms are not objects of evolutionary forces, but agents that co-constitute the affordances that shape evolution. Organisms enact evolution as they pursue their goals, negotiate their "affordance landscapes," and construct their conditions of existence (Walsh 2015: 241). He states:

[19] An experienced environment refers to the mediating interface between organism and the physical environment. What counts as an environmental cue (e.g., temperature, pressure, location, etc.) depends on the organism's sensory system and the active modulations performed by the organism (see Sultan 2015). Experienced cues are transduced into chemical and cellular processes (which regulate, e.g., gene expression patterns or microbiome composition), and lead to metabolic, morphological or behavioral changes. A difference in experienced environments between two organisms living in the same physical surroundings means that the environment is experienced differently by each organism (e.g., as favorable or unfavorable, as stressful or non-stressful). For discussion on experiential niche construction, see Baedke et al. (2021).

> Since its inception in the early 20th century, the modern synthesis theory of evolution has been guided by a methodology that explicitly prohibits explanations of phenomena in the natural world that appeal to the fulfillment of goals and purposes [. . .]. Increasingly, it is becoming apparent that the purposiveness of organisms, as manifest in the robust, reactive plasticity of their various systems, from gene networks to entire organisms, is pivotal to the process of evolution. (Walsh 2017: 257).

Recently, these emerging debates around an agency-focused extension of evolutionary biology have gained substantial funding support. An example of this is the research network "Agency, Directionality, and Function: Foundations for a Science of Purpose," which includes 24 different projects that should address the role of organismal purposiveness for evolutionary biology, ranging from theoretical models to empirical tests.

8.6 Conclusions

In recent years, many scientists and philosophers of science have called for a return of the organism in the biosciences. They have especially argued for expanding the standard population genetic framework of evolutionary biology by a more organism-centered account. This EES should focus less on genes and more on developing organisms and their active, reciprocal interactions with their environments. Unfortunately, this development lacks a clear historical understanding on which theoretical traditions it draws on. Almost exclusively, the EES debate has focused on contrasting the current attempts to highlight organisms' roles in evolution by juxtaposing this approach with that of the MS (construed in many different ways). We showed that taking this historical lens is rather limited, as the core epistemic claims of the organism-centered perspective of the EES actually did not emerge in the history of biology as a reaction to the MS. Instead, its most central ideas about organisms and the organism-environment relationship were developed independently from the MS and were widely debated in the early twentieth-century biology. This concerns especially three currently resurfacing epistemic roles that organisms should play in our explanations of the evolutionary process: organisms should allow (1) contextualizing parts (especially genes) in development, (2) focusing on reciprocal organism-environment relations (in contrast to, e.g., gene-environment relations), and (3) understanding the role of agency in evolution.

This new historiography, which links evolutionary debates in the early twentieth-century organicism, dialectical materialism and holism with contemporary discussions, allows to better understand the conceptual and theoretical framework underlying the EES. However, understanding the origin and theoretical presumptions of this explanatory framework does not necessarily mean that, *ipso facto*, this approach is feasible or desirable. In fact, it faces the challenge of how to integrate the newly highlighted explanatory role of the organism with the quite different explanatory standards of mainstream evolutionary theory, which largely focuses on genes and populations, but not organisms as causally efficacious units. In

short, we need to answer: when organism-centered explanations have more explanatory power and should be chosen over gene-centered explanations, and vice versa?

When we seek to add explanations of developmental and organismal causes, like developmental bias, phenotypic plasticity, niche construction, to the explanatory framework of evolutionary theory, we need to know due to which epistemic virtues organism-centered explanations are better and which tradeoffs between explanatory standards (like precision, sensitivity, proportionality, and idealization) we face when trying to integrate organismal and genetic accounts of evolution.[20] If organism-centered explanations do not meet criteria of explanatory power entrenched in the field (like a specific degree of precision, sensitivity, or proportionality) scientists will remain skeptical on whether they carry explanatory power and increase our understanding of evolution. Then, these critics might reject the integration of organismal and populationist views within a more pluralist framework of evolutionary causation. In addition, this perspective stresses that evolutionary biologists need to start reflecting not only on the evidence that supports genetic or organismal causes of evolutionary change, but on which explanatory standards they want their evolutionary explanations and models to hold. In other words, they need to decide whether they give the explanatory standards of early twentieth-century organism-centered accounts of evolution another chance or not.

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[20] For a detailed discussion of this challenge and how to solve it, see Baedke et al. (2020).

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9 Causes and Consequences of Selection: A Commentary on Baedke and Fábregas-Tejeda

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Abstract

In their chapter, Baedke and Fábregas-Tejeda take a historical approach to some of the concepts typically presented as a counter-point to the MS as characterized by EES proponents. Specifically, the authors focus on three EES concepts—a holistic approach to organisms and development, reciprocity in organism-environment relations, and the role of organismal agency in evolution—and trace their origins to a strong, early twentieth century, organismal movement in biological explanation that later declined, especially with the mid-century rise of reductionist molecular biology. They locate the motivations for the crystallization of the organismal movement, which arose in multiple English- and German-speaking scientific communities, in the tensions between vitalism and

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reductionist materialism around the turn of the twentieth century, even though the antecedents of this perspective on biology go back considerably further in time.

Keywords

Genetic determinism · Gene's-eye view of evolution · Organismal agency · evo-devo · Natural selection · Microevolution

In their chapter, Baedke and Fábregas-Tejeda take a historical approach to some of the concepts typically presented as a counter-point to the MS as characterized by EES proponents. Specifically, the authors focus on three EES concepts—a holistic approach to organisms and development, reciprocity in organism-environment relations, and the role of organismal agency in evolution—and trace their origins to a strong, early twentieth century, organismal movement in biological explanation that later declined, especially with the mid-century rise of reductionist molecular biology. They locate the motivations for the crystallization of the organismal movement, which arose in multiple English- and German-speaking scientific communities, in the tensions between vitalism and reductionist materialism around the turn of the twentieth century, even though the antecedents of this perspective on biology go back considerably further in time.

Though there were differences of emphasis and nuance among thinkers who constituted the organismal movement, Baedke and Fábregas-Tejeda outline some core similarities that permeated this perspective across nations. One major issue on which there was concordance among many thinkers was on how to conceive of the relationship between development and heredity, which, in EES terms, expresses itself as largely as a contrast between holistic organismal approaches to development and strong genetic control over developmental processes, the latter often ascribed to the MS. A second point on which many thinkers within the organismal movement concurred was that of conceiving organism-environment interactions as being inherently reciprocal, even though they often arrived at this view from very different philosophical starting points. Finally, the third point of wide agreement was the incorporation of organismal agency into evolutionary explanation. The authors flesh out these various themes in the early twentieth-century organismal biology, and also discuss the most likely reasons why these organism-centred ways of thinking about development, heredity, and evolution faded deep into the background over the second half of that century. Baedke and Fábregas-Tejeda apportion major responsibility for the decline in organismal thinking to advances in molecular biology and the concomitant shifts in funding priorities that prevented the organismal movement from becoming an institutionalized discipline. They also, secondarily, implicate the perceived gene-centric thinking of the institutionalized MS in the decline of the organismal movement. They then point out that there were calls, even before the appearance of a coherent EES movement, for the restoration of the organism to a central position in evolutionary explanation. Finally, they summarize how the EES has appropriated or resuscitated these three major organism-centred themes from the

organismal movement of the early twentieth century. Through their analysis, Baedke and Fábregas-Tejeda offer an alternative perspective to one which sees the EES as arising in response to the MS, or perceptions of the MS, suggesting rather that many of the core organism-centric approaches highlighted by EES proponents had in fact developed independently of the MS around the same time that the latter was beginning to crystallize. We agree that this is an important perspective that should not be lost in the EES-MS debates, and also note that many important concepts in evo-devo (e.g., developmental bias), similarly, have their roots in the ideas of Bateson (1894), de Vries (1905), and others, and were articulated well before the MS coalesced into a well-defined body of thought.

Unlike the authors, we are neither philosophers nor historians of biology, and we are happy to agree with their historical analysis, trusting it to be accurate. However, we have a slightly different perspective on the issues they discuss, deriving from our background in empirical evolutionary biology research (see also Vidya et al. 2022, this volume). We now discuss some thoughts on these issues that we believe complement the analysis of the authors, and perhaps offer some additional food for thought. In particular, we suggest that it is helpful, when thinking about explanations in evolution, to distinguish between the causes and consequences of selection in the context of micro-evolutionary change, and that such a distinction allows for a finer examination of the three issues highlighted by Baedke and Fábregas-Tejeda. By micro-evolutionary change, we mean changes resulting in either new variants of already existing traits (new character values in the morphology/taxonomy literature), or changes in the distribution of trait-variants within a defined population. By causes of selection, we mean factors or phenomena that play a role in determining total offspring production by individuals exhibiting different trait-variant combinations, whereas by consequences of selection, we mean factors or phenomena mediating between differential reproductive output of individuals exhibiting different trait-variant combinations, and the consequent changes in frequency of those trait-variants in the population. Though he did not refer to causes versus consequences of selection in this context, the distinction between the two was actually quite explicitly made by Darwin (1859, 1868), who recognized that selection primarily acted on individuals via differences in offspring production, but that it was heredity that linked the relative reproductive success of individuals bearing different subsets of trait-variants to whether those trait-variants would tend to increase or decrease in the offspring. Another way of looking at this distinction is to think of reproductive output as reflecting ecological success in the Darwinian 'struggle for existence', whereas changes in frequency reflect short-term evolutionary success in obtaining representation in coming generations. Consequently, micro-evolutionary change arises from an integration of both ecological and evolutionary success. We next examine the three concepts on which the authors focus in light of this distinction.

The distinction between causes and consequences of selection permits a finer dissection of whether, and in which circumstances, the individual organism is a meaningful unit of evolutionary change, in addition to its relatively uncontroversial role as the principal functional unit of biology. Selection arises as a result of the

interaction of individual organisms with their ecological context, leading to the realization of differences in reproductive output. Consequently, the individual organism, taken as a holistic entity operating within an ecological context with which it interacts in a many-to-many manner, plays a major role in determining the causes of selection. Given that organismal agency is almost a definitional attribute of being alive (Sultan et al. 2022), it is clear that organismal agency, therefore, also plays an often very important role in determining the causes of selection. The consequences of selection, however, depend critically on heredity, not necessarily gene-based, rather than ecology and functional biology. It is in this second phase of micro-evolutionary change that the role of organismal agency is negligible and, therefore, typically neglected altogether. The really important factor shaping the consequences of selection is the degree to which trait-variants are faithfully passed on to offspring, also termed 'transmission fidelity'. The holistic organism, other than its ultimate causal role in affecting which sort of hereditary system mediates the transmission of trait-variants to offspring, plays no proximate causal role here. A similar case can be made for reciprocity of interactions between the organism and its environment. Any such reciprocal interactions affect the causes but not the consequences of selection.

The contrast between gene- or population-based versus organism-based explanations in evolution that Baedke and Fábregas-Tejeda allude to in passing, as well as the relationship between development and heredity, are somewhat broader issues that, we believe, get clarified substantially by distinguishing between the role of genes in evolutionary explanation in the MS, largely though not exclusively through Fisher's (1918, 1930, 1941) work, and the subsequent 'gene's-eye view of evolution' associated with Williams (1966) and, most famously, Dawkins (1976). We suggest that much of the EES literature (e.g. as summarized in Laland et al. 2015), as well as writings on the gene's eye view of evolution (e.g. Okasha 2006; Ågren 2021), conflate the very different roles of genes in evolutionary explanation in the Fisher-MS view and in the Williams-Dawkins view, and that this conflation has contributed non-trivially to some almost unnecessary aspects of the EES-MS debates (for details, see Vidya et al. 2022, this volume). Briefly, in our opinion, the Fisherian or MS view did not ascribe causality to genes in terms of phenotypic attributes. Based on the fact that Mendelian genetics provided the only explanation of the process of hereditary transmission of trait-variants at the time, the Fisherian view, embodied in quantitative rather than population genetics, deployed statistical (not causal or material) effects of genes on the inheritance of phenotypes to permit one- or few-generation predictions of change in phenotypic trait-variant distributions under selection, especially when the phenotypes were complex, with poorly understood and typically polygenic underpinnings, sensitive to environmental effects. The complementary discipline of population genetics concerned itself solely with explaining change in genotypic distributions, which could be correlated with phenotypic distributions only for traits with a very simple genotype-to-phenotype mapping. The Fisherian view, thus, used statistical attributes of genes, abstracted from complex and environment-sensitive genotype-to-phenotype relationships, in order to explain the consequences of selection on complex metric traits. It is this

statistical nature of the Fisherian gene's eye view of micro-evolutionary change in terms of the consequences of selection that is often missed in much of the EES discourse. For example, when Baedke and Fábregas-Tejeda write 'organisms should allow . . . focusing on reciprocal organism-environment relations (in contrast to, e.g., gene-environment interactions)', they seem to be treating gene-environment interactions as a 'thing' belonging to the same logical category as organism-environment relations. However, this is not the case: a gene-environment interaction is one way of statistically (not materially or causally) quantifying the effects of a real organism-environment relation on variation in a phenotype. Similarly, a gene–gene interaction does not ascribe any causal import to the genes in question; it statistically describes the consequences of some physiological/biochemical interaction that affects a phenotype in a manner not explainable by adding up the independent effects of genotypes at those loci on the phenotype.

The Dawkinsian gene's eye view, on the other hand, seems to strongly ascribe a degree of causality to genes in the micro-evolutionary process, both in terms of the causes and the consequences of selection, and thus—very controversially—considers genes, in their capacity as replicators, to be units of micro-evolutionary change. In our opinion, it is only in the Dawkinsian and not the Fisherian gene's eye view that the organism is more or less replaced by the gene as the locus of micro-evolutionary explanation. In the Fisherian perspective, the gene is not a unit of micro-evolutionary change; it is at best a useful unit of book-keeping with regard to the consequences of selection, and that too, not in its capacity as a material entity, but only through a statistical abstraction of its phenotypic effects. The Dawkinsian view is often claimed to be a direct extension of Fisher's genetic thinking about the process of selection (e.g., Ågren 2021 and references therein), but we do not agree with these claims, believing them to arise from misunderstanding some of the nuances of Fisher's thinking. Hamilton's (1964a, b) development of inclusive fitness in the context of kin-selection, in our opinion, is situated somewhere in between the Fisherian and Dawkinsian points of view. Ågren (2021) notes that Dawkins was surprised and somewhat dismayed that Hamilton never quite gave up the individual for the gene as a locus of explanation, as opposed to book-keeping. Yet, it is also undeniable that Hamilton's (1964a, b) formulation served as a stepping-stone to the narrower Dawkinsian conception of the gene's eye view. It is often suggested that a common strand of genetic determinism flows through the thinking of Fisher, Hamilton, and Dawkins. It is, however, interesting to note in this context that critiques of both Hamilton's and Dawkins' conceptions of genes somehow driving organismal evolution in somewhat simplistic ways came from a population genetics perspective (e.g., Sober and Lewontin 1982; Matessi and Karlin 1984; Mueller and Feldman 1985; Nowak et al. 2010). We also note that critiques of the use of optimization thinking in evolutionary ecology, which typically rests upon an implicit assumption of a simplistic genotype-to-phenotype mapping, without gene-gene interactions, very similar to that in the Dawkinsian gene's eye view, have also typically come from a population genetics perspective (e.g., Rose et al. 1987). It is difficult to reconcile these observations with the notion that the Dawkinsian formulation was simply an extension of the Fisherian gene's eye view. We suggest that

part of the antipathy of many MS supporters to some EES arguments arises because the conflation of the Fisherian and Dawkinsian gene's eye views in the latter often leaves one with the impression that the baby is being thrown out with the bathwater.

The arguments of some of the early twentieth century thinkers of the organismal movement about treating heredity as a part of development within a holistic, organism-centred framework, mentioned by Baedke and Fábregas-Tejeda, actually seem to be a return to a position almost axiomatic till the late-eighteenth century (see multiple papers in edited volumes by Rheinberger and Müller-Wille 2003; Müller-Wille and Rheinberger 2007), till it began to be challenged in the early decades of the nineteenth century, especially through the work of Propser Lucas (Kendler 2021) and Imre Festetics (Szabó and Poczai 2019). The conceptual separation of the transmission of a trait from its generation through development was further developed in the work of Darwin (1868), and Galton (1872), reaching its apogee with Morgan (1926), ironically himself an embryologist. Once again, the separation of heredity from development is useful in examining the consequences of selection, because development is largely irrelevant in that domain. In the examination of the causes of selection, the major relevant phenomena are those of organismal biology and ecology. Neither heredity, nor development are relevant in that domain, although development, of course, gives rise to the organismal phenotypes that engage in the 'struggle for existence'. Development is at its most relevant in evolutionary explanation as a phenomenon that generates variation. The organismal properties and activities of those variations interact with the ecological context to yield corresponding reproductive outputs. In these two domains, we entirely agree that invoking genes as causal factors (genetic determinism) represents a tremendously worrisome epistemological narrowing of explanation in biology and evolution. Finally, it is largely heredity that determines the consequences of development, organismal biology and ecology for patterns of change over generations in the composition of populations. Thus, the three major biological phenomena of development, ecology and heredity play sequentially complementary roles in mediating micro-evolutionary change (Joshi 2005). In this perspective, individual organisms are important functional units of biology, development, and one subset of the process giving rise to micro-evolutionary change, the subset pertaining to the causes of selection. For the other subset of the micro-evolutionary process, it is neither helpful nor meaningful to consider the individual organism as a unit of micro-evolutionary change.

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Organisms and the Causes and Consequences of Selection: A Reply to Vidya et al.

10

Alejandro Fábregas-Tejeda and Jan Baedke

Abstract

In response to our chapter "The Organism in Evolutionary Explanation: From Early Twentieth Century to the Extended Evolutionary Synthesis," population biologists T. N. C. Vidya, Sutirth Dey, N. G. Prasad, and Amitabh Joshi press for making an explicit distinction between the *causes* and the *consequences* of selection, and further suggest that such a distinction weighs in the three explanatory roles ascribed to organisms in past and present evolutionary research that we recounted in our work: (1) contextualizing parts in development; (2) drawing attention to reciprocal organism-environment interactions; and (3) underscoring the role of agency in evolution. Here, we first provide an overview of their arguments and then offer a rejoinder to their position which, we think, does not correctly apportion the evolutionary significance of organismal development and activities. We argue that organisms are relevant both for the "causes" (and sources) and the "consequences" of selection, and for evolutionary dynamics and trajectories in general. Evolutionary biology cannot dispense with the successful populational models built with the mathematical tools and assumptions of quantitative and population genetics, but, at the same time, it also needs new organismal models that take into account development, agency, and organism-environment reciprocal interactions.

Keywords

Organisms · Natural selection · Causes of selection · Consequences of selection · Environment · Niche construction · Organismal agency

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In response to our chapter "The Organism in Evolutionary Explanation: From Early Twentieth Century to the Extended Evolutionary Synthesis," population biologists T. N. C. Vidya, Sutirth Dey, N. G. Prasad, and Amitabh Joshi have provided a good summary and advanced thoughtful comments to what we expound there. In the context of evolutionary explanations of micro-evolutionary change (e.g., the arrival of new variants of preexisting traits, changes in the populational distribution of trait variants), they press for making an explicit distinction between the *causes* and *consequences* of selection, and further suggest that such a distinction weighs in the three explanatory roles ascribed to organisms in past and present evolutionary research that we recounted in our text: (1) contextualizing parts in development; (2) drawing attention to reciprocal organism-environment interactions; and (3) underscoring the role of agency in evolution. Here, we first provide an overview of their arguments and then offer a rejoinder to their position which, we think, does not correctly apportion the evolutionary significance of organismal development and activities.[1]

10.1 The Evolutionary Stance of Vidya and Colleagues

Regarding the distinction between causes and consequences of selection, they propose the following operational characterization:

> By causes of selection, we mean factors or phenomena that play a role in determining total offspring production by individuals exhibiting different trait variant combinations, whereas by consequences of selection, we mean factors or phenomena mediating between differential reproductive output of individuals exhibiting different trait variant combinations, and the consequent changes in frequency of those trait variants in the population (Vidya et al. 2023: 151).

Vidya and colleagues contend that, in the purview of this division, reproductive output reflects ecological success in the struggle for existence every organism faces,

[1] In our commentary, we restrict our discussions to the points considered by Vidya and colleagues on the topic of the causes and consequences of selection, and how organisms intersect (or not) with those evolutionary processes. Nevertheless, we want to stress that they raise other important issues as well: we agree with them on the fact that, in many instances of the EES debate, the target of criticism has not been Modern Synthesis thinking per se (for example, the Fisherian view imbued in quantitative genetics), but rather the subsequent gene's-eye view of evolution stemming from Williams and Dawkins, and cognate theses such as gene-centrism and -determinism in development that took prominence in the last decades of the twentieth century. More nuanced historical appraisals of what is covered by the EES-derived neologism "Standard Evolutionary Theory," and when it misses its target, are surely needed and welcome. At the same time, it is important to stress that some EES proponents have waged specific critiques against the models of quantitative genetics, such as genetic variance–covariance matrices (see Pigliucci 2006), so it is not correct to claim that they only dispute population genetics. The general point of contention that underlines that what the EES is opposing or resisting to is not always clear in historiographic terms might still hold though.

and changes in trait representation across generations are indicative of short-term evolutionary success; hence, micro-evolutionary change is the net integration of ecological and evolutionary success in commensurate timescales. Furthermore, an upshot of embracing this distinction, they assert, can be found in the ability of scientists to make "a finer dissection of whether, and in which circumstances, *the individual organism is a meaningful unit of evolutionary change*, in addition to its relatively uncontroversial role as the principal functional unit of biology" (*Idem*; emphasis added). On this point, they argue that individual organisms, conceptualized as integrated wholes that develop and act in given ecological settings while sustaining manifold interactions with their environments, indeed have a role to play in shaping the causes of selection. For them, agency, in this context, is one of the capacities that organisms marshal, by virtue of them being alive as thermodynamically open systems, to determine the causes of selection. But beyond this role granted to organisms engaging with their conditions of existence (e.g., pursing intrinsic goals such as survival and self-maintenance), which for them is so ubiquitous it seems borderline trivial to overemphasize something of this sort in evolutionary science, Vidya and colleagues do not think it is justified to regard organisms as *bona fide* meaningful units of evolutionary change. This is especially evident, they tell us, when dealing with the consequences of selection: as these depend on heredity, the backdrop of how an organism develops and how/why it interacts with (or alters) its surroundings stops being relevant:

> It is in this second phase of micro-evolutionary change that the role of organismal agency is negligible and, therefore, typically neglected altogether. The really important factor shaping the consequences of selection is the degree to which trait-variants are faithfully passed on to offspring, also termed 'transmission fidelity'. The holistic organism, other than its ultimate causal role in affecting which sort of hereditary system mediates the transmission of trait-variants to offspring, plays no proximate causal role here. A similar case can be made for reciprocity of interactions between the organism and its environment. Any such reciprocal interactions affect the causes but not the consequences of selection (Vidya et al. 2023: 4).

10.2 Organisms as Evolutionary Agents

We can offer some counter-reflections to the arguments of Vidya and co-authors. To start, even if one were to fully accept their line of reasoning without posing objections and round off the debate on the evolutionary importance of organisms, there would still be important work to be done by scholars to understand the intensity and reach of organismal modification of the *causes* of selection. Some evolutionary biologists may want to abstract away the causes of selection and focus only on its consequences due to pragmatic considerations/constraints or for particular epistemic purposes, for instance, for modeling changes in trait distribution and frequency across generations, or for tracking the evolutionary consequences of gene-based fitness differences, mutation, and random or non-random assortments. However, these approaches do not afford a license to fully disregard the causes of selection in evolutionary explanations and the causal contribution that organisms have in altering

them through their development and activities: the causes and sources of selection (whether abiotic, biotic, or a mix of both) are indeed important in any complete explanation of a phenomenon that has natural selection as its foremost *explanans*. To gain a better understanding of certain evolutionary phenomena that have been the result of selection regimes, biologists should account both for the causes *and* the consequences of selection. Even under the most restricted of views, organisms would still have an important place in evolutionary theory as modifiers of selective pressures and total offspring production (in addition to being "the principal functional unit of biology," as Vidya et al. labeled them).

Second, and along these lines, it remains unclear whether Vidya and colleagues argue for the causal irrelevance or rather the epistemic irrelevance (or both) of the organism for explaining the consequences of evolution (see also Vidya et al. Chap. 17, this volume). *Prima facie*, their argument seems to be consonant with the vantage points of Dretske (1988) and Ramsey and Aaby (2022), who argue that we should distinguish between structuring and triggering causes of phenomena of interest. In evolutionary contexts, triggering causes operate on the individual-level through developmental processes and ecological interactions, and structuring causes shape the structure of population-level processes and alter population-level outcomes. But while Ramsey and Aaby (2022) allow for the possibility that individual-level causes, like those proceeding from niche construction and phenotypic plasticity responses, can causally structure and influence population-level consequences, Vidya and colleagues staunchly reject this opportunity. When fully cashed out in causal terms, their position turns out to be a puzzling one: they seem to suggest that the organismal causes of selection are *causally bounded* and *decoupled* from the consequences of selection if the former simply cannot impact the latter. For that to be the case, there would have to be an unsurmountable fissure between the triggering causes (of selection) and the structuring causes of diachronic population composition. How could these not be causally related, in some way or another? We come to see why they adopt that rationale once we realize that the populational outcome of selection is entirely framed by them in statistical terms and not causal ones (a common move when drawing and abiding by the distinction between proximate and ultimate *causes* in evolution, despite what their name suggest; for discussion and ways to move forward, see Otsuka 2015). In other words, they adopt a two-folded view of selection: causalist when it comes to the developmental causes of selection, but switching to a statisticalist frame of reference when it comes to the populational consequences of selection.[2] If their view is epistemically correct, then

[2]For most philosophers of biology, this would not be a common position to take in the opposition between "causalist" and "statisticalist" interpretations of natural selection, a debate that has raged in the last 20 years and is usually mounted in dichotomous terms: i.e., either one grants that natural selection can be effectively spelled out in causal terms all the way through and exerts causal influence over population change, or one concedes that natural selection is not really a process that exists in the world, but rather turns out to be a very convenient statistical summary of the genuinely causal processes taking a toll in the actual lives and deaths of individual organisms engaging and struggling with their conditions of existence (for steadfast statisticalists, the idea of "causes of

evolutionary biology (and the EES debate within it) has a serious dissociation problem: From it follows that classical population biology and the EES do not speak the same language when talking about the same term: "natural selection." The question would no longer be, using the terminology of Ramsey and Aaby (2022), one of (whether or) how to integrate "triggering" developmental *causes* and "structuring" populational *causes* in evolutionary processes. In its place, any narrative about developmental causes in evolution that attempts to inform populational consequences of selection would need to connect not only individual-level findings to population-level ones, but more importantly, to translate a causalist view of what makes natural selection to happen in the first place (i.e., what causes it) into a statistical view of what consequences that selection brings to population structure. Any attempt at integrating EES-type explanations with traditional evolutionary explanations of a particular phenomenon would require to switch the lens from a causal-mechanistic one into a statistical-populational one in the course of the same explanation. Why should that be the case? Is that the only alternative? Why is it warranted to request such a move and not being open to re-negotiate what should be included in an evolutionary explanation or what overall explanatory standards should prevail? While we agree that this is the outcome entailed by such a dualist view of natural selection (commencing from what causes it to what it statistically brings to trait frequency in populations), we are skeptical on whether this two-folded understanding of selection is the best way to go or even if it accurately represents the breadth of today's evolutionary theory, as well as the debates about how to extend its explanatory scope.

Third, once we allow—against Vidya and colleagues—the consequences of selection to be framed in causal terms, we come to see that the causes and sources of selection make a difference for evolutionary outcomes (not everything in this terrain is about "transmission fidelity" of heritable traits, as these authors proclaim):[3] for instance, when environmental variation is buffered by the activities of organisms (e.g., constructing components of their local environments, or picking suitable resources), selection gradients exhibit reduced temporal and spatial variation, and even weaker selection when compared to non-constructed, abiotic sources (see Clark et al. 2020). Laland et al. (2017) have argued that sustained rounds of niche construction processes (let us not forget, reliant on the activities of developing organisms) impose a consistent bias on selection by dependably generating and maintaining specific environmental states; this leads to predictions related to the consequences of selection depending on its sources (i.e., constructed, non-constructed, or mixed), for example, reduced variance in field measures of

selection," as Vidya and colleagues frame them would seem odd, to say the least; for them, natural selection is an abstract, higher order effect). For a good overviews of this debate, see Otsuka (2016) and Pence (2021); see also Walsh et al. (2017).

[3] Even in the restricted issue of transmission fidelity there is space to disagree with Vidya and co-authors: using causal graph theory, Otsuka (2015) has shown that developmental processes can have an impact on evolutionary responses by affecting one or more of the four components of the Price equation, including transmission fidelity.

responses to natural selection. An example of this can be found in the work of Kevin Laland and colleagues:

> Selection arising from niche-constructed aspects of the environment will have similar (if weaker) regularities and consequences to that observed in artificial selection, but significantly more regularity than natural selection arising from autonomous environmental factors. Responses to niche construction are likely to be qualitatively (or at least quantitatively) different from selection arising from autonomous environmental processes, leading to qualitatively different genetic responses and patterns of trait coevolution (Laland et al. 2017: 5).

Using quantitative genetics models, Fogarty and Wade (2022) have suggested that the pace of phenotypic change can be significantly different in the presence of niche construction, and that the physical changes organisms bring to their environments may alter trait heritability and the response of phenotypes to selection. Organisms produce substantial changes to their physical surroundings, select the environments in which they will live, and adjust their phenotypic constitution in response to shifts of conditions, and all these activities can have causal effects on an individual's phenotype–environment match, its fitness, and its individualized niche (characterized in terms of the environmental conditions that make possible survival and reproduction, and which refers to how organisms experience selective regimes; see Trappes et al. 2022; see also Müller et al. 2020). In general, in addition to affecting selection pressures, reciprocal causation loops instantiated between developing organisms and their environments can have several protracted evolutionary consequences, not least when organisms from different species are involved and interact (for discussion, see Baedke et al., 2021; see also Chiu and Gilbert 2020).

Fourth, in their piece, Vidya and co-authors seem fairly confident in asserting that development, ecological interactions (e.g., between organisms and environments), and heredity are completely separate biological processes and perform sequentially complementary roles in mediating micro-evolutionary change that depends on the action of natural selection. According to their stance, only the first two steps would be related to organisms, which could have an impact on the causes of selection, and the last one can be construed without an organismic context and pertains to the consequences of selection (see Vidya et al. 2023; see also Joshi 2005). The problem with this view, heuristically and explanatorily powerful as it surely is for evolutionary biology at large, hinges on the danger of turning epistemic *idealizations* (related to how scientists grasp, communicate, and generate knowledge by relying on certain simplifications and distortions) into an ontological view of how evolution *always* occurs and what fundamentaly *is*, and thus what deserves to be accounted for. The potential peril is one of "pernicious reification" of the explanatory and mathematical models used to convey evolutionary dynamics, which unavoidably derive from operations of abstraction to render the objects of study tractable: making use of an analogy, the maps employed by biologists (the simplified models tailored for

particular epistemic aims) can become the (evolutionary) world.[4] For instance, for traditional evolutionary models, populations are cast as ensembles of abstract (gene) types (Morrison 2002), an epistemic move which definitely does not capture the ontic correlate of a population in the world in its entirety: i.e., assemblages of conspecifics interacting with themselves, organisms from other species, and their complex and fluctuating surroundings (for an analysis, see Walsh 2019). Construing populations as ensembles of abstract gene types for certain explanatory purposes is nice and fine as long as gene dynamics are not treated as ontological equivalents of populations of living organisms. What sounds *prima facie* obvious is not always acknowledged explicitly by biologists: any population is more than its gene pool, and, as such, the population level is not exhausted by alleles and their evolutionary kinematics.[5]

Although not recognized very often, treating variation, heredity, and selection as perfectly separable components is a common stock idealization in the toolkit and training of evolutionary biologists: assuming that developmental sources of selectable variation are decoupled from their effects on fitness and heredity allows explaining adaptive change solely by appealing to fitness differences between reliably inherited gene types (Uller et al. 2020). Selective explanations (*sensu* Sober 1984) based on genetic fitness differences make desirable to exclude other influences on the evolutionary process (e.g., organismal activities), even when these might be potentially significant, because they could reduce the explanatory power of generalizations and stable inferences to many counterfactual scenarios of adaptive evolutionary change where background conditions could be different (for an analysis, see Baedke et al. 2020).[6] But underlying this *model* of evolutionary explanation lies the assumption that the processes that occur within (and stem from) individual organisms throughout their ontogenies constitute one set of causes, and the processes that influence populations (e.g., natural selection, drift) constitute another, wholly independent set of causes which should be given explanatory primacy and priority (Walsh 2019; see also Bourrat 2019). These idealizations prevent some developmental causes from being regarded as genuine evolutionary causes (Fábregas-Tejeda 2019).[7]

[4] On this point, Andrews (2021: 29) advances important general remarks: "Reification involves mistaking an aspect of a model—its structure, its construal, or the union of both—for an aspect of empirical data or the natural world; mistaking the math for the territory, so to speak. Reification also occurs when we take an analogical relationship to be a literal one, or when elements of a model's construal in its original domain of application get brought along parasitically into a novel domain in model transfer."

[5] In addition, "bookkeeping" about evolutionary change can, in principle, not only be provided by genes, but, for example, also through registering stable developmental or ecological niches that affect populations' trait stability.

[6] For a discussion on the explanatory role of invariance in theoretical population genetics, see Walsh (2015).

[7] In their contribution, Vidya and colleagues impute to us the mistaken view according to which "organism-environment" and "gene-environment" are ontological comparable relata. They suggest that we "seem to be treating gene-environment interactions as a 'thing' belonging to the same

In contrast, organism-centered models that have inspired EES thinking link ontogenetic processes with evolution and do not treat developmental variation, ecological interactions, and hereditary relationships as roundly dissociable and causally autonomous given that processes such as developmental bias, phenotypic plasticity, extra-genetic inheritance (e.g., epigenetic and ecological inheritance), and niche construction offer various cases in which developmental causes can direct and facilitate evolutionary change (for an analysis, see Uller and Helanterä 2019; Uller et al. 2020). EES-type models are most certainly not preferable for all explanatory standards in evolutionary biology (see Baedke et al. 2020), but for certain target systems and *explananda* they are indeed explanatorily adequate: for example, in instances where developmental niche construction takes a prominent role, with the behavioral and developmental repertories of organisms altering parent-offspring similarity or generating new variation, the working assumption that development, ecology, and heredity should be fully disentangled is harder to maintain (see, for example, Stotz 2017; Schwab et al. 2017; Dury et al. 2020; Aubernon et al. 2022). Some evolutionarily relevant interactions and phenotypic changes are actively mediated by organisms that shape their own developmental niches with intergenerational consequences (see, for instance, Laland and Sterelny 2006). Likewise, in the study of eco-evolutionary dynamics, perspectives that accentuate phenotypic plasticity from a developmental systems standpoint can sometimes be brought to the fore to predict how population responses might unfold and how they happen as they do, as these differ in their fundamental assumptions on what counts as causal processes that generate differential fitness and inheritance when compared to traditional, genotype-specified norm of reaction viewpoints (Smallegange 2022; see also Sultan 2017; Uller et al. 2020).

In sum, organisms are *causally* relevant both for the "causes" (and sources) and the "consequences" of selection, and for evolutionary dynamics and trajectories in general. Evolutionary biology cannot dispense with the successful populational models built with the mathematical tools and assumptions of quantitative and population genetics (once, of course, we become aware of their limitations and

logical category as organism-environment relations" (Vidya et al. 2023: 5). We certainly do not think in those terms and our discussion on idealization and abstraction in evolutionary models should make this contention more explicit: a gene-environment interaction has no ontic referent due to unbreachable scale-related and spatio-temporal discordances (e.g., a *token* gene inside the nucleus of any cell of a developing organism never interacts directly with the environment of said organism). What are featured in the models of quantitative genetics are the products of higher order abstraction (e.g., simplifications, surrogate variables, or mathematical identities) that can be analytically or explanatory useful for certain scientific tasks (gene-environment interactions being one among many abstractions featured in the models). Vidya and colleagues seem to be aware of this: "a gene-environment interaction is one way of statistically (not materially or causally) quantifying the effects of a real organism-environment relation on variation in a phenotype" (Idem). However, they assume that gene-environment interactions unproblematically apprehend (ontic) causal organism-environment interactions. One needs to do more work to show how organism-environment interactions get translated into, for instance, statistical gene-environment covariances. How can we link causal knowledge about the former to statistical models about the latter? These are not trivial questions that most evolutionary biologist simply take for granted in their praxis.

idealizations while avoiding pernicious reification), but, at the same time, it also needs organismal models (both conceptual and mathematical)[8] that take into account development, agency, and organism-environment reciprocal interactions (see, as promising cases in point, Brun-Usan et al. 2021, 2022; for discussion, see Baedke and Fábregas-Tejeda Chap. 8, this volume). Without a doubt, these will not be the holy grail that will explain everything in evolution and will not be free of some drawbacks, but at least they would help biologists address a broader range of *explananda* and contrast classes for these, and supply them with a developmentally-informed battery of *explanantia* (see Uller et al. 2020; Baedke et al. 2020). Beyond confrontational standoffs and disproportionate rhetorical strategies on both sides of the Extended Evolutionary Synthesis debate, we hope that one lasting outcome of these discussions will be the growing recognition inside the evolutionary biology community that natural selection and developmental processes *together* shape evolutionary trajectories. Keeping organisms outside of evolutionary explanations simply will not do.

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[8] We should clarify here that the notion of "model" in the philosophy of science not only encompasses mathematical models, but also different kinds of epistemic objects with differing virtues and aims: e.g., analogical models, mechanistic models, testing models, scale models, probing models, phenomenological models, heuristic models, didactic models, toy models, instrumental models, and a large etcetera (for a general introduction to models in science, see Frigg and Hartmann 2020).

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Part IV

The Structure of Evolutionary Theory: Beyond Neo-Darwinism, Neo-Lamarckism and Biased Historical Narratives About the Modern Synthesis

11

Erik I. Svensson

Abstract

The last decades have seen frequent calls for a more extended evolutionary synthesis (EES) that will supposedly overcome the limitations in the current evolutionary framework with its intellectual roots in the Modern Synthesis (MS). Some radical critics even want to entirely abandon the current evolutionary framework, claiming that the MS (often erroneously labelled "Neo-Darwinism") is outdated, and will soon be replaced by an entirely new framework, such as the Third Way of Evolution (TWE). Such criticisms are not new but have repeatedly re-surfaced every decade since the formation of the MS, and they were particularly articulated by developmental biologist Conrad Waddington and paleontologist Stephen Jay Gould. Waddington, Gould, and later critics argued that the MS was too narrowly focused on genes and natural selection, and that it ignored developmental processes, epigenetics, paleontology and macroevolutionary phenomena. More recent critics partly recycle these old arguments and argue that non-genetic inheritance, niche construction, phenotypic plasticity and developmental bias necessitate major revision of evolutionary theory. Here I discuss these supposed challenges, taking a historical perspective and tracing the arguments by critics back to Waddington and Gould. I dissect the old claims by Waddington, Gould and more recent critics that the MS was excessively gene centric and became increasingly "hardened" over time and narrowly focused on natural selection. Recent critics have consciously or unconsciously exaggerated the long-lasting influence of the MS on contemporary evolutionary biology and have underestimated many post-Synthesis developments, particularly Neutral Theory, evolutionary quantitative genetics and the power and generality of the

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Price Equation. Critics have also painted a biased picture of the MS as a more monolithic research tradition than it ever was and have downplayed the pluralistic nature of contemporary evolutionary biology, particularly the long-lasting influence of Sewall Wright with his emphasis on gene interactions and stochasticity. I argue that some of the criticisms of the MS and contemporary evolutionary biology are primarily meta-scientific, revealing the underlying identity politics of critics when pushing their alternative research agendas. It is still unclear what their proposed alternative research frameworks would entail and why the existing theoretical framework is insufficient. Finally, I outline and visualize the conceptually split landscape of contemporary evolutionary biology, with four different stably coexisting analytical frameworks: adaptationism, mutationism, neutralism and selectionism. I suggest that the field can accommodate the challenges raised by critics, although structuralism ("Evo Devo") and macroevolution remain to be conceptually integrated within mainstream evolutionary theory.

Keywords

Developmental bias · Extended evolutionary synthesis · Modern synthesis · Macroevolution · Mutationism · Neo-Darwinism · Niche construction · Non-genetic inheritance · Population genetics · Phenotypic plasticity · Quantitative genetics · Third way of evolution

11.1 Introduction

The Modern Synthesis (MS) of evolutionary biology was one of the most important scientific achievements in evolutionary biology during the twentieth century (Mayr 1993; Mayr and Provine 1998; Cain 2009). The MS formed gradually, through a series of influential research books and articles by Dobzhansky, Huxley, Mayr, Rensch, Simpson and several other biologists (Mayr and Provine 1998; Reif et al. 2000). An important early achievement was the formation of the *Society for the Study of Evolution* (SSE) in March 1946 and the establishment of its scientific journal *Evolution*. Cain (2009) has described the MS as a shift away from *object-based* (i.e. organisms) natural history to *process-based* (selection, gene flow, genetic drift) natural history. Biologists and natural historians from the MS and onwards sought to explain patterns within and among populations and species with current and past evolutionary processes. The shift towards process-based natural history was stimulated by the developments of mathematical population genetics in the decades before the MS, particularly contributions by Fisher (1930), Haldane (1932) and Sewall Wright (1931, 1932).

It is important to emphasize that the architects of the MS aimed to be *synthetic*. Accordingly the results of their efforts has sometimes been termed *"the synthetic theory of evolution"* (Reif et al. 2000). Specifically, Mayr and others repeatedly emphasized that the MS incorporated insights from several different fields, including

genetics, systematics, paleontology and natural history (Haffer 2007). This synthetic goal became evident in the famous debate between Mayr and Haldane about the utility and limitations of so-called "bean bag genetics" (Mayr 1959; Haldane 1964; Crow 2008; Dronamraju 2011). Mayr strongly criticized the mathematical population geneticists Fisher, Wright and Haldane for ignoring gene interactions in their theoretical models (Mayr 1959). Mayr instead emphasized what he felt was the more important contributions by himself, Dobzhansky and other empiricists and naturalists in the formation of the MS (Dronamraju 2011). In this famous debate with Haldane, Mayr clearly revealed that he erroneously thought that the mathematical population geneticists were not aware of gene interaction (epistasis), in spite of epistasis being central in Sewall Wright's Shifting Balance Theory of evolution (Provine 1986; Wade and Goodnight 1998; Steffes 2007). In fact, Wright's interest in genetic interactions, rather than simply additive effects of genes, is an example of early system-level thinking that could be viewed as a predecessor to systems biology today. Moreover, the fact that Wright, one of the founding fathers of modern population genetics, was interested in such interactions and system-level phenomena of organisms establishes a forgotten link between the organicist school (Peterson 2017) and early population genetics (Steffes 2007). This forgotten historical link contradicts Mayr's claim and sweeping characterization of Wright as a simple "bean bag geneticist" who was not aware of epistasis (Mayr 1959).

In retrospect, it is of course easy to point to many limitations of the MS, such that neither developmental biology nor ecology had any central roles (Antonovics 1987; Endler and McLellan 1988). This should not divert us from realizing that the aim of the MS architects was—indeed—a synthetic one (Reif et al. 2000) and the MS has clearly served its purpose, at least for sexually reproducing organisms (Novick and Doolittle 2019). Recent critics often describe the MS as more simplistic and monolithic than it ever was, and have frequently exaggerated the role of population genetics in the synthesis formation (Pigliucci 2007; Laland et al. 2014, 2015; Noble 2015; Müller 2017). These biased narratives about the MS have plagued many discussions about the state of contemporary evolutionary biology, which I discuss in this chapter.

11.2 What the Modern Synthesis Was (and Was Not)

A frequent claim made by critics of the MS is to equate it with "Neo-Darwinism" (Koonin 2009; Noble 2015, 2021). Ironically, this conflation is sometimes also made by defenders of the MS (Charlesworth et al. 1982, 2017; Hancock et al. 2021). This conflation between the MS and Neo-Darwinism is historically inaccurate (Reif et al. 2000; Chen et al. 2021) and can be traced to the late developmental biologist Conrad Waddington (Waddington 1957; Wilkins 2008; Peterson 2017) and the late paleontologist Stephen Jay Gould (2002). Waddington and Gould used the label Neo-Darwinism in a negative and condescending fashion when they talked about the MS. However, Neo-Darwinism is a historical term that was coined several decades before the MS by Romanes (Gould 2002) and was closely linked to August

Weismann's doctrine about separation of the germ line and the soma, i.e. the rejection of Lamarckian inheritance of acquired characters (Jablonka and Lamb 2007; Pigliucci 2009). As the name Neo-Darwinism implies, only one evolutionary force was recognized prior to emergence of mathematical population genetics: Natural selection (sexual selection was still not fully accepted). As emphasized by Lynch and other researchers, modern evolutionary biology and its predecessor MS, clearly allows for multiple evolutionary processes in addition to selection, specifically genetic drift, mutation and recombination (Charlesworth et al. 1982; Lynch 2007; Svensson and Berger 2019), contrary to claims by some molecular biologists like Eugene Koonin who incorrectly characterize the MS as just recognizing natural selection (Koonin 2009). While Neo-Darwinism only recognized the single evolutionary force (natural selection) that was discovered by Darwin and Wallace, the MS and evolutionary biology developed in to a pluralistic field that incorporated multiple evolutionary processes that were formalized by mathematical population genetics (Svensson and Berger 2019). Moreover, Mayr himself explicitly clarified that the MS was distinct from Neo-Darwinism (Haffer 2007; Pigliucci 2009) as did the paleontologist George Gaylord Simpson, another leading architect of the MS (Simpson 1949).

Nevertheless, the conflation of the MS and Neo-Darwinism is still commonly made by some critics of contemporary evolutionary biology. For instance, the physiologist Dennis Noble (Noble 2013) claims that *"The 'Modern Synthesis' (Neo-Darwinism) is a mid-20th century gene-centric view of evolution based on random mutations accumulating to produce gradual change through natural selection"*. Noble further claims that *"all the central assumptions of the Modern Synthesis (often also called Neo-Darwinism) have been disproved"* (Noble 2013). Noble further argues that an extended *"Integrative Synthesis"*—an entirely *"new conceptual framework"*—will *"replace"* the MS (Noble 2015). Similar confident claims have been put forward by the molecular microbiologist James Shapiro (Shapiro 2011). Shapiro and Noble launched *"The Third Way of Evolution"* (TWE) initiative a few years ago (https://www.thethirdwayofevolution.com/) that claims to provide a middle path (*"Third Way"*) between creationism and Neo-Darwinism. The enthusiasm for their project has—mildly put—not been overwhelming among evolutionary biologists (Charlesworth et al. 2017). To date, there are few leading evolutionary biologists who have openly embraced the TWE. TWE has generated more enthusiasm among a vocal minority of philosophers, such as Dennis Walsh and John Dupré, the latter who in 2012 characterized evolution as *"a theory in crisis"* (Dupre 2012). The sheer confidence by which some philosophers and critics of contemporary evolutionary biology predict that contemporary evolutionary biology will soon be replaced by an entirely new framework (details of which are very unclear) is remarkable, particularly as the majority of evolutionary biologists are not even aware of the existence of TWE and carry on their research as usual. Those who doubt this should join any of the regular evolutionary biology congresses organized by the societies ESEB (*European Society for Evolution*) and SSE (*Society for the Study of Evolution*) where little of this forthcoming paradigm shift announced by Noble, Shapiro, Walsh and Dupré has been visible during the past decade. The

impression one gets from the efforts by these biologists and philosophers is that they are trying to launch a culture war against contemporary evolutionary biology, by erroneously claiming that not much has happened since the MS and by repeatedly equating the latter with Neo-Darwinism. The MS is portrayed by these critics as a dogmatic monolith, and some of their criticisms are more meta-scientific than scientific. The poor historical scholarship among some of these critics and their inaccurate and biased characterizations of the MS suggests to me that the TWE is largely an identity political project rather than presenting any serious challenge to the current theoretical framework.

The main problem with Noble's and other similar criticisms of contemporary evolutionary biology are the biased and historically misleading characterizations of the MS (see e.g. (Noble 2013, 2015, 2017, 2021)), where the MS is not only conflated with Neo-Darwinism but also with the much later developments, such as Richard Dawkins's theory of the selfish gene (Dawkins 1976). Any serious student of the history of evolutionary biology should know that the MS emphasized evolving populations of organisms, or "population thinking" in Mayr's terminology (Haffer 2007). The integrative nature of the MS with its emphasis on evolving populations is therefore radically different from the more reductionistic perspective with emphasis on individual genes, developed by Williams (1966) and Dawkins (1976). The more reductionistic genic perspective, in turn, is closely associated with the emergence of sociobiology and modern behavioural ecology that developed several decades after the formation of the MS (Ågren 2016). Lumping these later scientific and conceptual developments together with the earlier MS neglects substantial differences between radically different research traditions.

The conflation of the MS, Neo-Darwinism and later schools of thought is by no means restricted to Noble and colleagues, but can sometimes also be seen in the writing of orthodox population geneticists and theoretical evolutionary biologists, such as Brian Charlesworth and colleagues (2017). One recent example is provided by Stoltzfus (2019), who in discussing the pioneering statistical approach to studying selection that was developed by Lande and Arnold (1983) argued that *"quantitative genetics is the branch of mathematical theory that most closely follows neo-Darwinian assumptions"* (Stoltzfus 2019; p. 57). This is a remarkable claim, considering that neither population nor quantitative genetics existed as scientific fields more than 150 years ago when the term Neo-Darwinism was first coined by Romanes. Today, the term Neo-Darwinism seems mainly to be used as a pejorative label of the MS by those who would like to see radical conceptual change in evolutionary biology (see e.g. (Koonin 2009; Noble 2015, 2021)), rather than as a descriptive term for a specific historical school of thought.

Another common but misleading characterization of the MS is to label it *"the Modern Synthesis theory"* (Müller 2017) or *"Standard Evolutionary Theory"* (SET) (Laland et al. 2015), implying a closed and rigid system and a formal theory, against which challengers revolt. To be fair, I have used the term SET myself when critically evaluating such claims (Svensson 2018). However, this was in response to the prior establishment of the term SET by Laland and colleagues (2015). Labelling the MS as a "theory" is, however, misleading, as it was a loose conceptual framework of *how to*

do science, rather than a formal theory (Cain 2009). Specifically, the establishment of the MS reflected a change in conceptual focus among biologists towards evolutionary processes operating within populations, away from the previous focus on object-based natural history and individual organisms (Cain 2009). It is telling that there are very few mathematical equations produced by any the leading architects of the MS or something that could be called theory in any meaningful or substantial way. To be sure, the MS relied on mathematical population genetics theory as one of several underlying frameworks (among other influences), however it was not equivalent to population genetics but went far beyond it (*contra* Müller 2017). In fact, the main architects behind the MS were organismal biologists and systematists like Dobzhansky, Mayr and Rensch and the paleontologist Simpson (Mayr 1993; Mayr and Provine 1998). Since the MS is a research framework of how to do science and a *perspective* rather than a formal theory, it follows that it cannot be replaced by any new theory, let alone a new paradigm, which has even been admitted by one of the leading critics of contemporary evolutionary biology; Massimo Pigliucci (Pigliucci and Finkelman 2014).

Another frequent characterization of the MS is that it is "gene centred" (Dupré 2021), implying that it exclusively focusses on allele frequency changes in a shared gene pool, ignoring organismal evolution (Laland et al. 2015). Although Mayr is sometimes claimed to have held this narrow gene-centric view, in his later writings, he clearly distanced himself from the narrow view that evolution could solely be reduced to allele frequency changes (Haffer 2007). In fact, one could probably argue the opposite: Mayr was sometimes not gene centric enough, and frequently revealed his remarkable weak knowledge about population genetics theory, as in the debate about bean bag genetics (Dronamraju 2011). The beanbag genetics debate showed that Mayr did not seem to understand the finer details of mathematical population genetics theory (Haldane 1964; Crow 2008) and revealed his lack of understanding that epistasis was central to Sewall Wright's thinking and his population genetic framework (Steffes 2007). Provine noted that something similar could be said about Dobzhansky's lack of understanding of the details of mathematical population genetics in his collaboration with Sewall Wright (Provine 1986).

Recently, Huneman (2019) reminded us that the MS was hardly as monolithic as critics like Pigliucci, Laland, Noble and others have claimed it to be. One could even question the unity and coherence between quite disparate research traditions within the MS (Svensson 2018; Huneman 2019). The MS can be characterized as containing two rather distinct research traditions: one adaptationist school focused on natural selection, primarily in the UK (Lewens 2019), exemplified by the collaboration between Ford and Fisher, and a more pluralistic school in North America, exemplified by the collaboration between Sewall Wright and Dobzhansky (Huneman 2019). In addition, we should not forget the German contribution to the MS with its more structuralistic focus on development (Reif et al. 2000). The Israeli philosopher Ehud Lamm notes in a critical book review that the MS was a complex evolutionary process that is now well behind us (Lamm 2018), and similar views have been expressed by some science historians (Reif et al. 2000; Cain 2009). Today, the MS mainly serves as a rhetorical figure and an argument by those calling

for radical conceptual change in evolutionary biology (Buskell and Currie 2017; Lamm 2018). Clearly, much of the debate about the MS has less to do with the synthesis per se and instead reveals that some reformers and critics are mainly engaged in an identity political culture war, where the MS is portrayed as more rigid and dogmatic than it ever was. The limitations of the MS are thus often used as an excuse to criticize contemporary evolutionary biology, including (real or perceived) gene centrism or reductionism (Pigliucci 2007; Noble 2013, 2015, 2017; Laland et al. 2014, 2015; Müller 2017).

11.3 Gould's Mixed Legacy: Strawman Arguments and Myths About the MS

Here, I will focus on two common strawman arguments against the MS that were raised repeatedly by Gould and—despite being refuted many times—continue to live on in ongoing debates. These two arguments are, first the so-called "isotropy assumption" about variation (Pigliucci 2019) and second, the claim that the synthesis "hardened" over time and became narrowly focused on natural selection, ignoring other evolutionary processes such as genetic drift (Gould 2002).

The isotropy assumption is the claim that the MS assumes that variation is equally likely in all directions (Pigliucci 2019). Taken to its logical extreme, the isotropy assumption would say that all variation is free, and that characters do not covary (Fig. 11.1a, b). In the more technical language of evolutionary quantitative genetics, it would be equivalent to claim that all the off-diagonal elements in the genetic variance-covariance matrix (**G**) (Steppan et al. 2002) are zero, i.e. the strong claim that traits are genetically uncorrelated with each other (Fig. 11.1b). One could visualize this supposed isotropy assumption as the off-diagonal elements of **G** being spherical, rather than ellipses (cf. Fig. 11.1b vs. d). Such an extreme view is obviously a caricature of both the MS and contemporary evolutionary biology. No leading evolutionary biologist has such a naive view of unconstrained variation, to my knowledge. On the contrary, it is clear that both Darwin and researchers working in the MS tradition were well aware of genetic correlations, correlated growth and correlated responses to selection (Charlesworth et al. 1982; Svensson and Berger 2019; Svensson 2020), revealed, for instance, by the rich literature on the evolution of allometric relationships (Bolstad et al. 2015; Tsuboi et al. 2018; Svensson et al. 2021). Yet, Pigliucci (2019) claims that this isotropy assumption of developmental processes and variation is a key feature of the MS, essentially re-iterating previous older arguments by Gould (2002). One wonders how Pigliucci deals with the fact that Julian Huxley—one of the architects of the MS—coined the term "allometry", which is a prime example of correlated variation and non-linear scaling relationships between traits (Huxley and Teissier 1936)? Did Huxley really assume isotropic variation? Indeed, the evolution of allometric relationships is a popular theme in contemporary evolutionary biology research (Bolstad et al. 2015; Tsuboi et al. 2018). If the isotropy assumption is so widespread as Pigliucci (2019) claims: why have then genetic correlations, correlated responses to natural selection and the

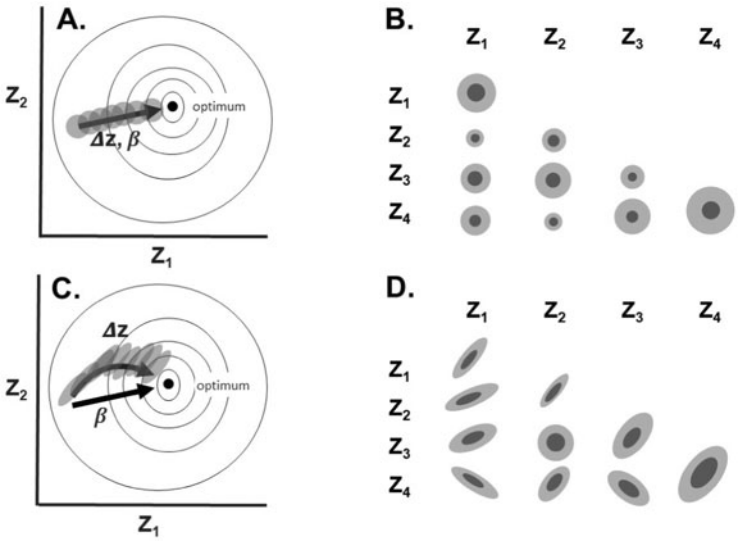

Fig. 11.1 (**a**) When two phenotypic traits (here denoted Z_1 and Z_2) are uncorrelated with each other (as shown by circles) and are evolving on an adaptive landscape with one peak (denoted "optimum") they will evolve in a straight direction towards the peak, and the evolutionary response to selection (Δz) will be aligned with the selection gradient (β). (**b**) A hypothetical genetic variance-covariance matrix (**G**: grey) and a mutational variance-covariance matrix (**M**: black) of four different traits (Z_1—Z_4). Shown are the standing genetic and mutational variances of traits (diagonal elements) and the standing genetic and mutational covariances (off-diagonal elements). This hypothetical variance-covariance matrix is *isotropic*, meaning that traits vary independently of each other and genetic variation is equally abundant in all directions (hence all elements are circular, and traits are uncorrelated with each other). Some evolutionary biologists have argued that this isotropy assumption has been the default assumption in the MS and in evolutionary genetics (Gould 2002; Pigliucci 2019). (**c, d**) In contrast to the isotropy assumption, phenotypic traits in natural populations are often genetically and phenotypically correlated with each other, shown here as variances and covariances as being elliptically shaped, rather than circular. In (**c**), we see how such genetic covariance between the same two traits as in (**a**) (Z_1 and Z_2) result in a biased and curved evolutionary trajectory that delays the time needed to evolve to the fitness optimum. (**d**) shows a more realistic genetic variance-covariance matrix and a mutational matrix, where traits can be either positively (e.g. Z_1 and Z_2), negatively (e.g. Z_1 and Z_4) or uncorrelated with each other (e.g. Z_2 and Z_3). Note that **G** and **M** are aligned in **D**, consistent with theory and empirical evidence suggesting that they are both shaped by the adaptive surface and correlational selection (Jones et al. 2014; Houle et al. 2017; Svensson and Berger 2019; Svensson et al. 2021)

evolution of genetic variance-covariance structures been the focus of so much evolutionary biology research for decades (Lande 1979, 1980; Lande and Arnold 1983; Zeng 1988; Schluter 1996; Steppan et al. 2002)? The inevitable conclusion here is that the isotropy assumption is neither an accurate characterization nor a strong argument against the MS or contemporary evolutionary biology. See Salazar-Ciudad (2021) and Svensson and Berger (2019) for further critique of the isotropy claim.

Gould further claimed that the MS hardened, more narrow in scope and exclusively focused on natural selection and that other evolutionary processes and stochasticity were downplayed over time (Gould 1983, 2002). This so-called "hardening of the Modern Synthesis"-argument is a popular and widespread myth that has frequently been re-iterated by several later authors (Pigliucci and Müller 2010a; Huneman 2019). In fact, this so-called hardening argument has seldom been questioned, but it appears to be accepted at face value among some biologists and philosophers who otherwise seem to maintain a critical distance from Gould (Huneman 2019). Here, I question Gould's claim that the MS hardened, in line with previous authors who have also critically dissected his highly biased historical narratives (Orzack 1981; Sepkoski 2012; Svensson 2020). I argue that the hardening myth of the MS was a deliberate exaggeration promoted by Gould to justify his own scientific project, aiming for an expansion and radical revision of evolutionary theory (Gould 1980). To secure his place in history, Gould pushed the hardening myth and other strawman arguments to paint a highly biased view of the MS as excessively adaptationist and dogmatic, while brushing under the carpet facts that ran counter to his views (Orzack 1981; Sepkoski 2012; Svensson 2020). An uncomfortable fact that does not fit Gould's narrative include the tension between "Wrightian" and "Fisherian" population genetics that was present from the beginning of the MS, and which has shaped evolutionary biology ever since, contributing to its pluralism (Orzack 1981; Provine 1986; Coyne et al. 1997; Wade and Goodnight 1998; Goodnight and Wade 2000; Svensson 2018; Huneman 2019). Clearly, the Wrightian tradition with its emphasis on stochasticity, genetic drift and gene flow has been a key part of the MS (Provine 1986), especially in North America (Huneman 2019), where Gould spent his entire academic career. The existence of the Wrightian tradition therefore partly refutes Gould's claim about the excessive adaptationism of the MS (Orzack 1981). Moreover, the examples Gould used as evidence for his claim that the MS hardened from its early formative years in the 1940s to later decades (Gould 2002) do not hold up, upon critical inspection.

Gould re-read original and updated versions of Dobzhansky's and Mayr's early synthesis-books (Dobzhansky 1937; Mayr 1942). He argued that there was a much stronger emphasis on selection in explaining patterns of genetic variation, polymorphisms and clines in nature in the later editions (Gould 2002). This stronger emphasis on selection was interpreted by Gould as an increasingly dogmatic attitude of these two major architects of the MS (Gould 1983, 2002). An alternative, but more plausible interpretation is that both Dobzhansky and Mayr changed their views in the face of new empirical evidence, rather than for ideological reasons or because they became more narrow minded. Dobzhansky, for instance, studied the dynamics of chromosomal inversion polymorphisms in natural populations of *Drosophila* (Dobzhansky 1970). The reason for studying these chromosomal inversion polymorphisms was that Dobzhansky collaborated with Sewall Wright and was interested in studying genetic drift and testing aspects of the Shifting Balance Theory (Provine 1986). Dobzhansky therefore picked (putatively) neutral markers like chromosomal inversions with the aim to study genetic drift. Dobzhansky and

many others should be excused in that they could not see how this seemingly arbitrary chromosomal character could be important to fitness or affect an organism's phenotype in the pre-DNA era. Dobzhansky therefore started with a neutral expectation, but soon—to his surprise and disappointment (!)—he found out that these chromosomal inversion polymorphisms fluctuated predictably with season and changing temperatures (Dobzhansky 1970; Provine 1986). These fluctuations implied that these chromosomal inversion polymorphisms were not selectively neutral and did not change in frequencies solely by genetic drift, as he had hoped (Provine 1986). Thus, Dobzhansky's empirical insight that selection was operating on these chromosomal inversion polymorphisms can hardly be characterized as a "hardening" or reflecting more dogmatic attitude where selection became overemphasized. Instead, it was rather the opposite: Dobzhansky clearly and at first *underestimated* the importance of selection. To the extent Dobzhansky updated his view and increasingly recognized the importance of natural selection, it was a hard-won empirical insight, in striking contrast to Gould who never did any field work himself on extant organisms in natural populations. Later work—on both *Drosophila* and many other organisms—has revealed that chromosomal inversion polymorphisms are often targets of strong natural and sexual selection with pronounced effects on organismal fitness (Noor et al. 2001; Kirkpatrick and Barton 2006; Kupper et al. 2016; Hooper and Price 2017; Faria et al. 2019). Thus, the late Dobzhansky was correct in upgrading the importance of selection, whereas the early Dobzhansky clearly overestimated the importance of genetic drift.

Gould (2002) further argued that another sign of the hardening of the synthesis was how Mayr changed his view of polymorphisms from being described as selectively neutral in his early book *Systematics and the Origin of Species* (Mayr 1942) to being mainly interpreted in adaptive terms in his later book *Animal Species and Evolution* (Mayr 1963). In Mayr's 1963 book such polymorphisms (e.g. colour polymorphisms) were characterized in adaptive terms and as being the target of selection. However, Mayr's changed opinion hardly represents any hardening view, as claimed by Gould, but rather his increasing insights about the importance of selection that the early Mayr clearly underestimated in 1942. Later empirical work on some of the classical colour polymorphisms that were considered neutral characters by Wright and contemporaries have revealed that they are more often than not targets of strong selection (Schemske and Bierzychudek 2001; Turelli et al. 2001). More generally, recent research on colour polymorphisms have revealed that such polymorphisms are often targets of natural or sexual selection and upon closer inspection are seldom neutral (Wellenreuther et al. 2014; Svensson 2017). The fact that both Gould and the early Mayr in 1942 assumed a priori that seemingly meaningless polymorphisms would be selectively neutral might reflect a lack of understanding of population genetic theory by both of them. In all populations of limited size, genetic drift will operate to a greater or lesser extent. The maintenance of polymorphisms and genetic variation therefore requires the operation of some selective mechanism, like overdominant selection or negative frequency-dependent selection (Svensson 2017). Thus, it is the *maintenance* of variation (i.e. polymorphisms) that requires a selective explanation, not the lack of variation

(Svensson 2017). In contrast, lack of variation and the attainment of monomorphism is the default expectation in all populations of limited size, an important aspect of population genetic theory that neither Gould nor the early Mayr seemed to have fully understood.

A third example of how Gould's claim about the hardening of the MS reflects increasing empirical insights and not just a dogmatic change in mindset is the study on Darwin's finches by the British ornithologist David Lack (Lack 1945, 1947). There were two versions of Lack's book: one monograph published in the series *Occasional papers of the California Academy of Sciences* in 1945 (Lack 1945) and another one only 2 years later, published by Cambridge University Press (Lack 1947), but with very different messages. In the 1945-version, Lack interpreted interspecific differences in bill size and bill shape mainly in non-adaptive terms, such as bills being selectively neutral and primarily functioning as species isolation mechanisms (Lack 1945). In contrast, in the 1947-version, Lack instead interpreted the same bill differences in ecological terms, as reflecting interspecific niche differentiation in terms of food resources (Lack 1947). The 1947-version was received and reviewed enthusiastically by Mayr, who held Lack in high regard and who emphasized his contribution to bring in ecology to the MS (Haffer 2007). Gould (1983) interpreted this shift in interpretation by Lack as another example of the hardening of the MS, presumably caused by Lack's strong personal connection with Mayr (Haffer 2007). However, it seems much more likely that Lack's changed view reflected a genuine change of mindset, from a non-adaptive a priori assumption that interspecific phenotypic differences are largely neutral and of little significance, to a more realistic ecological view where they at least partly contribute to enhance survival and reproduction in a species niche (Lack 1947). In retrospect, we know now, thanks to the impressive later empirical field work by Peter and Rosemary Grant, that the interspecific bill differences in Darwin's Finches are indeed targets of natural selection and affect inter- and intraspecific competition (Grant and Grant 2014). Therefore, the later 1947-version of Lack turned out to largely be correct (Lack 1947). In Lack's pioneering work we therefore rather see a careful and thoughtful naturalist who changed his opinion and adopted a more realistic view of phenotypic characters, away from an initially questionable assumption that these traits were simply neutral and without any ecological importance to survival and reproduction. Gould—unlike Lack—was a paleontologist and not a field biologist. Gould's lack of appreciation of ecology could explain why he did not understand and appreciate the importance of subtle and small phenotypic differences to organism's survival and reproduction.

Summing up this section: Gould's characterization of the hardening of the MS can be turned entirely upside down: it was a healthy shift away from the unfounded assumption that most characters were strictly neutral and without any importance to fitness to a more ecologically realistic view that even small phenotypic differences could be important. The changing perspectives of Dobzhansky, Mayr and Lack reflect a healthy updating of their views in the face of new empirical evidence more so than any dogmatic stance. In fact, one can instead criticize the MS from the opposite perspective: the architects of the MS did not pay enough attention to

ecology and might have underestimated the importance of studying natural selection directly in the field (Antonovics 1987; Endler and McLellan 1988). Antonovics (1987) pointed out that the architects of the MS typically did not bother to even measure natural selection in natural populations. Similarly, Endler and McLellan (1988) emphasized that few of the leading figures behind the MS worked in natural populations of non-model organisms. It was not until several decades after the MS that evolutionary biologists started to quantify natural and sexual selection in the field to fill in this missing gap (Lande and Arnold 1983; Endler 1986). Moreover, Mayr clearly underestimated the importance of natural selection and the importance of different environments on islands and mainlands in his now largely discredited theory of effect speciation through genetic revolutions (Barton and Charlesworth 1984; Haffer 2007). The founder effect speciation model—which Mayr was very proud of—is a strictly neutral model with little or no role for natural selection. The founder effect speciation model clearly illustrates that Mayr often rather *underestimated* the power of natural selection, *contra* the claims by Gould (1983) and others who in Mayr see a strong and dogmatic adaptationist. The myth that the MS hardened and that it only recognized natural selection is a historically questionable view that Gould promoted, but it continues to be re-iterated by some critics who argue that we need radical conceptual change of contemporary evolutionary biology (Laland et al. 2014, 2015; Müller 2017).

11.4 Extrascientific Criticisms of the MS: Adaptation Without Natural Selection?

Some past and recent criticism against the MS might not have only been scientifically motivated, but extrascientific motives could also partly have played some role (Futuyma 2017). Such extrascentific motives could be based on either ideology or religion, but they are often dressed up as criticism of reductionism, or decrying the lack of any room for purpose in evolution and in the MS, as exemplified by the writings by Noble (Noble 2013, 2015, 2017, 2021). Left-leaning biologists like Waddington and Gould were often critical of what they perceived as genetic reductionism, and many times they had some good points in raising these criticisms (York and Clark 2011; Peterson 2017). However, perhaps they went too far and were also for some ideological reasons opposed to population genetics? Waddington and Gould might for partly ideological reasons have exaggerated their critique of population genetics and downplayed its huge importance for the development and progress of modern evolutionary biology. Many public intellectuals and authors like Arthur Koestler openly flirted with Lamarckism during the twentieth century (Koestler 1971) because they felt that a Lamarckian world with acquired inheritance would be more progressive and more hopeful than the cold Darwinian world with no obvious room for any higher purpose (Futuyma 2017). The increased interest in transgenerational epigenetic inheritance and the possibility that such epigenetic inheritance might turn out to be adaptive can partly be explained by ideological leanings towards the Lamarckian temptation (Haig 2007). This Lamarckian

temptation still exists in the general public and even in a small minority of some vocal biologists. For instance, Eva Jablonka—a leading critic of the MS and a strong proponent of the EES—insists in using the term Lamarckian for phenomena like epigenetic inheritance (Jablonka and Lamb 2007). Jablonka was criticized for this by the philosopher Peter Godfrey-Smith and the biologists' Mary Jane West-Eberhard and David Haig (Haig 2007; West-Eberhard 2007; Godfrey-Smith 2007). The insistence that some variation—including mutations—are "directed" rather than random with respect to the organism's current needs (Godfrey-Smith 2007) is an old but discredited idea that never seems to go away, in spite of being firmly refuted in many experimental studies (Lenski and Mittler 1993; Futuyma 2017; Svensson and Berger 2019). Recent attempts to associate transgenerational epigenetic inheritance to Lamarckism does not hold upon closer critical scrutiny (Deichmann 2016; Loison 2018, 2021). Some of these molecular mechanisms are likely to have evolved by a standard process of natural selection and are therefore evolutionary outcomes, rather than evolutionary drivers (Loison 2018).

In light of the many failures during the twentieth century to prove a central role for adaptive Lamarckian inheritance in evolution, the time now seems overdue to bury both Lamarckism and Neo-Lamarckism (as well as Neo-Darwinism). Jablonka, Noble and others calling for an extension or expansion of evolutionary biology have certainly not helped their own cause by flirting with Lamarckism and directed variation. The (provocative) rhetoric by some critics of contemporary evolutionary biology and their insistence on pushing the Lamarckian angle is presumably the main reason why EES and TWE are still viewed with skepticism in large parts of the evolutionary biology community (Welch 2016; Charlesworth et al. 2017; Futuyma 2017; Svensson 2018). Noble's claim that conscious processes and other adaptive features in organisms evolved because they serve a purpose (Noble 2021) deliberately avoids referring to natural selection, the only scientifically known evolutionary process that can systematically increase organismal adaptation across generations and which can explain adaptive organismal design (Gardner 2013, 2017). Ironically, Noble's obsession with purpose puts him conceptually somewhat close to the ultra-adaptationists and Darwinists Grafen and Gardner in the so-called "Formal Darwinism" project (Grafen 2014; Lewens 2019a). The main but crucial difference is that Noble denies a central role of natural selection in explaining (apparent) design and purpose of organisms. Noble seems to search for some other explanation than natural selection of organismal adaptation and it is unclear if it is even a scientific one (Noble 2021). Similarly, some critics of the MS and contemporary evolutionary more or less openly admit that they aim to re-introduce metaphysical principles in biology by highlighting organismal "agency" (Walsh 2015; Buskell and Currie 2017; Dupré 2021), which the majority of evolutionary biologists, including the present author, firmly reject, unless such perceived agency is described as an outcome of natural selection. There are, however, many conceptual and philosophical problems associated with introducing agency into evolutionary thinking (Okasha 2018).

To the extent (apparent) purposeful organisms exist, evolutionary biologists explain their currently adaptive traits by the standard process of natural selection

that have operated on these traits in the past and which still operate to maintain current function. This has implications for the odd idea of *"adaptation without natural selection"* or *"adaptive evolution without natural selection"* (Pigliucci and Kaplan 2000; Kull 2014), which is the claim that some adaptive traits that increase organismal survival and/or success in a given environment are not products of, or cannot be explained by past or current natural selection. But adaptation without natural selection is an oxymoron, at least if we measure adaptation in terms of fitness or organismal performance. Any trait that enhances organismal fitness in a given environment relative to other trait variants will (per definition) be target of selection, and once the trait has reached its adaptive optimum, it will subsequently be maintained by stabilizing selection for its current utility (Reeve and Sherman 1993; Hansen 1997). Such traits might (or might not) have evolved for their current function, but current utility *implies* ongoing stabilizing selection (Hansen 1997). Hence, currently adaptive traits are (per definition) targets of selection, illustrating that adaptation without natural selection is a meaningless phrase. In addition, such traits could also have an evolutionary history of directional selection where they spread because of the advantages they confer today, and only such traits would count as "true" adaptations, according to Gould and Vrba (Gould and Vrba 1982). Traits for which current utility differ from the selective benefits that drove their original spread were labelled "exaptations" by Gould and Vrba (1982) and exaptations were claimed to be qualitatively different from "true" adaptations. However, the term exaptation is problematic, as it sets up an arbitrary one-generation distinction between the past and the present, as ultimately every trait must have evolved from another trait in the first place. Moreover, exaptation is a one-generation term only, as a trait that is maintained by selection for only one generation for its current function will (per definition) become an adaptation (J. A. Endler, personal communication). Some authors insisting on using the term adaptation without natural selection point to adaptive phenotypic plasticity as an example, which can generate a fit between organism and environment within a single generation (Pigliucci and Kaplan 2000) or they argue for plasticity-led evolution as an alternative to adaptive evolution by natural selection (Kull 2014). The latter idea is often termed "plasticity first" or described as "genes as followers, not leaders", referring to Mary Jane West-Eberhard's original suggestions (West-Eberhard 2003).

Upon critical scrutiny, the superficial arguments above do not provide any evidence at all against adaptive evolution being driven by natural selection. First, theory and empirical evidence have clearly shown that adaptive phenotypic plasticity and phenomena such as genetic assimilation can and are often targets of natural or sexual selection, show heritable variation and can evolve by the standard process of selection (Lande 2009; Chevin et al. 2010; Svensson et al. 2020). Second, the plasticity-first hypothesis and the idea of genetic assimilation of originally plastic changes is (as indicated by the term *"genetic"* before assimilation) is not an alternative to evolution by natural selection, but rather points to the possibility that *the initial adaptive change can be achieved by a plastic adjustment of the organism followed by natural selection on genetic variation that stabilizes the trait* (Price et al. 2003; Lande 2009). West-Eberhard (2003) herself has been quite clear that her idea

about plasticity-led evolution was not mean to be an alternative to genetic evolution by natural selection, but rather an *initiator* of subsequent evolutionary change, where phenotypes played a major role ("leaders") and preceed the genetic change that followed ("genes as followers"). Specifically, West-Eberhard explicitly defines evolution by stating (P. 32):

> Strictly speaking, the units that replicate themselves most precisely, and therefore have cross-generational effects that both reflect past differential reproduction and affect future reproduction, are genes. So genes are the most appropriate units of evolution.

This quote by West-Eberhard is interesting insofar it reveals a fairly traditional view on the definition of evolution, and she does clearly not see plasticity and genetic assimilation as alternatives to adaptive evolution by natural selection. Following the definitions by leading EES proponents (Laland et al. 2014, 2015), West-Eberhard, a leading proponent of plasticity's role in evolution, could thus be classified as "gene centric". Interestingly, West-Eberhard refused to co-author the papers by Laland et al. (2014, 2015) in their call for an EES. Her main objection was that these authors did not explicitly define evolution in terms of genetic change (Kevin Laland, personal communication). The quote above by West-Eberhard also reveals the problems of using the term "gene centric" for those being critical of the EES, as it is obviously possible to emphasize plasticity and phenotypes in evolution, but still be labelled as a gene centrist.

The objections above against adaptive plasticity exemplifying adaptive evolution without natural selection also apply to other adaptive within-generation phenomena. Such within-generation phenomena include thermoregulation and other regulatory behaviours, acclimation, various physiological responses, dispersal, habitat selection etc. (Huey et al. 2003; Edelaar et al. 2008; Edelaar and Bolnick 2019). Such adaptive within-generation modifications that increase an individual organism's fitness or even mean population fitness are ecologically important, but should be viewed as adaptive outcomes of evolution by natural selection (Fig. 5.2), rather than adaptations that formed without natural selection (*contra* Pigliucci and Kaplan 2000; Kull 2014). Once again, there is an important distinction between evolutionary processes leading to between-generation changes and evolutionary outcomes, as revealed in various adaptation expressed during the lifetime of individual organisms (Lynch 2007; Gardner 2013).

Finally, another class of extrascientific motives behind recent criticisms against the MS and contemporary evolutionary biology could be boredom. All evolutionary biologists are (luckily) not interested in the same things, and not everyone appreciates population and quantitative genetics. It is most likely one of the motivations behind Pigliucci's push for the EES, as exemplified in a critical dissection of Brian Charlesworth's views, who had expressed the opinion that most of the problems in evolutionary genetics had been solved: *"Well, perhaps, but some of us are not ready for retirement just yet"* (sic! P. 2744; Pigliucci 2007). The author of the present chapter has, in some discussions with leading proponents of the EES, encountered similar attitudes, for instance that *"reaction norms and population*

genetics are boring". Similar views were expressed by the evolutionary developmental biologist Sean Carroll in his book "*Endless Forms Most Beautiful: The New Science of Evo Devo*", where he declared that the development of form in butterflies and zebras was a more inspiring story to tell about evolution than changes in gene frequencies (Carroll 2006). Population geneticist Michael Lynch was, however, rather blunt in his dismissal of this criticism of population genetics and stated: "*Evolutionary biology is not a story-telling exercise, and the goal of population genetics is not to be inspiring, but to be explanatory."* (Lynch 2007). He further developed his criticism of Carroll and evolutionary developmental biology and the frequent claim that this new field would supposedly overturn evolutionary biology by stating that *"No principle of population genetics has been overturned by any observation in molecular, cellular, or developmental biology, nor has any novel mechanism of evolution been revealed by such fields."* (Lynch 2007).

Although I am not a population geneticist, I strongly agree with Lynch that *the primary goal of evolutionary biology is not to inspire but to explain.* For any theory that aims to be connected to the real world, we should prioritize explanatory power over beauty. I strongly suspect that my view is shared by most of my empirically oriented evolutionary biologist colleagues. The fact that not everyone gets inspired by population and quantitative genetics theory is not a strong or compelling argument that we need major conceptual change in our field.

11.5 Scientific Criticisms of the MS: From Waddington and Gould to the EES

It did not take a long time after the formation of the MS for the developmental biologist Conrad Waddington to express his discontent and bitterness against what he called *COWDUNG*, or *"Conventional Wisdom of the Dominant Group"* (Peterson 2017). Waddington even negatively labelled his former collaborator and co-author Haldane as a simple "Neo-Darwinist" (Peterson 2017). Waddington's decision to distance himself from Mayr, Dobzhansky, Haldane and other leading architects of the MS probably contributed to make his influence on modern evolutionary biology much less than it could potentially have been (Peterson 2017). Waddington was therefore not able to incorporate his views about epigenetics into the mainstream of the MS (Wilkins 2008). It is still an open question whether this was mainly the fault of Waddington himself—deliberately distancing himself from the mainstream—or due to attitudes from Ernst Mayr and the other synthesis architects (Peterson 2017).

In a similar vein, the late paleontologist Stephen Jay Gould distanced himself from the MS and Neo-Darwinism (Sepkoski 2012). Interestingly, Gould started his career as a fairly mainstream evolutionary biologist, building upon Ernst Mayr's rather orthodox theory of allopatric speciation (Mayr 1942). Gould and his collaborator Niles Eldredge incorporated this allopatric theory into their own theory of "punctuated equilibrium" (Eldredge and Gould 1972; Sepkoski 2012). After successfully establishing himself as a major player in the new and growing field of

paleobiology and mathematical models in macroevolution, Gould devoted a large part of his late career to popular science columns in the journal *Natural History* (Sepkoski 2012). Gould also developed his criticisms of the MS in many articles (Gould and Lewontin 1979; Gould 1980; Gould and Vrba 1982) as well as in his late magnum opus *The Structure of Evolutionary Theory* (Gould 2002), published in 2002, the same year as his death. In his critique of the MS and Neo-Darwinism, Gould made several claims that received strong criticisms from other evolutionary biologists (Orzack 1981). Some of Gould's critics argued that he used extensive strawman arguments against the MS (see Sect. 11.3 to justify his own scientific agenda aiming for a major paradigm shift in evolutionary biology (Sepkoski 2012; Svensson 2020). While few would question Gould's scientific contributions to paleobiology, his popular outreach and his admirable fight against creationism, a common (and still valid) criticism of his work is that he strongly exaggerated the rigidity of the MS (Sepkoski 2012). For instance, Gould's biased characterization of the MS as excessively deterministic and adaptationist and his claim that it ignored random factors and stochasticity (Gould 1980, 1981) received strong criticism by Orzack, Charlesoworth, Lande and Slatkin who also pointed to the influence of Sewall Wright on the development of the MS (Orzack 1981; Charlesworth et al. 1982).

Some of the arguments used by Gould—despite being repeatedly countered and in many cases refuted—have survived also after Gould's death, and they regularly resurface in ongoing calls about the necessity to extend the MS (Pigliucci 2007, 2009; Laland et al. 2015) as well as in more radical calls for the entire replacement of MS (Noble 2013, 2015, 2017; Müller 2017). Many critics re-iterate Waddington's and Gould's arguments in their calls for an entirely new synthesis, and argue that insights from developmental biology and epigenetic mechanisms (Table 5.1) in themselves necessitate a major revision, extension or replacement of the MS. While it is quite clear that we now live in a post-Synthesis period, it is striking how the same old arguments by Waddington and Gould resurface at regular intervals. In contrast, other relevant criticisms against the limited scope of the MS, such as its relative neglect of ecology (Antonovics 1987; Endler and McLellan 1988), are more seldom discussed.

11.6 Recent Challenges to the MS

In the previous sections, I have shown how Gould systematically mischaracterized the MS for several decades, making highly exaggerated claims about its strict focus on natural selection and downplaying its pluralistic nature. However, discontent with the MS was present from its early days, and a number of extensions, expansions and various "add-ons" have been suggested (Table 11.1). Some molecular, developmental and theoretical biologists even argue that the MS should be abandoned entirely or replaced, as it can no longer be fruitfully extended (Koonin 2009; Stoltzfus 2017; Müller 2017).

Table 11.1 A non-exhaustive list in chronological order, of different attempts and initiatives to extend, expand or replace the Modern Synthesis, pointing to new phenomena, discoveries and various "add-ons"

Name(s)	Term	Phenomena	References
C.H. Waddington	Extended synthesis	Epigenetics Genetic assimilation Genetic interactions	Waddington (1957) Wilkins (2008)
S.J. Gould	Expanded evolutionary theory	Hierarchical theory Species selection Nonadaptation Exaptation	Gould (1980) Gould (1982) Gould and Vrba (1982) Gould (2002)
J.A. Endler T. McLellan	A new synthesis	Source laws Ecology of natural selection Origin of variation	Endler and McLellan (1988)
M. Pigliucci G.B. Müller	Extended evolutionary synthesis	Evolvability Phenotypic plasticity Epigenetic inheritance Complexity theory High-dimensional adaptive landscapes	Pigliucci (2007) Pigliucci (2009) Pigliucci and Müller (2010b)
E. Koonin	"A postmodern state, not so far a postmodern Synthesis"	Horizontal gene transfer (HGT) Gene duplications Gene loss Neutral molecular evolution	Koonin (2009)
E. Danchin E. Jablonka M. Lamb	Inclusive evolutionary synthesis or expanded evolutionary synthesis	Non-genetic inheritance Cultural evolution Information theory	Jablonka and Lamb (2005) Jablonka and Lamb (2007) Danchin (2013)
D. Noble J.A. Shapiro	The third way of evolution (TWE) or integrated synthesis	Evo devo theory Plasiticty and accommodation Epigenetic inheritance Multi-level selection Genomic evolution Niche construction Replicator theory Evolvability	Noble (2013) Noble (2015) Noble (2017) Shapiro (2011)
K.N. Laland T. Uller M.W. Feldman K. Sterelny G.G. Müller A. Moczek E. Jablonka J. Odling-Smee	Extended evolutionary synthesis (EES)	Developmental bias Plasticity Non-genetic inheritance Niche construction	Laland et al. (2014) Laland et al. (2015)

Closer inspection of Table 11.1 leads me to two conclusions. First, calling for a new synthesis by pointing to limitations of the MS has been a popular and widely used tactic by critics for decades to express strong discontent and push for new ideas, as already noted and discussed by some philosophers and historians (Buskell and Currie 2017; Lamm 2018). In this context, the MS has mainly served as a justification for other grand projects, often also labelled "syntheses" of various kind. Second, the sheer number of phenomena that have been claimed to be missing from the MS is rather bewildering, and it often difficult to see what these different factors have in common (Table 11.1). For instance, in an early call for an Extended Evolutionary Synthesis (EES), Pigliucci listed *"evolvability, phenotypic plasticity, epigenetic inheritance, complexity theory and high-dimensional adaptive landscapes"* as phenomena largely unexplained by the MS (Pigliucci 2007). The last of these phenomena—high-dimensional adaptive landscapes—is odd, as this is a rather orthodox theoretical evolutionary genetic concept, developed by Sergey Gavrilets (Gavrilets 2004), who has clearly distanced himself from the EES (Gavrilets 2010). A few years later, Dennis Noble added *"replicator theory, genomic evolution and multi-level selection"* to his version of the *"Integrated Synthesis"* (Noble 2015). About the same time as Noble, Kevin Laland and colleagues restricted themselves to four phenomena in their version of the EES: *Developmental bias, plasticity, non-genetic inheritance* and *niche construction* (Laland et al. 2015). Before these recent initiatives, we saw that Gould called for incorporation of *species selection*, *hierarchical theory* and *macroevolution* in his proposed expanded version of evolutionary theory (Gould 1982). Already in the 1950s, Waddington highlighted *genetic assimilation*, *epigenetics* and *gene interaction* that he felt were missing from the MS (Waddington 1957; Wilkins 2008; Peterson 2017).

The sheer variety of disparate phenomena listed as challenges to the MS (Table 11.1) therefore gives the impression that different critics have compiled their own "laundry lists" of favourite topics that they feel have been duly neglected (Welch 2016). Or—to put it bluntly—many of these phenomena have little to do with each other, and more reflect the personal interests of critics and what they consider to be important. However, as material for a new synthesis, it is obviously not enough to list a number of interesting phenomena, but there must also be some common thread connecting them together in a convincing conceptual or theoretical framework. Otherwise, biology risks becoming what the physicist Ernst Rutherford dismissed as the mere "stamp collecting" of various facts, but with no theoretical coherence. It is therefore not entirely unexpected that the novelty and theoretical coherence of the EES has been questioned by some philosophers (Lewens 2019b; Buskell 2019, 2020; dos Reis and Araújo 2020) and evolutionary biologists (Welch 2016; Charlesworth et al. 2017; Futuyma 2017; Svensson 2018). A rather blunt recent criticism of the EES is that it is neither an extension nor a proper synthesis (dos Reis and Araújo 2020).

What should we make of all this? On the one hand, proponents of an EES have clearly highlighted some interesting phenomena that deserve to be studied more in depth, such as plasticity and non-genetic inheritance (Laland et al. 2015). On the other hand, skepticism towards a new synthesis based on these and other phenomena

prevails in the evolutionary biology community and the EES is far from being embraced by the mainstream. One impression one gets from Table 5.1 is that advocates of various extensions of the MS are conceptually split among themselves and have difficulties in finding common ground. In particular, while some EES proponents strive for a simple extension (Pigliucci 2007, 2009; Laland et al. 2015) more radical critics instead strive for "replacement", or something we might consider a true paradigm shift (Noble 2013, 2015, 2017). Whereas the former camp can be viewed as "reformers", the latter are better labelled as "revolutionaries". A second impression from Table 11.1 is that it is unclear why particular phenomena are highlighted as arguments for an extended synthesis (e.g., plasticity, developmental bias, niche construction and non-genetic inheritance; (Laland et al. 2015)), whereas other interesting topics like the link between microevolutionary processes and macroevolutionary patterns (Uyeda et al. 2011; Bell 2012; Hansen 2012; Svensson and Calsbeek 2012a; Arnold 2014) are not included. The link between micro- and macroevolution is even outrightly dismissed by some EES proponents (Müller 2017):

> A subtler version of the this-has-been-said-before argument used to deflect any challenges to the received view is to pull the issue into the never ending micro-versus-macroevolution debate. Whereas 'microevolution' is regarded as the continuous change of allele frequencies within a species or population. . ., the ill-defined macroevolution concept. . ., amalgamates the issue of speciation and the origin of 'higher taxa' with so-called 'major phenotypic change' or new constructional types. Usually, a cursory acknowledgement of the problem of the origin of phenotypic characters quickly becomes a discussion of population genetic arguments about speciation, often linked to the maligned punctuated equilibria concept. . ., in order to finally dismiss any necessity for theory change. The problem of phenotypic complexity thus becomes (in)elegantly bypassed. Inevitably, the conclusion is reached that microevolutionary mechanisms are consistent with macroevolutionary phenomena. . ., even though this has very little to do with the structure and predictions of the EES. The real issue is that genetic evolution alone has been found insufficient for an adequate causal explanation of all forms of phenotypic complexity, not only of something vaguely termed 'macroevolution'. Hence, the micro–macro distinction only serves to obscure the important issues that emerge from the current challenges to the standard theory. It should not be used in discussion of the EES, which rarely makes any allusions to macroevolution, although it is sometimes forced to do so.

This rather blunt dismissal of macroevolution by Müller (2017) is certainly not a view shared by other evolutionary biologists, including myself. Interestingly, this outright dismissal of macroevolution by Müller above reveals a view and a lasting legacy that the EES seems to have inherited from the MS, where macroevolution was sometimes simply viewed only as "microevolution writ large" (Sepkoski 2012), although it is important to emphasize that at least some representatives of the MS accepted macroevolution as an autonomous field of research, distinct from microevolution (Stebbins and Ayala 1981). The comment is also interesting as it reveals what Müller thinks is the challenging and interesting problem: the evolution of organismal complexity. For Müller and other critics of the MS and contemporary evolutionary biology, complexity is *the* big problem that we should focus on in explaining, rather than the micro- and macroevolution link.

For several reasons, I think Müller's viewpoint is mistaken and a dead end. First, complexity is by no means easily defined, let alone explained. In fact, some complexity at the molecular level might have little if anything to do with adaptive processes, such as natural selection, but can arise through neutral evolution alone. For instance, the theory of Constructive Neutral Evolution (CNE) postulates that the accumulation of neutral mutation could build up considerable complexity at the molecular level without any need for directional natural selection (Stoltzfus 1999; Muñoz-Gómez et al. 2021). There is some recent experimental evidence for CNE from a study of long-term evolution of vertebrate steroid receptors that have increased in complexity simply through neutral evolution (Hochberg et al. 2020). Such neutral evolution acted in a ratchet-like fashion, leading to a state where current complexity is maintained by purifying selection (Hochberg et al. 2020). I strongly suspect that Müller and other EES proponents with their strong focus on organismal phenomena are not very interested in such neutral evolution at the molecular level and its consequences. Müller (2017) and others seem to implicitly assume that complexity per se always needs some non-neutral explanation. That is not necessarily the case. Null models of evolution can successfully explain the evolutionary increase in both complexity and diversity (McShea and Brandon 2010). In contrast, natural selection is so far the only known evolutionary force that can systematically increase fitness across generations and that can convincingly explain the evolution of (apparent) purpose and adaptive features of organisms (Gardner 2017). Null models like the Neutral Theory do not seem to be held in high regard in the EES community and it is hardly mentioned in their writings (see e.g. (Pigliucci 2007, 2009; Laland et al. 2015)). This striking neglect in the EES community contrasts with much of mainstream contemporary evolutionary biology and the population genetics community where Neutral Theory still holds a central position (Kern and Hahn 2018; Jensen et al. 2019).

11.7 The Re-emergence of Mutation-Driven Evolution and Directed Variation?

The architects of the MS correctly dismissed several alternative but now firmly discredited evolutionary processes, such as the inheritance of acquired characters (i.e. Lamarckism), orthogenesis (i.e. the innate tendency of organisms to evolve in certain directions towards a "goal"), saltationism (evolution by large mutations) and the idea that mutations were the main drivers of evolution rather than natural selection ("mutationism") (Gould 2002). The idea that mutations were the main drivers of evolution was championed by early Mendelians like Hugo de Vries, Gregory Bateson and Thomas Hunt Morgan. These geneticists focused on mutations of large visible effects, such as eye colour and wing mutants in *Drosophila*, often with abnormal phenotypic effects (Gould 2002) but of questionable ecological importance for adaptive evolution in natural populations. These laboratory-based geneticists did apparently not understand or appreciate the importance of natural selection, as they studied organisms in strict laboratory settings, and they had little

direct research experience from natural populations. It was only when Dobzhansky entered the laboratory of Thomas Hunt Morgan that this experimental genetic laboratory tradition in the USA was merged with studies of natural populations that he was trained in from his early educational years in Russia and the Soviet Union (Gould 2002). As a result, Dobzhansky had a deep understanding of both genetics and natural history and he realized that although mutations were an important part of the evolutionary process, in themselves they could not achieve much without the aid of natural selection (Dobzhansky 1970; Provine 1986). Both Dobzhansky and other contemporary evolutionary biologists like Haldane understood that mutations were the ultimate source of novel genetic variation and they both wrote about the mutational process in the years immediately preceding the MS (Dobzhansky 1933; Haldane 1933). But these evolutionary biologists concluded—correctly as it later turned out—that mutations alone were unlikely to explain long-term directional evolution at the phenotypic level, in contrast to the claims of the early mutationists.

Given the strong experimental and empirical evidence against directed mutations (Lenski and Mittler 1993; Futuyma 2017; Svensson and Berger 2019) and the failure of the early mutationists to appreciate the power of natural selection, it is astonishing that some contemporary evolutionary biologists are pushing for a revival of mutationism or mutation-driven evolution (Stoltzfus 2006; Nei 2013; Stoltzfus and Cable 2014). Mutationism was closely connected to the theory of orthogenesis—the idea that internal factors were primarily responsible for evolutionary change and that the external environmental factors (*aka* natural selection) played only a minor role (Gould 2002; Stoltzfus 2006; Stoltzfus and Cable 2014). According to the early Mendelians and mutationists, large-effect visible mutations were important, and the role of natural selection was mainly to sort out the unfit variants. The mutationists contrasted such a negative role of selection with the mutational process that they felt was the real driver of evolutionary change. For good reasons, this view was firmly rejected by the development of quantitative genetics theory and empirical insights from plant and animal breeding (Fisher 1918). The early mutationists clearly overestimated the importance of mutations and underappreciated standing genetic variation and the creative role of natural selection, and mainly saw selection as a "sieve" that could only sort out the unfit (Beatty 2016, 2019). However, the sieve-analogy underestimates the importance of standing genetic variation for adaptation (Barrett and Schluter 2008) and modern views of natural selection emphasize its multivariate nature, and its more creative role in shaping the genetic and phenotypic correlation structure of organisms (Sinervo and Svensson 2002; Svensson et al. 2021).

Those who try reinstate mutationism and mutation as the main driver of evolutionary change are therefore likely to face strong resistance, for good historical and scientific reasons. No serious evolutionary biologist today would question that mutations is the ultimate source of novel genetic variation, and in neutral evolution (where selection is per definition is absent), such neutral mutation pressure can lead to directional evolutionary trends (Sueoka 1988; Svensson and Berger 2019). Moreover, mutation-driven neutral evolution can potentially result in increased molecular

complexity, as emphasized in Constructive Neutral Evolution (CNE), as discussed in the previous section (Stoltzfus 1999; Hochberg et al. 2020; Muñoz-Gómez et al. 2021). There is clearly a potential role for CNE at the molecular level. However, it is important to underscore that even if the initial buildup of such molecular complexity would be entirely neutral and mutation-driven and with no role for natural selection, as soon as these molecular complexes are affecting aspects of the organismal phenotype and thereby likely its fitness (e.g. cell physiology and other aspects of cellular performance), they would immediately and (per definition) become targets of purifying selection (Hochberg et al. 2020; Brunet et al. 2021).

Likewise, few evolutionary biologists deny that genetic effective population size (N_e) determines the efficacy of natural selection, which becomes weaker and less powerful when N_e is low, i.e. approaching neutrality (Lynch 2007, 2010; Jensen et al. 2019; Svensson and Berger 2019). It is uncontroversial to say that the likelihood of mutation bias leading to a fixation bias increases with the inverse of N_e, as selection then becomes weaker relative to genetic drift (Lynch 2007). These insights from standard population genetic theory are far away from the original claims by the early mutationists. Mutation bias is, however, unlikely to play an independent role in adaptive evolution, unless it is aided by genetic drift and/or selection (Lynch 2007; Svensson and Berger 2019). Recently, some researchers claimed, based on a mathematical model, that mutation bias can play an important role in adaptive evolution even in the absence of natural selection (Gomez et al. 2020). However, closer inspection of their model assumptions reveals that for this to work, they have to assume unrealistically high adaptive mutation rates (Gomez et al. 2020). It is therefore questionable how relevant this and other models of mutation bias without selection are for evolution in natural populations.

Most evolutionary biologists today view mutations as a stochastic evolutionary process with no directionality or purpose, with no foresight or any tendency for mutations to systematically increase organismal fitness across generations (Svensson and Berger 2019). This view has strong empirical support (Svensson and Berger 2019), although the representatives from the fringe movement TWE (James Shapiro and Dennis Noble) question this and claim a role for adaptive directionality, purpose and functionality of novel mutations (Shapiro 2011; Noble 2013, 2017). These authors claim that various aspects of genome organization and gene expression in organisms are clearly functional and that these functional aspects of the genome contradicts the traditional view of mutations as random (with respect to current utility and future adaptation; (Svensson and Berger 2019)). For instance, Noble (2017) argues that the existence of functionally significant targeted somatic hypermutations during the lifetime of individual organisms contradict the standard assumption in evolutionary theory that mutations are random with respect to fitness. Noble points to somatic mutations in the vertebrate immune system as an example of such adaptive design, where mutations seem to have purpose, indicating adaptive foresight (Noble 2017). However, both Noble and Shapiro conflate somatic mutations and changes within the lifetimes of individual organisms with germline mutations and evolutionary change across generations (Gardner 2013). Noble and Shapiro also overlook fundamental differences between somatic mutations and

germline mutations, such as that the former are higher (Moore et al. 2021), and are also targets of selection due to their strong link to lifespan and other life-history characteristics (Cagan et al. 2021). The existence of highly sophisticated molecular repair mechanisms, patterns of adaptive gene expression, methylation and other epigenetic mechanisms that Noble and Shapiro highlight is no evidence at all against natural selection operating on random mutational input (Gardner 2013). Instead, and much more likely, natural selection has operated on and shaped these molecular mechanisms and other aspects of genomic architecture (Sinervo and Svensson 2002; Svensson et al. 2021), including both somatic and germline mutation rates (Lynch 2010; Cagan et al. 2021; Moore et al. 2021). Molecular features at the genomic level are therefore shaped by selection, drift, mutation and recombination (Lynch 2007; Gardner 2013; Svensson et al. 2021), and should be viewed as evolutionary outcomes rather than as evolutionary processes in their own right (Loison 2018). Again, a common mistake made by some critics of contemporary evolutionary biology—not only Noble and Shapiro—is to conflate evolutionary processes with the products of evolution (Lynch 2007).

In contemporary evolutionary biology, the stochastic nature of mutations is often conceptualized as historical contingency reflecting the role of chance (Losos et al. 1998; Blount et al. 2018; Svensson and Berger 2019). Thus, adaptive evolution reflects the balance between the deterministic role of natural selection that systematically increases organismal adaptation across generations ("survival of the fittest"), and the nature and arrival order of novel mutations that selection can act upon, the latter often called "arrival of the fittest" (Wagner 2015). All else being equal, if selection operates on a character governed by multiple loci, those loci with the highest mutation rates are more likely to produce novel adaptive mutations that can be "seen" by selection and which subsequently will increase in frequency and become fixed through successive selective sweeps (Xie et al. 2019). A case in point is the adaptive evolution of pelvic reduction in stickleback fish (*Gasterosteus aculeatus*) after colonization and adaptation to freshwater habitats in postglacial lakes (Xie et al. 2019). Molecular studies have revealed that such pelvic reductions are achieved by recurrent deletions which are produced by *Pitx1* enhancer sequences that increase double-strand breaks (Xie et al. 2019). As a result, elevated mutation rates at this locus contribute to make genomic evolution highly parallel and more predictable, through repeated and adaptive phenotypic changes. However, the spread and fixation of these novel mutations are ultimately caused by natural selection in the new freshwater habitat (Xie et al. 2019). This example illustrates that elevated mutation rates alone are not sufficient to drive adaptive and parallel evolution, but natural selection plays a crucial role in the spread and fixation of novel variants. More generally, the role of mutational stochasticity, including the arrival order of novel mutations, has been recognized in contemporary evolutionary biology, both theoretically and empirically, e.g. in mutation-order speciation (Schluter 2009) and in research on historical contingencies (Blount et al. 2018). It therefore appears that those arguing for mutation bias as an entirely novel evolutionary principle (Yampolsky and Stoltzfus 2001; Stoltzfus and Yampolsky 2009; Gomez et al. 2020) might have somewhat exaggerated their cause. It is currently difficult to see

that mutation-driven evolution or mutation bias (Nei 2013; Stoltzfus and Cable 2014) would require a major revision of the current already pluralistic theoretical framework of evolutionary biology.

11.8 Developmental Bias, Niche Construction, Non-Genetic Inheritance, and Plasticity

The most recent challenge to contemporary evolutionary biology is the push for an EES (Table 11.1), as exemplified by the writings by Kevin Laland and colleagues (Laland et al. 2014, 2015). The EES group argues that the current theoretical framework with only four main evolutionary processes (selection, drift, recombination and mutation) based on population genetic theory (Lynch 2007; Svensson and Berger 2019) is incomplete, and fails to fully explain phenotypic evolution and organismal adaptation (Laland et al. 2014, 2015). Specifically, they argue that evolutionary theory needs to take in to account four additional processes that they claim have been neglected: *phenotypic plasticity*, *developmental bias, niche construction* and *non-genetic inheritance* (Laland et al. 2015). Although many evolutionary biologists agree with Laland and colleagues that these are important and interesting topics, it has been questioned if these phenomena are really the game changers they have been portrayed to be, and they do not necessarily require a novel conceptual framework (Welch 2016; Charlesworth et al. 2017; Gupta et al. 2017; Futuyma 2017; Svensson 2018). A major criticism is that these four factors are all possible to incorporate into the current theoretical framework. I will not re-iterate these criticisms in detail here, but briefly discuss why these four factors are not evolutionary processes of the same kind as the evolutionary forces in population genetic theory (Lynch 2007; Svensson and Berger 2019).

I suggest we can view these four factors as either *evolutionary outcomes* or *source laws*, and sometimes both, when there exist feedbacks between evolutionary outcomes and selection (Fig. 11.2). However, I argue that these four factors are not *consequence laws* like the traditional evolutionary processes of genetic drift, mutation, recombination and selection (Sober 1984; Endler and McLellan 1988) (Fig. 11.2). Here, I define source laws, following the definitions by Sober (1984), as the underlying causes of fitness differences, selection and mutation rates etc. Examples of source laws are temperature, radiation, predation, climate and most aspects of the external or internal abiotic or biotic environment organisms experience (Fig. 11.2). The source laws influence the consequence laws, which directly change the heritable composition of populations. Source laws, therefore, only indirectly influence the heritable composition of populations, but they are important as they are the ultimate factors causing fitness differences between phenotypes or genotypes (Sober 1984). Source laws therefore arise from ecological and physical conditions, morphology and physiology of organisms, whereas consequence laws are thus the evolutionary effects of these fitness differences (Sober 1984; Endler and McLellan 1988). Population genetic theory is a theoretical framework mainly focused on evolutionary forces, such as the consequence laws of selection, drift, mutation and

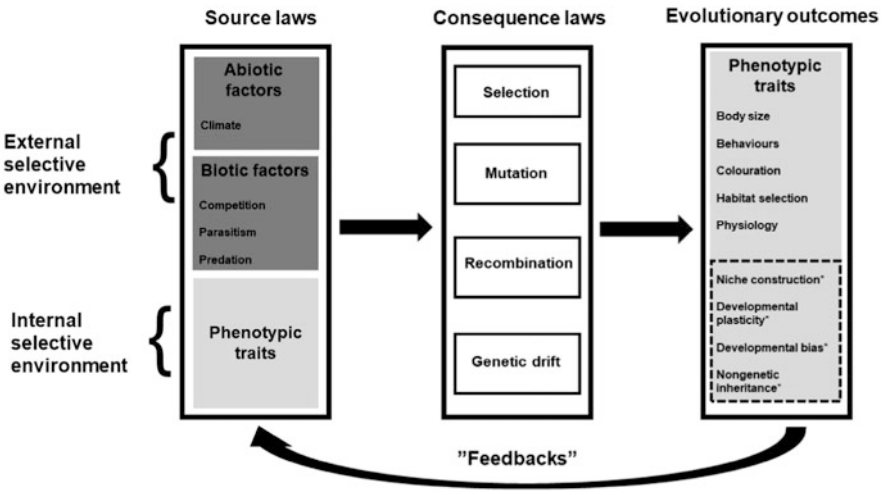

Fig. 11.2 Source laws, consequence laws, evolutionary outcomes and feedbacks. Source laws refer to the factors (intrinsic or extrinsic) behind the consequence laws (e.g. natural selection). Consequence laws have been a major focus of population genetics and evolutionary biology (Sober 1984; Endler and McLellan 1988). Extrinsic source laws are the abiotic (e.g. climate) and biotic factors (e.g. predation or competition) that generate selection pressures and are thus agents or causes of selection (Wade and Kalisz 1990). Source laws could also be phenotypic traits themselves that have ecological consequences (i.e., intrinsic factors). For instance, body size has cascading ecological consequences in terms of population size, starvation endurance and thermoregulation, and could therefore lead to novel selection pressures. Evolutionary outcomes are the products of the consequence laws, and such outcomes are adaptations as are the various phenotypic traits that are shaped by selection, drift and the other consequence laws. A special class of evolutionary outcomes is the four factors highlighted by the EES: developmental bias, plasticity, niche construction and non-genetic inheritance (highlighted with "*" within another box with dashed line). These evolutionary outcomes (but also other phenotypic traits) can feed back and generate novel selection pressures on organisms. That is, an evolutionary outcome of selection can subsequently also become a source law, through feedbacks and lead to reciprocal causation between selection and its products (Svensson 2018). For instance, the beaver dam is an evolutionary outcome or "extended phenotype" that changes the selective environment and influencing selection back on the beaver (Odling-Smee et al. 2003)

recombination and how these consequence laws change the heritable compositions of populations (Sober 1984). In contrast, the source laws deal with how variation in fitness arises and how fitness-trait covariance relationships change due to changes in the biotic and abiotic environment (Endler and McLellan 1988; Wade and Kalisz 1990). Source laws are typically studied within the domain of ecology, rather than belonging to population genetics (Brandon 1990; Wade and Kalisz 1990; Svensson and Sinervo 2000; Siepielski et al. 2017). Needless to say: a full understanding of evolution will require a deep understanding of both source laws and consequence laws, i.e. both of the ecological agents of selection and the evolutionary changes that follow from how selection and the other evolutionary forces operate on populations (Endler and McLellan 1988; Wade and Kalisz 1990).

The four factors highlighted by Laland and colleagues can therefore be viewed as source laws that influence the strength, direction or mode of natural selection (Fig. 11.2). For instance, phenotypic plasticity and various forms of habitat selection of organisms can counteract natural selection, as exemplified by adaptive thermoregulatory behaviours in reptiles and the so-called "Bogert effect" (Huey et al. 2003). In addition, but not mutually exclusive, these four factors can also be viewed as evolutionary outcomes or products of selection (Fig. 11.2). Under this alternative perspective, these four factors are adaptive traits that are shaped by current and past natural selection, but such traits can also shape future evolution on themselves. For instance, there exists a well-developed quantitative genetic theory of the evolution of phenotypic plasticity and reaction norms (Lande 2009; Chevin et al. 2010; Chevin and Lande 2011) that has also inspired empirical research in natural populations (Svensson et al. 2020). Under this view, phenotypic plastic traits are modelled and conceptualized as *function-valued traits* (Kingsolver et al. 2001), where trait values are not fixed but change with the environment (Stinchcombe and Kirkpatrick 2012). In this framework, reaction norms are viewed as composite phenotypes, and their intercepts and slopes can be treated as traits that are targets of selection (Lande 2009; Chevin et al. 2010; Svensson et al. 2020). The highly successful quantitative genetic research programme on phenotypic plasticity therefore partly contradicts the claims by Laland and colleagues that phenotypic plasticity is neglected in contemporary evolutionary biology. On the contrary, phenotypic plasticity has been a major research theme for decades, starting already in the 1980s (Via and Lande 1985).

Similarly, the argument that contemporary evolutionary biology neglects developmental bias and naively assumes isotropic variation, i.e. lack of correlations between traits (Gould 2002; Pigliucci 2019) is obviously incorrect (Fig. 11.1; see also Sect. 11.3 for more detailed critique). As a counter argument to this claim, one can point to an extensive body of population and quantitative genetic research exploring mutational pleiotropy (Lande 1980), correlational selection and its consequences for genetic correlations (Cheverud 1984; Phillips and Arnold 1989; Sinervo and Svensson 2002; Svensson et al. 2021) and the evolution of genetic covariance structures in general (Steppan et al. 2002). The term developmental bias does also have some inherent problems as development will nearly always be non-isotropic (Salazar-Ciudad 2021). In a quantitative genetic context, developmental bias may not even be meaningful or informative, as it adds very little to our current understanding (Svensson and Berger 2019). Insightful quantitative geneticists pointed out several decades ago that genetic variances and covariances estimated at the population level do not only reflect genetics alone, but also epigenetic and developmental effects as well as revealing the history of past ecology and selection (Cheverud 1984). Interest in developmental bias has its intellectual roots in structural explanations of animal form, based on physical principles, development and ideas about self-organization, as exemplified in the work by the pioneering work by D'Arcy Thompson book *"On growth and form"* (Thompson 2014), in the anti-selectionist views expressed by Goodwin in *"How the leopard changed its spots"* (Goodwin 2001), Lima-De-Faria in *"Evolution without selection"* (Lima-De-Faria 1990) and in Rupert Sheldrake's ideas about "morphogenetic fields" (Sheldrake

1995). The ideas in these and similar books are popular outside evolutionary biology circles but are based on misunderstandings and are sometimes grounded in metaphysical arguments. It is a common misunderstanding by these and other anti-selectionists that the physical principles behind morphological development contradict or can replace adaptive explanations of traits based on natural selection. Indeed, structuralists and other critics have failed to understand the crucial distinction between proximate explanations of phenotype formation and ultimate explanations for the evolution of adaptive complexity, as originally explained by Mayr (1961). Mayr's key insight was that proximate and ultimate causes were conceptually different but complementary questions, rather than being mutually exclusive. Mayr's distinction firmly established evolutionary biology as a legitimate research field, independent of functional biology, developmental biology and molecular biology (Dickins and Barton 2013; Conley 2019; Svensson 2020). Some advocates of the EES have questioned the proximate-ultimate distinction as a valid explanatory framework in evolutionary biology (Laland et al. 2011), but this has understandably encountered strong resistance from those who insist that this is still a useful conceptualization (Dickins and Barton 2013; Conley 2019). In contemporary evolutionary biology, internal factors like developmental bias (or developmental constraints) serve as a dispositional property of populations alongside with other dispositional factors like evolvability (Love 2003). Dispositional factors set the outer limits of the space within which selection operates (Maynard Smith et al. 1985). Viewed this way, developmental bias can also interact with selection to influence evolutionary trajectories (Schluter 1996). But developmental bias or developmental constraints, whether arising from principles of physics, genetics or development, is not an evolutionary force that can change the heritable composition of populations by itself (Maynard Smith et al. 1985), unlike the consequence laws of selection, drift, mutation and recombination (Sober 1984). Developmental bias is sometimes put on an equal footing and portrayed as an alternative to evolution by natural selection in explaining adaptive radiations (Brakefield 2006), but this is misleading. Developmental bias is not an evolutionary process that operates in isolation but rather this dispositional factor *interacts* with natural selection (Maynard Smith et al. 1985). For instance, genetic covariances can bias the evolutionary trajectory of a populations and delay the time until it reaches an adaptive peak (Schluter 1996) (Fig. 11.1a, c). However, in this scenario it is selection that drives the evolutionary change, not developmental bias or genetic covariances, which are not evolutionary forces, following Sober's definition (Sober 1984) (Fig. 11.2).

The third factor highlighted by Laland and colleagues is niche construction (Laland et al. 2015). This is the phenomenon by which organisms modify their local selective environments, such as earthworms modifying the surrounding soil structure or the classic example of the beaver building its dam (Odling-Smee et al. 2003). Odling-Smee et al. (2003) argued that such niche construction is a neglected evolutionary process and claimed that it deserved status as an alternative evolutionary route to organismal adaptation, on equal footing and as important as natural selection. While few evolutionary biologists would deny that organisms modify their local environments and many times in an adaptive fashion, only a minority view

such niche construction as an evolutionary process of equal importance as natural selection. Accordingly, the claim that niche construction is neglected has been questioned, and it has been pointed out that niche construction is neither neglected nor is it an evolutionary process (Dawkins 2004; Brodie 2005; Scott-Phillips et al. 2014; Gupta et al. 2017; Svensson 2018). Another frequent criticism is that niche construction is too broad a term that encompasses too many phenomena, including both adaptive modifications by organisms such as the beaver dam, but also non-adaptive effects, such as the creation of toxic waste products under crowded conditions (Dawkins 2004; Gupta et al. 2017; Svensson 2020). That organisms modify their selective environments and that they therefore are active evolutionary agents and not solely passive objects of selection is interesting, but this has also been recognized by many other evolutionary biologists outside the core niche construction literature (Levins and Lewontin 1985; Huey et al. 2003; Brodie 2005; Edelaar et al. 2008; Edelaar and Bolnick 2012, 2019; Svensson 2018). Niche construction is probably best viewed as a healthy reminder about the ecological context of evolution (Dickins 2020) and that organisms partly shape the adaptive landscape and the selection pressures they experience (Huey et al. 2003; Tanaka et al. 2020). Niche construction also reminds us that both source laws such as the ecological causes of selection (Endler and McLellan 1988; Wade and Kalisz 1990) and the consequence laws of population genetics (Sober 1984) are equally important parts of evolutionary research.

From an empirical viewpoint, niche construction could be incorporated as phenotypic intermediate traits in causal graphs (Fig. 11.3). Traits can influence fitness both directly by being targets of selection, but also indirectly, by influencing other traits (i.e. niche construction activities) (Otsuka 2019) (Fig. 11.3). Niche construction can therefore readily be incorporated in the contemporary theoretical evolutionary framework (Otsuka 2019). Powerful tools in the form of causal graphs, path analysis and structural equation modelling have been available for a long time, whereby information about both traits and selective environments can be incorporated in the same analysis (Kingsolver and Schemske 1991; Svensson et al. 2002; Morrissey 2014; Otsuka 2019). I suspect, however, that advocates of niche construction theory will not be entirely satisfied with these pragmatic empirical solutions to incorporate niche construction into evolutionary research.

Finally, the fourth factor highlighted by Laland and colleagues is non-genetic inheritance, sometimes called extra-genetic inheritance or extended inheritance (Laland et al. 2015; Bonduriansky and Day 2018). This includes a broad range of inheritance channels outside DNA, such as various forms of transgenerational epigenetic inheritance (e.g. methylation and histone modifications), social learning, maternal effects etc. (Bonduriansky and Day 2018). This rapidly developing field cannot be covered in full detail here (see Bonduriansky and Day (2018) for an excellent overview). Opinions about non-genetic inheritance range from it being viewed a major game changer that will require a substantial revision of evolutionary theory and an abandonment of the MS (Jablonka and Lamb 2005, 2007; Jablonka 2017) to those who consider it as an "add-on" that can easily be incorporated in the existing evolutionary framework as a proximate mechanism (Dickins and Rahman

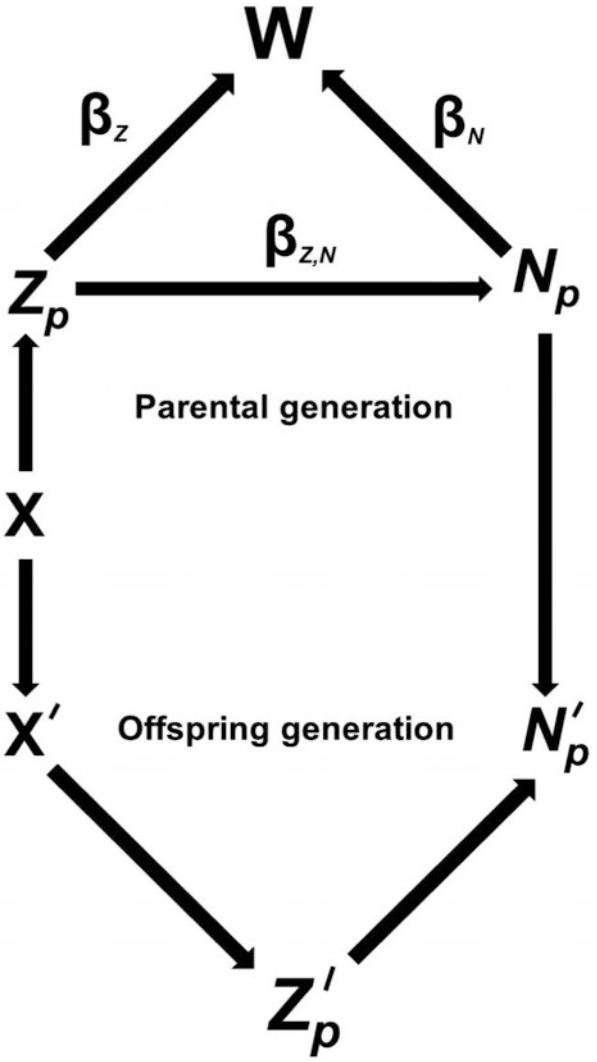

Fig. 11.3 Causal model and path diagram of the relationship between a standard phenotypic trait (Z_p: parental generation; Z_p: offspring generation), a niche construction phenotype (N_p: parent generation; $N_{p'}$: offspring generation), fitness (W) and genetic inheritance (X: parental generation genotype; X′: offspring generation genotype). Direction of arrows denote causal relationships. The phenotypic trait influences parental fitness directly (β_Z: direct selection gradient), but also indirectly, through the niche construction trait that subsequently influences fitness (i.e. the pathway $\beta_{Z,N} \times \beta_N$). In this example, the niche construction trait is not under direct genetic inheritance, although indirectly, through the genetic basis of Z_p. However, note that there is a pathway of non-genetic inheritance of the niche construction trait, since it influences the offspring environment in the next generation ("ecological inheritance"), e.g. the case of the beaver dam. Modified from Otsuka (2019)

2012), or viewed as an evolutionary outcome of selection (Loison 2018). It is important to emphasize that the analytical framework of population genetics can be readily modified to model and analyze selection on other heritable units than genetic alleles, including epialleles (Lu and Bourrat 2018). The quantitative genetic approach in the Price Equation can statistically capture effects of non-genetic inheritance on the resemblance between relatives (Frank 1995, 1997; Rice 2004) and can also be generalized to other inheritance systems (Luque 2017; Luque and Baravalle 2021). One strength of quantitative genetics is that is agnostic with respect to the heritable basis of traits (i.e. DNA vs. other mechanisms of inheritance) as it ignores genetic details (Steppan et al. 2002; Queller 2017). But it is worth

emphasizing that also the theoretical machinery of population genetics originated well before our understanding of the structure of DNA (Charlesworth et al. 2017), meaning that the population genetic analytical framework can be applied to non-genetic inheritance through other heritable channels, including epialleles (Lu and Bourrat 2018).

Somewhat paradoxically, therefore, the deliberate neglect of mechanisms and details in the quantitative genetics also makes it extremely powerful and flexible (Steppan et al. 2002; Queller 2017). However, this point does not seem to have been fully appreciated by all advocates of the EES. Proponents of the EES frequently portray contemporary evolutionary biology as being caught in a narrow tradition of one- or two-locus models of population genetics where the environment is deliberately excluded (Laland et al. 2015). This narrow portrayal of contemporary evolutionary biology ignores the many post-Synthesis developments and the central role quantitative genetics theory and empirical tools have played in evolutionary research. Work on phenotypic plasticity (Lande 2009; Chevin et al. 2010; Chevin and Lande 2011; Svensson et al. 2020) and the evolutionary consequences of variation in social environments and Indirect Genetic Effects (IGE:s) (Bailey et al. 2018) exemplify such post-Synthesis quantitative genetic research. Quantitative genetics theory and tools are therefore extremely flexible and versatile and can be fruitfully adjusted to study many of the problems that EES advocates have highlighted. Other examples of such research is the relationship between non-genetic and genetic inheritance (Greenspoon and Spencer 2018; Rajon and Charlat 2019), how trait interactions and intermediate traits such as niche construction can affect fitness (Morrissey 2014; Otsuka 2019) and how feedbacks from social or non-social environments jointly shape evolutionary dynamics (Hendry 2016; Bailey et al. 2018; Svensson 2018).

11.9 Where Are We?

Given the frequent calls for an expansion or extension of evolutionary theory (Table 11.1) and recent strong claims that the current evolutionary framework is incomplete, it might be worthwhile to step back a little and ask the same question as Ernst Mayr asked on Darwin Centennial Celebration in 1959 (Mayr 1959): *"Where are we?"*. In this chapter, I have critically reviewed the various attempts aiming to replace or extend the current evolutionary framework and the MS, which is claimed to still hold a strong influence on contemporary evolutionary biology (Table 5.1). My overview suggests that some of the more radical critics have failed to convince the majority of biologists that evolutionary theory is in crisis (Dupre 2012) and that the field is therefore is in need for major reform, even replacement (Shapiro 2011; Noble 2013, 2015, 2017; Müller 2017). As I have argued here, these claims paint a misleading picture of the current state of evolutionary biology and have grossly overstated the historical legacy and lasting influence of the MS. These critics have failed to appreciate the substantial changes to evolutionary biology that took place long after the MS was finished, such as the incorporation of the Neutral Theory of

Molecular Evolution (Kimura 1983; Kern and Hahn 2018; Jensen et al. 2019) and the growth and development of evolutionary quantitative genetics as a tool to study phenotypic evolution over both micro- and macroevolutionary time scales (Arnold 2014). The TWE project, in particular, has produced very little constructive contributions to the development of current evolutionary biology research. I anticipate that the TWE will continue to be a fringe movement outside mainstream evolutionary biology, for good reasons. TWE proponents have promoted a highly biased and mischaracterized view of the MS that is far from historical reality and does not paint a fair view of the richness and synthetic ambition of this historically important attempt to unify biology (Reif et al. 2000; Cain 2009; Lamm 2018). I fully agree with Cain (2009) who argues that we should stop talking about the MS as if it is equivalent to contemporary evolutionary biology, and instead view it as a historical period that is now firmly behind us.

Other critics like the EES proponents are more modest in their ambitions to push for conceptual change in evolutionary biology (Table 5.1) (Laland et al. 2015). The phenomena the EES proponents highlight are clearly worthy of study, although not necessarily the game changers they are sometimes portrayed to be. These phenomena are compatible and possible to study within the current flexible and pluralistic evolutionary research framework. I anticipate that evolutionary quantitative genetics will grow in popularity and importance and will become increasingly and flexibly applied to phenomena like non-genetic inheritance, niche construction, phenotypic plasticity and developmental bias, often in combination with other tools like causal graphs and path analysis (Otsuka 2019).

The diverse and somewhat split conceptual landscape of contemporary evolutionary biology today can be described as a series of partly overlapping research frameworks that coexist stably, namely neutralism, mutationism, selectionism and adaptationism (Fig. 5.4). These research currents and traditions are mainly focused on microevolutionary processes within and between populations but have not yet fully entered the macroevolutionary domain (Fig. 11.4). However, neutralist and selectionist perspectives are not restricted to population and quantitative genetics, but can be applied also to higher-level units as species, e.g. in evolutionary community ecology (Vellend 2016) and in ideas about species selection and random drift in macroevolution (Rabosky and Mccune 2010; Chevin 2016) (Fig. 5.4). Similarly to macroevolution, "Evo Devo" and other structuralist perspectives and research traditions, are still somewhat isolated from these four traditional research currents (Fig. 5.4). Evo Devo should probably be located close to mutationism, since this field is focused on questions about the origin of novel heritable variation (Fig. 5.4).

Researchers within each of these different traditional domains have partly different interests, and emphasize different evolutionary processes, namely genetic drift, mutation and selection (Fig. 11.4). The difference between selectionism and adaptationism might not be immediately obvious, but briefly, selectionists are mainly interested in *evolution as a process* and are following the tradition by Lande and Arnold (1983), whereas adaptationists are more interested *in adaptation as a state*, as exemplified by the work by Gardner, Grafen in the "Formal Darwinism Project" (Grafen 1988, 2014; Gardner 2017). Adaptationists like Grafen and

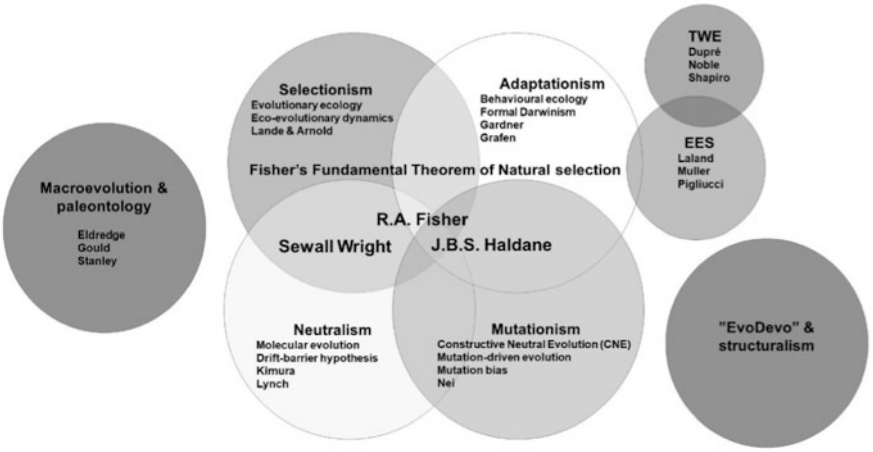

Fig. 11.4 The split conceptual landscape of contemporary evolutionary biology. Microevolutionary research can be classified into four different currents or analytical traditions shown in spheres: adaptationism, selectionism, neutralism and mutationism. These different currents are broad categorizations of different analytical frameworks and they are not completely separated, as indicated by the overlap between them. The three founders of mathematical population genetics (R. A. Fisher, J. B. S. Haldane and Sewall Wright) and their approximate positions are indicated. Fisher emphasized more strongly selection in large panmictic populations and adaptation of organisms, hence he overlap adaptationism and selectionism. Sewall Wright emphasized the interaction between neutral processes such as genetic drift and selection in subdivided population, hence he overlaps between selectionism and neutralism. Haldane was interested in the role of mutation in the evolutionary process and hence he can partly be put in the mutationist sphere. The "Formal Darwinism" school and "Neo-Paleyan" biologists like Grafen and Gardner is mainly a school found in the UK with a strong emphasis on adaptation as a state, rather than the evolution as a process. In contrast, the selectionist school is stronger in North America, and is more focused on the evolutionary process and is represented as the "Lande & Arnold"-school of measuring selection in natural populations. Neutralism is represented by Lynch and Kimura, whereas mutationism is represented by Nei. Finally, the two main challengers of the current evolutionary framework (the EES and TWE) are probably closer to the adaptationism current than any of the other schools. Macroevolution, paleontology and "Evo Devo" are still largely separated research domains from these microevolutionary currents, although their relative positions in the conceptual landscape are indicated

Gardner are more interested in organismal adaptive design and the products of evolution than in the evolutionary process behind such adaptations. This adaptationist current has been labelled "Neo-Paleyan biology", by the philosopher Tim Lewens and it is especially strong in the UK (Lewens 2019a). "Neo-Paleyan" refers to the Christian William Paley and other natural theologians in the UK prior to Darwin-era. Paley was made famous by Richard Dawkins in his popular science book *"The Blind Watchmaker"* (Dawkins 1986). The difference between adaptationism, selectionism, neutralism and mutationism illustrate the diversity of coexisting analytical perspectives in contemporary evolutionary biology. This diversity within contemporary evolutionary biology research contradicts characterizations of evolutionary biology as only allowing a single Neo-Darwinian perspective (Shapiro 2011; Noble

2013, 2015, 2017). The future will tell if and how the EES and the TWE will become integrated with one or several of these existing research currents. It seems to me that proponents of the EES and TWE are mainly focused on adaptationism but have less to say about evolutionary processes, and neither have they identified any convincing novel evolutionary process. These critics of contemporary evolutionary biology might therefore have more in common with the Formal Darwinists than they are willing to admit themselves (Fig. 11.4).

11.10 Looking Forward

Evolutionary biology is currently experiencing an exciting period with increasing amounts of large-scale genomic and phenotypic data and increased integration between neontological and paleontological approaches (Losos et al. 2013). Much of the current dramatic transformation of evolutionary biology is data-driven, whereas the basic theoretical and conceptual framework was established several decades ago, before, during and after the MS. For instance, adaptive landscape theory remains as a central organizing concept in contemporary evolutionary biology (Arnold et al. 2001; Gavrilets 2004; Svensson and Calsbeek 2012a), even though its theoretical foundations were laid out almost a century ago (Wright 1932). Similarly, quantitative genetics gave us tools like the genetic-variance covariance matrix (**G**) that still holds a central place in evolutionary theory and seems to grow in importance and popularity (Steppan et al. 2002; Queller 2017). Some philosophers and critics of the MS and contemporary evolutionary biology have rather bluntly dismissed both adaptive landscapes and **G**-matrices as inadequate or even misleading (Pigliucci 2006, 2008; Kaplan 2008). However, these critics failed to deliver any constructive alternative analytical approaches to study evolution. Their anticipated coming demise of the adaptive landscape and **G**-matrix evolution have accordingly not been fulfilled, and they clearly underestimated the explanatory power of these tools and approaches (Svensson and Calsbeek 2012b). In contrast to what these philosophers anticipated, adaptive landscape theory and **G**-matrix evolution are likely to remain for many years to come, largely because of the power and flexibility of these tools to link phenotypic patterns with underlying evolutionary processes (Arnold 2005). We see increasing efforts to extend quantitative genetic and population genetic theory and methodology to incorporate non-genetic inheritance, niche construction, phenotypic plasticity and other interesting phenomena that have been highlighted by EES proponents (Laland et al. 2015). These phenomena are increasingly being incorporated in the current research framework as various "add-ons" and refinements of existing theory (Day and Bonduriansky 2011; Bonduriansky et al. 2012; Greenspoon and Spencer 2018; Bonduriansky and Day 2018; Rajon and Charlat 2019). Thus, so far we have seen little of the radical conceptual change of evolutionary biology that some critics have claimed would be necessary (Pigliucci 2007, 2009; Noble 2013, 2015, 2017; Müller 2017). In short: gradual change has taken place and still happens to evolutionary

biology, and there is no sign of major overhaul or any forthcoming paradigm shift at the horizon, contrary to the claims by some critics like Noble and Shapiro.

11.11 Conclusions

In our largely data-driven era, it is important to step back, critically reflect on the historical roots of our field and think about the bigger conceptual picture. Many of the conceptual tools and theories we use in evolutionary biology have their intellectual roots in the MS-period from the last century. This does not mean that we still live in the MS era, despite frequent claims by critics of contemporary evolutionary biology (Noble 2015, 2021; Müller 2017). However, it is probably uncontroversial to state that theory development has not kept up the same pace as empirical developments during recent decades. The recent discussions and calls for various extensions to the current evolutionary research framework are therefore welcome, although my overview here suggests that critics have failed to convince the evolutionary biology research community at large that their proposed additions cannot be handled by the current framework. The challenge from the EES is a valuable reminder that it is not only is the spread of adaptive variants by selection that is interesting and important (as already emphasized in the traditional evolutionary framework), but so is also the origin of heritable variation through developmental mechanisms and plasticity, as well as source laws arising from ecology (Table 11.1; Fig. 11.2) (Endler and McLellan 1988).

A positive development of the recent discussions about the MS, EES and TWE is that the relationship between philosophy and evolutionary biology might become strengthened and re-vitalized. Clearly, philosophy of science has an important role to play in the conceptual and theoretical development of evolutionary biology. Early and foundational work by Elliott Sober clarified the relationship between source laws and consequence laws, and formalized concepts about evolutionary forces (Sober 1984), as did Dan McShea and Robert Brandon in more recent work (McShea and Brandon 2010). Likewise, philosophers like Samir Okasha and Peter Godfrey-Smith clarified issues about origin and consequences of multi-level selection (Okasha 2005; Godfrey-Smith 2009). These philosophers clearly knew both evolutionary biology and the history of the field, which presumably contributed to the success of their work.

Not all philosophy of biology has played that positive role, however, especially as some philosophers have uncritically re-iterated myths about the MS that were initially propagated by Gould and others, but which have already been refuted. I have critically scrutinized some of these myths in this chapter. The long-lasting legacy of Gould and some of his more controversial claims have not always been positive. These claims contributed to establish misunderstandings about the current state of evolutionary biology, one being that the field is in deep crisis (Dupre 2012). To avoid repeating such mistakes, philosophers should communicate and collaborate with both historians of science and evolutionary biologists (and vice versa of course). Otherwise, philosophers risk spending effort on small and insignificant

problems that are of little interest except to other philosophers. Some such problems might even be purely semantic, such as the odd idea that natural selection is not really an evolutionary process but just a statistical outcome of lower-level phenomena (Walsh et al. 2002; Otsuka 2016). Most evolutionary biologists probably consider such questions as rather esoteric and of little interest or relevance to the field. Attempts to re-introduce metaphysics in evolutionary biology (Dupré 2021), for instance, are unlikely to impress the evolutionary biology research community. Those arguing for organismal agency as an evolutionary process (Walsh 2015) but leave out natural selection as the most obvious explanation for apparent purpose or design (Noble 2021) are unlikely to convince the majority of evolutionary biologists. It is worth re-iterating that the only known evolutionary process that can systematically increase organismal adaptation across generations and lead to (apparent) purpose is natural selection (Gardner 2013, 2017), although some critics of contemporary evolutionary biologist insist that adaptation can be decoupled from natural selection (Pigliucci and Kaplan 2000; Kull 2014; Noble 2021). Accepting that natural selection is the only known evolutionary process that can systematically increase organismal fitness and adaptation across generations does not mean that we could not appreciate within-generation phenomena such as phenotypic plasticity, habitat selection and adaptive niche construction (Edelaar and Bolnick 2019). Crucially, however, such within-generation phenomena are not evolutionary processes, but evolutionary outcomes (Gardner 2013; Loison 2018, 2021). Furthermore, accepting natural selection as the only known evolutionary process that can systematically increase organismal adaptation across generations does not mean that we need to uncritically adopt a pan-adaptationist position where all organismal features are seen as highly adaptive, or that we need embrace the Neo-Paleyan adaptationist biology tradition (Lewens 2019a). There is still plenty of room for non-adaptative and maladaptive evolutionary processes like mutation, drift and recombination (Lynch 2007; Svensson and Berger 2019). The important point, however, is that these other evolutionary processes cannot systematically increase organismal fitness and adaptations across generations, like natural selection, although they can of course *decrease* fitness (Lynch et al. 1995; Svensson and Berger 2019). Increased understanding of evolution requires *both* bold new ideas and a deep and nuanced understanding of the rich history of the MS and how contemporary evolutionary biology has advanced over the past century.

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It Is the Endless Forms, Stupid: A Commentary on Svensson

12

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Abstract

In this commentary, I briefly consider the idea raised by Svensson of *source* laws and *consequence* laws in evolutionary biology, and use it to review what we might consider to be the canonical processes of biological evolution, focusing in particular on recombination. I then ponder why we have always seen such a variety of evolutionary explanations competing to explain the biodiverse world around us.

Svensson provides a masterful account of the history of the development of modern evolutionary biology, from the period comprising the Modern Synthesis through to the present day, and what this means for the claims of the Extended Evolutionary Synthesis. His chapter should become required reading for anyone interested in the whole notion of extending evolutionary theory, either as history or as a research project. And whilst I have borrowed his previous usage of Standard Evolutionary Theory (SET) as a compromise for the corpus of contemporary evolutionary theories and tests in my own chapter (Chap. 29), I recognise the limitations of any such formulation. As I hope my own chapter makes clear, I agree wholeheartedly that we do not have a body of inflexible theory, set immovably in stone, but rather an organising intellectual framework, built on the notion of biological evolution as a population genetic process. Within this framework, we see a dynamic collection of theories and observations, continually speaking to, and being challenged by, new explanatory evolutionary hypotheses, accreting knowledge as we go. In this brief commentary, I will consider just two aspects of Svensson's rich and detailed chapter,

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doing but little justice to the breadth of the chapter itself—of which I am in some awe—and the many different aspects of the structure of modern evolutionary biology he addresses. The first is the notion of source laws and consequence laws to help us navigate evolutionary processes, and the second is why we see such a diversity of evolutionary explanations.

Following Sober (1984), Svensson makes the case that understanding the difference between *source* laws and *consequence* laws can help us discriminate the kinds of explanations that we are trying to make, and about what kinds of phenomena. Source laws represent the intrinsic (organismal) and extrinsic (ecological) contexts in which organisms live, which determine how the consequence laws of evolutionary biology play out, shaping the changes in frequencies of DNA sequences in populations across generations that is evolution. These *consequences* are in terms of fitness differences amongst organisms that are non-random with respect to phenotype (i.e. selection), the genetic changes brought about mutation and migration between populations (i.e. gene flow), or the stochastic effects of phenotype-independent fitness differences, known as genetic drift. The four canonical processes of biological evolution are therefore mutation and gene flow, which generate genetic variation in populations, and selection and genetic drift, which sort that variation, non-randomly or randomly with respect to phenotype, respectively. All of these evolutionary consequences are shaped by the context—or source laws—which individuals in a given population experience, moment to moment, both intrinsically in terms of their own development and organismal physiology, and extrinsically in terms of the ecology they experience.

The keen-eyed reader will notice though that I have drawn a couple of small distinctions from Svensson in terms of my canonical processes. The first is the inclusion of gene flow (i.e. the migration of individuals and their genetic material between populations). This is a relatively minor difference between us, as if we just focus on a species with one single (panmictic) population, then gene flow is by definition not possible (and Svensson certainly appreciates the role of gene flow in evolution; see for instance Dudaniec et al. 2022). The second difference concerns the status of recombination. I would argue—using the distinction offered by source laws and consequence laws—that recombination is more of a *source* than a *consequence*. Recombination as a concept captures the ways in which different DNA sequences can be brought together in individuals, creating new genotypes. The first way in which new combinations can be made is via Mendelian independent assortment of chromosomes during meiosis. Importantly, this form of recombination need not lead to a subsequent change in the frequencies of DNA sequences (henceforth alleles) in a population. Indeed, we generally expect or assume a fair meiosis (not that meiosis is always fair, but that is another story, explained by natural selection). However, meiosis and the formation of gametes will in fact more than likely lead to a (very) small and stochastic misrepresentation of the alleles present in the parent, a process perhaps best captured under the consequence law of genetic drift (i.e. meiosis represents a source law, with genetic drift the consequence law in this instance).

The second form of recombination is when crossing-over occurs during meiosis, and homologous chromosomes swap physical material between them. Again though,

this need not involve change in the sequence of the resulting recombinant chromosomes. However, it often will, and in doing so it will be *mutagenic*. After all, translocations are a class of mutation (as are mis-repairs of bases during the resolution of a crossing-over event). As such, the crossing-over is a possible source of mutation, which is the *consequence* of the crossing-over. Thus, recombination certainly plays a hugely important role in shuffling alleles across chromosomes and generating new genotypes, but the evolutionary consequences of this shuffling are played out either as genetic drift or mutation. Longer-term, new allelic combinations are themselves likely to be very important in terms of natural selection, which is of course why sex and recombination is such a major part of our lives (e.g. Otto and Lenormand 2002). Finally, for completeness, not all gametes may have an equal chance of fertilising a gamete of the opposite sexual function, but if this is due to the phenotypic characteristics of either the sperm or the egg, then instead we have natural selection at the gametic level (so-called haploid selection, including intra-ejaculate sperm competition: Immler 2019). In summary, using the framework of source and consequence, I would argue that recombination is best viewed as a source, a way in which the four consequence laws of evolutionary process may play out. This is because, fundamentally, it only changes allele frequencies via mutation or drift, and so is one step removed from these consequences.

Why the diversity of evolutionary theories? The entangled bank is—or at least should be—the classic Darwinian metaphor. Whilst Spencer's "survival of the fittest" remains an impactful phrase, a short-hand for the non-professional, it is the entangled bank, rich and complex with animal and plant life, that best captures Darwin and his world-changing accomplishment. Darwin populated the final paragraph of arguably the most important book ever written with birds singing on the bushes, insects flitting around, and worms burrowing through damp earth. He saw ecology as the *driver* of biodiversity, originating species through the ecological competition for resources that generated natural selection, but also ecology as the *result* of that biodiversity, with ever more niches hammered into the economy of nature, leading to ever more kinds of ecological interactions. And one reason that we have never stopped developing new evolutionary theories is that we have never stopped encountering new kinds of biodiversity, new ways in which DNA and RNA replicators seek to propagate themselves in nature. This means that an evolutionary biologist has to consider the selfish DNA of transposable elements jumping from locus to locus in a genome, through to the rise and fall of countless lineages of life across the hundreds of millions of years of our planet's biological history. There have been endless forms, not just of life, but of ways in which life has struggled for existence, and evolved solutions to that struggle. These myriad solutions have allowed us to write our own narratives, as indeed Svensson hints, often emphasising one aspect or another of this diversity. For example, we can see the world either as a wonder of symbiosis and cooperation, or on the other hand a world dreadful with competition, all red teeth and red claws. We can see stunning adaptations to life and sex all around us, or view life as a long collection of accidental contingencies, with genomes drifting along with our planet through space and time. Many of these

narratives overlap and intersect, but sometimes they jar, and more thinking, broader perspectives, are often needed.

In truth, the sheer exuberance of nature requires many standpoints, and many ways of being an evolutionary biologist. We can dig up fossils, watch young animals learn to be, sequence countless genomes, or dream of impossible fitness landscapes populated with genes in quasi-linkage equilibrium. Any and all of these things can make you an evolutionary biologist. But it does mean that we often end up asking different kinds of questions. As such, what perhaps is extraordinary is not that evolutionary thinking is diverse, with individual biologists focusing on different questions, with different kinds of explanations, amongst their different study systems and across different levels of biological organisation. Rather, it is extraordinary that we have something resembling a coherent organising framework that binds us all together at all, an entangled bank of competing theories, alive and vigorous and changing over time. That framework of course comes from Darwin: descent with modification. Even though it took others to formalise the mechanisms of descent, including what genetic inheritance is and how it works, and even though we still argue over the ways in which modification can arise and the role of adaptive versus non-adaptive modification, it is Darwin's framework—encapsulated for many decades now as a population genetic process—that binds us together.

That diversity of life, and that diversity of evolutionary mechanisms by which populations evolve and diverge, is both our blessing and curse. We are blessed with puzzles and conundrums to solve, nuances of nature to argue over, but we are also cursed with the magnitude of what evolutionary biologists are attempting to do. Given the comparative simplicities of physics, chemistry, and molecular biology, evolutionary biologists face a task many orders of magnitude more difficult: we ask why the world is like it is, from individual nucleotides of DNA to the death-throes of the dinosaurs. For many scientists and philosophers outside of evolutionary biology it no doubt seems a hopeless task, and one that risks the storytelling that Svensson notes some critics of evolutionary theory have often emphasised. But your storytelling is my hypothesis making, and I am going to generate evolutionary predictions and test them.

Understanding and explaining biodiversity lie at the heart of evolutionary biology. It is what we teach and research if we are lucky enough to be evolutionary biologists. But perhaps we also need to think harder about the diversity of evolutionary explanations. That diversity is not a weakness of modern evolutionary biology, waiting to be resolved by any new synthesis, let alone the Extended Evolutionary Synthesis. Rather, it is central to understanding life. We have this amazingly simple idea: replicating organisms competing for life. From it has stemmed the organic world, which we cherish and puzzle over. And as with life, so with our necessary theories of life, a diversity of phenotypes begetting a diversity of theories. I suspect that it is no surprise that many of our greatest evolutionary biologists, from Darwin and Wallace onwards, were and are great naturalists, connoisseurs of the entangled bank and the profusion of life. Put simply, you need to see the insects flitting, and hear the birds singing on the bushes. And I too am drawn to the entangled bank, to smell the damp earth.

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Ecology, Agents, and the Causes of Selection: A Reply to Shuker

13

Erik I. Svensson

Abstract

Shuker points to some inconsistencies in my terminology about consequence laws in evolutionary biology, specifically that gene flow and recombination are not as easily classified as one gets the impression of after reading Sober (1984). I agree, and I also emphasize that evolutionary biology should not only focus on consequence laws, but also source laws arising from the ecological selective environments and the agents of selection that arise as a consequence of the organismal-environment interaction.

I thank David Shuker for his appreciative but also critical response to my chapter. I find myself largely in agreement with him about how to characterize the current state of evolutionary biology and the field's history. We are obviously both critical to those who have characterized evolutionary biology as a more monolithic field than it actually is, including frequent claims that it is "gene centric" (Laland et al. 2014, 2015) and therefore more or less indistinguishable from the field of population genetics (Pigliucci 2007). For evolutionary and behavioral ecologists like me, Shuker, and many others who regularly attend international conferences such as those organized by ISBE (*International Society for Behavioral Ecology*) and ESEB (*European Society for Evolutionary Biology*), such claims do not paint a fair picture of our complex, diverse, and sometimes even chaotic field. Evolutionary and behavioral ecologists like me and Shuker are mainly motivated by our interests in phenotypes, including behavior, morphology, and physiology, rather than single genes or DNA sequences. It is for exactly this reason that Shuker, I and many other

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evolutionary ecologists, behavioral ecologists and whole-organism biologists feel somewhat alienated by some of the criticisms from those who argue for an "Extended Evolutionary Synthesis," or EES. Specifically, neither their historical narratives nor their description of the current state of evolutionary biology capture the complexity and intellectual richness of our diverse research field (see Smocovitis and Svensson's chapters in this volume for in-depth discussion and criticisms of these simplified historical narratives).

However, in one sense I do think both Shuker and I should be somewhat sympathetic to some of the reformers, including those calling for an EES, even if we disagree with their historical narrative and their rather one-sided characterization of our field. They deserve our sympathy since some of these reformers call for an appreciation of organismal biology and the study of phenotypes in this heavily molecular era that is dominated by genomics and bioinformatics. It is true that evolution often can and is often characterized in population genetic terms, but evolutionary biology is certainly more than population genetics. Although I much appreciate the sharp and critical mindset of Michael Lynch (one of my favorite population geneticists) and Neutral Theory, I disagree with Lynch's claim that *"Nothing in evolutionary biology makes sense except in the light of population genetics"* (Lynch 2007). Evolutionary biology is so much more than population genetics. It is therefore somewhat ironic that both Lynch and Pigliucci—albeit from radically different perspectives—characterize the core of evolutionary biology solely in terms of population genetics (Lynch 2007; Pigliucci 2007)! If the EES debate has brought anything long-lasting and useful, it is this: the study of phenotypes and phenotypic evolution deserves more appreciation and a more central position in our field.

One of my general messages that Shuker largely seems to agree with is this: it is not only the consequence laws of population genetics (genetic drift, mutation, recombination, and selection) that is interesting and important to study, but also the source laws arising from the ecological conditions that organism's encounter that form their "struggle for existence" (Walsh 2015) that Darwin so poetically described in 1859 (Darwin 1859). The importance of ecology, agents, and causes of selection needs to be emphasized repeatedly in our field, since evolutionary biology is not—and should not—only be a field focused on DNA sequence evolution and population genetics. These points have been made many times before by several prominent evolutionary biologists and ecologists (Antonovics 1987; Endler and McLellan 1988; Wade and Kalisz 1990) but need to be re-iterated. In short, evolutionary biology is both about consequence laws *and* source laws, as they were originally defined by Elliott Sober (1984).

Finally, Shuker points out—correctly—that my list of consequence laws might be incomplete and should maybe also have included gene flow, whereas recombination could sometimes instead be viewed as a source law, given that it acts as a selection pressure behind, e.g., the evolution of sex and provides input in the form of novel genetic variation, alongside mutation. Again, I largely agree with Shuker. Thus, the distinction between what constitutes a source law and what constitutes a consequence law is not always as straightforward as implied by one reading of Sober

(1984). Nevertheless, the roles of recombination and gene flow as either source laws or consequence laws (or maybe a bit both?) clearly deserves to be discussed. How we view these processes also depend on whether we are focusing on a single population or a set of connected populations (as Shuker points out). In the latter case, gene flow should probably be included among the consequence laws.

To add additional complexity, other population genetic processes such as assortative mating might be important in evolution, but they do not change allele frequencies, only genotype frequencies (Sober 1993; Kirkpatrick and Nuismer 2004; Otto et al. 2008). Is assortative mating an evolutionary process, a source law, or a consequence law? This largely depends on how we define evolution (changes in allele frequencies, genotype frequencies or something different). This is clearly beyond the scope of this reply, but these and other conceptual problems in our field will keep philosophers of biology and evolutionary biologists busy for many years to come (Sober 1993). We should all be lucky and feel privileged to be part of such an intellectually exciting and conceptually rich field so that these questions remain to be discussed.

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Part V

Hypertextuality of a Hyperextended Synthesis: On the Interpretation of Theories by Means of Selective Quotation

14

David Haig

Abstract

Since classical times, the concept of final cause or of *telos* has had two aspects: the goal toward which something proceeds; and the thing's purpose or what it is good for. Darwin explained the origin of purposes by a process without a goal. Organisms have become adapted to their environments by natural selection of undirected (random) variation. Adaptationists are principally interested in understanding the purposiveness of living things. The relation of the Extended Evolutionary Synthesis (EES) to teleological reasoning is complex. On the one hand, its proponents downplay the importance of adaptation by natural selection and of teleological explanation in terms of purpose or function. On the other hand, they favor a more teleological evolutionary theory in which variation is not random but directed or biased toward what is needed.

Keywords

Teleophobia · Teleology · Information · Meaning · Difference · Purpose · Chicken and egg · Phenotype · Genotype

14.1 Introduction

> Or we may say: this egg is older than that fowl (the fowl having been produced from it); and on the contrary; this fowl existed before that egg (which she has laid). And this is the round that makes the race of the common fowl eternal; now pullet, now egg, the series is continued in perpetuity; from frail and perishing individuals an immortal species engendered. . . . And

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> whether we say, or do not say, that the vital principle (anima) inheres in the egg, it still plainly appears, from the circuit indicated, that there must be some principle influencing this revolution from the fowl to the egg and from the egg back to the fowl, which gives them perpetuity. (William Harvey 1651/1847: 285)

A specter haunts biology—the specter of final causes. Fear of this specter—teleophobia—stifles speech. Thou shalt not ask why. We wander without direction. Our time is out of joint. We hear strident calls for revolution and a return to past certainties. Partisans man the barricades. The forces of reaction mobilize to crush dissent. Things fall apart. The center cannot hold. The best lack all conviction, while the worst are full of passionate intensity. Is something rotten in the state of Darwinism?

From Darwin to Derrida (Haig 2020) is an extended argument about the origins of meaning and purpose. It is a defense of the use of teleological language in biology and an attempted reconciliation of biology and the humanities. Central to its argument is the concept of an interpreter, an entity that has evolved or been designed to use information in choice. The action chosen is the *meaning* of the information for the interpreter. This is a definition, not a discovery. *Texts* are a subset of meanings: interpretations *intended* to be interpreted; outputs chosen to be *used* as inputs to future interpretations. Other terms, from *environment* to *phenotype* to *gene*, are also given non-standard definitions. The book's readers will judge whether this reconfiguration of terms is a hyperextended synthesis that stretches common sense beyond its limits, causing injury and pain, or a chiropractic adjustment that restores flexibility to an immobile joint.

For a man with a hammer, every problem looks like a nail. For a peasant with a sickle, every problem looks like a head of wheat. My favorite hammer fits snugly in my hand: effects can be causes of causes when causes recur. Two truths universally acknowledged are that one should not confuse cause and effect and that a cause cannot come after its effect. These truths justify the rejection of final causes: the end (*telos*) of a process is an effect of the process and cannot be its cause. These truths deceive. Teleological language is justified in biology because natural selection confounds common understandings of cause and effect.

Exclusivity of cause and effect is unproblematic in statements about individual things: a particular egg is either laid by a particular chicken (the chicken is a cause of the egg) or develops into that chicken (the egg is a cause of the chicken), but it cannot be both (for completeness, one should add that an egg is neither cause nor effect of an unrelated chicken). However, what is true of relations among individual things (token-causation) need not be true of relations among kinds of things (type-causation), especially when kinds are related by reproductive recursion. An egg is both an effect of a chicken-that-was and a cause of a chicken-to-be. Eggs (considered as kinds) are both causes and effects of chickens (considered as kinds). Scientific laws are statements about kinds, meant to have general validity.

The hammer strikes. What is true of chickens and eggs is true of genotypes and phenotypes. Genotypes determine phenotypes by developmental processes.

Phenotypes determine genotypes via natural selection. A protein is a cause of its DNA sequence, the central dogma of molecular biology notwithstanding.

The hammer strikes again:

> Mechanistic and adaptive explanations adopt different concepts of the "causal" relation between genotype and phenotype. The mechanistic concept is the familiar view that genotype determines phenotype. . . . An organism's final form is seen as the outcome of a chain, or cascade, of molecular events. The second causal concept is that of natural selection. From this perspective, phenotype determines genotype. If one is to understand why particular DNA sequences, and not others, occur in modern populations, the answers will be found in the historical interaction of organisms with their environment (Haig 1992).

and again:

> Sequences that promote their own replication will be perpetuated, whereas sequences that are less effective replicators will be eliminated. The effects of a sequence may thus be included among the causal factors that account for the presence of the sequence in a gene pool. It is this causal feedback between genotype and phenotype — when combined with a source of genetic novelty (mutation)—that explains how a purposeless process (natural selection) can produce purposeful structures and functions (adaptation) (Haig and Trivers 1995).

Past phenotypes explain present genotypes. Effects reinforce their causes. Genes exist *for the sake of* their functions. Adaptations are final causes. The differential beating of hearts has shaped cardiac development via rhythmic effects on differential survival of bodies and replication of genes. Projecting nails can cause injury if they are not hammered home.

14.2 Engaging the Extended Evolutionary Synthesis

Many arguments in evolutionary theory are arguments about texts. Beneath the sound and fury, these are commonly semantic arguments about how words *should* be defined because words mean different things to different readers. In the framework of Haig (2020), the meanings of each text *for its interpreters* are simply how each interpreter interprets the text. When two interpreters disagree about what a text means there is no fact of the matter as to which meaning is true. The interpreters simply interpret the text differently. If they communicate their respective interpretations, then these communications are new texts intended to be interpreted by subsequent interpreters who may conclude that one interpretation is more useful or closer to the author's intended interpretation of the text. Such conclusions are themselves interpretations of the originary text. If the author is living, one can ask her about her intended meaning but her answer is simply her interpretation of what her earlier text means. This is the nature of discourse. Interpretations evolve dialectically.

Texts are neither true nor false, but some texts may be more useful than others. Observations of nature and of experiments are used to inform theories and justify

their conclusions. Evolutionary theory is grounded in facts of the world. A consensus of readers may judge that some texts are internally inconsistent or contradicted by facts, but the interpretation of "facts" is rarely theory-free. Such is the fitful nature of theoretical progress.

In recent years, much has been written about the need for an Extended Evolutionary Synthesis (EES) to correct astigmatisms of Standard Evolutionary Theory (SET). And much has been written in defense of the visual acuity of SET. Radical champions of transformative revolution man the barricades against reactionary defenders of piecemeal reform. Both sides have meaningful things to say *in their own terms*, and both sides willfully create straw men to criticize what the other side is trying to say. Neither side speaks with a single voice. The prospects for dialectical consensus are not promising. I have been a partisan in these debates, more reactionary than revolutionary, but want to step back to analyze what the argument is about. In this endeavor, I will interpret the texts of Kevin Laland and co-authors, neglecting other key contributors, in an attempt to identify areas of agreement and key points of friction.

Because of my long-held belief that genotypes cause phenotypes recursively, we seemingly stand on common ground when Laland et al. (2013a) proclaim: "Evolutionary biology would be better served by a concept of reciprocal causation, in which causation is perceived to cycle through biological systems recursively." And I applaud Laland et al.'s (2013b) reply to charges that reciprocal causation creates an explanatory mess:

> We agree that there is infinite regress of interlinked causal influences for any current event—if so inclined, researchers could trace causation back in time all the way to the big bang—but the suggestion that it is "not conducive to successful biological science" is a little overdramatic. The reciprocal causation stance is perfectly operational. It merely places the onus on researchers to make sensible judgments as to how far to trace back causality for the problem in hand.

Reciprocal causation is a leitmotif of the EES: there is a reciprocal relation between evolutionary and developmental processes; organisms are shaped by their environment and shape the environment; phenotypic plasticity has evolved and itself molds the evolutionary process; epigenetic and genetic inheritance modify each other, interchangeably; phenotypic accommodation promotes genetic accommodation reciprocally; and so on. All these strands can be considered different aspects of the reciprocal relation between genotype and phenotype that arises from reproductive recursion. Surely Laland and colleagues would agree that there is a reciprocal relation between adaptive function and evolved mechanism—functions are causes of evolved mechanisms—but this would be an accommodation too far. Laland et al. (2013b) write:

> Yet functions are not causes. Functions are descriptions of what characters are fashioned to do. The bird migrates *in order to* get better access to food or mates, but, as many previous researchers have pointed out, the outcome of a behavior cannot determine its occurrence.

Our disagreement over the causal role of functions is a point of friction that should be acknowledged.

It would be simplest to argue that Laland and colleagues are opposed to arguments from design whereas adaptationists, like myself, are comfortable with ascriptions of function and purpose. I might even accuse Laland and colleagues of teleophobia, but closer reading reveals that we agree on the apparent design of living things. We differ over whether natural selection is its predominant source or just one among a number of other neglected sources:

> It would be a mistake to assume ... that all semblance of design arises solely from natural selection of genetic variation. We foresee many characters that exhibit design features but are not biological adaptations (e.g. spandrels, exaptations, products of cultural evolution, as well as the appearance of design brought about through niche construction). (Laland et al. 2013b)

They accept the "semblance" or "appearance" of design but dissent from a view that design features arise "solely from natural selection of *random* variation." I have deliberately substituted the adjective "random" for "genetic" in this misquotation because I think our fundamental disagreements would remain if the first sentence read "It would be a mistake to assume that all semblance of design arises from natural selection of random variation, whether this variation is genetic, epigenetic, or cultural." The core of our disagreement is over whether natural selection of random variation is sufficient to explain the purposive features of living beings.

Ours is the latest version of an old argument. Many of the concerns, and some of the proposals, of Laland and colleagues are prefigured in Spencer (1893a) *The inadequacy of natural selection* (also see Spencer 1893b), and my adaptationist rejoinders are prefigured in August Weismann (1893) *The all-sufficiency of natural selection*. The semblance of design is undisputed. One has a choice: either one accepts natural selection as an adequate explanation or one seeks additional mechanisms. Defenders of SET believe natural selection is adequate but proponents of EES ascribe some design features to directed or non-random sources of variation, whether this variation is genetic, epigenetic, or cultural.

14.3 Differences that Make a Difference

Concepts of cause are heterogeneous and biologists regularly slip from one concept to another, with frequent ambiguity about which concept they are using in a particular sentence. A previous section emphasized the distinction between causal relations among individual things (token-causation) and causal relations among kinds of things (type-causation). An orthogonal distinction is between causation as mechanism and causation as difference-making. Mechanisms explain how *things* or events cause other things or events. Difference-making explains how *differences* between things or events cause different outcomes. Much of biology is rightly concerned with mechanisms but adaptation by natural selection is concerned with

difference-making. There can be no selection without a difference, just as there can be no choice without an alternative. A more subtle statement would be that selection marks the transition from *a difference* subject to choice to *a thing* that is chosen.

All choices involve choosing among differences *in the context* of things that are the same. I have defined a gene's *phenotype* as all differences it makes in the world relative to an alternative and its *environment* as all things that are the same relative to the alternative (Haig 2012). By these definitions, variation that does not have a genetic cause is part of the environment not of the phenotype. Natural selection involves feedback between two differences: a *genetic difference* that is the cause of a *phenotypic difference* that is the direct target of selection. The first difference is the difference-maker. The second difference is the difference-made. The things that are the same are the *environment* that selects (Haig 2012). Both phenotype and environment are defined relative to an alternative. If the alternative changes, then a different phenotype is under selection in a different selective environment. A former genetic difference that has become fixed in a population is part of the environment that selects among present genetic differences. For each particular genetic difference, other genetic differences are a variable part of its environment, experienced the same by both alternatives over the course of many generations (for a discussion of linkage disequilibrium, see Haig 2020: 83). Choices of nature convert phenotypic differences (that which is selected) into environmental samenesses (that which selects). DNA sequences are the textual record of past selection and an important part of the environment that selects (Haig 2014, 2020). The phenotypic *performance* of the genotypic *text* is judged in environmental *context*.

Organisms modify their environments and choose where to live. In one of the founding texts of niche construction theory, Lewontin (1983) decried the "impoverished view of the relation between gene, environment, and organism" that he saw as endemic to evolutionary theory (with emphasis added):

> What is left out of this adaptive description of organism and environment is the fact, *clear to all natural historians*, that the environments of organisms are made by the organisms themselves as a consequence of their own life activities.

A dialectical relation between organism and environment may be "nothing new" to a natural historian but "revolutionary" to a population geneticist. If your mathematical models of evolutionary change treat the environment as a constant to which gene frequencies respond, then the writing of coupled equations in which gene frequencies respond to the environment and the environment responds to gene frequencies can profoundly change your view of evolutionary dynamics. Whether one views niche construction as radically unsettling or the repackaging of old ideas may depend on one's prior *interpretation* of evolutionary theory. Both beliefs can be honestly held.

A definition of the environment as "*all things that are the same relative to a genetic difference*" has a natural affinity for "niche construction." The constructed niche (the evolved parts of the environment that selects) includes the cells and bodies in which genes reside as well as other parts of the genome that are either invariant or

randomly distributed relative to the genetic difference under consideration. Thus defined, the constructed niche is distinct from an extended phenotype which comprises differences a genetic difference makes in the world (Haig 2017). Perhaps we can agree on this.

14.4 Exaptations, Spandrels, and Constraints

> Although an organ may not have been originally formed for some special purpose, if it now serves for this end, we are justified in saying that it is specially adapted for it. On the same principle, if a man were to make a machine for some special purpose, but were to use old wheels, springs, and pulleys, only slightly altered, the whole machine, with all its parts, might be said to be specially contrived for its present purpose. Thus throughout nature almost every part of each living being has probably served, in a slightly modified condition, for diverse purposes, and has acted in the living machinery of many ancient and distinct specific forms (Darwin 1862: 348).

Darwin described new devices made of old parts as "specially adapted" and "specially contrived" for their present purpose. Gould and Vrba (1982) introduced exaptation as a "missing term in the science of form" to distinguish original function from current utility:

> [We propose that] features that now enhance fitness but were not built by natural selection for their current role ... be called *exaptations* and that adaptation be restricted, as Darwin suggested, to features built by selection for their current role (Gould and Vrba 1982: 4).

Exaptations included features that were previously adaptive but now employed for a new purpose, as well as features that were previously non-adaptive and now coopted for a purpose. What it would take for a feature to qualify as an adaptation rather than an exaptation is not altogether clear if adaptive tinkering is true of "almost every part of each living being." Darwin clearly did not restrict his concept of adaptation to the original function for which old wheels, springs, and pulleys had evolved. Therefore, Gould and Vrba appear to have misrepresented how he defined adaptation.

Spandrels and exaptations have similar connotations. Gould and Lewontin (1979) described spandrels as "necessary architectural byproducts of mounting a dome on rounded arches," and an example of the broader category of "architectural constraints." Gould (1997) later averred that he and Lewontin borrowed the architectural term to "designate the class of forms and spaces that arise as necessary byproducts of another decision in design, and not as adaptations for direct utility in themselves ... features arising as byproducts, rather than adaptations, whatever their subsequent exaptive utility ... the definition of spandrel includes both its origin as a necessary but consequential (and therefore 'nonadaptive') form and its availability for later (or secondarily adaptive), and potentially crucial, use." If spandrels are *necessary byproducts of another decision in design*, then they are an inseparable part of that decision. Should they be described as part of the adaptive feature or as non-adaptive? What non-rhetorical difference does it make?

Structural alternatives to spandrels (more strictly pendentives) existed for the architects of San Marco, but these alternatives had been tried and found wanting for the construction of large domes by earlier architects (Dennett 1995: 268–275; Mark 1996; Houston 2009). If a structure is chosen because alternative structures are more likely to collapse, is the chosen structure an adaptive solution or an architectural constraint? It depends on what you mean by these terms. In the context of evolutionary biology, most "structural constraints" have persisted for long evolutionary periods not because there has been no variation—malformed children are born every day—but because variants have been rapidly eliminated by, what has been called, negative selection. Morphological radiations of finches and honeycreepers on the Galapagos and Hawaiian archipelagos suggest that negative selection against structural variants is relaxed on islands. Among the "developmental constraints" on continents, of the morphological exuberance expressed on islands, has been the existence of other species with more functional morphologies.

Whether or not negative selection is considered part of the adaptative process, is one source of disagreement about the importance of adaptation by natural selection relative to other evolutionary processes. Consider a genetic variant X that undergoes a selective sweep in a population formerly fixed for variant Y. Once X has displaced Y, X is maintained by selection if it has higher fitness than new variants Z that arise by mutation. [1] The selective replacement of Y by X is known as positive selection whereas the failure of Z to replace X is known as negative selection. The functions of X relative to Y are the phenotypic differences X makes relative to Y that account for Y's selective replacement by X and the functions of X relative to Z are the phenotypic differences that X makes relative to Z that account for the selective elimination of Z. Gould and Vrba (1982) consider 'adaptation' to be restricted to functions established by positive selection and interpret negative selection as a 'constraint' on what can evolve. Adaptationists, on the other hand, consider adaptation by natural selection to include both positive and negative selection. This is not a disagreement of fact but of definition. Friction would be reduced, and a joint lubricated, if we recognized our different definitions.

Etymologically, "exaptation" is an old wheel slightly altered and specially contrived to serve a purpose previously served by "adaptation" or "preadaptation" whereas "spandrel" is an architectural term used to represent a non-adaptive "constraint." If all adaptations modify existing structures and are subject to architectural constraints imposed by these existing structures, do the newer terms serve any useful function? Clearly, they do if usefulness is measured by use: Google Scholar retrieved 1390 articles from the year 2019 using the search term "exaptation" and 1070 using the search term "spandrel" (although some of the latter are architectural rather than evolutionary uses). One rhetorical use of *exaptation* and *spandrel* has been to diminish what is ascribed to *adaptation* in polemics against adaptationism.

[1] Some mutations make *no functional difference* and are not subject to selection. I think of them as neutral variants of X.

A major motivation for introducing spandrels and exaptation as new terms in evolutionary biology was to distinguish evolutionary origin from current utility. Gould and Vrba's claim that a trait is an adaptation for its original function, but not its subsequent uses, is similar to the contested claim in the humanities that the meaning of a text is authorial intent, not subsequent interpretations, or the contested claim in legal studies that the meaning of the United States constitution is the original intent of the 'founding fathers' not how courts have subsequently interpreted the document. The problem of determining original intent in evolutionary biology is exacerbated by the lack of founding documents. Every feature is a reinterpretation of older features. Every genetic text is a rewriting of older texts (Haig 2020).

14.5 Purpose, Goal, and Direction

Ever since Aristotle, the notion of *telos* (final cause or end) has encompassed two somewhat different, but related, concepts. One is the usefulness of a thing, the other is the goal toward which a thing moves or develops: driving-in nails are the *telos* of a hammer; a chicken is the *telos* of an egg. The concepts are related because a carpenter has a goal in mind when she purposefully uses a hammer. For Aristotle, *telos* was internal to organisms: the potential chicken within the egg was actualized in development. The living thing held its end within itself (en*tel*echia).

In simplistic terms, Christianity replaced Aristotle's intrinsic *telos* with an extrinsic *telos*. Entelechies yielded to a creator. Thomas of Aquinas offered five arguments for the existence of God of which:

> The fifth way is based on the guidedness of nature. Goal-directed behavior is observed in all bodies obeying natural laws, even when they lack awareness. Their behavior hardly ever varies and practically always turns out well, showing that they truly tend to goals and do not merely hit them by accident. But nothing lacking awareness can tend to a goal except it be directed by someone with awareness and understanding; the arrow, for example, requires an archer. Everything in nature, therefore is directed to its goal by someone with understanding, and this we call God (Aquinas 1989: 13–14).

His was a teleological universe. Obedience to natural laws was evidence of guidedness and hence of a guiding hand. Without what we call God, all would be chaos. The antithesis of a lawful universe of *directed* events was a lawless universe of *random* events. The primary adjectival sense of random in the *Oxford English Dictionary* is "Having no definite aim or purpose; not sent or guided in a particular direction; made, done, occurring, etc., without method or conscious choice; haphazard."

The scientific revolution of the seventeenth century led to a general acceptance among natural philosophers that one could have physical law without a legislator, order without orders, direction without a director. For hard-minded physicalists, this meant the exclusion of final causes from science. Teleology became anathema. For some, God played a role in setting the mechanism in motion but then the universe unfolded without further intrusion. Natural theologians used the appearance of order

and purposefulness in nature, especially in living things, as evidences of a creator. Their argument from design combined both purpose and goal. Organisms possess purposeful parts. Purposes require minds with goals. Therefore, the purposive features of organisms prove the existence of a divine mind who has a goal (including for us).

The exclusion of final causes bore abundant fruit in physical science, but the exquisite order of living things, their goal-directedness, and occasional disobedience did not fit easily within a purposeless but lawful universe. Despite these appearances, many nineteenth-century biologists came to see living things as subject to physical law and therefore bereft of final causes. Vitalists demurred that there was something special about living things, some kind of internal drive or life force. Into this mix was thrown the theory of natural selection. Darwin (1859) gave a naturalistic account of contrivance in nature that dispensed with a mind or a goal. Two major interpretations of Darwin's import for final causes can be distinguished: the first, that natural selection *eliminates* purposes and goals; the second, that natural selection *explains* purposes without pre-existing goals.

14.6 Karl Ernst von Baer and *Zielstrebigskeit* (Goal-Directedness)

The eminent embryologist Karl von Baer addressed purpose (*Zweck*) and goal (*Ziel*) in a paper on paedogenesis (1866: 126). Many natural scientists condemned reference to goals. He felt called upon to justify his preference for teleological language against "this fear in front of purposes, or better goals—this teleophobia, as one might call it" ("*diese Furcht vor Zwecken, oder besser Zielen—diese Teleophobie, wie man sie nennen könnte*"). *Zweck* and *Ziel* could be used as synonyms, but von Baer preferred *Ziel*. He coined another term *Zielstrebigkeit* to designate the goal-directedness of living things. This term possesses strong connotations of inner initiative (German *streben* and English *strive* are orthologous).

That same year, Ernst Haeckel's *Generelle Morphologie der Organismen* presented its author's monistic, materialist synthesis of the science of organic form in which he stridently and uncompromisingly rejected all appeals to final causes in favor of efficient causes alone (Haeckel 1866: 98). Teleology had no place in scientific explanation. He called instead for a science of rudimentary organs, what one might call a theory of inaptitude (*Unzweckmäßigkeitslehre*) or dysteleology (*Dysteleologie*). For Haeckel, Darwin had banished forever the ghost of final causes: "*In Darwin's discovery of natural selection in the struggle for existence, we see the most striking proof of the exclusive validity of mechanically acting causes in the entire field of biology; we see in it the definitive death of all teleological and vitalistic judgments of organisms*" (Haeckel 1866: 100).

von Baer defended his own teleological views in an address delivered in December 1866 in which he derided Haeckel's materialism (*Über den Zweck in den Vorgängen der Natur*, republished in Baer 1876). In this address, von Baer explained his preference for *Ziel* and *Zielstrebigkeit* over *Zweck* and

Zweckmäßigkeit: *Zweck* implied conscious intent, and was properly restricted to human affairs or to nature as a whole (*die Gesammtheit der Natur*) (Baer 1876: 74); *Ziel* was more inclusive than *Zweck* because it did not presuppose consciousness but could include conscious *Zwecke* (Baer 1876: 82). von Baer employed a familiar metaphor for *Zielstrebigkeit*: when I desire to shoot an arrow into a target, the purpose is mine and stays with me, but the arrow moves with the absolute necessity to *its* goal without knowing the purpose (Baer 1876: 86). It is in this sense that the goal of an egg is the development of a new chicken (*Das Ziel des Eies ... ist die Entwickelung eines neuen Hühnchens*) (Baer 1876: 83). Haeckel, not Darwin, was the target of von Baer's opening fusillade. In Haeckel's view, according to von Baer, everything occurred in the world from absolute necessity (*absolute Nothwendigkeit*), leaving no room for chance (*Zufall*), purpose in nature (*Zweck in der Natur*), or free will (*freier Wille*). von Baer found absurd Haeckel's argument that physical necessity was incompatible with purpose and was incredulous at Haeckel's denial of a role for chance (ibid, p. 68ff).

von Baer shifted aim when he turned his rhetorical fire to the Darwinian hypothesis (*Zum Darwins Lehre* in Baer 1876). Haeckel had been targeted, in part, because of his rejection of chance (*Zufall*) but the Darwinian hypothesis, as von Baer had come to understand it, ascribed too great a role to chance. Chance was not creative. It had no goals. It could not create complex things. Darwinists, he argued, wanted to replace explanation by *Ziel* with explanation by *Zufall*. His embryological studies had convinced him of the cardinal importance of *Ziel*. *Zielstrebigkeit* acted from within, like Aristotle's *entelechy* (Baer 1876: 458, including footnote), rather than from without like Darwin's material causes. When he came to address the transmutation of species, von Baer wrote:

> But we must especially fight against Darwin's view of the entire history of organisms only as a result of material effects, and not as a development. It seems to us unmistakable that the gradual formation of organisms into higher forms and finally to man was a development, a progress towards a goal, which one may conceive as more relative than absolute (Baer 1876: 425; translation by Nyhart 1995: 118).

Ay, there is the rub. Few Darwinists, then or now, would dispute that embryonic development is goal-directed, but extrapolation of the goal-directedness of development to goal-directedness of evolutionary change aroused, and continues to arouse, Darwinian teleophobia. For von Baer, organisms had goals but not purposes; the evolutionary process was goal-directed; nature as a whole, not its parts, had a purpose. For Darwin, organisms evolved with goals and purposes, but natural selection had neither goals nor purposes.

Emil Du-Bois Reymond (1876: 23), on the other hand, believed that Darwin had shown how Nature could continually throw doubles even with unloaded dice. In physiology, teleology could be used as a heuristic principle ("*der Teleologie als heuristichen Principes uns zu bedienen*"). Because of the theory of natural selection, the anthropomorphic name *Zweckmäßigkeit* no longer had anything uncanny

(*unheimlich*) about it. He saw no need for exchanging the older term for *Zielstrebigkeit* as suggested by von Baer.

14.7 Directedness

> According to Teleology, each organism is like a rifle bullet fired straight at a mark; according to Darwin, organisms are like grapeshot of which one hits something and the rest fall wide (TH Huxley 1864: 568).

For many of von Baer's contemporaries, the principal explanatory antithesis pitted directed lawfulness (*Zielstrebigkeit*) against random lawlessness (*Zufälligkeit*). When Darwinists derived purposiveness (*Zweckmäßigkeit*) from undirected variation, it was easy to interpret them as claiming that the wonders of the living world had been assembled by chance. The implausibility of the creation of complex beings by repeated throws of dice remains one of the principal arguments of creationists against Darwinists and has been expressed to me privately by eminent biochemists and molecular biologists. Those unconvinced by Darwinism often desire a more *directed* process. But their argument from design is mistaken. Under the Darwinian hypothesis, natural selection, not mutation, is the creator of order and source of organismic direction.

Nineteenth-century diatribes are echoed in twenty-first-century tirades. Proponents of an EES ascribe an evolutionary role to directed variation that adds *zielstrebige* leaven to the *zufällige* dough. Orthodox Darwinists insist that mutational variation is undirected. What would it mean for variation to be *directed*? The noun *direction* comes from the verb *direct* whose oldest sense in the *Oxford English Dictionary* is "To write (something) directly or specially *to* a person, or for his special perusal." The meanings of words evolve. Compare the modern sense of direction in "He proceeded in a northerly direction" with the sense in "She has five students working under her direction." The adjective *directed* can be used in a passive sense of *has a direction* or an instructive sense of *was directed*. There are sufficient degrees of freedom of interpretation for authors to mean different things by "directed variation" and to yell at each other.

Every random variable has a mean and deviations from that mean. When an archer shoots at a target, she does not release the arrow in a random direction but, when she shoots repeatedly at the same target, the scatter of her shots are undirected deviations from her intended mark. If the target is moved, then she changes aim and releases her arrows in a new direction. Her shots will now exhibit random scatter around a new center. If she attempts to hit a moving target, then she must anticipate where the target will be when the arrow arrives. Her shots will show random variation around the spot where she aims but may show systematic deviation from the shifting goal if she poorly anticipates its future positions.

In the standard Darwinian account, archers are products of evolution by natural selection, but natural selection is not an archer. If the adaptive target has been stationary for a prolonged period then most genetic shots will be "on target" with

random scatter due to the slings and arrows of outrageous fortune. The evolutionary history of the mean is non-random but variation around the mean is random. If the target moves, then shots previously on target will miss the mark and mutant shots that "by chance" were closer to the new target will increase in frequency and be surrounded by a new halo of off-target variation. Past changes of utility, as judged by natural selection, determine the non-random location of the mean but mutational variants are random with respect to current or future utility. Natural selection accounts for evolutionary responses that track a shifting target, but most Darwinists would reject suggestions that mutation anticipates the direction of change that is needed. The adaptive fit of organisms to their environments remains on target by negative selection and shifts target by positive selection.

Although most Darwinists reject evolutionary goals, most are comfortable with organismic goals: actions taken *for the sake of an end* (purpose) proceed *toward an end* (goal). The directedness of development is explicable in terms of reproductive recursion. Development toward a goal is purposive because adult forms have transmitted genetic differences responsible for their distinctive development. Differences of development are the phenotypic effects of developmental genes. For each genetic difference, the factors that are the same are its developmental niche. Variation in development is winnowed by the constraints of this developmental niche. Adaptationists are also comfortable with contingent goal-directed behavior, with the twists and turns of a cheetah as it pursues a fleeing gazelle. The same is true of contingent goal-directed development. Adaptationists feel no discomfort when a branch grows toward the light. Adaptive phenotypic plasticity allows adjustment of aim by feedback from targets.

14.8 Directed Variation

In the nineteenth century, developmental and evolutionary change were often viewed as conceptually similar. The word *evolution* could be used for both processes. Indeed, "theory of evolution" was frequently used for the embryological hypothesis of preformation as opposed to epigenesis. Etymologically, *evolution* was an unrolling of what was already there, in the sense of the unrolling of a scroll. The chosen field of embryologists predisposed them to think of development as goal-directed because adult forms were readily conceived as the goals toward which embryos developed. By contrast, naturalists studied the fit of organisms to their natural environments and were predisposed to see adaptive purpose as the fundamental problem. As transmutational ideas gained traction, some embryologists argued that morphological change in developmental time and transmutation in evolutionary time were analogous goal-directed processes. Embryologists and naturalists were predisposed to attach different importance to *Zweck* and *Ziel*.

In the half-century after the *Origin of Species*, many biologists accepted transmutation of species but rejected natural selection in favor of directed mechanisms. Alternative evolutionary theories were modeled on organismal growth and development. Bowler (1979) provides an excellent review of late-nineteenth and early-

twentieth-century theories of directed variation (orthogenesis) including the tension between theories of internal and external direction. Charles Otis Whitman (1906: 44) strove to unify three competing theories of transmutation (with my annotated labels):

> Natural selection [*Zweck*], orthogenesis [*Ziel*], and mutation [*Zufall*] appear to present fundamental contradictions; but I believe that each stands for truth, and that reconciliation is not distant.

Orthogenesists stood accused of the sin of teleology but Whitman (1906: 45) offered absolution.

> If a *designer* sets limits to variation in order to reach a definite end, the direction of events is teleological; but if organization and the laws of development exclude some lines of variation and favor others, there is certainly nothing supernatural in this, and nothing which is incompatible with natural selection. Natural selection may enter at any stage of orthogenetic variation, preserve and modify in various directions the results over which it may have had no previous control.

Current debates can be considered a reprise of earlier disagreements about the reciprocal relations between evolution and development. One of the consistent claims of the EES is that SET has ignored and belittled developmental biology. Proponents of the EES posit that mechanisms internal to organisms shape evolutionary trajectories, reprising arguments of orthogenesists. Theodor Eimer (1897) wrote that "definitely directed and law-conforming evolution produces the *simultaneous* transmutation of numerous individuals of the same species." A similar belief in directed variation, and coordinated change, is a hammer in the hands of Laland et al. (2013b):

> In the standard account, genetic mutations (and novel phenotypes) are random with respect to direction, rate, and location, typically disadvantageous, and appear in a single individual. Conversely, in the developmental plasticity/bias account, genetic mutations can be non-random with respect to rate and location, while novel phenotypes can be directional, functional, and may appear in multiple individuals.

Their hammer strikes again (Laland et al. 2013b):

> The MS predicts that genetic mutations (and hence novel phenotypes) will be random in direction and typically disadvantageous, whereas the EES predicts that novel phenotypic variants will frequently be directional and functional.

and again and again (Laland et al. 2015):

> Facilitated variation . . . provides a mechanistic explanation for how small, genetic changes can sometimes elicit substantial, non-random, well-integrated and apparently adaptive innovations in the phenotype.

> Sources of bias in phenotypic variation [are] considered an important evolutionary process, which does not only constrain but also facilitate and direct evolution. Developmental bias is a major source of evolvability.

Laland and colleagues do not invoke supernatural causes when they speak of directed variation. They simply claim that biases in the direction of phenotypic variation facilitate adaptive genetic evolution via reciprocal causation. If evolved biases were undirected with respect to utility they would be more likely to impede adaptation than to facilitate it, under the assumption that there are more ways to go wrong than get better. The substantive claim appears to be that variational biases have evolved to point in the direction of what is needed under changed circumstances. In other words, novel phenotypes and adaptive innovations can arise from biases in the direction of variation that facilitate adaptive evolution. In this sense, the EES promotes a more teleological view of evolutionary change than favored by adaptationists who remain acutely sensitive to anything that sniffs of directed mutation.

Disagreement over "directed variation" is a major point of friction between defenders of EES and SET that might be salved by recognizing different *senses* of "direction" and being clear when one is referring to directedness of phenotypic or genotypic variation while acknowledging their reciprocal causation. Adaptive phenotypic plasticity is an undisputed phenomenon and it is also undisputed that the existence of phenotypic plasticity shapes the range of acceptable genotypic responses to environmental change.

14.9 The Poetry of Life

"Phenotype determines genotype" is a picaresque and quixotic epic of discursive difference-making. The varied effects of allelic variants, of p's and q's, in a complex world determine which variants become invariant. The judgment of nature in a single generation, whether a variant is Copied or Not Copied, may rest on a single difference made at a moment of crisis or many differences of different nature made over the course of a lifetime. Similar judgments of nature must be repeated, generation after generation in which variants make many differences (never precisely repeated), before one genetic variant replaces another. A variant's fate is determined by its overall effects relative to the alternatives. It is not a simple story, and a story unknowable for most details that made a difference, but it is the cause of why organisms do the things that they do, rather than something different. "Genotype determines phenotype" is a much simpler tale of molecular mechanisms. It dominates research and funding, but there is much that it leaves unexplained.

An organism's adaptations are evolutionary predictions of what will work in its world. Natural selection trains genetic networks on the ill-defined task of getting-by in the world, using past environments of past organisms as its training set. Replicative feedback from *evaluation* of past phenotypes by past environments *rewards* some variants and *punishes* others. The attribution of credit to smaller genomic parts

is achieved by the recursive randomization of variants at each locus against the background of variants at other loci with the apportionment of credit less effective for variants in strong linkage disequilibrium. (The genomes of asexual organisms are judged as wholes not by their parts.)

All parts work together in a well-crafted sentence. The combination of meiotic randomization with repeated trials has similarities to back-propagation methods used to assign "credit" in the training of the deep-learning networks of artificial intelligence. Under back-propagation, the adjustment of nodal strength is achieved by linear estimation of a node's contribution to successful performance as assessed by the training mechanism. A detailed knowledge of the connections of every node in a network gives little insight into how the network achieves what it does. Functionalities cannot readily be ascribed to individual nodes or localized parts but are distributed across the network. Natural selection similarly rewards additive contributions to the fitness of the genetic parts of highly non-additive networks. How these networks perform their adaptive functions and how individual parts contribute to organismal performance are likely to be difficult to understand, even with complete knowledge of the connections.

Artificial intelligence teaches difficult lessons. Deep-learning performs impressive feats of interpretation by transparent mechanisms for obscure reasons. The opacity of how these networks achieve what they do is a cautionary tale for systems biologists who want to make sense of a cell by describing all the connections and interactions of its genetic and biochemical networks. And it is also a cautionary tale for adaptationists who want to understand the genetic substrate of adaptive features. A difference of coat color may be readily assigned to a genetic difference in a pigmentation pathway but the genetic contributors to more complex adaptations are probably broadly distributed across gene regulatory networks and participate in many other complex adaptations. Knowing the network may provide less understanding than is hoped or hyped.

The training of artificial intelligences involves recursive adjustment of connections of ready-formed networks, comparable in some respects to learning in a single lifetime. Organisms, on the other hand, acquire form by recursive development. Form is generated anew each new generation. There must be a hereditary record of past choices of nature or all would be lost. This is the role of genes. They are the persistent presence of what worked in the past. Genes are *formal causes* of what makes organic materials one kind of organism rather than another. The environmental contingencies that wrote the genetic record of past successes are an organism's *final causes*. They determine what an organism is good at doing.

Purposeless processes of formless materials produce purposeful material forms as specified by the textual record of past choices of nature. Genes are the texts of nature that give form to the phenotypic performances of living things. This is not the straw man of genetic determinism: not all that goes into a successful performance of *Hamlet* can be found in Shakespeare's text. Genetic texts are neither sacred nor unchangeable. DNA synthesis is non-selective quotation with occasional misquotation. Negative selection removes corruptions of meaning by mutational misquotation, but the precise wording drifts with unintended slips of the pen, and meaning

evolves by positive selection as rare slips of the pen are tested and found good in the changing environment of critical reception of the text (Haig 2020).

Many arguments in evolutionary theory are disagreements about how to interpret what others have written. The underlying arguments can be difficult to follow beneath the blizzard of words. Hard-minded experimentalists say theoreticians are not doing real science, but experimentalists' interpretations of the outcomes of their experiments are often shaped by unquestioned philosophical commitments. The modern debate between advocates of EES and SET reveals unresolved tensions in biology. This essay evolved as I grappled with texts from more than a century of debate, some in a language I know poorly: Why does Kevin Laland deny functions are causes but favor directed variation? Why did Karl von Baer warn against teleophobia but oppose Darwinism? As a man with a hammer, I have pounded these puzzling protrusions by asking what they reveal about the authors' underlying teleological commitments. Each reader will judge whether I have hit these nails on the head. Every workman prefers her own tools.

The hypertextuality of evolutionary debates is no coincidence. We have evolved from a world of matter in motion—of material and efficient causes obeying physical laws—to a living world of information, meaning, and purpose; a world of goal-directed beings that obey the letter of physical law but not in spirit. Biology cannot do without concepts of purpose and goal, although some biologists twist their prose into knots to deny what they do in practice. The meaningful features of life are difficult to quantify: attempts to mathematize meaning are often changing the question. One of the final metaphors of *From Darwin to Derrida* identifies natural selection as a poet who means many things at once. An organism and a long non-coding RNA make sense in many ways. The poet tries the mutations finding words that work.

Telos—the reciprocal relation of purpose and goal, of *Zweck* and *Ziel*—is the distinctive feature of life, grounded in the recursive relations of phenotypes and genotypes and of chickens and eggs. Making sense of the poetry of life will not be easy. "Hypothesizing about adaptive rationales is easy to do badly, and difficult to do well" (Welch 2017). This is hard science. It is time we cured our teleophobia and worked together to develop and evolve a predictive and explanatory teleology of life. We should interpret each other's arguments kindly. We have a choice of hammers.

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Teleology, Organisms, and Genes: A Commentary on Haig

15

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Abstract

This piece discusses David Haig's chapter "Hypertextuality of a hyperextended synthesis: On the interpretation of theories by means of selective quotation." We agree with Haig's project to dismount teleophobia in the life sciences, provided scholars offer detailed and careful treatments of what it is meant by "teleology" and in which contexts it is warranted to bring in this explanatory mode. However, we think that Haig's approach suffers from several shortcomings. First, we maintain that Haig fails to make a distinction between different kinds of teleology. Second, we then argue that organism-related internal (not external) teleology is at the center stage of the EES debate, a point not underscored by him in his criticism directed to this conceptual framework. Third, we contend that the type-causal understanding of teleology advanced by him in his genetic-adaptationist framework is inappropriate. In contrast, we point to two important epistemic elements of teleological explanations that need to be spelled out (*i.e.*, which dependency relations are traced, and which *relata* are relevant in evolutionary contexts). Fourth, we discuss some drawbacks of Haig's views regarding causal reciprocity and raise the issue of the seemingly organism-deprived ontology of his position. In this sense, we argue that developing, acting organisms are critical difference-makers in evolution and this issue seems to be absent from his ruminations. Finally, we conclude and ask: What can evolutionary biologists actually build with Haig's 'hyperextended hammer'? .

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15.1 Introduction: A Zone of Agreement

Evolutionary biologist David Haig, best known for his works on genomic imprinting and intra-genomic conflict (see Haig 2002 for an overview), has recently published *From Darwin to Derrida: Selfish Genes, Social Selves and the Meaning of Life*, a book of essays that, among other things, discusses theoretical possibilities on the evolutionary origin of meaning and purposiveness from a decidedly gene-centered, adaptationist stance (Haig 2020). In his chapter in this volume, "Hypertextuality of a hyperextended synthesis: On the interpretation of theories by means of selective quotation," he puts forward ideas that complement the main theses defended in his book and offers an evaluation of some aspects of the Extended Evolutionary Synthesis (EES) framework of Laland et al. (2015).

Haig establishes zones of agreement with Laland and his colleagues, the multiple epistemological and ontological differences of their positions notwithstanding: for example, he grants the importance of "reciprocal causation" as a theoretical tenet in evolutionary biology. Here, we want to do the same right from the outset: As philosophers of biology, in what do we agree with Haig? We concur with him, first and foremost, on the need to fight *teleophobia* in biology, particularly when this standpoint is advanced from prejudice without a proper historical contextualization and a thorough philosophical qualification of what teleology means or what is entailed by a teleological explanation. [1] As many contemporary philosophers of biology (see, e.g., Walsh 2015; Aaby and Desmond 2021), we are in favor of re-assessing goal-directedness and agency, and developing conceptually careful treatments of teleology in biology (especially as it pertains to the structure of explanations of organismal behavior and some developmental phenomena). Haig finishes his chapter with a nudge in this direction: "It is time we cured our teleophobia and worked together to develop and evolve a predictive and explanatory teleology of life. We should interpret each other's arguments kindly. We have a choice of hammers" (Haig 2023: 231).

However, besides standing along Haig on the need to dismount biological teleophobia and re-appraise this multi-layered notion in biology, we do not find many more points of agreement with his theoretical views. We think that his hammer misses the target of this project in five important ways. First, we maintain that Haig fails to make a distinction between different kinds of teleology. Second, we then argue that organism-related (internal) teleology is at the center stage of the EES debate, a point not underscored by Haig. Third, we contend that the type-causal

[1] A first clarification is in order: This should not be confused with the notion, which we completely reject, that there is an overarching teleology in all of nature, a preordained *telos* in the universe that guides, for instance, the evolutionary history of life forms (for a discussion on this point, see below).

understanding of teleology advanced by him in his genetic-adaptationist framework is inappropriate. In contrast, we point to two important epistemic elements of teleological explanations that need to be spelled out (i.e., which dependency relations are traced, and which *relata* are relevant in evolutionary contexts). Fourth, we discuss some shortcomings of Haig's views regarding causal reciprocity and raise the issue of the seemingly *organism-deprived* ontology of his position. In this sense, we argue that organisms are critical difference-makers in evolution and this issue seems to be absent from his ruminations. Finally, we conclude and ask: What can evolutionary biologists *actually* build with Haig's 'hyperextended hammer'?

15.2 Disentangling Types of Teleology in Biological Explanations

Throughout his essay, Haig alludes to (and in part conflates) different kinds of teleology without making explicit distinctions. Prima facie, what groups distinct kinds of teleology under the same general umbrella or justifies keeping the same broad linguistic descriptor is that, when used with explanatory functions, they give an account of something by appealing to *final causes* or by reference to an *end* or *goal*. Many biologists and philosophers have tried to disentangle different senses of teleology to identify which of them are relevant for biological endeavors (for instance, see Mayr 1998). Here, we argue that any attempt to rebut teleophobia needs to start by differentiating four kinds of teleology that have been influential in the history of philosophy and biology:

Cosmic Teleology This corresponds to the view of a subtending *telos* in the cosmos (e.g., of a divine nature) that directs or controls the general state-of-affairs in a particular direction or toward the attainment of certain purpose (for discussion, see Henderson 1917). In the realm of the living, this could translate into steered evolutionary trajectories in accordance with a plan or changes in the inorganic or organic world directed toward the fulfillment of a specific goal (e.g., the continued survival of mankind). For example, the *Strong Anthropic Principle* enunciated by Barrow and Tipler (1986) posits that certain conditions of the universe (e.g., physical constants) have been fine-tuned so that the appearance of sentient observers inevitably happens in a planet with the characteristics and history like ours.

Intentional Teleology Under this view, a particular goal is mentally anticipated by an agent and this intention is relevant for triggering and controlling the performance of an action (e.g., the planned construction of a house or heating some *Glühwein* to endure winter). For many scholars working in philosophy of action (see Paul 2020 for an overview), human action is primitively characterized in terms of intention and this, in turn, is purportedly explained by a causal chain (e.g., of bodily movements) that ultimately can be traced back to an agent's mental states that give reasons for acting. When these discussions are extrapolated to biological systems more generally, Allen and Bekoff (1995: 15) speak of *teleomentalists*, namely those that "regard

the teleology of psychological intentions, goals, and purposes as the primary model for understanding teleology in biology. Teleomentalists interpret teleological talk literally only when conscious agents are involved."

External Teleology According to this viewpoint, an object is useful or purposeful for a different entity (e.g., sand soil for the growth of a pine). For some authors, the external character involved is two-fold: "(a) *the agent* whose goal is being achieved is external to the object that is being explained teleologically, and (b) *the value* aimed at is the agent's value, not the object's" (Lennox 1992: 325; emphases in original). This type of analysis is usually cast over machines and their constituent parts, or other human-made artifacts. For instance, a watch is said to be purposive insofar as it was designed and assembled for the attainment of a particular human goal (i.e., telling time), and, in that same line, the purpose of, say, its battery is to keep it ticking (see Goudge 1961: 192–193). The design stance of external teleology has also been commonly applied to organisms and their traits (for a philosophical analysis, see Lewens 2004), especially when these are seen as shaped and optimized in evolution by bouts of a blind, purposeless process: natural selection acting on random genetic variation (Dawkins 1986). In recent developments of this position, it has been argued that external teleology is best seen as a maximization of inclusive fitness under Grafen's Formal Darwinism (Huneman 2019).

Internal Teleology For this view, sometimes also referred to as *immanent teleology* (see (Lennox 1992: 326), the proper *locus* of teleological adscriptions are not parts or traits of an organism, but rather the organism (or another evolved entity) as a whole which possesses goals of its own. In this sense, organisms differ from machines given that their purposiveness is internal or immanent (Russell 1924: 267). Moreover, at least since Immanuel Kant, organic parts are understood to be reciprocally related to one another, so that they are causally relevant for their mutual persistence and the maintenance of the whole (e.g., the reciprocal interplay of organs in an organism). The intrinsic purposiveness of organisms (in contrast to the extrinsic purposiveness of machines, always set by an external designer) means that organisms, through their activities, pursue their own goals, such as surviving, reproducing, overcoming challenges throughout their life cycles, or simply maintaining their organization in manifold developmental and environmental contexts (for discussion, see Nicholson 2013). Internal teleology also refers to the goal-directed capacity of organisms to undergo compensatory morphological or physiological changes during their life cycles (Walsh 2008).

After outlining these different types of teleology, it is important to stress that cosmic teleology plays no role in today's biology. Likewise, intentional teleology, with the conscious and rational undertones we discussed, is rarely postulated seriously in evolutionary sciences. [2] As Haig covered in his chapter, embryologist

[2] In the history of biology, some authors have tried to restrict the concept of purposiveness to high-order intentional teleology in order to claim that there cannot be any form of teleology in biology

Karl von Baer suggested to use the German term *Ziel* rather than *Zweck* to avoid the conflation of biologically relevant forms of teleology with intentional teleology (for a detailed historical analysis of these concepts, see Toepfer 2004). On the other hand, external and internal teleology can come in various forms in biological explanations. Distinguishing them is crucial.

Some authors have contended that Darwin's theory provided grounds for integrating external teleology reasoning into biological thought, while others have suggested that he replaced teleological thinking through natural selection or banished it altogether from biology (as an example of two counter positions on the topic, see Lennox 1993; Ghiselin 1994). [3] Haig belongs to this tradition of marshaling external teleology through an adaptationist lens, but he is also prone to blend it with a rationalizing, intentional teleology: "[. . .] natural selection can be viewed as inductive reasoning about effective action: a gene's effects are hypotheses about what works in the world, with confidence in a hypothesis increasing with the strength of past associations with favorable outcomes. [. . .] The intentionality of natural selection is retrospective. But *what worked* in the past was observing the present and predicting the future" (Haig 2020: xxvi–xxvii; emphasis in original).

Despite the justified focus on external teleology through assessments of Darwinism since the late-nineteenth century to today (which of course can dispense with intentional teleology), a number of authors have stressed that we should not forget about (and better conceptually integrate) internal teleology. For example, E.S. Russell (1916: 232) highlighted: "Perhaps Darwin did not realise this inner aspect of adaptation quite so vividly as he did the more superficial adaptation of organisms to their environment." However, the integration of internal teleology into biological explanatory frameworks was (and it is still today) much more contested. From a perspective of the history of philosophy this is surprising, given that internal teleology has a rich history and various famous, vocal advocates. A very influential position was advanced by Immanuel Kant, strongly influenced by the embryological research of his time, in his *Kritik der Urteilskraft* (*Critique of the Power of Judgment*): "For a body [. . .] who should be seen by itself and based on its inner potential as a natural purpose [*Naturzweck*], its parts need to mutually bring about each other (their form and relations) as well as the whole through their causality" (Kant 1913 [1790/93]: 373; German original). "Self-organized beings" (organisms) actively maintain their internal organization through the reciprocity of their parts and through an active, regulated interaction with their environment. [4]

whatsoever. For example, the physiologist Johannes Müller (1969 [1824]: 66) said: "In nature, nothing that is studied by physiological investigations has a purpose. Everything in nature is there for its own sake. Only actions of humans have purpose" (German original). For discussion, see also Goudge (1961).

[3] For example, embryologist Julius Schaxel (1922: 272) described this purported replacement as: "The theory of natural selection is possibly the most peculiar construct in biology: for the sake of a mechanistic postulate, misjudged teleology is dissolved into history" (German original).

[4] Kant's position on internal teleology was regulative, serving only a heuristic function (without making ontological claims about the constitution of organisms), but organismal organization has

Some scholars have maintained that internal teleology is actually more basic than (Darwinian) external teleology. For example, theoretical biologist Ludwig von Bertalanffy (1932: I, 59) argued that "selectionism does in fact not explain organic wholeness, but rather presupposes wholeness in organisms' functions of life. Only because organisms are 'whole-maintaining' and 'persisting' [*dauerfähige*] beings, they can struggle for existence with one another. Darwinian chance means nothing but rejecting the insight into the laws of the development of organic 'purposefulness'" (German original). It must be said that the pushback against internal teleology in the early twentieth century resulted in large part from objections against vitalist concepts such as Hans Driesch's *Entelechie* or Henri Bergson's *élan vital* (on this point, see, for example, De Klerk 1979).

Unfortunately, in his chapter, Haig does not provide these kinds of conceptual distinctions and his neglect of internal teleology suggests to the reader that, from all these different kinds of teleology, only external teleology matters for evolutionary considerations and organismal purposiveness is easily glossed over.

15.3 Teleology and the Extended Evolutionary Synthesis

Coming back to contemporary debates in evolutionary biology and the role that teleology plays inside them, Haig argues:

> The relation of the Extended Evolutionary Synthesis (EES) to teleological reasoning is complex. On the one hand, its proponents downplay the importance of adaptation by natural selection and of teleological explanation in terms of purpose or function. On the other hand, they favor a more teleological evolutionary theory in which variation is not random but directed or biased toward what is needed. (Haig 2023: 231)

Is he correct on this assessment? How does the EES square with respect to teleology if we grant that there are different senses of this notion and not all them are brought to the fore in evolutionary explanations? To solve this puzzle, let us analyze where standard evolutionary theory and EES stand with respect to these four kinds of teleology. Both standpoints agree on many things: for one, on the thesis that cosmic teleology should never play a role in biology (which makes moving the EES closer to creationism problematic, see Haig: 12; see also Laland 2017: 317). [5] Moreover, both agree that intentional teleology (concurrently appealing to language, rationality, and high-order cognition as its foundation) across the tree of life is off the table for general theoretical discussions in evolutionary biology. Scholars in the EES

been spelled out as an intrinsic feature of organisms in different metabolic or thermodynamic ways by different authors, such as J.S. Haldane, L. v. Bertalanffy, or H. Maturana and F. Varela with their theory of autopoiesis. For a recent discussion on Kant's controversial legacy for biological science, see Gambarotto and Nahas (2022).

[5] Mayr (1992: 119) claimed that "[. . .] by the time of the Evolutionary Synthesis of the 1940s, no competent biologist was left who still believed in any final causation of evolution or of the world as a whole."

camp, even when writing about organismal agency, reject teleomentalism explicitly (see Sultan et al. 2022). Another point of agreement between them is that a naturalized external teleology referring to adaptation indeed matters in evolutionary explanations. [6] For example, within niche construction theory, a major component of EES-type explanations (see Fábregas-Tejeda and Vergara-Silva 2018), the fit between organismal traits and environmental demands (adaptation) is understood as a reciprocal product between the action of natural selection and the consequences of sustained rounds of niche construction performed by organisms (see Day et al. 2003).

The point of contention for EES advocates is that external teleology cannot account for all properties of organisms, especially for how they plastically develop and actively impact evolutionary trajectories (e.g., through niche construction or developmental bias). Thus, the EES framework tries to tie in with the long theoretical tradition of internal teleology. This view highlights that developmental processes and organisms' interactions with their environments are (to some extent) goal-directed and purposive:

> For reasons [. . .] that derive from consideration of the properties of life and the principles of thermodynamics [. . .], we view agency to be an essential and inescapable aspect of nature. [. . .] We emphasize that living organisms are not just passively pushed around by external forces, but rather they act on their world according to intrinsically generated but historically informed capabilities. Organisms are self-building, self-regulating, highly integrated, functioning, and (crucially) "purposive" wholes, which through wholly natural processes exert a distinctive influence and a degree of control over their own activities, outputs, and local environments. Indeed, organisms must have these properties in order to be alive [. . .] (Laland et al. 2019: 131–132).

This internal teleology, EES proponents argue, is not fully determined by an external teleology grounded in natural selection. [7] For one, function-talk associated with external teleology does not exhaust all that there is to be said about internal teleology (see Walsh 2008; see also Nicholson 2013: 671). Explanations that resort to internal teleology are needed to account for how organisms build, alter, and dynamically

[6] Given all these agreements, we think it is also not correct to follow Haig in his assertion that "Nineteenth-century diatribes [about teleology] are echoed in twenty-first century tirades" (12). Today's debates in evolutionary sciences ignore substantial forms of teleology (cosmic, full-blown intentional) that were crucial components of nineteenth-century debates. The parallel drawn by Haig, although rhetorically suggestive, turns out to be, to a certain extent, historiographically naïve.

[7] It is important to call attention to the fact that not all points of contention in the EES debate can be pinned down to an opposition between standard views that underscore external teleology in the form of adaptation by natural selection and a novel developmentalist, organism-focused view that highlights internal teleology instantiated in plastic, regulative processes. The EES debate has also been about integrating developmental processes, extra-genetic forms of inheritance, and niche construction into evolutionary theory in a central way, introducing different explanatory standards in evolutionary explanations, and questioning the nature of evolutionary processes and causation (see Uller et al. 2020; Baedke et al. 2020; Uller and Laland 2019 and chapters therein). The discussions touching on teleology do not exhaust the issues that have been raised by the EES.

regulate their parts (Walsh 2008). What Haig points out as a source of disagreement between him and EES proponents over the causal role of functions is something more than a mere point of friction: he is asking EES proponents to fully embrace external teleology and sideline internal teleology. Furthermore, he seems to wrongly assume that external teleology (sustained by function-talk and natural selection as the causal purveyor of adaptive design) engulfs organismal internal teleology. This is a category mistake.

As we come to see, the EES has no conflicting position on teleology (downplaying and highlighting it at the same time, as Haig suggests). [8] Instead, it builds on a different philosophical tradition, internal teleology, compared to the sources of most evolutionists that draw on views of naturalized external teleology. Whether EES-derived hypotheses that suggest that organismal agency in particular is another central process in evolution (see Sultan et al. 2022) are right or not, is open to theoretical debate and empirical research. However, we need to recognize that the internal teleology defended by them has nothing to do with cosmic teleology (or creationism), conscious, representationalist intentionality, or even vitalist views. It highlights the possibility that the often-contingent ways of how organisms constrain their internal parts during development and interact and shape their environments through niche construction are agential processes that could bias or even facilitate evolutionary change (Sultan et al. 2022; see also Walsh 2015). But what is entailed by teleological explanations of this sort?

15.4 What Constitutes a Teleological Explanation?

Many critiques have been waged on why teleological explanations are problematic in biology. Among the most recurrent past and present arguments (see Mayr 1998; Allen and Neal 2020), we find charges of vitalism, of incompatibility with naturalism and mechanistic explanation, of being onerously mentalistic (appealing to the action of minds where there are none, e.g., in natural selection), of being empirically intractable, and of resorting to the theoretically incoherent and metaphysically fraught concept of *backward causation* (whereby goals in the future cause the means to reach them in the past).

One of the main arguments of Haig's chapter is that teleological language is justified in biology "because natural selection confounds common understanding of cause and effect" (Haig 2023: 231). He further claims: "Genotypes determine phenotypes by developmental processes. Phenotypes determine genotypes via

[8] Neither does it have a radically different view on how variation is produced compared to the Modern Synthesis, especially when it comes to chance, randomness, and biases (see the analysis of Merlin 2010). For example, biased variation as defended by the EES, as Haig suggests, is not the same as (orthogenetic) *directed variation*. The notion of developmental bias merely "captures the observation that perturbation (e.g., mutation, environmental change) to biological systems will tend to produce some variants more readily, or with higher probability than others" (Uller et al. 2018: 949).

natural selection. A protein is a cause of its DNA sequence, the central dogma of molecular biology notwithstanding" (*Idem*). To spell out his position, he draws the distinction between token-causation and type-causation, stating that the latter is free from the illegitimacies of backward causation: "[. . .] what is true of relations among individual things (token-causation) need not be true of relations among kinds of things (type-causation), especially when kinds are related by reproductive recursion. An egg is both an effect of a chicken-that-was and a cause of a chicken-to-be. Eggs (considered as kinds) are both causes and effects of chickens (considered as kinds)" (Haig 2023: 231).

Many philosophers of science (us included) would disagree with Haig's perspective on the grounds that bona fide causal relations only obtain between particulars and, in that regard, would argue that all purported type-causal claims (those usually subsumed under the umbrella of *type-causation*, e.g., "A protein is a cause of its DNA sequence," "Eggs are both causes and effects of chickens") are actually *generalizations* concerning those *token* relations (see, for example, Woodward 2003; Hausman 2005):

> Although there is a distinction between type- and token-causal *claims*, it does not follow that there are two kinds of *causation*—type and token—or that in addition to token-causal relationships involving particular values of variables possessed by particular individuals, there is a distinct variety of causal connection between properties or variables that is independent of any facts about token-causal relationships (Woodward 2003: 35–36; emphasis in original).

Spelling out teleology in terms of type-causal claims does not seem to be an adequate way to steer the debate against biological teleophobia. If Haig's causal construal is inadequate (as it does not convey actual causal relations), how can teleology be framed satisfactorily? We think that a crucial component of teleological explanations gets sidelined in his ponderings. As Walsh (2008: 113) mentions: "Teleology is a mode of explanation in which the presence, occurrence, or nature of some phenomenon is explained by appeal to the goal or end to which it contributes." Here, we argue that it is of utmost importance to be clear about how teleological explanations, insofar as they appeal to *goals*, actually explain. Two central questions must be answered to begin addressing this issue in the biological sciences: (a) Which dependencies do teleological explanations trace? (e.g., Are these dependencies causal or modal in nature?); and (b) What are the relevant *relata* of teleological explanations for evolutionary contexts? In the remainder of this section we will address (a), and (b) will be covered in the next section.

An appeal to ends or goals does not mean to explain the presence, occurrence, or nature of a particular phenomenon by some unactualized, future context; in other words, spurious *token* backward causation can be ruled out. The important relation of normative requirement in a teleological explanation is not that between cause and effect, but the relation that holds between the *goals* of an agent and the *means* marshaled by it toward their attainment. This is what Aristotle referred to as *hypothetical necessity*: we explain a phenomenon teleologically if (i) it results from a goal-directed activity, and (ii) is hypothetically necessary for the attainment

of the goal (Walsh 2008). Against this background, means, for philosophers like Walsh (2018) and Fulda (2017), can be construed as the elements of an agent's repertoire (e.g., the behavioral and developmental capacities of an organism) that are conducive to the attainment of its goals (e.g., escaping the attack of a predator or polymorphically altering features of morphology or physiology to endure a change of seasonal conditions). Importantly, the relationship between goals and means (i.e., hypothetical necessity) is a *modal* relationship, not a causal one:

> Hypothetical necessity is not a causal relation—goals don't cause their means, they hypothetically necessitate them—but it is a natural one nevertheless. Hypothetical necessity entails that, without the action in question, the goal would not have occurred and, with it, the goal [. . .] occurs reliably. Its dual is the relation of conducing. Whereas ends hypothetically necessitate their means, means conduce to their ends. Conducing is not the same as causing; *m* conduces to *e* only if *e* is a goal and, under the circumstances, *m* would reliably cause *e* across a range of counterfactual conditions (Walsh 2018: 173; see also Fulda 2017).

Building from Woodward's interventionist view of scientific explanation (see Woodward 2003), Walsh (2008, 2015, 2018) has further argued that teleological explanations are explanatorily autonomous: they point to regularities and counterfactual dependencies between means and goals that cannot be reduced to standard causal-mechanistic explanations. In this view, teleological (modal) and mechanistic (causal) explanations are complementary to each other, as they provide access to different sets of counterfactual dependencies. In that sense, for example, it is possible to show that the goal-directedness of a system-as-a-whole has explanatory consequences that cannot be solely pinned down to the interactions of its parts (Walsh 2006, 2008).

Besides these issues with identifying the nature of relations traced by teleological explanations (in contrast to causal explanations), we also see some problems in how Haig discusses the *relata* of teleological explanations. This especially refers to the difference-makers in evolution.

15.5 Difference-Makers in Evolution or "Where is the Organism?"

In his chapter, Haig accentuates the importance of reciprocal causation as a theoretical cornerstone of the EES (for philosophical discussions on this topic, see Buskell 2019; Baedke et al. 2021):

> Reciprocal causation is a leitmotif of the EES: there is a reciprocal relation between evolutionary and developmental processes; organisms are shaped by their environment and shape the environment; phenotypic plasticity has evolved and itself molds the evolutionary process; epigenetic and genetic inheritance modify each other, interchangeably; phenotypic accommodation promotes genetic accommodation reciprocally; and so on. (Haig 2023: 231)

Nonetheless, he also claims that all of these reciprocal processes, from niche construction to plasticity-led evolution, "can be considered different aspects of the reciprocal relation between genotype and phenotype that arises from reproductive recursion." Haig incorrectly assumes that all forms of causal reciprocity vouched for in the EES are related to genotype-phenotype reciprocity. Haig's assessment notwithstanding, the proper *relata* of reciprocal causation within the EES are organism and environment, not genotype-phenotype. Only if an organism is a mere epiphenomenon of its genes would the assertion that organism-environment reciprocity is covered under genotype–phenotype reciprocity be warranted. Questionable as that standpoint might be (see, among multitude criticisms against subsuming all organismal processes and behaviors to genes, Moss 2004; Griffiths and Stotz 2013), it gives us an idea of the ontology underlying Haig's theoretical views. For him, genes are the central difference-makers in development and evolution. He resorts to somewhat idiosyncratic definitions to make this point clearer:

> I have defined a gene's *phenotype* as all differences it makes in the world relative to an alternative and its *environment* as all things that are the same relative to the alternative [. . .]. By these definitions, variation that does not have a genetic cause is part of the environment not of the phenotype. Natural selection involves feedback between two differences: a *genetic difference* that is the cause of a *phenotypic difference* that is the direct target of selection. The first difference is the *difference-maker*. The second difference is the *difference-made*. The things that are the same are the environment that selects (Haig 2023: 231; emphases in original).

With Haig's definition of the environment, as something static and unified (for a counter position, see Walsh 2021), it is difficult to see how an organism could be a difference-maker of environmental modification (as emphasized inside niche construction theory and the EES, see Laland et al. 2015). Under Haig's conception, the environment is never directly affected by an organism or an organismal phenotype without genetic mediation, as organisms and individual phenotypes are always merely effects brought about by genes, never difference-makers themselves.

Moreover, Haig reduces organismal purposiveness to a causal side-effect of the purposiveness of the genes that are driving development:

> The directedness of development is explicable in terms of reproductive recursion. Development toward a goal is purposive because adult forms have transmitted genetic differences responsible for their distinctive development. Differences of development are the phenotypic effects of developmental genes. [. . .]. Adaptationists are also comfortable with contingent goal-directed behavior, with the twists and turns of a cheetah as it pursues a fleeing gazelle (Haig 2023: 231).

We think that his stance is not warranted. For example, philosopher Samir Okasha has argued that there is only a restricted sense in which genes can be considered *bona fide* agents in evolution (see Okasha 2018: Chap. 2). This pertains to the view of *selfish genes* which postulates that these have the *ultimately goal* of outcompeting other alleles and using organisms as vehicles of self-replication (Okasha 2018: 45; see also Dawkins 1976; Haig 2012):

> [. . .] to say that genes "want" to further their own replication, or encode strategies that further that goal, is ambiguous. Is this the logical point that the spread of any gene is necessarily at the expense of its alleles, or the empirical point that some genes spread via mechanisms that harm their host organism? Only in the latter case are we compelled to treat the gene rather than the organism as the agent, in order to apply agential thinking. (Okasha 2018: 46)

In his chapter and throughout his life-long work, Haig has committed to a gene's-eye view of evolution, which has indeed been very influential and heuristically fruitful in the history of evolutionary biology. But becoming aware of the limitations of the gene's-eye view (e.g., being clear on the *explananda* it can tackle and establishing what falls outside its explanatory purview) is an important consideration for making the most out of this persuasive stance (for discussion, see Ågren 2021; see also Agar 1996). Okasha (2018: 50) has argued that the merit of the *genes-as-agents* view, in particular, is that "it enables us to make sense of intra-genomic conflict and its phenotypic consequences, and thus to extend the adaptationist paradigm to a range of phenomena that would otherwise be baffling." Phenomena such as sperm competition, sex-ratio distortions, transposable elements, and so on, have resisted traditional adaptationist logic and the gene's-eye view allows biologists to make sense of them. But one thing is to grant this and do research on these topics, and a very different one would be to conclude from that assessment that *all* the features of organisms (including their intrinsic purposiveness and agency) are the direct consequence of selfish gene action and gene selection.

In sum, we consider that Haig, in his evolutionary ontology, undervalues an important difference maker which cannot be reduced to its genes: the organism. Empirical examples of the organism as a difference maker in evolution abound in contemporary biological literature (see, e.g., Sultan 2015 and Baedke 2019 for overviews). Consider a case of agential niche construction (*sensu* Aaby and Desmond 2021) as a representative instance of the kinds of explanatory regularities that can be uncovered and traced through teleological explanations centered on organisms (see the previous section). Clark et al. (2020) have recently conducted a multispecies meta-study of hundreds of selection gradients measured in natural populations showing that, when environmental variation is buffered by the activities of organisms, selection gradients exhibit reduced temporal and spatial variation in and weaker selection compared to non-constructed sources. When discussing this study, Aaby and Desmond (2021: 47) ponder:

> The reason why this should be seen as an example of a regularity that is uncovered by a teleological account is that it points to a general dependence of *means* on *goals*, which moreover holds across a wide array of taxa. What are environment buffering activities *for*? The evidence suggests that organisms engage in such activities *in order to* reduce environmental variation. [. . .] The reduction of environmental variation in turn alters the selective environment to favor traits that are useful for regulation and control over environmental factors (emphases in original).

Rather than drawing on external teleology and gene-environment relations, this approach adopts internal teleology and focuses on the *relata* "organism" and "environment." Clarke et al. (2020) exemplify that this conceptual framework, centered on organisms as agents, can be a fruitful starting point for evolutionary research.

15.6 Conclusions: What Can Biologists Build with Haig's Hammer?

Once we have recognized that explanations that resort to internal teleology (in the form of organismal purposiveness and agency) can play important roles in evolutionary science, a future step would be to integrate them with views that highlight external teleology and adaptation in evolutionary biology. If we stayed within Haig's vantage point (in his own words, a *hyperextended synthesis*, albeit only confined to external teleology), we could not be able to advance this project (nor successfully fight teleophobia, for the reasons we adduced in the previous sections). While he correctly states that "[w]e have a choice of hammers" (Haig 2023: 231), this choice in his hands bears strong relativist undertones. Different "interpretations of evolutionary theory" or "beliefs can be honestly held", Haig tells us, merely depending on which etymological or rhetorical preferences we have in defining central concepts one way or another. As Haig has previously argued, for him, choosing between MS- or EES-like explanations is based on "emotional and aesthetic reasons", and anthropological outlooks "based on differences of preference and thinking style rather than matters of substance" (Haig 2011).

In contrast, we have argued elsewhere that, to move forward in the EES vs. SET debate, we first need to understand the different explanatory standards mobilized in their respective explanations and what these conflicting accounts bring to the table in terms of explanatory power (see Baedke et al. 2020; see also Baedke and Fábregas-Tejeda, this volume). Only after we have gained an understanding of these issues, theoretical integration could be at reach. Contra Haig's relativistic choice of hammer, we argue instead that evolutionary biologists should not only know which hammer to pick and when, but how to build a house by using different hammers at the right time. Wielding a sledgehammer for banging in a nail is rarely useful.

We would like to invite Haig to further elaborate on what his hammer brings to the table (not least to EES proponents) and what are the heuristically fruitful interfaces between his theoretical views and scientific practices. What could evolutionary biologists build with his hammer, besides spaces not haunted by the specter of teleophobia? We think this issue is important because, among other things, his type-causation view of teleology seems to be out of reach for practicing biologists. For example, Hitchcock and Knobe (2009, 591) have contended that only token causation provides appropriate targets for scientific interventions and experimental settings. Walsh (2018, 174) has argued that theories that deal with teleology and agency demand particular batteries of concepts and methods that are not shared with other biological theories. The challenge for biologists and philosophers of science is

still to come up with a good and robust theory of agency in biological contexts. Do Haig's views form a full-fledged theory of teleology and agency?

We can agree with him that teleophobia in biology should be avoided, but we disagree that only external teleology should shape evolutionary reasoning. Here we argued that, to fight teleophobia, we must be clear on which type of teleology biologists are referring to and how teleological explanations, insofar as they appeal to goals, actually explain. We maintained that two central questions need to be addressed: Which dependencies are traced by teleological explanations? What are the relevant *relata* of teleological explanations for evolutionary contexts? On the first point, we argued that the dependencies traced are not type-causal, as Haig asserts, but rather modal in nature: goals hypothetically necessitate their means, means conduce to their ends across a range of counterfactual scenarios. On the second point, we showed that, in the teleological explanations vouched for by EES proponents, their central *relata* are organism and environment (e.g., in agential niche construction). We exposed that organism-related internal teleology is at the center stage of the EES debate and that embracing the gene's-eye view of evolution does not change the fact that organisms can figure as important difference makers in the biological world.

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A Token Response: A Reply to Fábregas-Tejeda and Baedke

16

David Haig

Abstract

I find myself strongly agreeing with Fábregas-Tejeda and Baedke's criticisms of Haig's views on teleology. Perhaps the only major point upon which we disagree is whether these are Haig's views. I have no privileged access to the inner workings of Haig's mind, but I have talked with him regularly over the years and therefore possess some insight into how he hoped he would be interpreted and how he might have responded to these criticisms. But who am I to say? In talking of persons, it is often convenient to have some collective term for a set of tokens that exhibit a degree of temporal and causal continuity. For convenience, I refer to one such collection of tokens as myself and attach to this collection the labels Haig and I. As an ephemeral temporal token of Haig, I offer you this brief response.

Keywords

Type causation · Token causation · Teleology · Genes · Organisms

I find myself strongly agreeing with Fábregas-Tejeda and Baedke's criticisms of Haig's views on teleology. Perhaps the only major point upon which we disagree is whether these are Haig's views. I have no privileged access to the inner workings of Haig's mind, but I have talked with him regularly over the years and therefore possess some insight into how he hoped he would be interpreted and how he might have responded to these criticisms. But who am I to say? In talking of persons, it is

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often convenient to have some collective term for a set of tokens that exhibit a degree of temporal and causal continuity. For convenience, I refer to one such collection of tokens as myself and attach to this collection the labels Haig and I. As an ephemeral temporal token of Haig, I offer you this brief response.

I wholeheartedly agree with Fábregas-Tejeda and Baedke's assertion that type-causal claims are *generalizations* from multiple instances of token relations. To take just one example, "genetic drift" is not an instance of token causation. It is a summary way of talking about many instances of token-causal events without having to descend into the unknowable narrative of precisely what happened. Similarly, "natural selection" is not an instance of token causation but another generalization from a complex concatenation of token-causal events. Although we cannot know the precise history of all token-causes, we can nevertheless say useful things about the consequences of drift and selection. And one of the effects of selection is that gene-tokens have come to cause token-effects that are "good for" gene-tokens of that kind. Darwinian final causes are "efficient ways of talking about efficient causes" (Haig 2022, p. 236).

My account of Darwinian teleology is based on generalizations across collections of token-causal events in which token-causes have token-effects. There is no backward causation. When a gene-token replicates, one token is replaced by two tokens and, for convenience, we can label the progenitor token and its progeny as tokens of the same kind or type. Because gene-tokens of the same kind are physically indistinguishable, they will tend to have similar token-effects which we can similarly label as effects of the same kind. Effects of this kind are causes of effects of the same kind via the causal mediation of gene-tokens. Thus, some *effects* of gene-tokens *causally* contribute to the replication of tokens of the same kind and thereby contribute to the repetition of effects of the same kind. These kinds of token-effects are what gene-tokens are "good for."

In this account, the reason why genes are important is that they constitute an archival record of past choices of nature. The useful information that accumulates in successful genetic lineages comes *from the environment* that has preserved these lineages and rejected others that carried different information. One of the most important parts of this environment is the organismal bodies in which genes reside. These bodies are the constructed niches of genetic denizens who have become adapted to the needs of the organisms in which they reside. Bodies, the selective environments that we call organisms, undergo evolutionary change in response to changes both within and without.

> All choices involve selection among things that are different against a backdrop of things that are the same. In a "choice" of nature, that which is different is *phenotype* and that which is the same is *environment*. Evolution by natural selection is a process that converts phenotype, that which is selected, into environment, that which selects. It is a process by which large events, at the level of ecology and organismal behavior, determine fine structure, at the level of molecular gene sequences. It is the means by which the macrocosm shapes the microcosm. (Haig 2020, p. 99)

Fábregas-Tejeda and Baedke describe my ontology as "organism-deprived" and my view of the environment as "static." They also berate me for my "neglect of internal teleology," and my exclusive reliance on external teleology. These are positions that I did not immediately recognize as my own. Genes, considered as material tokens, are parts contained within organisms and describing the teleology of genes as external to organisms is potentially confusing. Fábregas-Tejeda and Baedke define external teleology as present when "an object is useful or purposeful for a different entity" and internal teleology as present when an "organism as a whole . . . possesses goals of its own." My position on these matters is that organisms have goals that are not necessarily the goals of their genes, and genes, considered as strategic groups of material tokens, can possess goals that are not necessarily goals of the organisms in which they temporarily reside. This is the tension between selfish genes and social selves expressed in *From Darwin to Derrida*'s subtitle.

Is this teleology internal or external? I think of genes and organisms as possessing immanent teleology—an agent's ends reside within itself—but these ends have been shaped by the environment and are responses to that environment. The drama of evolution by natural selection is this interplay between internal and external change. Can we rephrase the question?

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Part VI

The Darwinian Core of Evolutionary Theory and the Extended Evolutionary Synthesis: Similarities and Differences

17

T. N. C. Vidya, Sutirth Dey, N. G. Prasad, and Amitabh Joshi

Abstract

In this chapter, we evaluate debates surrounding calls for an Extended Evolutionary Synthesis in light of the Darwinian core of evolutionary theory, which was somewhat broader than the Modern Synthesis. We suggest that Darwin's nuanced operationalization of natural selection rested upon two innovations: the atomization of individuals into trait-variants, and a reconceptualization of heredity in terms of transmission of trait-variants. Darwin also implicitly differentiated between the causes and consequences of selection, noting that while selection acts on individuals, it is actually trait-variants that are consequently differentially transmitted, and the species that is eventually modified. This is important because the individual, with inherencies and agency, is largely relevant only when examining the causes of selection, with trait-variants being the more appropriate

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unit for studying its consequences. Consequently, we emphasize the importance of restricting the use of 'fitness' to one-step change in trait-variant frequency, instead of also using it for lifetime reproductive success of individuals, or even trait-variants. Fitness, thus defined, is always inclusive, circumventing much unnecessary debate. We also present a schematization of explananda in evolutionary biology and suggest a framework for the comparative evaluation of factors affecting evolutionary change. We further suggest that the controversial 'gene's eye view of evolution' is best seen as not one, but two distinct views, one Fisherian and the other Dawkinsian, and that conflating them has led to considerable unnecessary debate. In conclusion, we suggest that it is helpful to view received evolutionary thought as an evolving set of explanations, intertwined with one another to varying degrees, rather than a distinct, static Modern Synthesis. This leads to our viewing various processes and factors affecting the origin, dynamics and patterns of prevalence of variants at various levels of biological organization, as representing differing but complementary parts of a complex, nuanced, multifarious and evolving standard evolutionary theory.

Keywords

Modern synthesis · Natural selection · Trait-variants · Darwinian fitness · Reproductive output · Transmission fidelity · Transmission efficiency · Gene's eye view of evolution

17.1 Introduction

In this chapter, we discuss some issues that often come up in the context of debates between the supporters of the Modern Synthesis (henceforth, MS) and the Extended Evolutionary Synthesis (henceforth, EES). These issues primarily pertain to the (i) role of the individual in evolutionary explanations, (ii) nature(s) of Darwinian fitness (henceforth, fitness), (iii) often neglected non-genetic interpretations of quantitative genetic theory, (iv) relationships between different evolutionarily important factors and (v) role of development in evolutionary explanations. When discussing contentious issues, it is helpful to be clear about one's potential biases, points of view and focus at the outset. Our backgrounds are in various areas of biology, and all four of us are empirical evolutionary biologists also interested in, and engaged with, theory. Between us, we work primarily on the evolution of life histories, dispersal, social organization, adaptations to crowding, population stability and sexual conflict. One of us works primarily in the wild, on large mammals and birds, while the other three work with laboratory systems of microbes or dipterans. We also share an interest in many issues in the history and philosophy of evolutionary biology. More to the point, all of us would self-identify as having been trained, and presently working, within the broad framework of the MS. Nevertheless, we are appreciative of, and sympathetic to, many aspects of the calls for an EES, although we find some aspects of the EES discourse—both on history and mechanisms—to be

somewhat muddled and often overhyped. Here, we discuss some aspects of the EES-MS debate in the historical context of the changes in evolutionary thinking from Darwin's times to the present. We also try to place this ongoing debate within the broader context of what evolutionary biology needs to explain. In this attempt, we have tried, as far as possible, to set our biases aside and follow the sentiment expressed in this Urdu couplet by Nabraas Akbarabadi:

کھیل ہے یہ سب نظریے کا، اے بیخُد، چھوڑ دے
ہر نظریے کا تقاضہ توڑ کر بن جا نظر
khel hai ye sab nazariye ka, ai Bekhud, chhor de
har nazariye ka taqaaza tor kar ban ja nazar
(Leave aside illusions born of many different points of view:
Break the shackles of perspective, be vision personified!)

Although discomfort with the perceived restrictive nature of some of the views that were eventually codified in the MS of the mid-twentieth century was intermittently articulated, both before (e.g., Bateson 1894; de Vries 1905; and the work of Woltereck, Nilsson-Ehle, Johannsen, Romashoff and Timoféeff-Ressowsky, discussed in Sarkar 1999, 2006) and after the synthesis (e.g., Goldschmidt 1940; Waddington 1953; Eldredge and Gould 1972; Gould and Lewontin 1979; Stanley 1979; Dey and Joshi 2004), the present form of calls for an EES took clearly discernible shape only in the early twenty-first century, roughly coinciding with the publication of 'Evolution: the Extended Synthesis' by Pigliucci and Müller (2010). The EES, which is claimed to be a significant extension to the mid-twentieth century MS, is an umbrella term used to cover at least four somewhat distinct, though overlapping, aspects of evolutionary thinking: (1) an additional focus on non-genic inheritance, including epigenetic, cultural and ecological inheritance; (2) supposedly novel conceptualizations of evolutionary forces, such as niche construction and developmental or mutational bias; (3) a rethinking of the logical status of various evolutionarily important factors, including natural selection, niche construction and developmental or mutational bias; and (4) a renewed emphasis on keeping the individual organism, with inherency and agency, at the centre of evolutionary thinking (Laland et al. 2015; Newman 2022a, b). Over the past decade or more, there has been considerable debate about many of the claims made by EES proponents (e.g., Laland et al. 2014; Gupta et al. 2017a, b; Feldman et al. 2017; Charlesworth et al. 2017; Svensson 2018; Dickins and Dickins 2018; Buskell 2019; Lewens 2019; Dickins 2021). In general, these arguments juxtapose EES with MS, and there is as yet no general consensus on whether EES marks a seriously consequential extension to the MS, or whether the phenomena highlighted by EES are readily accommodated within the MS.

In this chapter, we examine various aspects of the EES-MS debate by focussing on what we label the Darwinian Core of evolutionary theory (DC), encompassing the views of Darwin on evolution as contained in his books and other writings. In our delineation of the DC, we emphasize not only aspects that are very well recognized, such as the assertion that natural selection is the major driver of adaptive evolution, but also important aspects that have often not received much attention, such as why

development was relegated to the periphery of evolutionary explanation by Galton (1872), long before the marginalization of development from heredity by Morgan (1926) and others (the latter discussed by Sarkar 2006), and how the atomization of the individual (*sensu* Gould and Lewontin 1979) was actually a largely unrecognized but nevertheless fundamental component of what Mayr (1955, 1959, 2004) regarded as one of Darwin's greatest contributions, and somewhat controversially termed 'the shift from typological to populational thinking' (for detailed critiques of this interpretation by Mayr, see Greene 1992; Amundson 1998, 2005; Winsor 2006a, b; Hey 2011). The point we wish to make is that there are not just many similarities but also quite a few differences between DC and MS, with the latter representing a slightly narrower conceptualization of the evolutionary process. We use the term MS, adopted from the title of Huxley's (Huxley 1942) book, to refer to the consensus view of the key elements of the evolutionary process—putting together insights from Mendelian genetics, cytogenetics, population and quantitative genetics, studies of genetic and chromosomal variations in nature, natural history, systematics and palaeontology—that crystallized during the period between 1918 and 1950 (Rao and Nanjundaiah 2017; Sarkar 2017). The MS, it should be noted, was slightly broader than Neo-Darwinism, a view of evolution heavily influenced by the views of Weismann (1889, 1893a, b, 1902) on the primacy of natural selection and the impossibility of the inheritance of acquired characters, that developed in the few decades after Darwin (Reif et al. 2000). Unfortunately, the term Neo-Darwinian Synthesis was also often used later as a synonym for MS, potentially creating confusion for one first encountering this literature (Reif et al. 2000).

At this point, we would also like to take a step back and take a broader view of the domain of evolutionary biology and locate the issues discussed in this chapter in the context of that bigger picture. Evolutionary biology attempts to provide explanations for the hitherto puzzling observations of the 'relatedness of species, diversity of species, and adaptedness of species' (Rose 1998). Darwin (1859, 1868, 1871), to varying degrees, provided explanations for all three of these ubiquitous observables of the living world: for relatedness and diversity through descent with modification, with selection playing a role in promoting diversification of new species, and for adaptedness through selection. Darwin, however, focussed disproportionately on explaining adaptedness, possibly because that was the aspect often stressed when arguing for the role of a creator in the origin of life forms (e.g., Paley 2008). Adaptedness was, in fact, a principal concern of the uniquely British natural theology tradition, and this might explain why subsequent British evolutionists have given far more attention to explaining adaptation compared to, say, the origins of form (Ågren 2021).

We elaborate upon the tripartite explanandum above, to list out some overarching categories of issues that evolutionary biology must address, and to locate the DC, MS and EES within this schema to better examine their inter-relationships. Mirroring the dichotomous categories of microevolution and macroevolution, potentially bridged by speciation, we examine how evolutionary biology needs to explain issues of origin, increase and persistence of phenotypic variations that give rise to observed spatio-temporal patterns of variations at different levels of biological

Table 17.1 One way of categorizing the major explananda that need to be addressed by a science terming itself evolutionary biology (see text for details). We will henceforth refer to these six categories by their combinatorial labels, '*micro/macro-origins/dynamics/patterns*'

	Attributes and consequences of phenotypic variations		
Nature of phenotypic variations	1. Origins	2. Dynamics of relative abundance	3. Resultant patterns in time and space
A. Macro-evolutionary	1A) '*macro-origins*' How do macro-evolutionary variants (forms) arise? Are certain variants more/less likely to occur in different contexts?	2A) '*macro-dynamics*' What are the factors/mechanisms affecting the dynamics of relative abundance of different macro-evolutionary variants (forms) over a given time span?	3A) '*macro-patterns*' How do 1A and 2A result in different spatio-temporal patterns in the diversity of macro-evolutionary variants (forms)?
B. Micro-evolutionary	1B) '*micro-origins*' How do micro-evolutionary trait-variants arise? Are certain variants more/less likely to occur in different contexts?	2B) '*micro-dynamics*' What are the factors/mechanisms affecting the dynamics of relative abundance of different micro-evolutionary trait-variants over a given time span?	3B) '*micro-patterns*' How do 1B and 2B result in different spatio-temporal patterns in the diversity of micro-evolutionary trait-variants within species?

organization. For our purposes, we find it helpful to think of six such categories of issues that any science calling itself evolutionary biology needs to address, three each for macro-evolutionary and micro-evolutionary phenotypic variations, respectively (Table 17.1).

Like most schemata in biology, this is a fuzzy rather than a clearly and unambiguously delineated organization of explananda. We believe, nevertheless, that this is a useful schema, and one to which we will return repeatedly. Here, we explain the sense in which we are using some of these terms and make a few general points about how different types of evolutionary explanation map onto this schema.

We use the term macro-evolutionary phenotypic variations to refer to the appearance of either new traits altogether, e.g., horns in a hitherto hornless species, or new variants of existing traits that are well beyond the known range of distribution of trait-variants of that trait, e.g., a phytophagous insect that can utilize a novel food plant species belonging to a different angiosperm family than the plants normally used by that insect species. This is also sometimes termed the appearance of novel forms (e.g., Carroll 2005). By trait-variants, we mean alternative versions of a trait; our usage mirrors the sense in which Darwin (1859, 1868) used the terms 'characters' and, more frequently, 'variations'. By micro-evolutionary phenotypic variations we mean the appearance of new trait-variants of an existing trait close to, or within the range of known variation in that trait. Since speciation, according to the biological species concept for obligate sexually reproducing species, involves

reproductive isolation as a definitional criterion (e.g., Howard and Berlocher 1998), we note that species defined thus can originate through, and be separated by, either micro- or macro-evolutionary variations.

All the six categories above encompass elements of both process and pattern, albeit to considerably varying degrees. The categories '*macro-origins*' and '*micro-origins*' include considerations of how new variations arise at different levels, yielding novel forms or trait-variants, as also those of patterns in how various variations differ in the likelihood of their arising at a given time and population. In recent times, diverse investigations spanning both these categories are often integrated into discussions of the origins of evolutionary innovation (e.g., Erwin 2021). The categories '*macro-dynamics*' and '*micro-dynamics*' include considerations of the time-dynamics, across varying time scales, of absolute or relative numbers, biomass, or other relevant measurables, of alternative macro- or micro-evolutionary variations within an ensemble, including persistence or extinction. The categories '*macro-patterns*' and '*micro-patterns*' focus on the patterns in the distributions of macro- and micro-evolutionary variations across space and time that result from processes under '*macro-origins*' and '*macro-dynamics*', and under '*micro-origins*' and '*micro-dynamics*', respectively. It should be noted that chance plays a role in practically all the processes across these six categories. We speculate that the separation between changes happening at these two levels of variation—micro- versus macro-evolutionary—was perhaps even more blurred during the early stages of the evolution of life on earth, and perhaps still is today in protists and monerans that exhibit a level of organismal complexity that is similar to what was probably the case in early evolution. In simple, often unicellular, species, it is likely that far more mechanisms are shared between '*macro-origins*' and '*micro-origins*', and under '*macro-dynamics*' and '*micro-dynamics*', respectively, than is the case in more complex metazoans.

We further note that evolutionary biology since Darwin has addressed these six categories in an uneven manner. The category '*macro-origins*' is presently largely the domain of evo-devo and was relatively neglected for several decades in the twentieth century (Amundson 2005). Although we now better understand how developmental genetic networks (e.g., Salazar-Ciudad et al. 2001; Salazar-Ciudad 2009) and also non-genetic, often physical, properties of cells and organisms (e.g., Salazar-Ciudad et al. 2003; Newman and Bhat 2009; Bhat et al. 2016; Tickle and Urrutia 2016; Newman 2021, 2022a, 2022b) can shape the origins of new forms, the level of detail and generality with which we understand issues in this category of explaining origins of variation is somewhat less than that in the category '*micro-origins*'. Discussions of developmental bias, developmental constraints and the role of development in shaping the morpho-space anisotropically (e.g., Salazar-Ciudad 2021) also fall largely within '*macro-origins*', although they are also conceived of as affecting processes and outcomes here categorized as '*macro-dynamics*' and '*macro-patterns*', as part of a perspective primarily informed by palaeontology, systematics and biogeography in the past, and supplemented today by molecular phylogenetics, phylogenomics and phylogeography. It is also worth stressing here that selection is an important, though by no means only, process particularly in

'*micro-dynamics*' phenomena and, therefore, helps shape patterns mostly in the category '*micro-patterns*'. It is not clear how significant a role selection plays as a '*macro-dynamics*' process, although it is likely to be far less pervasive than its role as a '*micro-dynamics*' process (Newman 2022a).

Our understanding of the origin of novel trait-variants ('*micro-origins*') has progressed quite a bit since Darwin's unsuccessful attempts to grapple with this vexed issue through his theory of pangenesis (Geison 1969; McComas 2012). A large proportion of the explanations for '*micro-origins*' phenomena derives from genetics, involving both mutations in the broadest sense, including chromosomal changes and changes in gene expression (e.g., Dobzhansky 1937; Graves Jr. et al. 2017; Seabra et al. 2018; Fitzgerald and Rosenberg 2019; Barter et al. 2020; Dowle et al. 2020), as well as the recombinational shuffling of standing genetic variation, especially for quantitative traits (Teotónio et al. 2009; Mueller et al. 2013; Matuszewski et al. 2015; Phillips et al. 2018; Hickey and Golding 2021; Kawecki et al. 2021). The now fairly well-accepted role of phenotypic plasticity in preceding and facilitating adaptive evolutionary change (discussed in detail in Pfennig 2021) also has a bearing on issues in the category '*micro-origins*'. There is also, more recently, input from evo-devo towards understanding the origins of the kind of variation relevant to micro-evolutionary change, variously termed devo-evo (Prasad and Joshi 2003; Joshi 2005) or micro-evo-devo (Nunes et al. 2013). Issues in the category '*micro-dynamics*'—the domain of classic micro-evolutionary dynamics as affected principally by mutation, migration, selection and drift—are by far the most studied and well-understood, compared to those in the other five categories of our schema in Table 17.1. The category '*micro-dynamics*' is also largely the only category that Darwin's (1859, 1868, 1871) work successfully addressed in any detail, through his enunciation of the principle of natural selection; his attempt to illuminate '*micro-origins*' processes via pangenesis did not persist very long. The bulk of the work done on '*micro-dynamics*' issues today lies within the domain of population genetics and genomics, quantitative genetics, and ecology, in particular evolutionary ecology. The category '*micro-dynamics*' was also the primary focus of the MS, although it also attempted to incorporate issues of speciation into its purview. Issues in the category '*micro-patterns*' have also been covered in a lot of MS work, in conjunction with '*micro-dynamics*' issues. One unfortunate consequence of the preponderance over time of '*micro-dynamics*' and '*micro-patterns*' explanations, compared to most other categories in this schema, has been the tendency of textbooks of evolution to often convey the impression that the issues dealt with under these two categories essentially cover a very large part of the domain of evolutionary explanation.

The origin of species, despite the eponymous title of his book, was not really addressed by Darwin (1859) at all, save to express the hopeful view (his 'principle of divergence') that, consonant with his uniformitarian beliefs, '*micro-dynamics*' processes would, over long spans of time, aided by geographical separation, result in the origin of new species and, thus, eventually result in variations of the category '*macro-origins*'. Not surprisingly, given the conceptual centrality of species as a category in many areas of biology, speciation—a term coined by Cook (1906)—has

attracted the interest of researchers from evo-devo, systematics, palaeontology, phylogeography, ecology, population genetics, and quantitative genetics, largely during the second half of the twentieth century (e.g., Mayr 1982; Koeslag 1995; Howard and Berlocher 1998; Gavrilets 2003; Baker 2005; Via 2009). Patterns in species diversity can result from a complex interplay of '*micro-patterns*' and '*macro-patterns*' processes, but work on these issues has not been as extensive as that on speciation (e.g., Cracraft 1982; Vrba 1984; Jablonski 2008), perhaps because processes in the categories '*macro-origins*' and '*macro-dynamics*' are not as well characterized as those in the categories '*micro-origins*' and '*micro-dynamics*'. We have preferred to ignore the category of species in our categorization of phenotypic variation (Table 17.1) because phenotypic variation both within- and among-species can span from micro-evolutionary to macro-evolutionary. Therefore, for example, a possible categorization of variation within species, across species and higher taxa would tend to obfuscate an appreciation of processes acting on substantially different types of variation.

We next briefly explain our priorities in, and motivation for, writing this piece, harking back to the poetic sentiment expressed in the first paragraph of this section. In terms of the schema in Table 17.1, three of us (SD, AJ & NGP) work almost entirely within the domain of '*micro-dynamics*', whereas TNCV works primarily within the '*micro-dynamics*' and '*micro-patterns*' categories. We have chosen not to comment on every aspect of the EES-MS debate in detail, especially those already extensively and clearly discussed in the literature, preferring to focus on a sub-set of issues that we believe are neglected, or at least under-appreciated, in this debate. We agree that inheritance, especially cultural and ecological inheritance, can often be non-genetic (e.g., Jablonka and Lamb 2005; Helanterä and Uller 2010; Danchin et al. 2011, 2019; El Mouden et al. 2014; Prasad et al. 2015; Bonduriansky and Day 2018; Jablonka and Noble 2019; Adrian-Kalchhauser et al. 2020), and that both these forms of inheritance have a major role to play in the evolutionary process. Although there is now some evidence for trans-generational epigenetic inheritance (e.g., Jablonka and Raz 2009; Klosin and Lehner 2016), as well as for its underlying mechanisms (Fitz-James and Cavalli 2022), in a few systems, we think that it would be premature to attempt an assessment of how common or rare it is in nature. Moreover, there is relatively little evidence as yet for persistent trans-generational epigenetic inheritance over large numbers of generations. We agree that extended phenotypes and niche construction are important phenomena in evolution, but we disagree with many of the claims made by niche construction proponents. This last issue has been already discussed in detail, and we refer the interested reader to a triptych of critique, response and counter-response (Gupta et al. 2017a, b; Feldman et al. 2017). Over the past two decades, we have been, almost in equal measure, excited, enlightened, frustrated and disappointed by various facets of the EES literature. We believe that at least some of the issues under debate lack the level of clarity one would have hoped for, while others have not been discussed in sufficient detail, or at all. Our approach in this chapter, consequently, is that of a metaperspective (*sensu* Hester and Adams 2014) rather than a review, although we have also tried to provide a reasonable and eclectic, though by no means exhaustive,

entry into the relevant literature. We hope that our efforts will contribute to some enhancement of the clarity with which we, as a community, describe, discuss and debate the structure of evolutionary thought.

17.2 The Darwinian Core (DC) of Evolutionary Theory

We have been guided in writing this chapter by a belief that a deeper and more nuanced appreciation of the past often facilitates an improved understanding of the future, eloquently expressed by Allama Iqbal thus:

سامنے رکھتا ہوں اِس دورِ نِشاط افزا کو میں
دیکھتا ہوں دوش کے آئینے میں فردا کو میں
saamne rakhta hoon is daur-e-nishaat-afzaa ko main
dekhta hoon dosh ke aaine mein fardaa ko main
(The golden age that has gone by, is always in my heart and mind;
And in that mirror of the past, I see the future times outlined)

In this spirit, we now outline some aspects of the DC that we think have not received as much attention as they should have. Darwin (1859) is universally acclaimed for two major contributions that comprehensively changed biological thinking: (i) marshalling a compelling body of evidence for the occurrence of evolutionary change, which has never been seriously doubted since, thereby explaining why species and higher taxa appear to be connected by genealogical relationships, and (ii) providing a potent mechanism—natural selection—for adaptive evolutionary change. These two contributions went a long way towards explaining the diversity, relatedness and adaptedness of species, even though Darwin's 'hypothesis of natural selection', unlike his 'hypothesis of descent', gained widespread acceptance only several decades after it was first put forward (Gayon 1998). Another important contribution of Darwin's, according to Mayr (Mayr 1955, 1959), was to usher in a shift from typological or essentialist thinking to populational thinking among biologists. This assertion of Mayr's was strongly critiqued, and it was pointed out that most influential biologists pre-Darwin were largely not essentialist in their thinking (Greene 1992; Amundson 1998, 2005; Winsor 2006a, b; Hey 2011). Mayr (2004), however stuck to his claim, and this assertion of a shift in thinking, after Darwin, from typological to populational mode is still commonly encountered in textbooks, and in books on evolution aimed at a general audience (e.g., Rose 1998). We suggest that the relevant shift in Darwin's thinking was actually one from typological/essentialist to variational mode in the very limited context of how hereditary transmission mediates selection. We return to this issue after mentioning what we think are some very major and unappreciated contributions of Darwin to evolutionary thought, because this shift from a typological to variational understanding of heredity is intertwined with both contributions.

In our opinion, neither the notion of descent with modification, nor that of selection, by itself qualifies as a profoundly novel intellectual contribution by Darwin, though both were undoubtedly important and consequential. The general

idea of descent with modification had been expressed frequently in Europe, in both biological and general circles, over the century preceding Darwin (Freeman and Herron 2013). Similarly, notions approximating the idea of natural selection to varying degrees can be seen, over a span of about 2300 years, in the writings of Empedocles (Gottlieb 2000), Lucretius (Campbell 2003), Nasir al-Din Tusi (Alakbarli 2001), and, closer to Darwin and Wallace's time, of W. C. Wells and Patrick Matthew (Freeman and Herron 2013), and of H. G. Bronn, unfortunately known to much of the Anglophone world only as the translator of Darwin's (1859) book into German (Gliboff 2008). Indeed, in a footnote on the first page of 'An historical sketch of the progress of opinion on the origin of species previously to the publication of the first edition of this work', added as a preface to most editions (after the second) of *The Origin of Species*, Darwin quotes Aristotle, who himself is paraphrasing Empedocles only to disagree with him, and notes that, '*We see here the principle of natural selection shadowed forth*'.

We believe that, more than the idea of natural selection, it was its operationalization by Darwin in a particularly useful manner that constituted a very significant and novel intellectual contribution. This operationalization, in turn, rested upon two major conceptual innovations: the atomization of the individual into traits, and the reconceptualization of heredity as needing to explain not only the perpetuation of holistic types but also the generation and transmission of trait-variants. These innovations of Darwin's are often erroneously ascribed to either genetics as it emerged in T. H. Morgan's lab, or the MS (e.g., Gould and Lewontin 1979; Allen 1985; Amundson 2005). We note that these same two conceptual innovations also informed the work of Mendel around the same time. Not surprisingly, therefore, these innovations eventually led to development being excluded from explanation of both transmission genetics and micro-evolutionary change. This novel conceptualization of heredity, influenced by the experience of breeders, was one of Darwin's most unappreciated contributions to the discipline that later became known as genetics. We will discuss the consequences of Darwin's atomization of individuals and reconceptualization of heredity for how we conceive of and use the notion of fitness in a later section. Here, we focus on Darwin's reconceptualization of heredity and then delineate what we believe to be the constituents of the DC.

Darwin's thinking on heredity and evolution was influenced by natural history, biogeography, systematics, medicine and breeding. The first three influences were reflected in his setting up of the problem of evolution by recognizing that species and higher taxa appeared to be connected by genealogical relationships and, moreover, appeared to be well adapted to their respective ecological contexts and lifestyles. His solution to the problem—the mechanism of natural selection—was almost entirely inspired by analogy to breeding (in sharp contrast to Wallace, who did not believe domesticated animals to be relevant to understanding natural selection: Gayon 1998), whereas his views on heredity were influenced by developments in both medicine and breeding, especially in the late eighteenth and early nineteenth centuries (a detailed account of thinking about heredity in this period can be found in the papers in Rheinberger and Müller-Wille (2003), and Müller-Wille and Rheinberger (2007)). Prior to the late eighteenth century, ideas pertaining to heredity

were vague and diffused, with no specific focus on the transmission of variations, and heredity was considered inseparable from reproduction, thus falling within the domain of embryology. A corollary to this was that heredity was viewed primarily as ensuring the stability of the type via the transmission of similarities that unified all individuals of a species or variety. Elements of this view lingered on into the late nineteenth century alongside more specific conceptualizations that viewed heredity, in the sense of transmission, as a phenomenon distinct from embryology and physiology (Churchill 1987), and both approaches to the vexed problem of heredity can be seen in Darwin's writings.

The key conceptual developments in the study of heredity before Darwin, which culminated in the early- to mid-nineteenth-century writings of Prosper Lucas (Kendler 2021) and Imre Festetics (Szabó and Poczai 2019), were a focus on the transmission of variations to offspring, as well as a conceptualization of heredity as a distinct phenomenon, with its own 'laws', requiring to be explained in its own terms, rather than as a subsidiary component of reproduction, physiology, or embryology (Churchill 1987; López-Beltrán 1992, 2003; Wood 2003; Szabó and Poczai 2019; Kendler 2021). The significance of these developments for the subsequent study of both genetics and evolution is reflected in the fact that both Darwin and Mendel independently realized the importance of atomizing individuals into trait-variants, and treating the transmission of trait-variants as distinct and independent of their expression. It is now known that Mendel was aware of Darwin's work in considerable depth (Fairbanks 2020), but not vice versa (Sclater 2006), and there is no evidence that they were influenced by each other on the related issues of atomization and distinguishing the transmission of trait-variants from their phenotypic manifestation.

Darwin (1859, 1868, and essays printed in Darwin 1909) was interested in both the origin and transmission of trait-variants, and considered a spectrum of types of possible variation within a species, ranging from the continuous, effectively rendering each individual unique, through small discontinuous but widespread variations, implying that many individuals in a population could share very similar trait-variants that might be advantageous under some environments, to discontinuous 'sports' of larger effect, arising in one or a few individuals (Bowler 1974). In the absence of any clear knowledge of the mechanism by which variations arose, he believed axiomatically that variations were ubiquitous, generated almost continuously, and typically heritable (Bowler 1974; Gayon 1998). Moreover, Darwin (1859, 1868) believed variations to arise from the interaction between an organism and its environment, with a subsequent inheritance of the acquired characters. Darwin's writings on heredity are often somewhat vague and muddled, even self-contradictory at times, especially when he explores the relationship between the origin, expression, and transmission of trait-variants, and, therefore, between development and heredity, in his theory of pangenesis (Geison 1969; Gayon 1998; McComas 2012). This is undoubtedly because he was grappling with fundamental issues and concepts for which there was little empirical support, and which were imbued by much confusion at the time. Unfortunately, this ambiguity means that one can usually find specific

quotes from Darwin's writings that can be deployed to support whichever side of the development-heredity argument one wishes to bolster.

It has often been suggested that Darwin treated development and hereditary transmission as a unified whole (e.g., Winther 2000; Amundson 2005), but we suggest that this is a misrepresentation, especially if we examine Darwin (1868), and not just Darwin (1859), as also argued persuasively by Gayon (1998). Basically, Darwin (1868) rested his case for treating development separately from the transmission of trait-variants to offspring (heredity, in his words) on the phenomenon of reversion, or atavism, which refers to a character in a pedigree often skipping one or more generations before 'reappearing'. Darwin interpreted this not so much as evidence for reversion to a varietal type, as was common at the time, but rather as a strong indication that individuals were mosaics of characters (trait-variants), some expressed during development, and others latent. Latent characters, though not expressed were, nevertheless, transmissible to offspring, suggesting that development and heredity could be delinked. Summing up, Darwin (1868) wrote that, therefore, reversion '... proves to us that the transmission of a character and its development, which ordinarily go together and thus escape discrimination, are distinct powers...'. This argument for the separation of development and hereditary transmission of trait-variants was later reinforced even more explicitly and graphically by Galton (1872). The notion of individuals being a mosaic of trait-variants, of course, also arises naturally from the experience of breeders. This view was developed clearly by Darwin (1859, 1868; also see his 1844 essay in Darwin 1909, and some of his writings collected in Barrett 1977), emphasizing that the breeders' adage of 'like begets like' was not an expression of the conservative perpetuation of an overall varietal 'type' during reproduction (e.g., a crow gives rise to a crow), but rather a statement of the heritable nature of preferred trait-variants among individuals within a variety that could be independently selected for (e.g., one can successfully select for larger beak size in a given variety of crows). The separation of development from heredity should not, however, be taken to imply that Darwin considered development to be unimportant to evolution, as opposed to its being irrelevant to understanding the transmission of trait-variants from parents to offspring, and its evolutionary consequences. When considering large scale variations among related taxa within lineages, Darwin (1859) focused on changes in ontogeny, drawing on the tradition of comparative morphology and embryology, and even tried to interpret the principle of recapitulation in terms of differing selection pressures acting on different stages of the ontogeny. Moreover, Darwin also noted that growth correlations—his term for correlations among traits resulting from developmental processes operating during ontogeny—could cause micro-evolutionary change without selection, as well as constrain the ability of selection to effect micro-evolutionary change.

When discussing selection and the gradual modification of species or varieties by the accumulation of variations, however, Darwin retained his primary focus on specific, largely independent, and small-scale trait-variants (Howard 2009; Deichmann 2010), and also emphasized that bearing favourable trait-variants essentially improved the chance that an individual would survive better and reproduce

more than others who did not bear those trait-variants (Darwin 1859, 1868; also see his 1844 essay in Darwin 1909, and some of his writings collected in Barrett 1977). As pointed out by Gayon (1998), Darwin seems to have clearly seen that while selection acts on individuals within a species or variety, it is actually trait-variants that are consequently differentially transmitted to the offspring generation, and the species or variety that is eventually modified by the differential accumulation of subsets of trait-variants over generations. This is a view that has successfully withstood the test of time, and also highlights the substantial difference between Darwin's conception of selection and those of Alfred Russel Wallace (Darwin and Wallace 1858), who thought that selection acted primarily between varieties rather than individuals, or Herbert Spencer (1893), who conceived of selection as acting on individuals considered as a whole rather than on atomized trait-variants. There are some further aspects of Darwin's very nuanced conception of selection that are worth mentioning. Darwin (1859, 1868, 1871) clearly realized that selection acted on individuals that differed in their reproductive success, and that reproductive success could be achieved through better survival or greater reproduction, or both. Linking the relative reproductive success of individuals bearing different subsets of trait-variants to whether those trait-variants would tend to increase or decrease in the offspring generation was the intervening process of hereditary transmission. As Darwin put it in his essay of 1844 (reproduced in Darwin 1909), 'Can it be doubted, from the struggle each individual has to obtain sustenance, that any minute variation in structure, habits, or instincts, adapting that individual better to the new conditions, would tell upon its vigour and health? In the struggle, it would have a better chance of surviving; and those of its offspring which inherited the variation, be it ever so slight, would also have a better chance. Yearly more are bred than can survive; the smallest grain in the balance, in the long run, must tell on which death shall fall, and which shall survive. Let this work of selection on the one hand, and death on the other, go on for a thousand generations, who will pretend to affirm that it would produce no effect, when we remember what, in a few years, Bakewell effected in cattle, and Western in sheep, by this identical principle of selection'. Thus, the hereditary transmission of trait-variants was a crucial component of selection, together with the twin struggle for survival and mates. Not having any knowledge of the mechanisms of heredity, Darwin was essentially agnostic regarding the origin of trait-variants but assumed that the transmission fidelity (Box 17.1) of trait-variants was sufficiently high that it ensured that greater reproductive success, on an average, of individuals bearing a particular trait-variant in one generation would translate into an increased representation of that trait-variant in the offspring generation. Darwin, moreover, also appreciated that selection could operate through the enhanced reproductive success of close relatives, rather than that of the individual under consideration, and offered this insight as a possible explanation for the seeming paradox of altruistic sterility in honeybee workers (Darwin 1859).

Box 17.1 Transmission Fidelity and Change in Frequency of Trait-variants

Darwin's conception of natural selection can be viewed as an algorithm that maps the ecological success of trait-variants in the struggle for existence, as reflected in their reproductive output, on to the evolutionary success of those trait-variants, as measured by a one-generation change in relative abundance, through the intervention of heredity. Thus, it is heredity, gene-based or otherwise, that drives the degree of concordance between relative reproductive output of a trait-variant and the one-generation change in its relative frequency. A reasonably strong concordance between ecological and evolutionary success is required for selection to result in adaptive evolutionary change. Key to this role of heredity is the degree to which offspring resemble their parent(s) with regard to the trait under scrutiny. In this context, we define transmission fidelity using a simple example of discrete generation uniparental inheritance, with no difference in survival to reproduction and total offspring production among individuals exhibiting different variants of that trait.

Let there be m possible variants of a trait among individuals in a population, with frequencies $0 \leq f_i \leq 1$ ($i = 1..m$). Upon reproduction, assume that individuals exhibiting the i^{th} trait-variant produce, on an average, a fraction x_{ii} of their offspring exhibiting the same trait-variant, with the remainder $(1 - x_{ii})$ exhibiting one of the other trait-variants, potentially including those with zero frequency in the parental generation. Then, $0 \leq x_{ii}\ 0 \leq 1$ is the transmission fidelity of the i^{th} trait-variant.

Next, consider the frequency of the i^{th} trait-variant in the next generation. This will depend upon not just the transmission fidelity of individuals exhibiting the i^{th} trait-variant, but also the frequency of the i^{th} trait-variant among the offspring of individuals exhibiting all other trait-variants. Let x_{ij} be the probability that an individual exhibiting trait-variant i in the parental generation produces an offspring exhibiting trait-variant j ($j = 1..m$, but $\neq i$); clearly $\sum_{j \neq i} x_{ij} = 1 - x_{ii}$. Then, the frequency of the i^{th} trait-variant in the next generation will be given by $f'_i = f_i x_{ii} + \sum_{j \neq i} f_j x_{ji}$. The point to be noted is that, even in this simple example with constant x_{ii} and x_{ij} over generations, and equal survival to reproduction and total offspring production by all individuals, regardless of which trait-variant they exhibit, the frequency of a trait-variant in the next generation will depend not just on its frequency in the previous generation and its transmission fidelity, but on the frequency of all other trait-variants in the previous generation and their respective probabilities of producing offspring exhibiting the focal trait-variant, which partly depend on their respective transmission fidelities.

In more realistic scenarios, changes in the frequency of trait-variants will be driven by differences among trait-variants in survival to reproduction, as well as in reproductive output. Moreover, for many inheritance systems, including

(continued)

Box 17.1 (continued)
the familiar gene-based Mendelian one, both x_{ii} and x_{ij} will often be frequency-dependent and, thus, liable to change over generations as frequencies of trait-variants change. Transmission fidelities may also change based on the mating system, i.e., random mating, assortative mating, inbreeding, etc. In general, high transmission fidelities will result in positive correlations between ecological and evolutionary success of trait-variants. Conversely, for many sets of x_{ii} and x_{ij} values, ecological and evolutionary success of trait-variants can be uncorrelated or even negatively correlated, thus generating the possibility that transmission fidelity patterns in some cases might even negate the effects of higher reproductive output on frequency increase. These points are elaborated further in Box 17.2.

While he believed that selection acted on individuals most of the time, Darwin (1859) did not rule out the possibility, especially in social animals, of selection tending to increase the representation in a species of trait-variants that enhanced the survival and persistence of social groups. One can view Darwin's conception of natural selection as providing a sort of algorithm which can accommodate multiple component mechanisms in varying contexts, rather than a specific mechanism. This algorithm, independently of the underlying mechanisms, serves to map the ecological success of trait-variants in the struggle for existence, as reflected in their reproductive output, on to the evolutionary success of those trait-variants, as measured by a one-generation change in relative abundance, through the intervention of heredity, as long as heredity ensures reasonably high transmission fidelity of the trait-variant in question. This crucial role of heredity in mediating adaptive evolutionary change can also be thought of as linking the causes of selection to their consequences for the composition of a population (*sensu* Joshi 2005). Essentially, Darwin's conception of selection has not really been improved upon in the next one and half centuries except to apply its logic to phenomena unknown in Darwin's time, such as meiotic drive or transposable genetic elements, or to add mathematical detail to our appreciation of its consequences. It is in this sense that we think that, more than just the idea of natural selection, it was Darwin's nuanced elucidation of the myriad ways in which it could operate that constituted a major intellectual innovation.

In light of the above discussion, we now list what we believe to be the important constituents of what we refer to as the DC, in language more in consonance with our times than Darwin's.

1. Species arise from pre-existing species (descent with modification) and can diverge from one another over time (principle of divergence), thus explaining the diversity and relatedness of species.
2. Heredity must explain not just the transmission of similarities common to all members of a species, but also the transmission of individual trait-variants.

3. Individuals can be usefully thought of as a mosaic of reasonably independent trait-variants. Trait-variants are ubiquitous, arise almost continuously, in ways affected by the environment, and tend to be transmissible to offspring with fairly high and similar fidelity.
4. Organisms are typically involved in a struggle for existence, involving competition for resources, refuge from enemies, and mates. Certain trait-variants can confer advantages in this struggle to the individuals bearing them, or to relatives of those individuals.
5. Ecological success in the struggle for existence is ultimately measured by offspring production (also termed reproductive success). Though it is individuals that reproduce, one can meaningfully consider the average reproductive success of all individuals bearing a particular trait-variant as the reproductive success of that variant in comparison to that of other alternative variants of the same trait.
6. If the reproductive success of a trait-variant is higher than those of alternative variants of the same trait, the trait-variant will increase in representation in the next generation (this follows from 3, above). If the conditions that facilitated its higher reproductive success prevail over a long time, it may even entirely replace alternative variants of the same trait in a population.
7. Points 4, 5 and 6, above, constitute the typical process of selection, as commonly understood, as it operates among individuals. Selection can, however, also operate among groups in some situations. Selection provides an explanation for the adaptedness of species and is the major, but by no means the only, factor playing a role in micro-evolution and speciation.
8. Selection among individuals includes both differential reproductive success and, possibly differential transmission fidelity of the alternative trait-variants. Darwin's (1868) views on the latter are not very clear, but he, nevertheless, implicitly considered heredity to be an integral component of the selection process (for a very different and, in our opinion, erroneous view on this issue, see Bourat 2015).
9. Considerations of development are not relevant to understanding hereditary transmission of trait-variants. They can, however, be important for understanding the origin of large-scale variations among species or higher taxa, as well as some instances of micro-evolutionary change via growth correlations that can act independently of, or antagonistically to, selection.
10. Processes like selection, that can result in differentiation among populations, can also drive speciation over long periods of time (uniformitarianism). Change within a species via selection is typically slow (gradualism) (for a nuanced discussion of this issue, see Sober and Orzack 2003).

Of the ten points making up the DC, only Darwin's gradualism and uniformitarianism have largely failed the test of time. In all other respects, the DC has not been seriously challenged, though many of its tenets have been considerably elaborated and added to in the past one and half centuries.

17.3 The Crystallization of the Modern Synthesis (MS)

از حُسنی ملیحی خود، شوری بہ جہان کردی
هر زخمی و بِسمِل را، مصروفی فُغان کردی
az husn-e-maleeh-e-khud, shorey ba-jahaan kardi
har zakhmi-o-bismil ra, masroof-e-fughaan kardi
(With piquant beauty, you did raise, a tumult spanning worldly space
Thus shrinking anguished, injured souls, to lamentation's forlorn face
– Maulana Jami)

We now turn to the relationship of the MS to the DC. The scientific history of the MS, and its foundations, consequences, and shortcomings, have already been extensively discussed over the past few decades (e.g., Mayr and Provine 1980; Antonovics 1987; Gould 2002; Sarkar 2004; Amundson 2005; Rose and Oakley 2007; Plutynski 2009; Pigliucci and Müller 2010; Stoltzfus 2017; Charlesworth et al. 2017; Dickins and Dickins 2018; Huneman 2019; Dickins 2021), and we will, therefore, restrict ourselves to highlighting certain aspects of the MS-DC relationship that we believe warrant greater attention than they have hitherto received. Darwin and the MS are separated by about 60–80 years, and, during this time, there were several consequential developments in the attempt, inspired by Darwin, to understand evolution in terms of an interplay between ecology and heredity. On the one hand, there was a new focus on interpreting findings in natural history, biogeography, palaeontology and systematics in terms of evolutionary principles and, on the other, attempts to interpret the principles of heredity and selection statistically, rapidly yielding ground in the early 1900s to Mendelian genetics. Advances in development did not play a very major role in this phase of the growth of evolutionary thought. Ironically, August Weismann's attempt to provide an explanation for differentiation of cell types in the course of embryonic development led to a further separation of development and heredity, and also seemed to rule out the possibility of the inheritance of acquired characters due to the sequestration of the germplasm early in development (Weismann 1889, 1893a; but see also Winther 2001), thus leading to a narrowing of the DC that was only partly ameliorated in the MS. Weismann (1893b, 1902) also insisted on the primacy of selection over heredity in evolution, in the context of critiques of the efficacy of selection in bringing about evolutionary change (Galton 1877, 1889, 1894; Spencer 1893). A good account of this phase in evolutionary thought, termed Neo-Darwinism by George Romanes and others, is given by Forsdyke (2001).

The most consequential development between Darwin and the MS that substantially determined the form the MS took was undoubtedly the rediscovery of Mendel's work in 1900, followed by the linking of Mendel's hypothetical factors (genes) to chromosomal locations, largely through work in T. H. Morgan's laboratory in the early decades of the twentieth century (Schwarz 2008). Darwin had placed heredity centre stage in the study of evolution, even though his theory of pangenesis did not last, being discredited experimentally by Galton (1871) shortly after its full exposition by Darwin (1868). The early decades of twentieth-century genetics not only cemented heredity in this central position in evolutionary

explanation but also completed the already substantial exclusion of developmental considerations from our understanding of heredity, a process ironically led by T. H. Morgan, an embryologist who was initially opposed to the ideas of both Mendel and Darwin (Allen 1985; Amundson 2005; Sarkar 2006, 2017). Once the principles of transmission genetics had been verified, and extended from families to populations, it became crucial—in light of the tension between heredity and selection in preceding decades—to ascertain whether the Darwinian conception of selection was in fact compatible with the now known mechanism of heredity (Sarkar 2004; Joshi 2017b).

In many respects, the MS represented a conceptual narrowing of the scope of evolutionary thinking embodied in the DC, even though it was factually more expansive, incorporating new findings from heredity, evolutionary ecology, palaeontology and systematics. This narrowing is also reflected in the view that large parts of the MS that incorporated genetics into the view of adaptive evolutionary change through selection, especially the work of Ronald Fisher and Sewall Wright, are better described as constituting a reduction rather than a synthesis (Sarkar 2004), although that distinction is not very relevant to our purposes in writing this chapter. We now examine some of the ways in which the MS differed from the preceding DC, and emphasize certain aspects of the conceptual shifts involved, which we believe are important to understanding several sources of confusion in the evolutionary discourse over the past many decades, especially those surrounding the so-called gene's eye view of evolution (Ågren 2021). We do this by listing once again the ten major constituents of the DC, along with a brief explanation of how the MS changed or did not change each of them, and then offering some thoughts on how to resolve some of these confusions.

1. Species arise from pre-existing species (descent with modification) and can diverge from one another over time (principle of divergence), thus explaining the diversity and relatedness of species.
   Essentially unchanged in the MS.
2. Heredity must explain not just the transmission of similarities common to all members of a species, but also the transmission of individual trait-variants.
   Accepted implicitly, in a much narrower form, in the MS through the incorporation of Mendelian transmission genetics in families and populations. MS, unlike DC, incorporated a specific mechanism—genes and principles of their transmission—of heredity. Thus a more generalized view of heredity was narrowed down to a specifically Mendelian one.
3. Individuals can be usefully thought of as a mosaic of reasonably independent trait-variants. Trait-variants are ubiquitous, arise almost continuously, in ways affected by the environment, and tend to be transmissible to offspring with fairly high and similar fidelity.
   In the MS, trait-variants are often characterized at the level of the genotype or karyotype, rather than phenotypically. Genotypic/karyotypic trait-variants are ubiquitous, arise almost continuously by mutation and changes in the structure and number of chromosomes during meiosis/gametogenesis. The MS differs from the DC in ruling out the inheritance of acquired characters

in the Lamarckian sense, though some aspects of the environment are thought to play some role in generating mutational variation. By basing inheritance solely on Mendelian genetics, the MS implicitly takes a more nuanced stance on transmission fidelities than the DC: in Mendelian genetics, transmission fidelities can vary among genotypic variants and are typically frequency-dependent (Box 17.2). *Thus, in the MS, it is implicit that the positive relationship between reproductive success and increase in frequency of a variant can break down as a result of frequency-dependence and mating system (see also point 8 in this list). In the DC, Darwin's characterization of the 'powerful principle of heredity' implicitly assumed that transmission fidelities of all trait-variants are* > 0.5. *It is not clear whether Darwin thought that transmission fidelities could vary among trait-variants. A good discussion of some issues pertaining to transmission fidelity can be found in Frank* (2012).

4. Organisms are typically involved in a struggle for existence, involving competition for resources, refuge from enemies, and mates. Certain trait-variants can confer advantages in this struggle to the individuals bearing them, or to relatives of those individuals.

   Essentially retained in the MS, albeit with a slightly more abstract view of 'competition', including that between allelic or genotypic trait-variants, as opposed to competition between individuals.

5. Ecological success in the struggle for existence is ultimately measured by offspring production (reproductive success). Though it is the individual that reproduces, one can meaningfully consider the average reproductive success of all individuals bearing a particular trait-variant as the reproductive success of that variant in comparison to that of other alternative variants of the same trait.

   Essentially retained in the MS, with trait-variant often being construed more specifically as a genotypic variant.

6. If the reproductive success of a trait-variant is higher than those of alternative variants of the same trait, the trait-variant will increase in representation in the next generation (this follows from point 3 in this list). If the conditions that facilitated its higher reproductive success prevail over a long time, it may even entirely replace alternative variants of the same trait in a population.

   Essentially retained in the MS, with trait-variant often being construed more specifically as a genotypic variant, but with the caveat that the relationship between reproductive success and increase/decrease of a trait-variant can be complex (see also Box 17.2, *and point 3 in this list). Since genic heredity follows Mendelian rules, transmission fidelities of genotypic trait-variants are frequency dependent, permitting the maintenance of stable polymorphisms, the existence of unstable polymorphisms and sensitivity to initial conditions, and complex and often counter-intuitive behaviour of genotypic frequencies under selection and different mating systems.*

7. Points 4, 5 and 6, in this list, constitute the typical process of selection as it operates among individuals. Selection can, however, also operate among groups in some situations. Selection provides an explanation for the adaptedness

of species and is the major, but by no means the only, factor playing a role in micro-evolution and speciation.

Essentially unchanged in the MS, but with a somewhat greater emphasis on the primacy of selection, and of selection acting among individuals rather than groups, than in the DC. The MS also paved the way for an appreciation that selection can sometimes operate at levels below the individual, too (Lewontin 1970).

8. Selection among individuals includes both differential reproductive success and possibly differential transmission fidelity of the alternative trait-variants. Darwin's (Darwin 1868) views on the latter are not very clear, but he, nevertheless, implicitly considered heredity to be an integral component of the selection process.

   Essentially retained in the MS, in a stronger form than in the DC, but very well disguised, and not reflected explicitly in how selection is discussed. Because of its commitment to exclusively genic heredity, the MS depicts micro-evolutionary dynamics in a manner that makes it hard to see the implicit effects of transmission fidelity on change in genotypic or allele frequencies (see also Box 17.2*, and points 3 and 6 in this list). This problem is exacerbated by the large-scale deployment in population genetics of models that track micro-evolutionary change through allelic rather than genotypic frequencies, at least when random mating can be assumed. The benefit of tracking alleles rather than genotypes is that the number of state variables is reduced. The drawback is that, because allelic variants have a transmission fidelity of 1, unless mutation is invoked, the role of transmission fidelity as an integral part of the selection process, and the effects of mating system on transmission fidelity, are rendered implicit and invisible. Moreover, the discourse in much population genetics-based MS writing, though not in behavioural ecology, tends to consider selection as operating on viability by default, treating sexual selection or fecundity/fertility selection almost as afterthoughts. This further obfuscates the roles of heredity and mating system, because differential reproductive success of genotypes in viability selection models arises entirely through genotypic differences in viability, and the effect of transmission fidelity is subsumed into the non-linear, frequency-dependent marginal allelic fitness terms.*

9. Considerations of development are not relevant to understanding hereditary transmission of trait-variants. They can, however, be important for understanding the origin of macro-evolutionary variations among species or higher taxa, as well as some instances of micro-evolutionary change via growth correlations that can act independently of, or antagonistically to, selection.

   Essentially unchanged in the MS, but with a greater tendency to see development as irrelevant to the origin of macro-evolutionary variations among species or to micro-evolutionary change.

10. Processes like selection that can result in differentiation among populations can also drive speciation over long periods of time (uniformitarianism). Change within a species via selection is typically slow (gradualism).

Essentially unchanged in the MS.

Box 17.2 Transmission Fidelity in Population and Quantitative Genetic Models

In this Box, we use the formalism from Box 17.1 to illustrate how the gene-based Mendelian mechanism of heredity for trait-variants coded for by genotypes at one locus results in a frequency-dependent, type-variant-specific pattern of transmission fidelities. Moreover, the pattern of transmission fidelities is potentially affected in a type-variant-specific manner by the mating system. We also discuss, for polygenic trait-variants, the relationship between transmission fidelity of a trait-variant and its breeding value.

Consider three trait-variants (1,2,3), coded for by genotypes A_1A_1, A_1A_2, and A_2A_2, respectively. Let their frequencies in a given generation be f_1, f_2 and f_3, respectively. We assume no differences among trait-variants in survival to reproduction, or reproductive output. If the mating system is that of complete selfing, the transmission fidelities, and proportion of offspring exhibiting each of the other possible trait-variants, are given by:

$$x_{11} = 1; x_{12} = 0, x_{13} = 0$$

$$x_{22} = 0.5; x_{21} = 0.25, x_{23} = 0.25$$

$$x_{33} = 1; x_{31} = 0, x_{32} = 0.$$

Note that, in this case, transmission fidelities differ between trait-variants 2 and 1,3, but are constant across generations for all three trait-variants. Moreover, even in the absence of differences in expected reproductive output, f_1 and f_3 will increase over generations, relative to f_2, because

$$f'_1 = f_1 + 0.25f_2$$

$$f'_2 = 0.5f_2$$

$$f'_3 = f_3 + 0.25f_2.$$

This change in the phenotypic composition of the population is driven entirely by the differences in transmission fidelity across trait-variants, and is non-adaptive in that it does not result in any increase in the average expected offspring production of the population.

Now, consider another non-adaptive example involving the same trait-variants, but under a random mating system. Now, the transmission fidelities, and proportion of offspring exhibiting each of the other possible trait-variants, are frequency-dependent, and given by:

(continued)

Box 17.2 (continued)

$$x_{11} = f_1 + 0.5f_2; x_{12} = f_3 + 0.5f_2, x_{13} = 0$$

$$x_{22} = 0.5; x_{21} = 0.5(f_1 + 0.5f_2), x_{23} = 0.5(f_3 + 0.5f_2)$$

$$x_{33} = f_3 + 0.5f_2; x_{32} = f_1 + 0.5f_2, x_{31} = 0.$$

Therefore, the trait-variant frequencies in the next generation are given by

$$f'_1 = f_1(f_1 + 0.5f_2) + 0.5f_2(f_1 + 0.5f_2)$$

$$f'_2 = 0.5f_2 + f_1(f_3 + 0.5f_2) + f_3(f_1 + 0.5f_2)$$

$$f'_3 = f_3(f_3 + 0.5f_2) + 0.5f_2(f_3 + 0.5f_2)$$

These equations for change in trait-variant frequency will result in Hardy–Weinberg equilibrium in one generation when the following are satisfied:

$$f_1 = (f_1 + 0.5f_2)^2$$

$$f_2 = 2(f_1 + 0.5f_2)(f_3 + 0.5f_2)$$

$$f_3 = (f_3 + 0.5f_2)^2.$$

These two simple examples of an alternative mathematization of basic one-locus population genetics models serve to demonstrate several points about transmission fidelities under Mendelian heredity. First, differences in transmission fidelity across trait-variants can lead to changes in the phenotypic composition of a population even in the absence of differences in relative reproductive output (fitness) across trait-variants. Second, transmission fidelities can change if the mating system changes. Third, equilibria in trait-variant frequency ($f'_i = f_i$ for all i) can arise because losses of similar phenotype offspring of one's own ($f_i(1 - x_{ii})$) can be exactly offset by gain of similar phenotype offspring through the reproduction of other type-variants ($\sum_{j \neq i} f_j x_{ji}$), for all i, j. Finally, if we consider typical one-locus selection models, which assume differences in relative reproductive output (fitness) across trait-variants, inequalities between the various $f_i(1 - x_{ii})$ and $\sum_{j \neq i} f_j x_{ji}$ can similarly result in equilibria in trait-variant frequency by exactly cancelling out the fitness differences among trait-variants; this is what happens in the canonical case of overdominance for fitness in a one-locus model. More generally, in selection models, inequalities between the various $f_i(1 - x_{ii})$ and $\sum_{j \neq i} f_j x_{ji}$, which change over generations due to frequency-dependence of the x_{ii} and x_{ji} terms, interact with among-trait-variant fitness differences in shaping

(continued)

Box 17.2 (continued)
the dynamics of trait-variant frequencies, thereby underscoring the role of transmission fidelity as an integral part of the selective process.

The above examples assume discrete trait-variants arising from different genotypes at a locus, and the broad implications generalize to traits governed by a small number of loci. If we consider quantitative (polygenic) traits, then trait-variants are continuous rather than discrete, with each phenotypic value constituting a distinct trait variant. In such cases, the transmission fidelity of a trait-variant is closely associated with its breeding value, the deviation of the mean phenotypic value of its offspring from the overall population mean. Transmission fidelity of a trait-variant would then be reflected by the deviation of the mean phenotypic value of the offspring of all individuals exhibiting that trait-variant (these individuals may have different underlying multi-locus genotypes) from the phenotypic value for that trait associated with the common trait-variant of the parental individuals.

Two main points we wish to stress here, when considering Mendelian heredity, are that (i) transmission fidelity affects how differences in reproductive output among trait-variants translate into changes in their frequencies in complex frequency- and mating system-dependent ways, resulting in diverse patterns of dynamics that will not necessarily culminate in the fixation of the trait-variant with the greatest reproductive output, and (ii) this important role of transmission fidelity in the selective process is implicit and largely hidden in the standard mathematization of population and quantitative genetics models, especially when they use allele rather than genotypic frequencies as state variables because, in the absence of mutation or migration, an allelic variant has a transmission fidelity of 1, even though genotypic variants do not.

We note, in conclusion, that the Price (1970) equation also explicitly incorporates the notion of transmission fidelity in its apportioning phenotypic change to a sum of terms representing selection (differential reproduction) and transmission fidelity, respectively. A similar exercise to the one above, that interprets population genetics models from the perspective of the Price equation, can be found in Box 2 of Joshi (2020).

As we can see, the differences between the MS and the preceding DC are neither very large nor substantive in a conceptual sense, although some statisticalist philosophers of evolution have a somewhat different view (see Walsh et al. 2017). The MS retained Darwin's (1859, 1868, 1871) crucial atomization of individuals into traits, his realization that development was largely irrelevant to understanding either heredity or micro-evolutionary change, and his central insight that differential reproductive success of trait-variants would, thanks to heredity, translate into altered representation in subsequent generations. Elements of the DC that were not included in the MS were the Lamarckian inheritance of acquired characters, the recognition of group selection in the classic sense as potentially important in some evolutionary

scenarios, the possibility of a variety of mechanisms of inheritance, and the appreciation that development may have a major explanatory role in issues surrounding the origin of macro-evolutionary variations, the divergence among species, and some instances of micro-evolutionary change. Surprisingly, the MS strengthened the claim of both gradualism and uniformitarianism, especially in its genetic expressions (e.g., Dobzhansky 1937), even though these were not conceptually crucial to the Darwinian *weltanschaung*.

The MS differed from the DC in having an explicit mechanism of heredity in the form of Mendelian genetics. While this helped show that the mechanism of natural selection was indeed compatible with heredity (Fisher 1930; Wright 1931, 1932; Haldane 1932), it also had some, perhaps unintended, consequences that have resulted in considerable ongoing confusion about the units and levels of selection, in addition to ruling out non-genic forms of inheritance. The MS, especially in its population genetics *avatar*, substituted the more specifically construed genotypic trait-variants for phenotypic trait-variants and, moreover, when a random mating assumption could be deployed, typically modelled the dynamics of genotypic trait-variants at the allelic rather than the genotypic level. It also introduced the concept (s) of fitness, which was heuristically useful but also led to a lot of confusion (e.g., Kimbrough 1980; Matthen and Ariew 2002; Sober 2001; Ariew and Lewontin 2004; Roff 2008; Orr 2009) of the kind that Wittgenstein (1921/fresh Eng. transl. 1994) had warned about. The use of allelic level trait-variants and fitness as reproductive success diverted attention from the essential nature of selection acting among individuals, including transmission fidelity as an integral component, and also from the effects of frequency and mating system on transmission fidelity (see Box 17.2). In addition, this focus on allelic variants also gave rise to an entirely avoidable and long-lived debate about whether the individual or the gene (allele) is the most appropriate unit of selection (e.g., Okasha 2006; Ågren 2021), by facilitating what is often termed the gene's eye view of evolution. To our mind, this is a misleading contrast: the crucial difference is between individuals and trait-variants, and this was introduced as early as in the DC. We suggest that, in this DC perspective, there is no dispute about the biological units relevant to the causes and consequences of selection, respectively. For understanding the causes of selection, the relevant unit is the individual, whereas for studying the consequences of selection, it is the trait-variant, and not the individual. The contradistinction of the individual to the gene, that happens very commonly in the units of selection debates, merely, and unhelpfully, mapped the original individual versus trait-variant contrast onto a broader and extremely contentious debate about genetic determinism versus free will, or agency. We discuss this issue, and its consequences for the conceptualization of fitness, in greater detail in a later section. The levels of selection debate about whether selection acts primarily on individuals or groups was eventually, after a few decades of extreme antipathy to group selection, resolved, especially with the development of multi-level selection theory (Frank 2013 and references therein; Okasha 2006; detailed accounts in Lewontin 1970; Sober and Wilson 1998).

Interestingly, and in a striking reminder that the episodes in the history of ideas are often as inexplicable as those in the history of states, the MS involved two very

different treatments of the earlier biometric work on selection and heredity by Rafael Weldon and Karl Pearson by the same individual—Ronald Fisher. The work of Weldon and Pearson was itself a development of earlier statistical insights from Galton, but differing from it in significant ways, especially regarding his doubts about the efficacy of selection in the face of heredity (discussed in Joshi 2017a, b), On the one hand, Fisher's (Fisher 1918) treatment of traits affected by a large number of genetic loci with small individual effects on a phenotype effected a reduction of biometry to Mendelian genetics (Sarkar 2004), whereas on the other (Fisher 1930, 1941), it gave rise to what was, although couched in explicitly genetic terms, essentially a phenotypic theory of Darwinian micro-evolutionary change (i.e., quantitative genetics e.g., Mather 1943), in stark contrast to the overall highly genetic bias of the MS. We discuss this in the next section.

17.4 Quantitative Genetics as a Phenotypic Theory

ہوئی مِنّتکشِ تابِ شُنیدن داستاں میری
خموشی میں ہوئی مدفُون ہر آہ و فُغاں میری
hui minnatkash-e-taab-e-shuneedan dastaan meri
khamoshi mein hui madfoon har aah-o-fughaan meri
(My story begs a listening crowd, that hears with comprehending skill
Till then, my sighs and forlorn cries, lie buried in this silence, still
– Nabraas Akbarabadi)

Several years ago, we had expressed our surprise that quantitative genetics does not appear in any comprehensive or meaningful way in the EES-MS debate, despite its essentially constituting a phenotypic theory of micro-evolutionary change that does not necessarily assume genic inheritance, and being far more inclusive and flexible than population genetics in this role (Joshi 2005; Prasad et al. 2015). With the notable exception of work by Etienne Danchin and colleagues (e.g., Danchin et al. 2011, 2013, 2019), and some specific attempts to integrate epigenetic inheritance and quantitative genetic analyses (e.g., Spencer 2002, 2009; Santure and Spencer 2011; Banta and Richards 2018), the situation is unchanged. We think this is odd because quantitative genetics actually incorporates or addresses some of the issues that EES proponents often accuse evolutionary genetics of overlooking. We reiterate some of these aspects here and also discuss how, like population genetics, quantitative genetics also tends to obscure some facets of the role of transmission fidelity in micro-evolutionary change.

To describe quantitative genetics as dealing with the inheritance of polygenic or continuous traits, as textbooks tend to do, is about as fair as describing the phenomenal Brazilian footballer Ronaldo as someone who ran about a field kicking a ball. It is an accurate, but ultimately trivial, description that fails to capture the essence of the achievement involved. Quantitative genetics grew out of Fisher's (1918) demonstration that the statistical results of Karl Pearson and the biometricians on the phenotypic correlations between relatives were consistent with Mendelian genetics, on the assumption that continuous phenotypes could result from the effects of many

genes with individually small phenotypic effects. The validation of previous work on heredity and evolution, by showing it to be consistent with Mendelian principles, was an urgent and significant concern in the years following the rediscovery of Mendel's work. However, although it was couched in specifically genetic terms, quantitative genetics essentially provided a phenotypic theory of micro-evolutionary change (Joshi 2005; Prasad et al. 2015; Queller 2017). The concept of the breeding value (additive genetic value) of an individual with a given phenotypic value in a certain population and environment was effectively a way of operationalizing transmission fidelity in the absence of any knowledge of the details of the genotype to phenotype mapping, thus distilling out the consequential essence of the complex polygenic mechanism of heredity (see also Box 17.2). Transmission fidelity could thus be combined with reproductive success of individuals with differing phenotypic values, to yield evolutionary change in the location of the mean of the phenotypic distribution of that trait in that population and environment, due to selection. Even in the specific context of an underlying Mendelian genetic model, the additive genetic value of an individual accounts for the statistical effects of dominance and epistasis within its genome on offspring phenotype, something that is often not appreciated. Essentially, the Breeders' equation in quantitative genetics describes the one-step shift under selection in the mean of a phenotypic distribution for a continuous trait as $R = h^2.S$, where R is the response, reflecting the one-step change in mean phenotypic value, h^2 is the ratio of additive genetic variance (the variance in breeding values among individuals) to the phenotypic variance, and S is a measure of the strength of selection. In this formulation, h^2 and S effectively reflect transmission fidelity of trait-variants with different phenotypic values (Box 17.2), and their reproductive success, respectively. The original formulation of breeding value (or additive genetic value) by Fisher was for a case of random mating (Falconer 1985), but the logic can be extended to non-random Mendelian mating systems (Muralidharan and Jain 1992a, b), or even to systems with arbitrary non-genic mechanisms of heredity, by re-defining breeding value as a transmission fidelity metric for trait-variants and quantifying it appropriately. Thus, the quantitative genetics framework has the flexibility to explain micro-evolutionary change under non-genic inheritance through its inclusion of a transmission fidelity perspective (Danchin et al. 2011, 2013, 2019), and could be fruitfully used very generally across diverse systems, even though this flexibility is often hidden behind its explicitly genetic presentation. A systematic elucidation of when a generalized quantitative genetic framework will or will not suffice to capture micro-evolutionary dynamics under non-genic inheritance could be a fruitful avenue of further research.

Another point worth noting about the quantitative genetics formulation is that it explicitly includes the phenomenon of phenotypic plasticity, something the MS is often accused of ignoring. The partitioning of phenotypic value of an individual into a genotypic and an environmental value, and a stochastic error term ($P = G + E + e$) incorporates the notion that the same genome can give rise to different phenotypic values for a trait in different environments, the textbook definition of phenotypic plasticity. A genotypic value by environmental value interaction (G x E interaction)

implies genetic variation for phenotypic plasticity, and a G x E covariance of the beneficial sort can constitute adaptive phenotypic plasticity.

Finally, we stress that continuous traits affected by many multi-allelic loci of individually small phenotypic effect have a tremendous ability to generate multiple trait-variants (individuals with different phenotypic values for that trait) through the shuffling of standing within- and among-locus genetic variation alone (Teotónio et al. 2009; Mueller et al. 2013; Matuszewski et al. 2015; Phillips et al. 2018; Hickey and Golding 2021; Kawecki et al. 2021). One outcome of this is that even a sample of relatively few genomes from a population can rapidly regenerate the full pre-sampling phenotypic distribution. Therefore, the criticism that available phenotypic variation in a population may not be isotropic (*e.g.*, Salazar-Ciudad 2021) might often not hold true for continuous traits within populations, at least in the sense of availability of variants, even if not in the sense of a uniform distribution of the probabilities of their occurrence.

17.5 The Nature(s) of Fitness, and a Micro-Evolutionary Red-Herring

هر لهذه به شکلی بُتان عیّار بر آمد، دِل بُرد و روان شُد
هردم بہ لباسی دیگران یار بر آمد، گہ پیر و جوان شُد
har lehzeh ba-shakl-e-butaan ayyaar bar aamad, dil burd o nihaan shud
hardam ba-libaas-e-digaraan yaar bar aamad, geh peer-o-jawaan shud
(The Beloved, in artful varied forms, does steal my heart and then depart
One moment young, another old, in myriad garbs; this is his art
– Maulana Rumi)

Although the exact origins of the term 'fitness' are hard to pinpoint, both the term and the concept featured repeatedly in the work of Karl Pearson and, by the time the MS was being announced (Huxley 1942), were an important part of the micro-evolutionary lexicon and conceptual tool-kit (Gayon 1998). However, fitness has been used in multiple senses in the MS and later, resulting in manifold confusions that reflect its ultimate origins in Spencer's (Spencer 1864) misplaced rejection of trait-variants as the units of selection in favour of whole individuals, implicit in his coining of the most unfortunate phrase 'the survival of the fittest'. The crux of the problem is that fitness, even in its correct and restricted micro-evolutionary context, is variously defined on both individuals and trait-variants (phenotypic, genotypic or allelic), and can be used to mean the reproductive success of an individual, the average reproductive success of individuals exhibiting a specific trait-variant, the one-step change in frequency of a trait-variant, or the long-term expected evolutionary success of a trait-variant or lineage. Thus, fitness is used both as a causal predictor of subsequent changes in relative representation of different types in a population, as well as a descriptor of those changes (Ariew and Lewontin 2004). Indeed, fitness, like Rumi's 'artful Beloved' seems to appear before us in varied forms and disguises at different times and places. Textbooks exacerbate this confused state of affairs by often defining fitness, towards the earlier part of the book, as

the reproductive success of individuals, without mentioning that it is but one of the senses in which the term is used, and then, ironically, proceeding to use fitness in one or more of its other senses later on. Such a use of fitness for different sorts of attributes of entities at various levels of biological organization is clearly undesirable, as has been repeatedly pointed out (Kimbrough 1980; Matthen and Ariew 2002; Sober 2001; Ariew and Lewontin 2004). Yet, with the notable exception of the work of Earnshaw-Whyte (2012), no resolution has been offered beyond a cogent argument that fitness cannot possibly do justice to the myriad roles we expect it to play (Ariew and Lewontin 2004). Here, we outline the contours of what we believe is a long overdue and useful resolution.

The following discussion pertains only to micro-evolutionary change in frequencies of trait-variants due to selection, falling under the category of '*micro-dynamics*' issues in the schema presented in Table 17.1. Indeed, strictly speaking, it is best to restrict consideration of selection to situations where entities at or below the level of a species are being considered. The concept of selection implicitly includes a notion of competition, albeit often in a broadly metaphorical sense, and it is not clear whether entities at the level of higher taxa can be meaningfully thought of as being in competition. We will mostly restrict ourselves to discussing selection at the level of an individual, as contrasted to a trait-variant, as that is the comparison about which much confusion has arisen in the past. The two most crucial questions that need to be addressed to clarify the confusions about fitness are: (i) whether fitness is better conceived of as an attribute of an individual, or is it more useful to think of fitness as ascribable, on an average, to a trait-variant as an abstract entity (collection of all individuals in a population exhibiting that trait-variant)? and (ii) whether fitness is better conceived of as a measure of reproductive success (e.g., lifetime offspring production) or as reflecting a one-step change in frequency (the time-step will typically, but not always, be a generation) of the relevant entity type? Our answer to these questions is that it is best to think of fitness as reflecting the change in relative representation of a trait-variant in the population. Indeed, we believe that the individual is not much more than a red-herring in the context of trying to understand and depict micro-evolutionary change (i.e., the consequences of selection), and one that has led to tremendous confusion in evolutionary discourse, as we discuss below.

The popularity of defining fitness as an attribute of an individual, reflecting its reproductive success in a given ecological context, seems to arise from the intersection of a comfort with agential thinking and a failure to differentiate between the relevance of agency in different biological contexts and at different levels of biological organization, for example cells versus individual organisms (Okasha 2018). It appears that the tendency to ascribe agency to humans, animals, plants and even inanimate objects has deep roots in the human mind (e.g., Dennett 2006; Lindstrøm 2015), and it could perhaps have arisen through what Rose (1998) termed 'immanent Darwinism'. However, it needs to be recognized that the agency of a living individual, or of its constituent cells, is largely only relevant in the contexts of ecology (including successful reproduction) and development, but not in the domain of explaining the dynamics of micro-evolutionary change. This is because individuals, considered holistically, are effectively a unique constellation of variants

of many different traits and, as such, have no continuity across generations, unlike the trait-variants themselves. The agency of an individual can, therefore, affect its reproductive success, but not any meaningful measure of micro-evolutionary dynamics, because the transmission fidelity of any of a unique set of trait-variants is zero, by definition. A unique individual may produce many offspring, but none of them will be the same as the parent, except in the case of asexual reproduction. This might be termed the 'infinite individuals problem' for sexually reproducing species: if individuals are phenotypically unique, then any explanation of micro-evolutionary dynamics at the level of the individual will be restricted to a description of how one set of unique individuals was replaced by another set of different, equally unique, individuals in the next generation. For this reason, we believe, as did Darwin, drawing upon the experience and practices of breeding, that it is best to focus on trait-variants, not individuals, if our analyses are to have any chance of explaining patterns in micro-evolutionary change arising as a consequence of selection.

Having settled upon the trait-variant as the appropriate focus of an analysis of micro-evolutionary dynamics, we now consider whether reproductive success or a one-step change in the frequency of trait-variants constitutes a better way of thinking about fitness. The reproductive success of a trait-variant can be equated to the average reproductive success of all individuals in the population who exhibit that variant, while the one-step change in frequency quantifies the difference, across a generation, in the representation of that trait-variant in the population, relative to other variants of the same trait. Defining fitness as reproductive success may at first sight appear to satisfy the scientist's inherent *ceteris paribus* privileging of *a priori* prediction over *post facto* description, because fitness differences among trait-variants can then be thought of as predicting changes in their frequency over generation. However, in this context, all else is rather emphatically not equal. Differential reproductive success of trait-variants is positively correlated with relative representation in the next generation only under the implicit DC assumptions that transmission fidelities of trait-variants are typically high, and similar in magnitude (see also Box 17.2). Thus, the ability of fitness defined as reproductive success to serve as a predictor of change in frequency is not inherent in the measure. The only other benefit of defining fitness as the reproductive success of a trait-variant is that it preserves the notion that fitness is an intrinsic attribute of a type, or to be more precise, of the interaction between the biological characteristics of a type and its ecological context. Thus, we can treat fitness, as textbooks typically do, as a type attribute, and consider frequency-, density- or sex-dependent fitnesses to be special cases. While this usage confers the comfort of familiarity, we do not believe this is helpful, any more than the tendency of genetics textbooks to treat epistasis as a 'deviation' or 'exception' to Mendel's laws is. If, on the other hand, we define fitness as the one-step change in the frequency of a trait-variant, there are several conceptual benefits.

First, fitness of the trait-variant now incorporates not just reproductive success but also transmission fidelity, which renders explicit the connection between fitness and the process, as opposed to the act, of selection. When a breeder trying to develop a variety with large body size chooses the biggest individuals in a population to breed

from, that is an act of selection, which may or may not yield a response depending upon the level of additive genetic variance for body size in that population. At the same time, the entire process of generating the variety with larger average body size than its ancestors, encompassing both the act of selection, and the response to it, is also referred to as selection: this is what we are terming the process of selection. Our point is that the act of selection involves only differential reproductive success, whereas the process of selection requires differential *heritable* reproductive success, thereby encompassing the act of selection, transmission fidelity, and the consequent response to selection. We suggest that a concept and definition of fitness that reflects the process of selection is preferable to one that merely reflects the act of selection, even though the difference is only one of perspective.

Second, because the one-step increase in the frequency of a trait-variant depends on the interaction between bearers of alternative variants of that trait for survival, refuge and reproduction, fitness defined thus is always frequency-dependent. Because this measure of fitness includes transmission fidelity, it follows that fitness defined thus is also always dependent upon the mechanism of inheritance (genic or otherwise) as well as on the patterns of interaction among individuals that can alter the trait-variants they bear. These interactions, in situations of genic inheritance, constitute the mating system, i.e., the set of probabilities of individuals with trait-variant i mating with individuals bearing trait-variant j (i, j, $= 1..n$, if there are n variants of that trait in the population); in cases involving cultural inheritance, interactions would be reflected in the likelihood of an individual bearing cultural trait-variant i passing on i to an individual that earlier exhibited cultural trait-variant j, via learning, in its broad sense. In all such interactions, not just means but also variances will have consequences for the resulting micro-evolutionary dynamics. This manner of defining fitness, therefore, also opens up the possibility of a more general unified theory of selection that is agnostic to the mode of inheritance, something which a definition of fitness as reproductive success does not easily support, though quantitative genetics successfully took some steps in that direction. In essence, this is what the Breeders' Equation in quantitative genetics achieves, by combining fitness as reproductive success (in the S-term) with transmission fidelity pattern (in the h^2-term for univariate selection, or the $\mathbf{G}$-matrix for multivariate selection), although this is not immediately obvious from the form of the equation because the h^2-term and the $\mathbf{G}$-matrix are formulated in explicitly genetic terms, though they need not necessarily be so. This approach becomes more clearly apparent in the Price (1970) equation, with its ascribing of phenotypic change to the sum of terms representing selection (differential reproduction) and transmission fidelity, respectively. Similar approaches for understanding dynamics in diverse non-biological systems as generalized Darwinian processes are also now being explored (e.g., Reydon and Scholz 2015), potentially justifying Haeckel's expectation that Darwinian thinking would become important even in disciplines beyond biology (Richards 2008), and Price's (Price 1995) desire to do for selection what Claude Shannon achieved for information.

Third, and perhaps most importantly, once fitness is defined as a one-step change in trait-variant frequency, fitness is always inclusive, unless transmission fidelities of

all trait-variants equal 1. This is because the fitness of a trait-variant accrues either through the reproduction of individuals bearing that trait-variant (direct fitness), or through the reproduction of individuals bearing another trait-variant, but with transmission fidelity less than 1 (indirect fitness). This is a more satisfying property for fitness, compared to the situation at the individual level in which fitness can be either direct or inclusive, depending on social context, and will likely reduce the confusion that surrounds the debates around kin-selection and inclusive fitness (reviewed by Frank 2013; Birch and Okasha 2015; Kramer and Muenier 2016).

In contrast to these benefits of treating fitness as one-step frequency change, any advantage of defining fitness as reproductive success accrues only if we define fitness on individuals. However, as we have seen, that definition cannot properly capture the essence of the process of selection due to the infinite individuals problem. Consequently, we believe that there is a strong case for restricting the use of fitness to one-step frequency change in alternative variants of the same trait, and not using fitness to also refer to reproductive success, or to individuals. We stress that we are not suggesting that measuring and thinking about the lifetime reproductive success of individuals is not important to understanding microevolutionary change: its importance is entirely retained in our perspective. All we are suggesting is that we not label the lifetime reproductive success of an individual as its 'fitness', restricting the use of that term to the one-step change in the frequency of a trait-variant. We next touch upon some of the various confusions that would be dispelled by doing this.

One of the most contentious issues in micro-evolution in the past half-century has been the gene's eye view of evolution (recent book-length review by Ågren 2021), initially popularized by Dawkins (1976), though its antecedents go back to Williams (1966) and, some argue, to Fisher (1930) and Hamilton (1964a, b). We discuss whether or not Fisher's (1918, 1930, 1941) conceptualization of the role of genes in micro-evolutionary dynamics can be justifiably considered a key part of the Dawkinsian gene's eye view of evolution in the next section, restricting ourselves here to the implications of our perspective on fitness for certain aspects of the gene's eye view debates. In addition to the debates around the gene's eye view of evolution, there has been a slightly more narrowly focussed debate around kin-selection and inclusive fitness (of individuals) in the context of the evolution of altruism, a debate that began just a few years after Hamilton (1964a, b) first published his detailed treatment of the problem (reviewed by Frank 1998, 2013). We believe that these long-standing debates are less substantial than the papers addressing them might lead one to believe, and that they arise partly from confusions resulting from the idiosyncrasies of classical population genetics modelling, and some of the confusions about fitness and the role of individuals in micro-evolutionary dynamics discussed above, in addition to the fact that there are often multiple approaches to formulating a problem, with the choice of formulation often being driven by familiarity and convenience. Long-standing debates in ecology and evolution often have their roots in such conceptual confusions and imprecise use of terms; debates on more straightforward issues tend to get resolved relatively quickly (Kitcher 1987; Joshi 2022).

One unfortunate consequence of the greater visibility of population genetics (over quantitative genetics) in explanations of micro-evolutionary dynamics under selection is that, because population genetics models typically treat trait-variants at the allelic rather than phenotypic level, the contrast between individual and trait-variant has been translated into a contrast between individuals and genes. Thus, discussion of how to best model micro-evolutionary change has become conflated with the debate between genetic determinism and agency or free will in humans, reflected onto non-human species (e.g., Walsh 2015; Sultan et al. 2022). Because of this conflation, the genes versus individuals debate inflames passions to a degree that the more accurately focussed debate about trait-variants versus individuals would probably not. While it is true that a gene's eye view narrative of micro-evolutionary dynamics is often accurate as long as there are no significant gene-by-gene interaction effects on phenotypes, it breaks down in the face of such interactions due to the complex behaviour of marginal allelic fitnesses (Sober and Lewontin 1982). When a gene's eye view is applied to situations of micro-evolutionary dynamics that do not involve phenotypes with a simple genotype to phenotype mapping, as is the case in most evolutionary ecology studies, it tends to collapse into a vague belief that the transmission fidelity of the trait-variants is high because they are gene-based. This is actually untrue, because transmission fidelities of genetically encoded (genotypic) trait-variants can differ among variants, are typically frequency-dependent, and are therefore also dependent on the mating system, causing them to change in complex ways as the frequencies of the trait-variants change (see Box 17.2). This has historically been one factor contributing to the discomfort of many evolutionary geneticists with a lot of optimization-based explanations in evolutionary ecology (e.g., Rose et al. 1987), although this discomfort also partly derives from the tension between static and dynamic approaches to modelling (Frank 1998, Chap. 12). Despite its limitations, however, the gene's eye view has undoubtedly been successful in illuminating several aspects of the micro-evolutionary process. We suggest that the success of the gene's eye view, in terms of both book-keeping and heuristic value, is not so much from its focus on genes *per se*, but on genes *qua* trait-variants, albeit defined at the allelic level, as opposed to individuals, bringing with it the added benefit of often being able to assume a transmission fidelity of 1, a point that appears not to have been widely appreciated (e.g., by Okasha 2006; Ågren 2021).

The evolution of altruistic behaviours is another area where failure to clearly distinguish between the roles played by individuals and trait-variants in micro-evolutionary change has led to considerable confusion. When Hamilton (Hamilton 1964a, b) first worked out his genetical theory of social evolution, he did all his analysis at the level of trait-variants defined at the level of genotype or allele, deploying population genetics models under some simple assumptions about the genetic underpinnings of the relevant trait-variants. Yet, he sandwiched all his analysis of change in frequency of genetic trait-variants between an introduction and a discussion section that treated the entire issue in terms of individuals and their reproductive success, making the connection through the fact that a genetic trait-variant could increase in frequency if the altruist's behaviour increased the reproductive success of other individuals with whom it shared alleles identical by descent

(e.g., genetic relatives or kin), even at the cost of its own. This renders these extremely important papers somewhat disconcerting and difficult to read. This tendency of Hamilton's to analyze problems in social evolution at the level of trait-variants, but then present the ideas at the level of individuals, is attested to from personal experience by Frank (2013), and has led to much debate over the meaning of kin-selection and inclusive fitness, often tending to obscure the fact that kin-selection is a testable hypothesis whereas inclusive fitness is an aid to doing genetic book-keeping at the level of individuals, rather than alleles. Frank (1998, 2013) has discussed many aspects of these debates at length, especially highlighting how this emphasis on an, in our view entirely avoidable, individual's-eye view led to a misleading focus on kinship, or overall genetic similarity between individuals, as opposed to more narrowly focussed genetic or phenotypic similarity with respect to specific relevant traits, which in turn led to much debate about the relative merits of kin-selection versus multi-level selection book-keeping when studying social evolution. Such confusion between genome wide-similarity and genetic similarity at specific relevant loci is also found in interpretations of the cost of sex as being that of genome dilution (Williams 1975; Shields 1988), based on the misplaced belief of the relevance to the evolution of reproductive mode of the genome dilution occurring because asexual mothers share the whole genome with offspring, as compared to sexual mothers who share only half their genomes (Joshi and Moody 1998). This common but unfortunate urge to explain microevolutionary dynamics at both the level of the individual (or multi-locus genome) and the trait-variant (or one-locus genotype) permeates much of the discourse in evolutionary biology, particularly in behavioural ecology, even on topics not involving altruism, and creates confusion, especially for beginning students.

To sum up, we believe that we gain nothing but comfort, and lose considerable clarity, when we try to explain micro-evolutionary dynamics by 'thinking of an individual as acting so as to enhance its fitness', direct or inclusive. We suggest that it would be better if we stuck to explaining micro-evolutionary dynamics at the level of trait-variants. The debates about direct versus inclusive fitness, kin- versus individual-selection, and individuals versus genes, are all, to our mind, partly a consequence of failing to appreciate the underlying conceptual structure of genetic models of micro-evolutionary dynamics, especially the fact that all fitness in the sense of one-step change in trait-variant frequency is inclusive, except when modelled at the allelic level of trait-variants in the absence of mutation (which is what happens in most simple models of population genetics). The distinctions between direct and inclusive fitness, or kin- and individual selection, for example, seem to us to largely be artefacts of trying to tell the story at the level of individuals rather than trait-variants, whereas all underlying analysis is actually at the latter level. We also suggest that an inclusion of transmission fidelity into the definition of fitness better reflects the process of selection, and helps focus attention on the role of mating system and mechanisms of heredity in mediating the micro-evolutionary outcomes of differences in reproductive success among trait-variants. One advantage that population genetics brought to the MS, as compared to the DC, was a better implicit appreciation that transmission fidelity of trait-variants had a complex

dependence on trait-variant frequency, mating system and the details of the hereditary system, and that, therefore, the trait-variant with the highest reproductive success would not necessarily rise to very high frequency, an insight often not appreciated fully in evolutionary ecology (Rose et al. 1987), especially when deploying optimization models to explain the evolution of alternate trait-variants (strategies). Yet, because population genetics models are often framed and, more importantly, analyzed at the level of allelic rather than genotypic trait-variants, in systems with random mating and no mutation, the crucial role of transmission fidelity in mediating micro-evolutionary outcomes of differences in reproductive success among variants is often obscured and difficult to immediately discern (see Box 17.2). That is why we recommend a focus on fitness defined as one-step frequency change, at the level of trait-variants rather than individuals, as a prescription for enhanced clarity in our engagement with issues of micro-evolutionary dynamics. We note, however, that fitness defined as one-step change in frequency must still be calculated in diverse ways for different evolutionary problems, depending on context (e.g., Roff 2008). Our prescription has much in common with the statisticalist perspective of some philosophers of evolution (see esp. Matthen and Ariew 2002; Walsh 2007; Earnshaw-Whyte 2012; Walsh et al. 2017), although their work is often not that familiar to many researchers in evolutionary biology: the terminology they use can sometimes differ from that of evolutionary biologists, and their work is typically published in the literature on philosophy rather than evolutionary biology.

17.6 One Gene's Eye View of Evolution, or Two?

آفاقها گردیده ام مهری بُتان ورزیده ام
بِسیار خُوبان دیده ام لیکِن تُو چیزی دیگری
aafaaq-ha gar deede-am, mihr-e-butaan warzeede-am
bisyaar khoobaan deede-am, lekin too cheez-e-deegari
(I wandered till the ends of worlds, endured the love of idols, too
Of all the beauties I did see, I never found one quite like you
– Amir Khusro)

Since the rediscovery of Mendel's laws in 1900, there have been many, and varied, attempts to integrate Mendelian genetics into our understanding of the evolutionary process, some more helpful and pervasive than others. However, to our mind, the manner in which Fisher (1918, 1930 and esp. 1941) conceptualized and analyzed the role of Mendelian genes in mediating adaptive micro-evolutionary dynamics under selection remains uniquely elegant, insightful and consequential for our understanding of this aspect of the Darwinian conception of the evolutionary process. In particular, we believe that it is neither helpful nor accurate to conflate the Fisherian conceptualization of the role of genes in evolution with the later gene's eye view of evolution, deriving largely from the work of Williams (1966) and Dawkins (1976); here, we briefly explain the reasoning behind this assertion.

Over the past few decades, the so-called gene's eye view of evolution (for a recent book-length review, see Ågren 2021) has been at the centre of many criticisms of the MS made by proponents of the EES. For example, EES proponents often raise concerns that the MS ignores the importance of organismal agency and inherency in evolutionary explanation, that genes rather than individuals have been considered as the appropriate units of selection and micro-evolutionary dynamics, that genes have been imbued with an almost causal role in mediating micro-evolutionary change, and that, in general, genes seem to have more or less displaced the organism as the central focus of micro-evolutionary explanation. Typically, this canonical account of the gene's eye view of evolution is depicted as constituting an integral part of the MS. In this section, we argue that the typical depiction of the gene's eye view of evolution as arising from the merging together of population genetics and the Darwinian principle of natural selection, especially in the work of Fisher (1918, 1930, 1941), is a somewhat misleading and overly simplistic rendering of what should be, in fact, a far more nuanced account. We suggest that it is more appropriate to think in terms of two distinct gene's eye views of evolution, one primarily Fisherian and the other primarily due to Dawkins (1976) and, to a considerable degree, G. C. Williams (1966). In our opinion, the substantial differences between these two gene's eye views of evolution are often glossed over and, consequently, the two are conflated. This conflation often makes it appear as though critics of the gene's eye view of evolution are rejecting not just the Dawkinsian perspective, but also much of the population or quantitative genetics perspective deriving from the work of Fisher and others. This, not surprisingly, results in strongly worded responses from those who identify with the MS and find the Fisherian gene's eye view to be very useful in understanding many aspects of the evolutionary process. We note that many of these people, including us, do not find the Dawkinsian gene's eye view particularly helpful and, indeed, believe that it can often be misleading. It is worth observing in this context that one of the earliest and most cogent critiques of the Dawkinsian perspective came from the viewpoint of classical population genetics (Sober and Lewontin 1982). Moreover, among evolutionary biologists, it is typically the developmental biologists and population geneticists that are the least favourable to the Dawkinsian gene's eye view of evolution, while behavioral ecologists are largely supportive, a strange combination if the Dawkinsian and Fisherian gene's eye views were indeed substantially similar in conception and nuance.

We find it useful to compare the Fisherian and Dawkinsian gene's eye views along the twin axes of the distinction between the causes and consequences of selection, on the one hand, and what exactly they seem to have been trying to achieve through their work, on the other. The first statement in the preface of *The Genetical Theory of Natural Selection* (Fisher 1930) provides an instructive contrast. Fisher (1930) begins his book with the declaration, 'Natural selection is not Evolution'; Dawkins' (1976) arguments in *The Selfish Gene*, can reasonably accurately be summarized as proclaiming that natural selection is, in fact, more or less equivalent to evolution. This contrast is not surprising, given the differences in their training and in the general state of evolutionary thought in their respective times, and the fact

that, consequently, their principal aims were quite different. Fisher, trained as a mathematician and, working at a time when it was critical to show that the Darwinian principle of natural selection was entirely compatible with the recently understood principles of Mendelian heredity, primarily focused on the consequences of selection, no doubt because a lot of the controversy over selection in the preceding decades had been about whether selection could actually be efficacious in promoting adaptive evolutionary change in the face of heredity, the latter often being thought of as a conservative mechanism opposed to change in the phenotypic composition of the population (discussed in detail by Gayon 1998). Dawkins, trained as an ethologist and working in the period just after the heated group versus individual selection debates sparked off by Wynne-Edwards (1962), understandably had interests spanning both the causes and the consequences of selection, and seems to have been primarily interested in convincing behavioural ecologists to stop thinking in terms of group selection and focus, instead on individuals, especially through the lens of asking what was good for their genes. The similarities between the approaches of Fisher and Dawkins, unlike their differences, are fairly inconsequential, being limited to a shared focus on adaptive evolutionary change and on genes as mediating the consequences of selection. They both also seemed to believe that the roles of mutational or developmental bias in micro-evolutionary dynamics were typically small enough to be safely ignored, which may not necessarily be appropriate. While undertaking this comparison of the two gene's eye views, we note also that Fisher's (1918, 1941) quantitative genetics perspective does not seem to have influenced Dawkins' (1976) thinking to any appreciable degree. This is significant because the 'average-effect' conceptualization of the response to selection by Fisher (1941) effectively allowed an approximate black-boxing of the complex and diverse ways in which gene-by-environment covariances and interactions arise when individual organisms have to make a living in the complex natural world with which they reciprocally interact. As a result, the complexity of the causes of selection could be meaningfully set aside, while he focused on how formulations reflecting transmission fidelities (i.e., breeding values and their variance, V_A, and the notion of heritability, h^2) could be used to understand the consequences of selection, using trait-variants as a focus. Dawkins (1976), on the other hand, appears to have implicitly dealt with situations of traits affected by one or a few genes, wherein the genotype-to-phenotype map was simple. This is why the Dawkinsian gene's eye view breaks down in the presence of gene-by-gene interactions, as pointed out by Sober and Lewontin (1982). This difference of perspective between the Fisherian and Dawkinsian gene's eye views is non-trivial: Fisher (1918, 1941) grappled with, and suggested means for approximately resolving, the complex effects of polygenic control of traits, and the multi-faceted interactions between organisms and their environments, on patterns in the transmission fidelity of trait-variants, whereas Dawkins (1976) did not. Instead, Dawkins appears to have assumed, much like Darwin, that transmission fidelities would typically be sufficiently high and similar across trait-variants so as to ensure good correspondence between reproductive and evolutionary success.

We now examine these two versions of the gene's eye view of evolution in the context of the four common concerns raised by those uncomfortable with the reductionism seemingly implied in them. The critique that the gene's eye view ignores the importance of organismal agency and inherency in evolutionary explanation applies primarily to the Dawkinsian gene's eye view. The Fisherian gene's eye view focuses on genes (as trait-variants) in the specific context of modeling the consequences of selection. Fisher's (1918, 1941) conceptualizations do also implicitly take into account complex and environment-dependent genotype-to-phenotype maps that arise due to interactions between organism and environment, and this is of course where the inherency and agency of individual organisms plays a role. This point is often missed, in our opinion, because Fisher's statistical resolution of this complexity into a measure of the resulting transmission fidelity under Mendelian inheritance rendered his treatment of the consequences of complex organism-environment interactions implicit and, therefore, not immediately apparent.

The next common critique that genes rather than individuals have been considered as the appropriate units of selection and micro-evolutionary dynamics is also largely pertinent only to the Dawkinsian gene's eye view. In Fisher's work, the gene is an appropriate unit of understanding and tracking micro-evolutionary dynamics within the domain of understanding the consequences of selection. It is only in Dawkins' work, perhaps more in rhetoric than his thinking, that it often appears that genes are being promoted as an appropriate unit of selection in the context of trying to understand both the causes and the consequences of selection. We reiterate that, in our opinion, the appropriateness and utility of genes (strictly, mostly alternative alleles, sometimes alternative one- or a few-locus genotypes) as units on which we can base our understanding of the consequences of selection derives from their being trait-variants, as opposed to individuals, and not from their being genes *per se*. For understanding the causes of selection, the appropriate and useful unit of selection is the individual organism (or in some cases the entire multi-locus genome), and not the trait-variant, whether phenotypic, genotypic or allelic in nature.

Similarly, the critiques that genes have been assigned an almost causal role in mediating micro-evolutionary change, and that they sometimes seem to have displaced the organism as the central focus of micro-evolutionary explanation, are also germane only to the Dawkinsian gene's eye view. Again, Dawkins' (1976) rhetoric, if not necessarily his underlying thinking, does often appear to imply that genes are somehow integral even to considerations of the causes of selection. Even if this was not, perhaps, his intent, this is often the impression left on students when they first read *The Selfish Gene*. This appearance of the gene having supplanted the organism as the central focus of micro-evolutionary explanation, not surprisingly, elicits an impassioned response from at least a plurality, if not an absolute majority, of evolutionary biologists who are exquisitely cognizant of the role of individuals, and their reciprocal interactions with their environments, in shaping the causes of selection. We think it unfortunate that many evolutionary biologists, nevertheless, do not seem to appreciate the irrelevance of individual organisms to considerations of the consequences of selection.

For the reasons articulated above, it is our view that the conflation of the Dawkinsian and Fisherian gene's eye views of evolution is not just inaccurate, misplaced and misleading, but has also contributed substantially to both the content and harsh tenor of some aspects of the EES-MS debates. Unfortunately, the Dawkinsian gene's eye view, because of its conflation with its Fisherian counterpart, is often wrongly taken to represent a central tenet of the MS. While the Fisherian gene's eye view was indeed central to the MS conception of the micro-evolutionary process, the Dawkinsian view is not. Indeed, the Dawkinsian gene's eye view is far more extreme and simplistic than its Fisherian counterpart, and often clearly inapplicable to '*micro-dynamics*' processes, which is why most population geneticists fail to ascribe to it any great significance in the context of micro-evolutionary explanation. An appreciation of this point would, we think, dissolve one aspect of the EES-MS debates altogether, and, in a lighter vein, developmental biologists and population geneticists would find themselves on the same side of a debate, with the narrow viewpoint of *The Selfish Gene* on the other. Finally, we would like to, nevertheless, stress that there is one domain of evolutionary explanation in which the Dawkinsian gene's eye view is valid and holds entirely, as also highlighted by Ågren (2021). This is, entirely unsurprisingly, the domain of understanding the evolutionary dynamics of selfish genetic elements within-individuals, and the patterns of their prevalence within- and among-individuals. We find it very fitting that a gene's eye view identified with *The Selfish Gene* (Dawkins 1976) should actually be appropriate and useful for understanding evolutionary changes in patterns of prevalence of selfish genetic elements. In our opinion, however, an evolutionary understanding of the dynamics of selfish genetic elements is more a vindication of the appreciation that any replicator can act as a unit of selection in the appropriate context, than a vindication specifically of the Dawkinsian gene's eye view of evolution in its entirety. This is not to say that the Dawkinsian view is not helpful in understanding evolution; however, its usefulness seems to derive from, and is restricted to, certain features it has in common with the Fisherian gene's eye view, such as the focus on trait-variants rather than individuals for understanding the consequences of selection, and an appreciation that selection as a process is more generally applicable than specifically to organismal evolution.

17.7 The Evolutionary Shaping of the Distribution of Phenotypes

پھرا کرتے نہیں مجرُوحِ اُلفت فِکرِ درماں میں
یہ زخمی آپ کر لیتے ہیں پیدا اپنے مرہم کو
phira karte nahin majrooh-e-ulfat fikr-e-darmaan mein
ye zakhmi aap kar lete hain paida apne marham ko
(They wander not in search of cure, Love's injured souls are firm and calm
To all the myriad wounds they nurse, they are themselves a soothing balm
– Allama Iqbal)

Before we begin to sum up by taking a comparative look at the DC, MS and EES in the next section, we would like to briefly share some thoughts on a certain perspective on the sixfold schema we introduced at the beginning of this chapter (Table 17.1); we think this perspective may permit the development of a framework within which we can examine claims about the logical relationships of different evolutionarily relevant phenomena such as selection, mutation, transmission fidelity, hybridization, developmental bias, or niche construction, as well as discuss issues about proximal versus distal causes of variation. We develop these ideas here primarily in the context of micro-evolutionary change, but we hope that this framework will be fully extendable in detail to macro-evolutionary change at higher levels of biological variation, too.

We suggest that the frequency distribution of phenotypic trait-variants in a population at present can be viewed as having arisen from a sequence of past alterations to distributions ancestral to the focal one. If we ignore for a moment the critical analysis of treatments of developmental bias by Salazar-Ciudad (2021), using our focus on within-population trait-variants of the scale typical of micro-evolutionary change as our excuse, we can say that a multitude of processes, culminating in one, some, or all of selection, mutation, migration and drift in the immediately preceding generation affect the present distribution. Selection here includes both selection bias, resulting from differential reproductive success, and transmission bias, via the pattern of transmission fidelities, across trait-variants. The penultimate distribution that these processes act on is, of course, generated by the cumulative effects of these processes over multiple preceding generations in this populational lineage, underscoring the fact that these processes affect both the input and output distributions at any generational time-step. Factors like mutational bias (Stoltzfus and McCandlish 2017; Cano and Payne 2020), and developmental inherencies and biases at the level of micro-evolutionary variants (Nunes et al. 2013; Salazar-Ciudad 2021; Newman 2022a, b) will also play a role in shaping these distributions of phenotypic trait-variants, but the magnitude of this role with respect to micro-evolutionary variations seems at present difficult to estimate empirically.

If we now consider earlier ancestral distributions in this populational lineage, they too would have been shaped by phylogenetic effects of even more ancestral lineage (s) which, in turn are likely to have had their own phenotypic distributions shaped in part by these same processes of selection, mutation, migration and drift, as well as the inherencies and biases intrinsic to biological systems at various levels of organization. In some cases, if a lineage splits, for whatever reason, the initial differences between daughter lineages could be of the '*micro-origins*' type, with subsequent adaptive evolution in different ecological contexts sometimes inducing further divergence in their respective phenotypic distributions that would qualify as being of the '*macro-origins*' type. In other cases, the daughter lineages could remain separated only by '*micro-origins*' level variations, even after the passage of considerable evolutionary time, especially if the split did not involve the daughter lineages thereafter living in very different ecological contexts. However, in the case of lineage splits occurring early in the evolutionary history of life-forms, often these

splits could result from '*macro-origins*' level differences to begin with. Thus, the newly established daughter lineages could often differ not just in their distributions of a very similar set of trait-variants, but often by having two distributions encompassing a fairly different set of trait-variants, resulting in the saltational origin of new species or higher taxa. In such macro-evolutionary lineage splitting events, the alteration of the distribution, both in terms of the domain of variants represented as well as their associated frequencies, would likely be achieved through processes other than selection, involving primarily developmental mechanisms falling within the '*macro-origins*' category of our schema in Table 17.1. Similarly, inter-specific hybridization events could effect sudden large changes to phenotypic distributions with respect to both the domain of variants represented as well as their associated frequencies and, once again, developmental processes would be important in determining the nature of successful hybridization events and their effects on the distribution of trait-variants in the initial population of the resulting hybrid species. A good discussion of the kinds of developmental and other phenomena that would fall within our '*macro-origins*' category (Table 17.1) can be found in Newman (2022a, b).

In classical population genetics models within the MS, both mutation and migration effectively reduce the transmission fidelities of allelic variants, the former explicitly and the latter implicitly. Therefore, selection, construed as encompassing differences in transmission fidelity among trait-variants, in addition to differences in their reproductive success, can also be conceptualized to include the effects of mutation and migration by subsuming their effects into transmission fidelity, constituting one locus of fuzziness in the boundary between processes in the '*micro-origins*' and '*micro-dynamics*' categories (Table 17.1). Selection in the broad sense just described is, of course, also tempered by drift, reflecting not just sampling errors but all kinds of stochasticity in the transmission of trait-variants to the next generation (strictly speaking, migration can be subsumed into selection when it is trait-variant-dependent, and into drift when it is trait-variant-independent, but that distinction is not crucial for the present discussion), We note that the cumulative effects of this broad-sense selection are largely restricted to ancestor-descendant lineages within species, pertinent to the '*micro-origins*', '*micro-dynamics*' and '*micro-patterns*' categories (Table 17.1). Development, on the other hand primarily acts to create entirely new ancestor-descendant lineages, effects pertinent to the '*macro-origins*' and perhaps '*macro-dynamics*' categories (Table 17.1), even though it may also have some, relatively smaller, role to play in generating trait-variants of the micro-evolutionary, within-lineage, kind through the kinds of mechanism considered in devo-evo or micro-evo-devo (*sensu* Prasad and Joshi 2003; Nunes et al. 2013, respectively). On the whole, though, it seems to us that key developmental processes tend to be relatively more conserved within- than between-lineages. Consequently, the interventions of development and broad-sense selection in the evolutionary process appear to be largely distinct, although constituent processes of both may well interact within our category of '*micro-dynamics*'. We do not have much feel for processes under the '*macro-dynamics*', and '*macro-patterns*' categories (Table 17.1), but our feeling is that these are not particularly

well understood, especially the latter, and we hope that future investigations will both refine and extend our understanding in this regard.

If we look at the two other major phenomena invoked in the calls for an EES in the context of this framework, it is clear that they play somewhat subsidiary roles in the evolutionary process, relative to both broad-sense selection and development, in the sense discussed above. Both non-genic inheritance and niche construction would appear to get assimilated into selection in this broad sense. Non-genic inheritance essentially affects the pattern of transmission fidelity of trait-variants, an integral component of broad-sense selection. Of course, non-genic inheritance also opens up the possibility of inheritance of characters acquired via environmental effects through epigenetic or ecological inheritance, or through cultural inheritance by learning. Cultural inheritance also enables horizontal (among members of the same cohort within a generation), oblique (from parental generation individuals to non-offspring), and reverse (from offspring generation individuals to parental generation individuals) inheritance, in addition to vertical (parent to offspring) inheritance which is the only form available under genic inheritance, especially in metazoa, if we discount the low frequency of horizontal gene transfer in such taxa. Cultural inheritance of the symbolic kind also permits inheritance over time-steps much larger than one generation: aspects of our behavioural phenotype can be altered by reading Aristotle or Rumi. However, in terms of the logical structure of the framework described above, these myriad corollaries of non-genic inheritance do not disturb the conception of selection in the broadest sense, being assimilable into the pattern of transmission fidelities. They can, however, affect rates of change of frequency of trait-variants very substantially, especially in the case of cultural inheritance. Nevertheless, we should add the caveat that our speculations in this regard are those of outsiders; none of us is technically a student of cultural inheritance or learning. It also seems to us that a detailed survey of the implications of each of these corollaries for how processes in the '*micro-origins*', '*micro-dynamics*' and '*micro-patterns*' categories (Table 17.1) play out in evolution is not as yet available, although this could also be an expression of our ignorance of the relevant literature. In comparison to non-genic inheritance, niche construction seems to play an even more supporting role, in that its effects on broad-sense selection are quantitative rather than qualitative, unlike the effects of non-genic inheritance. In the absence of accompanying non-genic inheritance of the niche constructing phenotype, niche construction by itself does not affect the pattern of transmission fidelities of trait-variants. Its primary effect is merely to increase the reproductive success of its bearers, by modifying the environment to be more suitable for their survival or reproduction.

Examining the various evolutionarily relevant phenomena in this framework thus reveals that development, non-genic inheritance and niche construction have very different logical relationships with selection in terms of how and in which specific context they exert their effects on the distribution of trait-variants within- and among-lineages. Development acts largely orthogonally to broad-sense selection and, in this sense, may well be considered a phenomenon belonging to the same logical category as selection, broadly conceptualized. In terms of its effect on within-

lineage trait-variant distributions, it is also more distal than broad-sense selection, which is far more proximate. Non-genic inheritance can be subsumed into broad-sense selection but can have major, qualitative-grade effects on the outcomes of broad-sense selection. Niche construction can also be subsumed into broad-sense selection but, by itself, is likely to have smaller, quantitative-grade, effects on the outcomes of broad-sense selection, compared to non-genic inheritance. It is, thus, clear that the three major elements that make up the calls for an EES are actually very different in the manner in which they affect important aspects of evolutionary explanations.

17.8 The Extended Evolutionary Synthesis (EES) in the Context of the Darwinian Core (DC) and the Modern Synthesis (MS)

اُڑائے کُچھ ورق لالہ نے، کُچھ نرگِس نے، کُچھ گُل نے
چمن میں ہر طرف بِکھری ہوئی ہے داستاں میری
udaaye kuchh waraq lale ne, kuchh nargis ne, kuchh gul ne
chaman mein har taraf bikhri hui hai daastaan meri
(Tulip, Narcissus, and Rose, all took some pages from my book
And garden-wide did spread them: thus, my tale immortal did become)
– Allama Iqbal)

We have already compared the MS and the DC in a previous section in order to assess how much they differed, and in what specific manner. We now examine the major issues raised in the calls for an EES and try to fit them into the framework established in earlier sections. The main issues that proponents of the EES feel were neglected in the MS are the role of development, non-genic inheritance, and niche construction, respectively, in evolutionary explanation (summarised in Laland et al. 2015). There is also a discomfort with the perceived emphasis of the MS on gradualism and uniformitarianism (e.g., Eldredge and Gould 1972; Gould 2002; Salazar-Ciudad and Jernvall 2005; Newman and Bhat 2009; Beatty 2022). We are also uncomfortable with gradualism and uniformitarianism, and with the lack of consideration of non-genic inheritance and developmental (as opposed to merely developmental genetic) mechanisms in mediating micro-evolutionary change. On the other hand, we believe that many of the claims of both the neglect and conceptual importance of niche construction in evolutionary thinking are exaggerated and often misplaced (Gupta et al. 2017a).

We are also somewhat uncomfortable with the use of the term ‘synthesis’, in the context of both the MS and, even more so, the EES. At least in the sense of Hegelian dialectics (Maybee 2020), ‘synthesis’ implies a dialectical combination of antithetical elements into a coherent whole (see also Sarkar 2004). As we see it, the development of evolutionary thinking after Darwin has been more of a steady accretion of new facts and insights around a relatively unchanged essence embodied in the DC, rather than a sequence of syntheses. Before the calls for an EES took on the largely self-assigned accoutrements of a heresy, about 10–12 years ago, an alternative term ‘Standard Evolutionary Theory’ (SET) was also used for the MS,

emphasizing the fact that it was an evolving set of explanations, some closely, and others more loosely, intertwined with one another (Kuschera and Niklas 2004; Pigliucci and Kaplan 2006). In the minds of many of us who broadly identify our research as falling within the MS paradigm, the MS actually represents something more like an evolving SET constituting a sort of 'framework theory', rather than the often narrow and dogmatically gene-centric way in which it is represented in many textbooks; Antonovics (1987) actually referred to the evolutionary milieu in the 1970–1980s as a dys-synthesis! We suspect that the well-known 'phylogenetic inertia' seen in textbooks has played a role here, because some of the early textbooks of evolution were written by researchers closely associated with Dobzhansky, who espoused a fairly narrow, gene-based, gradualist and uniformitarian view of evolution, even equating evolution with a gradual change in allele frequencies in a population (e.g., Dobzhansky 1937). In fact, the received textbook view of the MS was inordinately influenced by Th. Dobzhanky and E. Mayr (also largely committed to gradualism: Meyer 2005), whereas the somewhat more nuanced and differing perspectives of people like J. B. S. Haldane, G. G. Simpson, and G. L. Stebbins did not receive that extensive a representation. In contrast to the impression left by textbooks, we have scarcely ever encountered the gradualist and uniformitarian positions among practising evolutionary biologists who self-identify with the MS paradigm in a broad sense, as opposed to the narrow textbook sense.

We look forward to a more meaningful incorporation of developmental perspectives, both evo-devo and devo-evo (or micro-evo-devo), as well as non-genic inheritance (especially cultural and ecological), into evolutionary thinking about problems pertaining to both the origin and prevalence of micro- and macro-evolutionary variations. In particular, we think that a developmental perspective is likely to yield better explanations in the future not just of the '*macro-origins*' and '*micro-origins*', but also the '*macro-dynamics*', and '*macro-patterns*' categories (Table 17.1), by enhancing our understanding of developmental effects on parameters associated with lineage splitting and within-lineage phyletic change. Developmental considerations can also play a role in explanations within the '*micro-dynamics*' and, therefore, '*micro-patterns*' categories (Table 17.1), as Darwin had recognized with his emphasis on growth correlations, and we hope that future work in micro-evo-devo will prove fruitful in this regard. Non-genic inheritance directly impacts our understanding of phenomena under the '*micro-dynamics*' category, with the effects of oblique, horizontal and reverse inheritance on micro-evolutionary dynamics likely to be a rich field of inquiry. We further believe that such an enhancement to evolutionary thinking will very much be in the spirit of the DC which was fairly agnostic about mechanisms underlying heredity and variation, albeit out of a necessity born of ignorance. In the spirit of Joseph Felsenstein's assessment of the contributions of R. A. Fisher to population genetics, we suspect that evolutionary biology for quite a long time to come might justifiably be described as 'an exercise in writing footnotes to Darwin'. We also prefer not to think of the steadily expanding domain and detail of evolutionary explanation as constituting an evolutionary theory. It is more like an intertwined mass of multiple growing theories, but also with diverse interstices. In this sense, we agree with the

sentiment that we need an enlargement of evolutionary explanation, not another 'synthesis' (Antonovics 1987; Stoltzfus 2017). Given this view, we think it is unfortunate that EES, especially in the writings of certain people, has been projected as being somehow antithetical to the Darwinian view of evolution. This is accomplished by treating the MS as synonymous with Darwinian thought, ignoring some of their differences with regard to factors other than selection and heredity, and then depicting the MS in an extremely narrow manner, effectively setting up a straw-man. The fact that textbooks often also depict the MS quite narrowly unfortunately contributes to the acceptability of this rhetorical tactic. This has sadly, but not surprisingly, often provoked somewhat dogmatic and intransigent responses from many who broadly identify with an evolving SET rooted in Darwin's crucial insights.

Unfortunately, scientific disciplines with an elaborate and well-articulated set of views on their principal questions can often react like religious orthodoxies bolstered by venerated canonical texts, turning inwards and protecting their borders from incursion (Kitcher 1987; Joshi 2005, 2014). The other side of this coin is the continuing attraction of what we term the 'Galileo syndrome'—we scientists often like to see ourselves as champions of heretic interpretations of the natural world, struggling against the oppressive obduracy of the orthodoxy. Together, these two phenomena tend to result in discussion giving way to debate, and often dispute, thereby constraining rather than facilitating intellectual progress.

17.9 Summary and Conclusions

کھول کر آنکھیں میرے آئینۂ گُفتار میں
آنے والے دور کی دھُندلی سی اِک تصویر دیکھ
khol kar aankhein mere aaina-e-guftaar mein
aane waale daur ki dhundli si ik tasveer dekh
(Behold in the mirror of my words and rhymes:
A shadowy picture of the coming times
– Allama Iqbal)

In some ways, the EES-MS debates are reminiscent of the old Indian parable of the blind men and the elephant, with each arguing for a different identification of the animal based on which part of it they happened to touch (first recorded from Buddhist sutras, English translation on pgs. 93–96 of Strong 1902). Evolutionary biology covers a vast domain and the evolutionary process essentially encompasses the whole of biology, trifurcated, in one perspective, among the three major interacting phenomena of development, ecology and heredity (Joshi 2005). There is more than a little element of people talking past each other in the EES-MS debate, with both sides often tending to conflate their set of insights and understanding with the entirety of evolutionary explanation. Even the very notion of an 'extended' evolutionary synthesis seems to implicitly assume that a single, comprehensive, and unified 'theory of evolution' is not just desirable, but possible. We think that it is unlikely that we can have such a unified theory, which would be nothing less than a

'unified theory of everything' in biology. The effects of evolution, as argued persuasively by Rose et al. (2005), appear to be local rather than global, suggesting that perhaps the only grand generalization possible about evolution is that it occurs, although this realization obviously does not preclude useful theorizing about sub-phenomena within evolution.

As a consequence of the above, we believe that it is worthwhile to compare different sub-phenomena within evolution based on whether, and how, they impinge upon the origin, dynamics or patterns of prevalence of micro- or macro-evolutionary variations (see Table 17.1). We suggest that selection is relevant, and very important, to understanding the dynamics of, and patterns in, the prevalence of alternative trait-variants ('*micro-dynamics*' and '*micro-patterns*' in Table 17.1). Development or mutation, which exercises its phenotypic effects through development, on the other hand, are relatively more relevant than selection when addressing questions about the origin and, to a lesser degree, the dynamics, of micro- and macro-evolutionary phenotypic variations. Niche construction, contrary to many claims, primarily plays a role, along with many other ecological processes, in modulating the action of selection. Moreover, the relevance and centrality of selection in evolutionary explanation is largely restricted to explanations of adaptive micro-evolutionary dynamics; it may not even make sense to think of selection among alternative life-forms as an analogous process to selection among trait-variants (what would one make of the question whether an annelid bauplan had higher fitness than an arthropod one, given that they would normally occupy very different ecological niches?). Thus, selection does have a fairly important position, relative to several other phenomena, but only in a subset of evolutionary explanation, in the context of explaining the prevalence of trait-variants subject to processes affecting micro-evolutionary dynamics. Species selection may be similar to selection among trait-variants, but it is not yet very clear as to how widespread it is, or indeed the degree to which its mechanisms can be considered analogous to those through which selection operates on trait-variants within species. Critically evaluating the possibility of selection among species, or among life-forms, is difficult in the present state of our knowledge of the relevant phenomena. Some kind of generalized process, in the sense of altering the available phenotypic space, however, does seem to occur at multiple levels of biological organization, although it is not clear that one can equate this phenomenon with selection in its classical micro-evolutionary sense. It is also important to appreciate that the simplistically sweeping and all-encompassing manner in which the MS is unfortunately often described in textbooks of evolution is actually far removed from the much more nuanced views of most of us who work within the domain of, and identify our research programmes with, the MS.

We also think that it is high time we revisited some of the ways in which we have conceptualized fitness and selection because focussing on transmission fidelities as an integral part of fitness, and restricting the use of the term fitness to the one-step rate of increase of trait-variants, actually clears up quite a few existing confusions in the field, by illuminating their underlying cause. Similarly, we believe that discussions of the role of the individual in evolutionary explanation would benefit from a consideration of which specific phenomenon in evolution one is trying to

explain. Individuals are important foci for considerations of development, and of the effects of ecological context on survival and reproduction, i.e., for questions pertaining to the causes of selection. However, in trying to explain the prevalence of alternative trait-variants (i.e., the consequences of selection), focusing on individuals is a distraction that has already led to considerable confusion within the field over the last many decades, especially in evolutionary ecology. We believe that debates between those who think that individuals are important to evolutionary explanation, and those who do not, have largely been unproductive, as have the debates about the units of selection, precisely because there has been insufficient attention paid to what exactly it is about the evolutionary process that one is trying to explain through a consideration of an individual or a trait-variant, respectively.

We should also point out that, given our focus on the EES-MS debates, we have entirely ignored, including in our categorization in Table 17.1, one very important perspective on the evolutionary process: that of coevolution. Species interactions, antagonistic and mutualistic alike, not only shape the evolution of individual species but, in many ways, are integral to most instances of selection and, indeed, life itself (Thompson 2005, 2013). It is perhaps not entirely a coincidence that some of the earliest tests of Darwinian explanations of evolution in nature, as opposed to the laboratory (Dallinger 1878), were in the context of species interactions: mimicry (Bates 1861; Müller 1879), and pollination (Müller 1873). With this caveat out of the way, we now adumbrate what we feel are the 11 major take-home messages from what has been discussed in this chapter. We will then conclude with some thoughts about the way ahead for the resolution, or rather the dissolution, of the EES-MS debate.

The main points that we would like readers to take from this chapter, starting from the most general and proceeding to the more specific, are the following:

1. One can meaningfully think of diverse areas and aspects of evolutionary explanation as mapping onto a schema organized into six categories of questions pertaining to the origins, dynamics and patterns of prevalence of micro- and macro-evolutionary phenotypic variations (Table 17.1). Most concerns of the DC and MS are seen to be about explaining the dynamics and prevalence of trait-variants ('*micro-dynamics*' and '*micro-patterns*' in Table 17.1), whereas the more cogent aspects of the calls for an EES mostly focus on explanations of the origin (and secondarily, to a lesser degree the dynamics) of variations, primarily, but no longer exclusively, at the level of macro-evolutionary phenotypic variations.
2. In addition to marshalling a compelling body of evidence for evolution in the sense of species being related through ancestor–descendant relationships, and describing natural selection as a potent mechanism for adaptive evolutionary change, Darwin also made three other very significant, and somewhat underappreciated, contributions that shaped subsequent evolutionary thinking: the five together constitute the conceptual crux of the DC. One was to reconceptualize heredity by shifting its focus from the preservation of types across generations to the transmission of variation among individuals, even siblings.

The others were to focus on trait-variants rather than individuals (the atomization of the individual), and on mechanisms for changes in their prevalence. It was these three latter contributions that permitted the development of a theory of micro-evolutionary dynamics, and also initiated the consequential emancipation of heredity, in the sense of transmission, from the confining embrace of both development and the individual, a process eventually completed almost 65 years later by T. H. Morgan and others.

3. Overall, the MS represented a narrowing of the DC, though not as greatly as Neo-Darwinism (see discussion in Forsdyke 2001), largely because of the need to show that evolutionary explanations of the mechanisms for change in the prevalence of trait-variants were consistent with the newly re-discovered principles of Mendelian genetics. The DC was more open to varied mechanisms for both the generation of trait-variants and their transmission to offspring. However, some elements of the MS, like quantitative genetics, were actually broader and more nuanced than is often thought to be the case. The phenomena considered under the aegis of the EES calls are best seen as complementary to those emphasized in the MS, in some cases, harking back to the broader, more eclectic, DC.
4. Since quantitative genetics is typically encountered only cursorily, as a small part of a population genetics course, it has been largely unrepresented or misrepresented in the EES-MS debate. Many critiques of the MS (for perceived errors of omission) are actually seen to be misplaced in the light of a clearer understanding of the essential nature of quantitative genetics. Despite its name, quantitative genetics constitutes, *inter alia*, a phenotypic theory of micro-evolutionary dynamics that permits the many complexities of the genotype to phenotype map, including phenotypic plasticity, and gene-by-gene and gene-by-environment interactions, to be distilled into the genetic variance-covariance matrix which, in turn, is what mediates, via transmission fidelities, between selection acting on individuals and the consequent evolutionary change.
5. Quantitative genetics is consistent with Mendelian genetics, but can also be deployed to explain micro-evolutionary dynamics in systems with arbitrary systems of inheritance, so long as the analogues of heritability or the genetic variance-covariance matrix can be delineated. In this sense, quantitative genetics constitutes a far more general theory of micro-evolutionary dynamics than population genetics, which is limited by the twin assumptions of Mendelian inheritance and simple control of the relevant phenotypes by one or a few genetic loci. A better appreciation of the nature of quantitative genetics would render some EES-MS discussions more meaningful and useful.
6. At present, the term fitness is variously used to quantify the reproductive success of an individual, the average reproductive success of individuals exhibiting a specific trait-variant, the one-step rate of increase of a trait-variant, and the long-term evolutionary success of a trait-variant or lineage. We strongly suggest that the use of the term fitness should be restricted to the one-step rate of increase of a trait-variant. This usage explicitly reflects the important role of transmission fidelity as a fundamental part of the process of selection, linking variation in

reproduction of individuals exhibiting different trait-variants to differences in prevalence of those trait-variants. As a corollary, we believe it is not helpful to talk about the fitness of individuals as a synonym of their reproductive success, since this only results in confusion, particularly noticeable in the literature on kin-selection (see 8, below).

7. Individuals, with their inherencies and agency, are important to explanations pertaining to ontogeny and ecology, since it is the individual that reproduces and interacts with its abiotic and biotic surroundings (i.e., the *causes* of selection). Yet, a theory of micro-evolutionary change in the prevalence of different trait-variants (i.e., the *consequences* of selection) cannot be built at the level of the individual, since every individual, considered holistically as a complex multi-trait phenome, is essentially unique. This implies that, at the level of individuals, it will be possible to only describe the replacement over time of one set of unique individuals by another set of completely different unique individuals.
8. Focusing on trait-variants as the meaningful units on which micro-evolutionary change can actually be described and quantified also highlights the misconceived nature of the units of selection arguments focused on individuals versus genes/genotypes. A phenotypically unique individual cannot exhibit a change in frequency over multiple generations: it can only exhibit a one-time change from being alive to being dead. The consequential issue, therefore, is whether to consider phenotypic or genotypic trait-variants as the units of micro-evolutionary change in any given scenario, and the choice will depend on context. As a result, ascribing fitness to individuals is not helpful (see 6, above), except to assuage a deeply ingrained discomfiture that we experience when unable to ascribe agency to objects that play a role in our explanations of the world and cosmos. Focussing on trait-variants as the units of micro-evolutionary change, together with limiting the use of the term fitness to the one-step rate of increase of a type-variant, also entails the desirable consequence of rendering all fitness inclusive, thereby eliminating a major source of confusion and debate—the unnecessary distinction between direct and inclusive fitness.
9. Development is important, indeed crucial, to a large subset of evolutionary explanations, especially those dealing with issues pertaining to the origin of phenotypic variations. Nevertheless, a detailed understanding of development, or of the complex genotype to phenotype map, is largely unnecessary for constructing and deploying a meaningful and useful theory that can approximate patterns in the dynamics and prevalence of trait-variants. Unfortunately, since work on the prevalence of trait-variants has historically constituted a very large proportion of research on evolution, a misleading impression that development is unimportant to explaining evolution has sometimes been created, especially in textbooks.
10. It is useful to think of not one, but two gene's eye views of evolution, that should not be conflated. The Dawkinsian (Dawkins 1976) and the Fisherian (Fisher 1918, 1930, 1941) gene's eye views of evolution differ substantially, and it is only the latter that is integral to the MS.

11. Heredity, in the broad sense of a mechanism(s) inducing positive correlations between parent and offspring phenotypes, or even phenotypes of interacting individuals not related to one another (as in cultural inheritance), is important in evolution because it connects the behavioural or reproductive success associated with a trait-variant to its consequent prevalence. As a mediator of transmission fidelity, heredity is, in fact, inseparable from both fitness and selection. It is important, however, to break out of our twentieth-century epistemological straitjacket that conflated heredity with the strictly parent-offspring transmission of genes. Epigenetic, ecological and cultural inheritance all have potentially important roles to play in evolution and can serve to link not just parents and offspring, but also unrelated individuals within and across generations. More importantly, differences in the kinds of transmission fidelity patterns of trait-variants that are primarily passed on by one or the other alternative mechanism of inheritance can greatly impact observed patterns of micro-evolutionary dynamics, even if the ecological factors associating some benefit with those trait-variants remain unchanged.

To sum up, our view on the EES-MS debate is that there is actually relatively little to debate about, barring rhetoric, if we get past our parochial sub-disciplinary viewpoints and take a much broader view of the domain of evolution. For example, the complaint that the MS does not address the origins of form, often made in the evo-devo literature, is akin to reprimanding evo-devo for not shedding light on the dynamics of allele frequencies under the joint effects of mutation, drift and selection. Existing theories of micro-evolutionary dynamics do not even try to address the origins of form (contra Newman 2021). It should be possible to appreciate that while development is very relevant to questions about the origin of macro- and even micro-evolutionary variations, it can nevertheless be safely ignored when addressing most questions about the prevalence of alternative micro-evolutionary variants, at least to a good level of approximation. We need to appreciate that phenomena highlighted in the MS, and in the calls for an EES, respectively, have their primary focus on different categories of questions outlined in the schema in Table 17.1, and that approaches focused on different phenomena are, thus, complementary rather than conflicting, and that none of these approaches has a claim to represent either the whole of evolutionary biology, or its most important components. We believe that the EES-MS debate has been exacerbated by the changing cultural milieu of science, in which 'marketing' has become increasingly crucial to how impactful any piece of work will be assessed to be (Joshi 2014; Gupta et al. 2017a). This leads inexorably to exaggerated claims to novelty and generality, as well as to rhetorical flourishes that serve to obfuscate rather than emphasize similarities or complementarities across approaches. Dialogue, unfortunately, becomes difficult when the participants are largely talking down to, or past, one another. Yet, evolution is far bigger than all of us and, indeed, than all of our sub-disciplinary biases and viewpoints, and meaningful dialogue across the diverse sub-disciplines that make up evolutionary biology is what is really needed at this time. It might, therefore, be advantageous now to abandon the EES-MS dichotomy altogether, and discuss various processes and

factors affecting the origin, dynamics and patterns of prevalence of variants, at various levels of biological organization, as differing but complementary parts of a complex, nuanced, multifarious and evolving SET, in the spirit of Bob Dylan (2014), when he wrote in the song 'Caribbean Wind': '*. . .there ain't a thing you can do about it, so let us just agree to agree*'.

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Evolution Is Bigger than All of Us: A Commentary on Vidya, Dey, Prasad, and Joshi

18

Vassiliki Betty Smocovitis

Abstract

This commentary responds to the historical reckoning offered by Vidya et al, and their more pluralistic view of evolutionary theory, which is best viewed as an "intertwined mass" of multiple growing theories instead of the simplistic rendering offered by the EES. It also comments on their use of the "Galileo Syndrome," the propensity of some scientists to champion their arguments—and themselves—by manufacturing oppressive orthodoxies which enable them to martyr themselves or elevate themselves as heretics.

Keywords

Evolutionary synthesis · Evolutionary theory · History · Perspectivism · Pluralism · Rashomon effect · Galileo syndrome

Evolution is indeed bigger than all of us, as T. N. Vidya, Sutirth Dey, N. G. Prasad, and Amitabh Joshi state in a chapter of exceptional eloquence and profound insights. As a discipline devoted to the study of evolution, at least the organic aspects, evolutionary biology appears to have undergone explosive growth since the 1940s when a self-aware and self-identified community of evolutionists first came together to found the first international society and a journal and celebrate the emergence of a new discipline (Chap. 2). It is not just that it has "grown," of course, so much as it has accepted, embraced, and assimilated novel approaches and methodologies from a diverse range of areas, while it has reached out to medicine and agriculture and

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even more recently to areas like robotics and AI. Meetings and conferences associated with organizations have grown too, and more and more specialized workshops and symposia are being held. It is for good reason that some now speak of theoretical versus applied evolutionary biology because the very character of the science has altered becoming more enriched as evolutionary theory is meeting more and more practical needs.

But with all the excitement, dynamism, and success, there has come a cost: an increasing heterogeneity leading to incoherence and at times an inability to communicate in a meaningful way. This has increasingly led to tensions between subdisciplinary areas, as well as individuals upholding one or another point of view. It is not as though the 1940s were dominated by unity as well as a strong consensus, as I have argued in my own work (Chap. 2 and see Smocovitis 2020); indeed, a stunning diversity of scientific opinion existed then, but that has only grown in time, and as the history recedes from memory, the tensions, friction and struggles get streamlined, diminished, and even erased so as to make the trajectory of science appear unidirectional, inevitable, and the emerging theory unified, as well much more clearly defined. What gets lost are the twists and turns, the fits and starts, the complex process by which scientists work at consensus building, if it is ever really attained. It is, in short, more of a complicated mess than is normally indicated in too many textbook histories.

Though they do not go to great length to discuss it, Vidya et al. know all this. It is there undergirding their chapter, aspects surfacing at key moments to bolster their claims. They build on it, and wisely, they begin their paper by acknowledging their own point of view, their bias, or rather biases, since this is a multi-authored paper, as they tackle arguments made for an extended evolutionary synthesis (EES) (Pigliucci 2007; Pigliucci and Müller 2010a). Their approach is simple, straightforward, and especially relevant because the EES is grounded in a historical argument (Lewens 2019). They set forth to trace the conceptual history of evolutionary theory beginning with Darwin, singling out the core of his theory, which they term the Darwinian core (DC), and then take special care examining it during the period of the evolutionary synthesis or the modern synthesis (MS). The goal is to compare the fate of that DC as it morphed into the MS. They then move to the EES to scrutinize the argument made by its advocates: that the MS served to constrain or hinder the development of evolutionary science. Vidya et al. want to know whether the MS can accommodate the EES or if the EES is a proper extension of the MS. They tell us explicitly that what they hope to offer is a "meta-perspective" of sorts rather than just a review, one that will seek an answer to the relationship between the MS and the EES, and again to determine if there is any real merit to the claims made by the EES. Finally, instead of focusing on the differences between the DC, the MS, and the EES, they also focus on similarities, finding the points of agreement between all three. This latter methodologic point, i.e., focusing on the points of agreement is a smart move since it enables them to get at the rootedness of the theory, putting flesh on those unvarying elements that persist instead of being taken away by looking only for the differences (see my reply to Svensson, Chap. 4).

Vidya et al., begin with the belief that the claims made by EES are "muddled" and "often overhyped," with which I agree. They also draw on an impressive body of literature including careful reading of historical scholarship as well as engaging philosophical perspectives. I applaud them for this, especially as it reflects the kind of substantive engagement with the past that I wish advocates of the EES would also do. Indeed, as I see it, a serious engagement with history would change the narrative pushed by the EES, especially the one retold in the 2010 books that ground the EES (Pigliucci and Müller 2010a, b). Nor do Vidya et al. make wild or erroneous claims about the past. They are modest, insightful and focus their inquiry beginning with an examination of Darwin's work and what they call the "trait-variant," which refers to "individuals exhibiting difference in alternative versions of one or a few traits." This, they say, "mirrors" the way in which Darwin used the terms "character" or "variant." Although it is a kind of presentist tool (Darwin did not use this term) "trait-variants" subsequently enable them to trace the conceptual history with greater ease and allow them to avoid several pitfalls or controversies in the history of evolution such as for example getting bogged down over disputes about the relevant unit of selection or evolution at the same time it enables them to understand microevolutionary change. Provocatively, they state that "individual" is not much more than a "red herring" when trying to understand microevolutionary change (I would very much love to see Ernst Mayr's reaction to that).

They are right about a great deal of the history that they cover, starting with the fact that there was little that was original with Darwin's view of natural selection; others had conceived of similar mechanisms before, but what Darwin did was to effectively operationalize natural selection, showing us how it would work. They are right about the emphasis on adaptation especially in the nineteenth century because of the dominance of the natural theology tradition that saw design in nature, and that sought to connect with the designer by understanding the designer's works. Their discussion here is especially lucid as well as convincing and they are right that natural selection itself has not altered hugely except in our appreciation of its consequences as gained through the application of mathematics, or in being applied to phenomena we know now like meiotic drive or transposable genetic elements, though I would add that the creative versus eliminative dimensions of natural selection has cycled off and on in the thinking of many evolutionists too. They take us through developments at the turn of the century after the "rediscovery of Mendel" and the need to bring the newer Mendelian genetics together with Darwinian selection theory and then take us through to the important events associated with Julian Huxley's book of 1942 traveling under the banner of the modern synthesis (MS). Comparing the DC with the MS, they make a stunning observation at least in my reckoning: the latter represents a "slightly narrower" conceptualization of the process of evolution. This is of course consistent with William B. Provine's argument for the "evolutionary constriction" that the evolutionary synthesis conceptually involved more of a narrowing, and ejection of alternative theories operating in evolution (Provine 1989), Lamarckism or the inheritance of acquired characters, being one of the most well-known of these. And intriguingly they rue the emphasis given to population genetics over quantitative genetics and argue for thinking in

terms of "trait-variants" which grant greater clarity to discussions pertaining to microevolution overall.

In comparing the DC with the MS and the EES, furthermore, Vidya et al. single out some of the major issues raised by calls for the EES by Laland et al. in their 2014 paper: the neglect of development, non-genic inheritance, and niche construction (Laland et al. 2014). They also add that the emphasis on gradualism and uniformitarianism discomfits them and agree that the MS's lack of consideration of non-genic inheritance and developmental mechanisms in microevolution is of concern, but do not accept arguments that niche construction is as conceptually important or as neglected as advocates of the EES seem to think. They call for the need for further work in development as well as non-genic inheritance and overall believe that much of this will address lapses in the original DC which was unable to incorporate development or heredity because these had been incompletely understood in Darwin's day. Vidya et al. continue the comparison and make several important claims. For example, that the term "synthesis" no longer seems applicable and indeed might even be confining overall since it implies a dialectical combination of elements that do not reflect the structure of evolutionary theory. That theory is more like an "intertwined mass" of multiple growing theories. They question the way in which the MS has been depicted simplistically and narrowly in textbook accounts, denying the plurality and complexity of a theory that itself evolves and that still has roots in Darwin's many insights. Indeed, the centerpiece of their own thinking emerging from this historical examination is that there has been a steady "accretion" of new facts and insights that continue to add or build on the DC. In short, Vidya et al. see continuity as well as growth in multiple directions consistent with a pluralistic and network-like theory embracing and accepting new disciplines, methods, and entire areas of research.

I cannot possibly disagree with any of these conclusions, since they seem so consistent with the historical record as I understand it and as other commentators have recently noted, coming at this from many directions (Scheiner and Mindell 2020). I would only add that others have called for a similar end to the term "synthesis" (Chap. 11) and that its use has actually diminished, slowly replaced by the term "integration" and its cognates, starting in the 1960s. Evolutionary biology itself became included in the larger category of "integrative biology," which also included developmental biology. Integrative biology itself has a fascinating history that has yet to be written.

Vidya et al. are also correct that disciplines have their own cultures of sorts and that the dynamic between them is evident when different perspectives are involved. They make a delightful reference to what they call the "Galileo syndrome," alluding to the propensity of scientists to champion their arguments—and themselves—by manufacturing oppressive orthodoxies which enable them to martyr themselves or elevate themselves as heretics. There is more than a kernel of truth to this: Stephen J. Gould, as one example, got a lot of mileage out of this with his claim that the synthesis "hardened" around a selectionist orthodoxy, at the same time he resurrected Richard Goldschmidt as the brilliant and insightful "heretic" (Chap. 4). This kind of bold historical-sociological claim, I think does help us understand some

of the dynamics behind the more hyperbolic claims associated with EES. But I am especially awed by Vidya's final claim that "evolution is bigger than all of us" as a way of understanding that we all may have plausible claims about the natural world and evolutionary change. This does remind me of the struggles common to "Rashomon effect" that I bring up in my reply to Svensson (Chap. 4), but here it is "souped-up" and made even more profound by the reference to the old Indian parable of the impaired or blinkered vision of men trying to describe an elephant. Indeed, this rings very true, and here I will end by saying that the last time I saw an analogy like this drawn in evolutionary biology was in a paper of 1965 by G. Ledyard Stebbins arguing for peace and perspectivism while systematists and plant evolutionary biologists were being rocked by the encounters with the newer molecular biology (Stebbins 1965). Instead of an elephant, he chose a creature more closely associated with evolution, a giraffe. He wrote:

> Biologists and laymen alike often try to answer such a question as: "Why does the giraffe have such a long neck?" The answer which a biologist would give to this question would depend upon his training and interests. A field naturalist might reply: "Because this fits the giraffe to his environment by enabling him to get food from the leaves of tall trees." A Darwinian evolutionist would say: "Because, in the remote past, certain animals with longer necks were better able to reach food, and so produced more offspring, to which they transmitted their longer necks." The answer of a developmental biologist might be: "Because, in the embryonic and fetal development, a large quantity of growth hormones becomes concentrated in the neck region, thus stimulating the excessive development of the seven neck vertebrae and the tissues associated with them." Finally, a modern molecular geneticist would be likely to answer: "Because a part of the code in the DNA of the giraffe's nuclei carries information for a long neck." (Stebbins 1965, p. 104)

Stebbins continued with this:

> When we look over these answers one after the other, we can easily see that none of them is actually wrong, and none of them is more basic or important than the others. Each is essentially correct but incomplete. Surprisingly enough, however, one can easily find sophisticated, high level discussions of similar biological problems in which one of these types of answers is considered to be the only significant biological one, and the others are minimized or completely neglected. Even when the need is recognized for these different approaches to such basic problems of biology, each type of answer is often regarded as sufficient in itself and separate from the others. The more we learn about the nature of life, however, the more we realize that these different avenues of approach are intimately connected with each other, and that each of the types of answers given above is incomplete unless it takes into account the facts which have led to the other answers (Stebbins 1965 p. 104).

Though this paper on the "gene to character transformation" was written in the middle of a heated debate some 60 years ago, Stebbins's insights still ring true, and I hear a similar call for perspectivism in Vidya's very satisfying account assessing the EES.

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Why Evolution Is Bigger than all of Us: A Reply to Smocovitis

19

T. N. C. Vidya, Sutirth Dey, N. G. Prasad, and Amitabh Joshi

Abstract

When reading the appreciative and accurate summary of our chapter by Smocovitis, we were struck, among other things, by her choice of one of our, somewhat cliched, rhetorical flourishes for the title of her commentary. This led us to ponder upon the special quality of evolution that makes this cliche particularly apt and resonant, and led to this musing inspired by her commentary on our chapter, particularly its title. We suspect that a major reason for this is the dual existence of evolution as both a 'character' (Urdu: کردار/*kirdaar*) that embellishes, and a 'perspective' (Urdu: نظریہ/*nazariya*) that informs, biology. Smocovitis emphasizes in her summation, invoking the 'Rashomon Effect', that 'we all may have plausible claims about the natural world and evolutionary change'. We entirely agree with this assertion and suggest that this stems from

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thinking of evolution as a perspective far more than from evolution as a character. As a character, evolution is a discipline within biology, with a reasonably well-defined set of tools—observational, experimental, and conceptual—which it deploys to understand the diversity, relatedness, and adaptedness of life forms, even as it has grown explosively since the 1940s, as Smocovitis puts it. As a perspective, evolution transcends the domain of biology, as first explicitly predicted by Ernst Haeckel: Smocovitis brings up how an evolutionary perspective informs agriculture, medicine, and even robotics and AI. In a way, this dual existence is consonant with the original lexical root of evolution as an unfolding: just as specific individual characters unfold in the course of a play, so too, overall, does the script, based on the perspective in which it is embedded.

Keywords

Evolution as perspective · Evolution as character · Evolution in pedagogy · Ernst Haeckel · Theodosius Dobzhansky

When reading the appreciative and accurate summary of our chapter by Smocovitis, we were struck, among other things, by her choice of one of our, somewhat cliched, rhetorical flourishes for the title of her commentary. This led us to ponder upon the special quality of evolution that makes this cliche particularly apt and resonant and led to this musing inspired by her commentary on our chapter, particularly its title. We suspect that a major reason for this is the dual existence of evolution as both a 'character' (Urdu: کردار/*kirdaar*) that embellishes, and a 'perspective' (Urdu: نظریہ/*nazariya*) that informs, biology. [1] Smocovitis emphasizes in her summation, invoking the 'Rashomon Effect', that 'we all may have plausible claims about the natural world and evolutionary change'. We entirely agree with this assertion and suggest that this stems from thinking of evolution as a perspective far more than from evolution as a character. As a character, evolution is a discipline within biology, with a reasonably well-defined set of tools—observational, experimental, and conceptual—which it deploys to understand the diversity, relatedness, and adaptedness of life forms, even as it has grown explosively since the 1940s, as Smocovitis puts it. As a perspective, evolution transcends the domain of biology, as first explicitly predicted by Ernst Haeckel (Richards 2008): Smocovitis brings up how an evolutionary perspective informs agriculture, medicine, and even robotics and AI. In a way, this dual existence is consonant with the original lexical root of evolution as an unfolding: just as specific individual characters unfold in the course of a play, so too, overall, does the script, based on the perspective in which it is embedded.

[1] We mention the Urdu words here because that was the language in which this thought first came to one of us while discussing this, and because '*kirdaar*' has nuances that 'character' or 'role' do not fully capture, including the actions of the individual playing that 'role', and also their intrinsic nature and motivation. Moreover, '*nazariya*' always transcends '*kirdaar*' in Urdu poetry.

The spirit of trying to understand the history of evolution permeates this very interesting volume and, again, we suspect it is evolution as perspective that makes this endeavour both so interesting and so important. Smocovitis alludes to the diversity of views that went into the making of the Modern Synthesis (MS) and the importance of understanding the various tellings of its history. One way of assessing the history of evolution leading up to and after the MS is through the dual existence of evolution as perspective and character. In a sense, Darwin (1859, 1868, 1871) took a nascent character-level study of evolution, embellished its character substantially, and, importantly, also gave evolution the attributes of a perspective. The MS, in many ways, was primarily a phase of increasing crystallisation, elaboration and sophistication of evolution as a character. This phase included, as Smocovitis points out, the early stages of the establishment of evolutionary biology as a formal discipline, with all its institutional, administrative and funding implications, a process that continued well beyond the announcement of the MS (Huxley 1942). Evolution as a perspective, as embodied in what we have termed in our chapter the Darwinian Core (DC) of evolutionary theory, however, was not much altered during the twentieth century from what it had been shortly after Darwin. Since the MS, empirical knowledge of many aspects of biology relevant to various sub-disciplines of evolution as character has grown substantially, almost exponentially. This growth of empirical knowledge is what has, in part, sparked off the many calls for an Extended Evolutionary Synthesis (EES) in recent decades (e.g. Pigliucci and Müller 2010; Laland et al. 2015). We suspect that a contributory factor to some of the EES-MS debates is a lack of explicit realization of these twin attributes of evolution as both character and perspective. We see the calls for an EES as lying primarily in the domain of evolution as character. Moreover, many of us who self-identify as working within the MS paradigm, see the MS primarily through the lens of evolution as perspective, encompassing within it multiple sub-disciplines operating in the domain of evolution as character. We wonder whether some of the disconnect between proponents of the EES and supporters of the MS arises from the fact that EES proponents often do not seem to clearly see that the calls for an EES pertain to evolution as a character, not as a perspective. Supporters of the MS, who see the MS as primarily reflecting evolution as a perspective, established in the DC, and relatively unchanged in its perspectival sense through the events leading up to the MS and beyond, in turn, wonder why EES proponents see their views as diametrically opposed to the MS when the latter is a perspective that has already accommodated multiple twists and turns of evolution as character, as varied evolutionary sub-disciplines have arisen and grown with the explosion of empirical information at multiple levels of biological organization (see chapters by Svensson, and by Smocovitis, in this volume). We think it is the perception that EES proponents are calling for substantial change in evolution as perspective that strikes many of us on the other side of the debate as being unwarranted. Perhaps a better delineation and distinction between these twin attributes of evolution as character and perspective would help people on both sides of the EES-MS debates to see many aspects of the debates afresh and appreciate that the differences between the EES and

MS viewpoints are not as great as they seem, because at the level of evolution as perspective, the differences blur considerably.

Smocovitis also seems to agree with our assertion that the way the MS is presented in many textbooks is simplistic and narrow and perpetuates a view of evolution that is open to critique. Here, too, we think that most textbooks, and consequently many syllabi for evolutionary biology courses, focus predominantly on evolution as character, without explicitly stating this limitation. This creates problems when students conflate the twin attributes of evolution as character and perspective and assume that the textbook version they encounter in an introductory course pertains to both. For example, Dobzhansky's much-quoted assertion that nothing in biology makes sense except in the light of evolution clearly pertains to evolution as perspective. His other much-quoted view, which has not stood the test of time well, that evolution can be equated to changes in allele frequency, is also often found in textbooks, but pertains to a sub-domain within evolution as character. Textbooks typically mention both, without a trace of irony about the inconsistency and incommensurability of these two statements, and with no acknowledgement that they pertain to different attributes of evolution (perspective and character), and that the former is broadly acceptable, whereas the latter is just plain wrong.

We should also add that these twin attributes of evolution as character and perspective make the field particularly vulnerable to being deployed for evil ends by the unscrupulous. It is precisely in its form as perspective that evolutionary thought can and has been deployed in the service of hateful socio-political ideologies; in its form as character, evolutionary thought is considerably more innocuous and relatively unsusceptible to depraved applications that negatively impact human society, compared to, say, virology as a character.

In conclusion, we point out that, on the one hand, the twin attributes of evolution as both perspective and character are intertwined in our metaphor, as reflected in the couplet below.

کھنڈہر ہے ہر نظریہ بس، بِنا کِردار کے

بِن نظریے کھوکھلا کِردارِ صد رنگین ہے

khandahar hai har nazariya bas, binaa kirdaar ke

bin nazariye khokhlaa kirdaar-e-sad-rangeen hai

(Viewpoints with no characters embodying them are empty ruins

Characters with no perspective to define them, hollow shells
– Nabraas Akbarabadi)

On the other hand, however, there is an asymmetry between them, both literal and metaphorical. Evolution as perspective constitutes a transcendent attribute, whereas

evolution as character is, by definition, a subsidiary, though also very necessary, attribute. A ruined building can be inhabited again and spring back to life, perhaps becoming even more resplendent than it was; a character without perspective, or with a limited perspective, is essentially an exile without a home (Urdu: خانہ بدوش/ *khaana-ba-dosh*, lit. carrying one's house on one's shoulders). Perhaps we should start viewing elements of the MS and EES as aspects of different characters enriching a common perspective that goes all the way back to the DC.

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Part VII

Inclusive Fitness: A Scientific Revolution

20

António M. M. Rodrigues and Andy Gardner

Abstract

Proponents of the "Extended Evolutionary Synthesis" argue that the current state of evolutionary biology departs from what was established in Modern Synthesis to such a degree that a new synthesis is needed. They present a "laundry list" of complaints concerning the core focus and assumptions of the Modern Synthesis and argue that the perspective of evolutionary biology must be shifted and these core assumptions relaxed in order to incorporate a plethora of new evolutionary factors. However, we contend that this revolution is already well underway, in the form of the inclusive-fitness research programme. We provide an overview of the inclusive-fitness revolution, charting its origins, explaining its core concepts and outlook, and describing the ways in which it has developed into a fully fledged and extraordinarily productive programme of scientific research. We then consider the apparently neglected processes and perspectives from an inclusive-fitness viewpoint. We conclude that progress in evolutionary theory is facilitated by focusing research attention on areas where there is a relatively poor fit between theoretical predictions and empirical observations, rather than complexifying models in pursuit of extra realism for its own sake.

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20.1 Introduction

In the last few years, several researchers have suggested that evolutionary biology requires a rethink in the form of an "Extended Evolutionary Synthesis" (EES; Pigliucci and Müller 2010, Laland et al. 2015, Müller 2017). Proponents of the EES argue that the current state of evolutionary biology departs from what was established in the Modern Synthesis (Huxley 1942) to such a degree that a new synthesis is needed. They present what has been described as a "laundry list" (Welch 2017) of complaints concerning the core focus and assumptions of the Modern Synthesis and argue that the perspective of evolutionary biology must be shifted, and these core assumptions relaxed in order to incorporate a plethora of new evolutionary factors (Table 8.1). In short, they call for revolution.

Our contention is that this revolution is already well underway, in the form of the inclusive fitness research programme (Hamilton 1964; Frank 1998; Bourke 2011; Gardner and West 2014). The concept of inclusive fitness arose in the 1960s and therefore postdates the Modern Synthesis of the 1940s. In its nearly 60 years of productive interplay between theoretical and empirical science, the inclusive fitness research programme has already incorporated much of the laundry list of supposedly neglected evolutionary factors. It is therefore surprising that proponents of the EES have variously mischaracterised (Wilson 2010), sidelined (Pigliucci and Müller 2010) or outright ignored (Müller 2007; Pigliucci 2007; Laland et al. 2014; Müller 2017) inclusive fitness theory, or even sought to characterise it as part of the edifice of the Modern Synthesis itself (Laland et al. 2015).

In this chapter, we provide an overview of the inclusive fitness revolution, charting its origins, explaining its core concepts and outlook, and describing the ways in which it has developed into a fully-fledged and extraordinarily productive programme of scientific research. We then consider each of the items of the EES "laundry list" in turn, showing how these apparently neglected processes and perspectives have been readily addressed within the framework of inclusive fitness. In doing so, we reveal a sharp contrast between the scientifically driven inclusive fitness revolution, on the one hand, and the philosophically driven EES movement, on the other, suggesting that progress in evolutionary theory has been facilitated by focusing research attention on areas where there is a relatively poor fit between theoretical predictions and empirical observations, rather than on needlessly complexifying models in the pursuit of extra realism for its own sake.

20.2 The Inclusive Fitness Revolution

The problem of adaptation is an ancient one but was expressed particularly clearly and urgently by William Paley in his landmark book *Natural Theology* (Paley 1802), which had a profound influence on Charles Darwin's thinking. Paley framed the problem of adaptation in terms of how to explain the apparent design of biological organisms, which he defined in terms of "contrivance and relation of parts"—that is, concerning the way in which each part of the organism appears intricately devised to carry out some purpose, and the way in which all parts of the organism appear devised to carry out the same purpose (Gardner 2009). Paley particularly highlighted the human eye, and its component parts, as a clear example of exquisite design, and Darwin likewise gave the eye special attention in *The Origin of Species* (Darwin 1859) when explicating how his theory of natural selection provided an explanation for this contrivance and relation of parts.

Darwin's theory of natural selection is based on the empirical observation that individual organisms vary, including in ways that are heritable. Accordingly, those variations in organismal characters that are associated with greater survival and fecundity will tend—by virtue of their bearers enjoying greater lifetime reproductive success—to accumulate in natural populations. Consequently—Darwin argued—subsequent generations of biological organisms are expected to appear increasingly well designed to maximise their reproductive success, as each and every one of their heritable characters becomes increasingly contrived as if for this purpose. Darwinism thereby provides a scientific theory for the process and purpose of organismal design (Gardner 2009).

However, Darwin (1859, pp. 192, 236–242) realised that there were clear examples of exquisite biological design that could not readily be explained by the above principle. In particular, he discussed the adaptations of sterile worker insects that are neither borne by members of the reproductive castes nor passed onto the sterile workers' offspring—for these individuals have no offspring. At first sight, it appeared that there was no possibility for natural selection to have moulded these adaptations. But, by drawing an analogy with the artificial selection practices of animal breeders, Darwin offered a solution to this problem. In situations where a desirable trait—such as delicious meat—cannot be assessed until after the individual has been killed, animal breeders understood that they may nevertheless have an opportunity to perform artificial selection indirectly, by prioritising the close kin of the killed individual for breeding, as relatives are expected to share heritable tendencies in common. In relation to the social insects, Darwin suggested that natural selection could also work indirectly, through the survival and fecundity of the workers' fertile family members, such that if the worker traits improved the reproductive success of their kin, then these too could be moulded through the action of natural selection.

Darwin's core logic of adaptation by natural selection was formalised by R. A. Fisher in *The Genetical Theory of Natural Selection* (Fisher 1930) as the "fundamental theorem" of natural selection. This reveals that the direct action of natural selection on the average of individual fitness is equal to the heritable variance in

fitness, which can never be negative, and hence the fundamental theorem provides a maximisation principle in which natural selection is always working to improve the individual's Darwinian fitness. Crucially, in setting out his assumptions, Fisher (1930, p. 27) explicitly excluded indirect effects whereby "an animal favours or impedes the survival or reproduction of its relatives", understanding that such kin effects need not result in the individual appearing to maximise its own Darwinian fitness, and perhaps feeling that there was no corresponding maximisation principle to be recovered under such circumstances (Gardner 2017). However, he did return to this indirect form of selection in his discussion of the evolution of anti-predator distastefulness in gregarious insect larvae (Fisher 1930, p. 158–159), suggesting that this is driven by a selective advantage enjoyed by siblings; this passage appears to represent the first quantitative use of the kin selection coefficient of relatedness (Best et al. 2018).

More than a century after Darwin had set out the basic logic, indirect selection finally received a formal, comprehensive, population genetics treatment through W. D. Hamilton's work on the evolution of altruistic behaviour (Hamilton 1963, 1964), and was given the name "kin selection" (Maynard Smith 1964). Hamilton showed how direct and indirect selection aggregate as a simple sum to give the overall action of natural selection, such that individual traits are expected to be moulded under their combined action, and he clarified that the impact that an individual's traits have on the fitness of her relatives translate into the action of indirect selection in proportion to their degree of relatedness. These insights are encapsulated in "Hamilton's rule" (Hamilton 1963, 1964, 1970; Charnov 1977), which in its simplest form states that the condition for a trait to be favoured by natural selection is that the sum of the fitness impact upon self ($-c$) and the product of the fitness impact upon a social partner (b) and the relatedness to this social partner (r), exceeds zero (i.e. $-c + br > 0$). As Hamilton's focus was mainly upon altruism, the impact of an individual upon her own fitness is often described in terms of "cost" and her impact on her social partner's fitness is often termed a "benefit", but more generally Hamilton's rule applies just as readily to mutually beneficial, selfish, or even spiteful behaviours (Hamilton 1964, 1970; West et al. 2007).

Kin selection can be conceptualised in two different ways (Fig. 20.1). The "personal fitness" (or "neighbour-modulated fitness") approach fastens attention upon a focal recipient and describes how her personal fitness is modulated by her own traits and also those borne by her social partners (Hamilton 1964; Frank 1998). Under this view, the cost term ($-c$) describes the impact that the individual has upon her own reproductive success and the benefit term (b) describes the impact the individual's social partner has upon her reproductive success. The idea here is that natural selection favours those heritable traits that are associated with higher fitness, and this association can be positive even if the trait directly reduces the individual's fitness (described by $-c$) so long as carriers of the heritable trait tend to have social partners who also bear the trait (described by r), and these social partners provide a sufficiently large benefit to her (described by b). Accordingly, under the personal fitness view of kin selection, the coefficient of relatedness functions as a statistical constraint that exists between the heritable traits of social partners, and which acts as

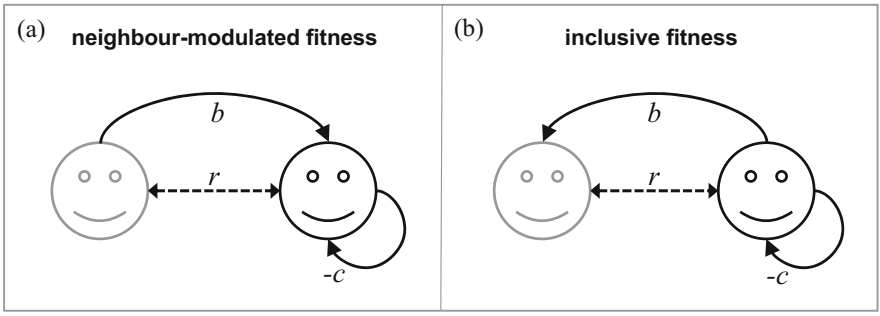

Fig. 20.1 Alternative formulations of kin selection. (**a**) The personal fitness approach fastens the attention on a focal recipient (black), her impact ($-c$) upon her own fitness, and the impact (b) of her social partner (grey) upon her own fitness, with the coefficient of relatedness (r) representing a statistical constraint that exists between the heritable traits of social partners. (**b**) The inclusive fitness approach fastens the attention on a focal actor (black), her impact ($-c$) upon her own fitness, and her impact (b) upon the fitness of her social partner (grey), with the coefficient of relatedness (r) representing a measure of the value that the actor places upon her social partner

a confounding effect such that the correlation between trait and fitness that drives natural selection does not reflect straightforward causation.

Alternatively, the "inclusive fitness" approach to kin selection fastens attention upon a focal actor and describes how her trait modulates her own fitness and the fitness of her social partners (Hamilton 1964; Frank 1998). Under this view, the cost term ($-c$) describes the impact that the individual has upon her own reproductive success, the benefit term (b) describes the impact the individual has on her social partner's reproductive success, and relatedness (r) enters into the calculus not as a constraint but as a measure of the value that the actor places upon her social partner. This reframing of kin selection recovers a direct causal pathway between trait and fitness, by changing the definition of fitness itself. The individual's inclusive fitness is defined as her personal fitness, minus all the effects of her social partners, and plus all the effects she has on her social partners, with each of the latter effects being weighted by her genetic relatedness to these recipients.

In developing the concept of inclusive fitness, Hamilton recovered a maximisation principle for natural selection that applies irrespective of whether kin selection is present or absent. Natural selection can be framed both from personal fitness and inclusive fitness perspectives: the overall action of natural selection with respect to any heritable trait can be viewed either in terms of that trait's correlation with personal fitness or with inclusive fitness—both approaches give exactly the same result. But personal fitness does not yield a maximisation principle, simply because the individual does not have full control over her own reproductive success, such that if she were viewed as striving to maximise her personal fitness then her only means of doing so would be by maximising her direct fitness, and this would not account for the confounding effects of kin interactions. In contrast, the individual does—by definition—have full control over her inclusive fitness, and hence can be validly viewed as striving to maximise this quantity (Grafen 2006; West and Gardner

2013). That is, Hamilton not only revolutionised the way we think about the process of adaptation—by formalising the logic of indirect selection and placing it on the same footing as direct selection—but also revolutionised the way we think about the purpose of adaptation—by revealing that organisms are not expected to appear designed to maximise their personal fitness but rather they are expected to appear designed to maximise their inclusive fitness.

Hamilton's work on kin selection was initially formalised by means of a simple population genetics model that made various simplifying assumptions about gene action, including additivity and weak selection. However, he subsequently made use of George Price's (1970) eponymous theorem to provide a much more general derivation (Hamilton 1970), and this approach has subsequently given rise to what is often termed the "regression" approach to Hamilton's rule, which is understood to apply as generally as the theory of natural selection itself (Orlove and Wood 1978; Queller 1992; Frank 1998; Gardner et al. 2011). Accordingly, in its general form, the theory of kin selection allows for heritable variants of large phenotypic effect, with concomitant strong and/or frequency-dependent selection, although in particular applications simplifying assumptions are often employed for the sake of analytical tractability. Price's theorem is celebrated for its substrate neutrality, such that it applies to genetical and non-genetical evolutionary change alike (Frank 1995; Price 1995). But less appreciated is that Hamilton's rule, too, can thereby be framed in general terms that transcend genetics. For example, the above account of Hamilton's work on kin selection has been framed without explicit reference to genes, and indeed Price's theorem allows for the logic of kin selection to be formalised under the assumption of blending inheritance, yielding exactly the same form of Hamilton's rule and with individuals placing the same value upon the reproductive success of their relatives (Gardner 2011).

A crucial step in the historical development of inclusive fitness was the way in which the re-derivation of Hamilton's rule from Price's theorem clarified the relationship between kin selection and group selection. During the first half of the twentieth century, evolutionary geneticists understood that natural selection was in principle a multi-level process, with Sewall Wright (1931) placing much emphasis on inter-demic selection as a facilitator of adaptation under his "shifting balance" view of evolution. Partly on account of Wright's work, much of the study of social evolution during the middle years of the century was in thrall to the idea that natural selection drives the evolution of adaptations that function to maximise the overall fitness of the population or species, with many apparently altruistic behaviours being explained away on the basis of confused and confusing "for the good of the species" thinking (Wynne-Edwards 1962; Lorenz 1963). Inclusive fitness theory provided an alternative explanatory framework for such behaviours and in a way that truly reflected how natural selection operates. Yet, Price (1972) showed that his theorem also provides a rigorous account of how selection operating at the between-group level can contribute to the overall action of natural selection, albeit alongside selection operating at the within-group level, which will often be stronger and sometimes acting in the opposite direction. That Price's equation underpins the theories of both kin selection and group selection has allowed the conceptual links

between the two theories to be clarified, and 50 years later most social evolution researchers understand that the two theories are not in opposition and simply provide alternative ways of describing the very same process of natural selection (Hamilton 1975; Leigh 2010; Frank 2013; Gardner 2020).

Just as the original motivation for the theory of inclusive fitness was strongly empirical, and stemmed from an apparent mismatch between the predictions of classical Darwinism versus the observations of puzzling organismal adaptation, so too have the many successful applications of inclusive fitness theory been driven by a tight interplay of theoretical and empirical considerations. A striking example is the study of sex allocation, wherein the classic prediction that natural selection favours an even investment of parental resources into offspring of both sexes—on account of individuals of the rarer sex tending to leave more descendants than individuals of the more common sex (Fisher 1930)—is flagrantly disregarded by many chronically inbreeding arthropods that exhibit highly female-biased sex ratios, and this mismatch stimulated the theory of "local mate competition" (Hamilton 1967), concerning the inclusive fitness consequences of sex allocation when mating groups are made up of close relatives. This close interaction of theoretical and empirical research has continued unabated, with the result that the theory of sex allocation enjoys excellent empirical support, and those areas within which the fit between theory and data is less strong quickly receive research attention such that the underlying biology rapidly becomes illuminated (West 2009).

The concept of inclusive fitness was developed to recover a fitness measure that the individual organism could be viewed as striving to maximise, yet it may also be usefully applied to the level of single genes to illuminate their evolutionary interests (Gardner and Welch 2011). For the most part, fair meiosis aligns the inclusive fitness interests of genes inhabiting the same body, such that the whole organism can be viewed as a unified inclusive fitness maximiser (Leigh 1977). However, genes residing in the same body may in some circumstances have divergent inclusive fitness interests, resulting in intragenomic conflict (Burt and Trivers 2006; Gardner and Úbeda 2017). One way in which this may happen is if different genes follow different rules of inheritance—such as nuclear genes that are inherited biparentally versus cytoplasmic genes that are inherited uniparentally—which may, for example, lead to disagreements over sex allocation on account of these genes disagreeing as to the valuation of sons versus daughters (Werren et al. 1988). Alternatively, even if inheritance is fair and Mendelian, genes may come into conflict with each other owing to their bearer engaging in social interactions that modulate their inclusive fitness in different ways. For example, if an individual engages in social interaction with her maternal half-siblings, then whereas her maternal-origin genes would maximise their inclusive fitness by inclining her to behave relatively altruistically her paternal-origin genes would maximise their inclusive fitness by inclining her to behave relatively selfishly, on account of the former being more related than the latter to these social partners (Haig 2002). The resulting parent-of-origin conflict is predicted to result in the self-imposed silencing of one of the genes, i.e., "genomic imprinting", a phenomenon that is difficult to account for in terms of individual-level advantage (Haig 2002).

The kinship theory of genomic imprinting does not only illuminate patterns of gene-level adaptation but also patterns of organism-level maladaptation. In particular, the effective haploidy of imprinted loci renders the individual especially vulnerable to mutations that would otherwise be recessive under diploidy, and the tension that exists between genes in conflict means that when mutational or epimutational perturbations occur the phenotypic effects can be drastic. Accordingly, genomic imprinting is associated with an array of debilitating human disorders—particularly concerning fertility, pregnancy, and infanthood, wherein conflicts over resource allocation are expected to be rife—with the associated clinical pathologies having patterns that are predictable in light of the inclusive fitness interests of maternal-origin versus paternal-origin genes (Wilkins and Úbeda 2011). For example: Prader–Willi syndrome is associated with a deletion of a gene cluster in chromosome q15 being inherited from the individual's father, and involves a clinical phenotype in children that makes sense in terms of reducing the amount of resource to be extracted from the mother, such as lack of appetite; whereas Angelman syndrome results from the very same deletion being inherited from the individual's mother, and involves a clinical phenotype in children that makes sense in terms of increasing the amount of resources to be extracted from the mother, such as frequent waking at night to feed (Haig and Wharton 2003; Haig 2014).

Conversely, shifting up a level of biological organisation from the individual organism, inclusive fitness also illuminates the major transitions in individuality (Maynard Smith and Szathmáry 1995; Bourke 2011). These transitions can be viewed as occurring when what was previously a group of separate individuals evolves to such a high degree of coordination and complexity—i.e. exhibiting the contrivance and relation of parts that provides the hallmark of design—that they are more usefully regarded as higher-level individuals in their own right. Examples include repeated evolutionary transitions from unicellular to multicellular life and from solitary individuals to eusocial colonies. Mirroring the principle that it is divergent inclusive fitness interests of genes that foment intragenomic conflicts which undermine the adaptive integrity of the organism, major transitions in individuality are best understood in terms of the inclusive fitness interests of social partners coming into such close alignment that conflicts between them are, at least in many respects, extinguished (Gardner and Grafen 2009). Accordingly, complex multicellular life has only arisen in lineages where a single-cell bottleneck ensures clonal relatedness between constituent cells (Fisher et al. 2013), and eusociality has only arisen in lineages where colonies are headed by a single, singly mated female, which ensures that the inclusive fitness value of rearing a sibling is equal to that of rearing one's own offspring (Hughes et al. 2008; Boomsma 2009). Thus, the theory of inclusive fitness explains the broadest scale evolutionary patterns through the history of life.

20.3 The Laundry List in the Light of Inclusive Fitness

Returning to the "laundry list" of neglected evolutionary processes that have been highlighted by proponents of the EES as necessitating a revolution in evolutionary biology, we now provide an assessment of these claims from an inclusive fitness perspective. The details of the list vary between tellings, as different researchers naturally have different emphases and different complaints to raise as to the ways in which they perceive that evolutionary biology has been pursued. Accordingly, we focus on the list given by Kevin Lala (formerly Laland) and colleagues in *The extended evolutionary synthesis: its structure, assumptions and predictions* (Laland et al. 2015), as the authorship of this paper includes many of the core contributors to the EES movement and thereby promises to provide a definitive account (Table 20.1).

A core complaint made by proponents of the EES is that conventional evolutionary biology focuses attention upon an arrow of causation that flows from environment to organism and does not give due attention to causal pathways running in the reverse direction, such that the possibility for "reciprocal causation"—whereby organisms both shape and are shaped by their environments—is neglected (Laland

Table 20.1 Extended evolutionary synthesis core assumptions and views from inclusive fitness theory

Extended evolutionary synthesis core assumptions (Laland et al. 2015)	The view from inclusive fitness theory
"Reciprocal causation (organisms shape, and are shaped by, selective and developmental environments)"	A core motivation for the theory of inclusive fitness is that organisms shape, and are shaped by, their selective and developmental environments—i.e., social interaction.
"Organism-centred perspective"	A core motivation for the concept of inclusive fitness is to provide an organism-centred perspective on social adaptation.
"Variable rates of change"	Inclusive fitness theory allows for mutational steps of arbitrarily large effect, and its whole-organism outlook reaffirms that the "gradualism" versus "saltationism" controversy concerns the process of adaptation rather than the process of evolution.
"Inclusive inheritance"	The logic of inclusive fitness holds even under non-genetic systems of inheritance, such as blending, and it explains the evolution of epigenetic effects.
"Non-random phenotypic variation"	Inclusive fitness theory predicts patterns of mutational and epimutational maladaptation, including by showing how these align with fitness interests of conflicting agents.
"Macro-evolution"	Inclusive fitness theory explains major transitions in individuality, which represent the most fundamental events in the unfolding of the history of life.

et al. 2015, Müller 2017; Table 20.1). This comes as a surprise to those working within the inclusive fitness research programme, as the basic motivation for this topic of study is to understand the consequences of an evolving population being part of its own selective environment.

A simple but striking illustration is provided by the evolution of dispersal. In seeking to understand why an individual would go to the effort to relocate from one place to another, often at considerable cost, a traditional approach has been to imagine that the prospects for the individuals are—at least on average—better at their destination than at their point of origin. However, the kin-selection analysis of Hamilton and May (1977) showed that even in stable, saturated environments, whereby the reproductive prospects for an individual at home are no better nor worse than anywhere else in the population, a substantial proportion of individuals are nevertheless expected to disperse because, were they to instead remain in their natal patch and succeed in achieving reproductive success, this would come at the expense of their kin who are competing for the same reproductive resources. Indeed, in their simple model involving a single, asexually reproducing breeder in each patch, Hamilton and May showed that even if dispersal is associated with almost-certain death nearly one-half of all individuals are expected to disperse, despite this reducing their reproductive opportunities to essentially zero, in order to improve the prospects of their surviving family members. More generally, for sublethal costs of dispersal, Hamilton and May's analysis captures the reciprocal causality of kin competition modulating selection for altruistic dispersal and altruistic dispersal modulating the intensity of kin competition, such that as the overall rate of dispersal increases then resource competitors become less related to each other, which reduces the kin-selected benefit of dispersal, and hence the overall rate of dispersal always stabilises at an intermediate level at which these forces exactly balance out (Frank 1986, 2013).

Beyond simple models of single organismal traits, the theory of inclusive fitness also illuminates complex, multi-trait, long-term, open-ended evolution. For example, the sperm-storage capabilities of the ancestors of the social hymenoptera permitted an iteroparous lifecycle to be compatible with a strictly monogamous mating system, such that an individual's relatedness to her maternal siblings is exactly equal to that of her own offspring, and hence her inclusive fitness is maximised by giving up her own reproductive success in order to improve the reproductive success of her mother provided there are any efficiency benefits of such cooperative breeding (Hamilton 1964; Boomsma 2007, 2009, 2013). The ensuing evolution of a non-reproductive helper caste with concomitant division of labour and efficiencies of scale have given rise to elaborate insect societies, in which the selection pressures exerted upon individuals and their traits are qualitatively different from those experienced by their non-social ancestors, and which have been associated with the further elaboration of phenotypes, including the evolution of multiple non-reproductive castes and the conquest of previously inaccessible ecological spaces (Boomsma and Gawne 2018).

A further core complaint made by proponents of the EES is that conventional evolutionary biology is focused on genes and has lost sight of the organism, including as an active participant in its own evolutionary fate (Laland et al. 2015,

Müller 2017; Table 20.1). Again, this claim is surprising to those working within the inclusive fitness research programme because, as discussed above, the recovery of a whole-organism adaptive rationale in the context of social interactions among relatives was precisely what motivated the development of the concept of inclusive fitness in the first place. Hamilton (1964) initially framed his analysis of the action of kin selection in terms of the individual's personal (or "neighbour-modulated") fitness, whereby indirect fitness effects are described from a recipient-centred perspective, but he went on to reframe the theory in terms of inclusive fitness, in which attention is fastened on a focal actor who wields full control over the maximand that captures her evolutionary interests. Inclusive fitness is firmly rooted into the whole-organism perspective of Paley (1802) and Darwin (1859), who both sought to explain the same contrivance and relation of parts manifested by organisms, though in rather different ways. Indeed, we find it surprising that proponents of the EES would seek to champion the whole-organism view whilst simultaneously arguing against the "pre-eminence" of natural selection (Laland et al. 2015), given that it is from the logic of natural selection that the concept of whole-organism fitness is derived and hence it is difficult to see how, in a hypothetical scenario in which natural selection is overpowered by non-Darwinian forces, biological organisms manifesting a unity of purpose could arise at all.

Insofar as the concept of inclusive fitness has also been applied at the intra-individual level, to understand the interests of single genes, the motivation for this has also largely stemmed from a whole-organism perspective, whereby the gene's-eye view has been taken in order to illuminate instances of organismal maladaptation (Burt and Trivers 2006; Gardner and Úbeda 2017). Inclusive-fitness interests are not perfectly aligned within genomes, and even genes obeying the rules of autosomal, mendelian inheritance can come into conflict with each other, as illustrated by the kinship theory of genomic imprinting whereby an individual's maternal-origin and paternal-origin genes disagree as to how their carrier should behave towards her matrilineal versus patrilineal relatives, resulting in parent-of-origin specific gene expression and a concomitant range of debilitating human disorders (Haig 2002; Wilkins and Úbeda 2011). Yet the detection of such intragenomic conflicts also provides evidence that organisms are, on the whole, close to their inclusive-fitness optima, because it is only when the genes' carrier is close to her optimum that they are actually favoured to pull her phenotype in different directions (West and Gardner 2013).

Proponents of the EES also highlight the importance of "variable rates of change", which they contrast with the assumption of "gradualism"—defining this in terms of the phenotype evolving via multiple, small steps—that they say characterises current evolutionary biology thinking (Table 20.1). This, too, is surprising to those working within the inclusive fitness research programme. The inclusive-fitness framing of natural selection, and its encapsulation in the form of Hamilton's rule, emerges directly from Price's (1970) theorem (Hamilton 1970, Queller 1992, Frank 1998, Gardner et al. 2011; Box 20.1) and thereby enjoys a vast scope of application. There is no requirement that mutations must always be of small effect, or that mutations of larger effect cannot be favoured by natural selection

(Gardner et al. 2011). Of course, particular analyses often do make use of simplifying model assumptions and streamlined methodologies for the purpose of tractability, simplicity and transparency, and on this basis the assumption of small allelic effects is sometimes employed in order to bring the powerful tools of differential calculus to bear on a particular problem (Taylor and Frank 1996; Gardner et al. 2011). Insofar as these simplifications enable the derivation of clear and testable predictions, then they are scientifically valid, and it is good practice to combine these approaches with methodologies that enable the relaxation of such assumptions albeit at the price of reduced transparency—such as individual-based simulations—in order to check that the results are robust and not artefacts of the simplifying assumptions (e.g., Rodrigues and Gardner 2012).

Box 20.1 Price's Equation, Kin Selection, Inclusive Fitness and Multi-Level Selection

Price's (1970, 1972) equation provides a general description of evolutionary change. The following exposition is based on that of Gardner (2020). Price's equation states that the change in the population average value of any trait of interest z is given by:

$$\Delta \mathrm{E}(z) = \mathrm{cov}(w, z) + \mathrm{E}(w\Delta z) \tag{20.1}$$

where w is relative fitness, Δz describes the difference between a parent's and their offspring's trait values, and cov denotes a covariance and E an expectation—with both of these descriptive statistics taken over all individuals in the population.

The covariance term on the right-hand side of Eq. (20.1) represents the part of evolutionary change that is due to selection, i.e. arising from statistical associations between trait and fitness, and the expectation term on the right-hand side of Eq. (20.1) represents the part of evolutionary change that is due to transmission, i.e. arising from imperfect heredity of trait values between parent and offspring. Accordingly, Price's equation isolates and formally defines the separate selection—$\Delta_{\mathrm{S}}\mathrm{E}(z) = \mathrm{cov}(w,z)$—and transmission—$\Delta_{\mathrm{T}}\mathrm{E}(z) = \mathrm{E}(w\Delta z)$—components of evolutionary change.

The action of selection can be expressed in terms of least-squares linear regression, as $\Delta_{\mathrm{S}}\mathrm{E}(z) = \beta(w,z)\mathrm{var}(z)$, where $\beta(w,z) = \mathrm{cov}(w,z)/\mathrm{var}(z)$ is the least-squares linear regression of fitness against trait value and var(z) measures the variance in trait value. This highlights that for selection to act in relation to any trait there must be variation in that trait and there must be a nonzero slope to the least-squares linear regression line fitted through the fitness and trait values. So long as there is variation present in the trait (i.e. $\mathrm{var}(z) > 0$) then the condition for selection to favour an increase in the trait is that its marginal fitness is positive (i.e. $\beta(w, z) > 0$).

(continued)

Box 20.1 (continued)

Kin selection emerges directly from Price's equation simply by adding the trait values z' of social partners as an additional predictor of fitness. Marginal fitness is then given by $\beta(w, z) = \beta(w, z|z') + \beta(w, z'|z)\beta(z', z)$, where: $\beta(w, z|z') = -c$ is the effect of the focal individual's trait on her own fitness, holding the social partner's trait constant; $\beta(w, z'|z) = b$ is the effect of the social partner's trait on the focal individual's fitness, holding the focal individual's trait constant; and $\beta(z', z) = r$ is the coefficient of relatedness, describing the statistical association between the traits of social partners. The condition for selection to favour an increase in average trait value is therefore given by $\beta(w, g) > 0$, or

$$-c + br > 0 \tag{20.2}$$

i.e., Hamilton's rule of kin selection (Hamilton 1964, 1970; Queller 1992). Note that this derivation of Hamilton's rule has focused on the individual's personal fitness and considered how it is modulated by the traits of her social partners (including herself), and therefore represents the "personal fitness" approach to kin selection. The alternative "inclusive fitness" approach to kin selection is obtained by rewriting marginal fitness as $\beta(w,z|z') + \beta(w',z|z')\beta(z', z)$, where w' is the relative fitness of the actor's social partner. The marginal fitness $\beta(w,z|z') + \beta(w',z|z')\beta(z',z)$ thereby describes the inclusive fitness effect of the trait.

An alternative approach to social evolution is the "multi-level selection" approach, which emerges by partitioning Price's equation in a different way:

$$\Delta_S E(z) = \mathrm{cov}_{i\in I}\left(E_{j\in Ji}\left(w_j\right), E_{j\in Ji}\left(z_j\right)\right) + E_{i\in I}\left(\mathrm{cov}_{j\in Ji}\left(w_j,\ z_j\right)\right) \tag{20.3}$$

where every group in the population has been assigned a unique index $i \in I$ and where every individual in group-i has been assigned a unique index $j \in Ji$. The first term on the right-hand side of Eq. (20.3) represents the covariance of group-average fitness and group-average trait value across all social groups in the population and thereby describes the action of between-group selection, and the second term on the right-hand side of Eq. (20.3) represents the average within-group covariance of individual fitness and individual trait value and thereby describes the action of within-group selection.

It is important to clarify that such considerations of phenotypic change proceeding at variable rates, in fits and starts and in jumps of small and large size, are in fact tangential to the real issues underpinning the "gradualism" versus "saltationism" controversy. This debate concerns not phenotypic evolution as such, but rather the origin of adaptation—i.e., the appearance of design, as manifest in contrivance and relation of parts. The "saltationism" stance is that the de novo appearance of design can occur in a single step, e.g., the human eye arising fully formed as the result of a

single mutation, whereas the "gradualism" view is that such adaptations arise in multiple steps. The saltationism stance can be rejected purely on grounds of probability: although in principle a single mutation could result in the de novo appearance of an organ as complex as the human eye, this is highly improbable, and the cumulative action of natural selection, working over multiple generations, retaining those variants that lead to improvement and disposing of those that do not, represents a much more efficient route to biological design. Inclusive fitness theory, which has emerged from the wider study of whole-organism complex adaptation, reaffirms that Darwinism—rather than saltationism—explains the design of organisms (Gardner 2013).

The proponents of the EES also object to the conventional focus on genetic inheritance, which they claim is too narrow (Laland et al. 2015; Table 20.1). They counter with the need for a broader understanding of inheritance, deemed "inclusive inheritance", that shifts the focus from genes to other forms of non-genetic inheritance, which may take place at other levels of biological organisation. This statement, again, is surprising when viewed from an inclusive fitness perspective. First, the fundamental logic of inclusive fitness theory assumes neither genetic nor particulate inheritance. As described above, Price's equation provides a general statement about the action of selection, irrespective of the form of inheritance. Further, it shows that inclusive fitness gives an exact description of the action of natural selection even under the assumption of blending inheritance (Gardner 2011). Second, far from ignoring them, inclusive fitness theory actually predicts and explains patterns of epigenetic effects. A notable example of this explanatory power of inclusive fitness theory is the kinship theory of genomic imprinting (Haig 2000, 2002), which predicts the evolution of epigenetic parent-of-origin effects modulating the phenotypes of offspring.

The nature of phenotypic variation has also been under intense scrutiny within the EES literature (Table 20.1). Proponents of the EES programme have rejected what they describe as the classic view of evolutionary biology that there is "no relationship between the direction in which mutations occur—and hence the supply of phenotypic variants—and the direction that would lead to enhanced fitness" (Laland et al. 2015), and instead propose that developmental bias means that mutations are more likely to occur in some directions than in others. This overlooks the extent to which inclusive fitness theory not only accommodates but also explains and predicts patterns of mutational bias, precisely by considering the fitness consequences of different phenotypes. As discussed above, the kinship theory of genomic imprinting predicts parent-of-origin specific gene expression as an evolutionary consequence of differences in the inclusive fitness interests of maternal-origin versus paternal-origin genes residing in the same genome with respect to certain social phenotypes (Haig 2002; Gardner and Úbeda 2017). Accordingly, inclusive fitness considerations imply that mutational effects will be particularly strong with respect to these social phenotypes. Moreover, as the direction of genomic imprinting—e.g., maternal expression and paternal silencing, or vice versa—is predicted to depend upon the direction of the intragenomic conflict between maternal-origin versus paternal-origin genes, so too are the phenotypic effects of different classes of genetic and epigenetic

mutations predicted to depend on the inclusive fitness interests of these conflicting genes (Haig and Wharton 2003; Haig 2014). Although originally developed to explain the adaptive "wildtype" design of organisms, the theory of inclusive fitness also provides a predictive framework for understanding patterns in relation to mutant phenotypes (Gardner and Úbeda 2017).

Finally, proponents of the EES have identified macroevolution as a topic for which they feel classic evolutionary theory provides an inadequate account, such that a revolution in understanding is required in order to explain phylogenetic patterns (Table 20.1). In particular, they suggest that the accumulated action of the canonical microevolutionary processes of natural selection, mutation, random drift and gene flow operating within evolving lineages cannot explain patterns at this higher level (Laland et al. 2015). Once again, such claims are puzzling from an inclusive-fitness perspective, as understanding of the major transitions in evolution—representing the broadest-scale patterning that exists across all the domains of life—features the principles of the theory of inclusive fitness at its core (Maynard Smith and Szathmáry 1995; Bourke 2011). As discussed above, major transitions in individuality from prokaryotic to eukaryotic cell, from unicell to multicellular organism, and from solitary individual to eusocial colony occur when the inclusive-fitness interests of previously free-living individuals are reconciled and aligned to such a degree that the social group is elevated to a new level of individuality in its own right (Gardner and Grafen 2009). This body of theory explains why, for example, obligate eusociality has only ever evolved in the context of strict female monogamy, and hence why it has been restricted to those rare lineages in which strict female monogamy is facilitated by pre-existing features such as sperm storage (Boomsma 2013), with decisive implications for macroevolutionary patterns concerning ecological success, the evolution of complexity and rates of speciation.

20.4 Conclusions

Proponents of the EES programme have highlighted a number of complaints concerning various aspects of the evolutionary process that they feel have been neglected by evolutionary biology to such an extent that the entire foundations of the discipline are in need of a rethink. Here we have considered their complaints from the vantage point of inclusive-fitness theory, showing that some of these apparently neglected factors (i.e. reciprocal causation and the role of the organism) were key motivators for the development of the concept of inclusive fitness and that the others (i.e. variable rates of change, inclusive inheritance, non-random phenotypic variation and macroevolution) are all accommodated—and, indeed, illuminated—by the theory of inclusive fitness. We are not suggesting that there is no need for further work on these issues, but we are surprised that the proponents of the EES have overlooked the accomplishments of the inclusive fitness programme, and that they have failed to acknowledge the successes of inclusive fitness theory in relation to addressing the shortfalls of the Modern Synthesis. More generally, consideration of the origin and subsequent developments of the inclusive fitness programme has

yielded insights into what makes for a successful revolution within evolutionary biology. In contrast to the EES approach, which has been to quibble with model assumptions and demand that researchers should incorporate more complexity for the sake of "realism", the theory of inclusive fitness has instead focused on identifying mismatches between theoretical predictions and empirical observations, as these mismatches draw our attention to the areas where our understanding is less complete and enable us to work productively towards putting that right.

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Phenotypes, Organisms, and Individuals: A Commentary on Rodrigues and Gardner

21

Thomas E. Dickins

Abstract

Rodrigues and Gardner have focused upon a key set of claims from Laland and colleagues, who advocate for an extended evolutionary synthesis (Laland et al. Proc R Soc B Biol Sci 282:20151019, 2015). Laland et al. focus their paper on a list of contrasting assumptions from the Modern Synthesis and an extended evolutionary synthesis (EES). It is this list that Rodrigues and Gardner take issue with, claiming that inclusive fitness theory has a response to all the purported inadequacies the extension seeks to solve. Their very straightforward question is why has inclusive fitness theory been ignored by those seeking extension?

Keywords

Phenotypes · Organisms · Individuals · Inclusive fitness theory

Rodrigues and Gardner have focused upon a key set of claims from Laland and colleagues, who advocate for an extended evolutionary synthesis (Laland et al. 2015). [1] Laland et al. focus their paper on a list of contrasting assumptions from the Modern Synthesis and an extended evolutionary synthesis (EES). It is this list that Rodrigues and Gardner take issue with, claiming that inclusive fitness theory has

[1] This paper has been cited 973 times and is therefore a good candidate for a key EES position paper (Google Scholar search conducted on 7 March 2022).

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a response to all the purported inadequacies the extension seeks to solve. Their very straightforward question is why has inclusive fitness theory been ignored by those seeking extension?

A superficial trawl of the EES literature shows instances where inclusive fitness is invoked, usually in discussion about how a trait might contribute to fitness maximization (Laland 2004; Helanterä 2011; Danchin et al. 2011; Laland et al. 2016). More interestingly Otsuka has adopted a Lakatosian perspective and discussed how inclusive fitness theory should be seen as a modification to the outer belt of Modern Synthetic theory to deal with issues such as altruism—in doing so he calls for similar modifications as a part of the effort to extend (Otsuka 2019). This is a somewhat deflationary take on the EES, but still Otsuka fails to note the detail of the work under inclusive fitness theory that Rodrigues and Gardner have highlighted.

One possible reason for not properly addressing inclusive fitness theory might relate to Dawkins' discussion about individuality developed as a part of his extended phenotype argument (Dawkins 1978, 2008). Dawkins was concerned to make clear that modelling individuals as fitness maximizers can run theorists into difficulty and he pushed hard on the interpretation, undoubtedly shared with Hamilton, that inclusive fitness was the outcome of actions taken by an individual, not a property of an individual. Moreover, he was keen to reinforce the view of strategic genes (or replicators) copying themselves across generations.

Here is an example from Dawkins of the kind of error he was concerned with:

> It is often pointed out that some coefficients of relationship are exact while others are probabilistic. For instance the coefficient between brothers is ½ but this "is an average figure: by the luck of the meiotic draw, it is possible for particular pairs of brothers to share more or fewer genes than this. The relatedness between parent and child is always exactly ½." … Gibson … correctly stated this point, but then went on to draw an incorrect inference. She supposed that an adult might invest in a son rather than in a full sibling because nature might prefer "a sure thing (relatedness = 0.5 as in the case of the son) to gambling (average relatedness = 0.5 as in the case of the siblings)." But only an individual could see the son as a 'sure thing'. From the point of view of a single gene determining parental or brotherly behaviour, the son is no more a sure thing than the brother: both are gambles with 50% odds. (Dawkins 1978, p. 64)

For Dawkins, this results in the error of seeing the adaptation in terms of an individual benefit. Much of Dawkins' effort in this work was to remove the individual from evolutionary biology. His view was that individuals are not replicated, they are idiosyncratic, and therefore not the appropriate unit to measure evolutionary change with. Instead, replicators needed to have the properties of fecundity, fidelity, and longevity. This fastidious approach to terminology and units of selection is not necessarily in conflict with inclusive fitness theory, and Rodrigues and Gardner clearly lay out the genes' eye view at play, demonstrating its theoretical potency with discussion of the major transitions and genomic conflict among other areas.

The focus upon major transitions in some EES quarters causes them to contrast a traditional view of individuals "defined as functionally integrated and spatiotemporally delimited wholes" (Pigliucci and Finkelman 2014, p. 515) with the view of

individuality that Rodrigues and Gardner defend. For Rodrigues and Gardner "major transitions in individuality are best understood in terms of the inclusive fitness interests of social partners coming into such close alignment that conflicts between them are, at least in many respects, extinguished" (this volume, p.x). Pigliucci and Finkelman point to Mayr's view that selection operates on (traditional) individuals, and that gene frequency changes are a statistical artefact of this process. Under Mayr's view there is one level of selection. Equally, under Dawkins' view there is only one level of selection, which is the gene. So, Pigliucci and Finkelman claim, what the Modern Synthesis gave was a monistic view of selection, with some discussion about which level it should be (individual or gene). The interpretation of major transitions that Rodrigues and Gardner adopt would most likely be seen as a part of this monistic tradition. Individuality is the outcome of aligned genetic interest via cooperation.

Pigliucci and Finkelman (2014) contrast the monistic view with the EES perspective that places selection at different levels, leading to potential fitness conflicts between component levels and the individual whole to which they contribute. [2] This they describe as a form of pluralism. Mechanisms that decouple fitness outcomes across the component levels but allow individuality include "the division of labor, cooperation between different kinds of individuals, and policing activity among them" (p. 514). Given this I think the likely reason that the EES has ignored inclusive fitness theory is that it was not built on the foundation of multilevel selection theory (Okasha 2006). And yet, as Gardner has made abundantly clear elsewhere, no prediction from multilevel selection theory has been made that cannot be redrawn in Hamiltonian terms (Gardner et al. 2011).

Pluralism creates additional structures to do scientific work but, given the mathematical equivalence noted in the preceding paragraph, one must wonder why this position is held. Pigliucci and Finkelman (2014) argue that this is not a case of total opposition between Modern Synthetic monism and the pluralism of the EES, but rather one of theoretical modification. That is a view in keeping with Otsuka's (2019) observations derived from Lakatos. But modification to what end? If standard evolutionary theory provides an account of individuals at various levels of description that turns out to be mathematically equivalent to the multilevel renditions, which in turn make no novel predictions, what is the gain? Ultimately, I think this reduces to a question of taste, but taste impacted by a position on development.

Within the EES community many have taken issue with the gene-centric position associated with Gould's hardening phase of the Modern Synthesis (Gould 2002). Much of this criticism has focused upon the concept of the instructional gene, a gene that controls all development leaving no room for other sources of phenotypic variation (Oyama 2000; Kirschner and Gerhart 2010; Newman 2010; Sultan 2017,

[2] Somewhat oddly they give cancer as an example, rather than noting this as a distinct dysregulation of cooperation within an individual with potentially disastrous consequence. This suggests there is not a process for determining error in these kinds of models, although they see this as a conceptual difference in how to think about individuality.

2019) and this in turn has been related to Mayr's separation of developmental processes from those of evolution (Laland et al. 2011). [3] For the EES, evolution is not solely about gene frequencies, but also the emergence of novel phenotypes and this is seen as something that genes alone cannot do. As a result some EES advocates have called for the introduction of a mechanistic theory of form within evolutionary biology (Pigliucci 2007; Pigliucci and Müller 2010).

This emphasis upon phenotypes is also an emphasis upon organisms and thus individuals. It is entirely possible that Dawkins would include some of the extant references to inclusive fitness within the EES literature in an updated version of *The Extended Phenotype*. His argument would be simply that the EES sees adaptation as an individual benefit, and this is wrong. It is also possible that those scholars arguing for extended or inclusive views of inheritance (e.g. (Danchin et al. 2011)) can see the replicator focus of Dawkins more clearly but are seeking to extend the list of replicators to include such phenomena as epigenetic marks. Whilst the longevity status of such marks varies (Skvortsova et al. 2018), Dawkins was open to the possibility of other replicators (Dawkins 1978). Conceptually this would not be an extension of evolutionary theory, but merely an addition to the mechanisms capable of delivering evolution.

The EES rejection of gene-centrism is not simply a claim that the gene is not fully instructional in development, nor one that there are other potential replicators. It is also an attempt to place the organism as an explanatory unit in its own right (Baedke 2019, Chap. 8). The much quoted claim that genes should be seen as followers of phenotypic innovation has become a motto for this perspective (West-Eberhard 2003). What this means is that development, affected by environmental influence, can produce novel phenotypic variation which meets and changes a selection regime. The claim is that developmental sources of this response should be considered equal causal partners with genes *in evolution* and so this reduces to a claim for causal parity. Development is often expanded to include organismic activity (behavior) as we see in Niche Construction Theory (Uller and Helanterä 2019), for example.

Otsuka presented the Price Equation and decomposed it into its reproduction and selection portions (Otsuka 2015). He then applied causal graph theory to show how epigenetic and related processes could be incorporated in the reproductive part (as heredity in the breeders' equation, a subset of Price) and niche construction into the selection component. By using Price, a famously substrate neutral equation designed to capture evolutionary outcomes, Otsuka has neatly demonstrated where the EES applies its various arguments. It is clear from his causal graphs that the link between an epigenetic mark (C) and a phenotypic value (Z) provides a route for developmental response, and he includes environmental inputs to cause the mark and to affect the phenotype (E_C, E_Z) (for example, $E_Z \rightarrow Z \leftarrow C \leftarrow E_C$). The detail of those developmental processes is an empirical matter. Similarly for the role of agents in niche construction, where the phenotype affects value change in environmental

[3] It should be noted that Dawkins' view of the individual as idiosyncratic incorporates a developmental view due to his commitment to genes as catalysts (Dawkins 1989).

resources (R) and those new resource values are passed on to the next generation (R') to affect their phenotype (Z') (for example, $Z \rightarrow R \rightarrow R' \rightarrow Z'$).

In the last example, the phenotype is a set of actions that changes environmental resources in a way that then impacts on the phenotype of the offspring generation. The conceit is that this kind of agency can cause a change in mean phenotype value within the population ($\Delta\bar{Z}$), which is the outcome for the Price Equation and a measure indicating evolution has happened. But, as Otsuka makes plain, this is mainly a selection effect. Whilst the organism affects resourcing through its expressed phenotype, the downstream effects of that alter selection dynamics and affect the offspring generation. Selective niche construction then cannot be the reason the EES argues that natural selection should not be seen as preeminent. But the EES also runs a concept of *developmental niche construction* that relies upon an inclusive model of inheritance that incorporates various mechanisms of transmission.

> With an inclusive notion of inheritance, explaining the complementarity between organisms and environment requires us to work through a sequence of events in which developmental niche construction is both a cause and a consequence of evolution... For example, individuals may exploit a new food resource by behavioural innovation (that is, within-generation plasticity). If offspring learn how to forage by observing and imitating parents (that is, a form of non-genetic inheritance), the new behaviour may persist, with more or less fidelity, down generations. If individuals that make use of the new resource have higher fitness, the result of natural selection should be increased canalization of the behaviour's acquisition in ontogeny, and hence a more reliable inheritance... Although this process likely would involve genetic changes, gene-frequency change follows the acquisition and inheritance of the novel behaviour, and the latter therefore provide part of the explanation for why the population adapts to the new resource... This is not a hypothetical mechanism for adaptive divergence. For example, cross-fostering of chicks between great tits and blue tits demonstrate that imitation of parents can contribute to reliable inheritance of 'species-typical' foraging behaviours (Uller and Helanterä 2019, p. 358)

From this quotation we can see a desire to explain adaptation (complementarity between organism and environment) and to include the idea that genes follow the phenotype. Here organisms are creating a developmental niche that impacts upon selection, and then leads to genetic changes via canalization processes. But those organisms can be understood just as Rodrigues and Gardner wish us to, via inclusive fitness theory, the range of plasticity in their behavioural responses can be understood as a reaction norm, and the outcomes of persistent behaviour in a given direction can lead to change in selection dynamics.

It is not immediately clear that the individuals at the heart of niche construction theory are any different from those defined as *traditional* (Pigliucci and Finkelman 2014). There does not seem to be a requirement for the multilevel selection position, and all that is claimed in developmental niche construction is that behaviour can alter selection pressure indirectly through imitation, etc., rather than through direct alteration of the physical environment as in selective niche construction. Moreover, the fact of social learning mechanisms requires an evolutionary account and I am

certain Rodrigues and Gardner could readily sketch one within the inclusive fitness framework.

To conclude, I too was puzzled by the lack of engagement with inclusive fitness theory and Rodrigues and Gardner have made transparent how this perspective has enabled engagement with the EES laundry list (Laland et al. 2015). It is possible that it is simply due to a deep commitment to multilevel selection theory, but to date there is no clear extra-theoretical or empirical gain to be had from this perspective. To that end, I have suggested this might come down to taste and a preference for this multilevel view could be based in the developmental criticisms of the genes' eye view. But again, it is not clear that developmental views radically alter standard theory or demand a multilevel position. In the end the deflationary view of the EES, that sees inclusive fitness theory as a modification to the core of the Modern Synthesis is perhaps the answer. To that end inclusive fitness is not seen as a part of the historical effort captured by the synthesis, but rather a theoretical entailment of it. Given that the strong anti-gene-centric views at play in much of the EES, we have a group of scholars still searching for an alternative structure that will later replace the core of the Modern Synthesis and perhaps its entailments.

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On Monism and Pluralism: A Reply to Dickins, T. E.

22

António M. M. Rodrigues and Andy Gardner

Abstract

Dickins has made some thoughtful suggestions as to why the important contributions of inclusive fitness theory have not been more celebrated by the proponents of the Extended Evolutionary Synthesis, considering the extent to which inclusive fitness theory has accommodated and illuminated—and, indeed, been motivated by—their "laundry list" of supposedly neglected evolutionary factors. We agree that this oversight could be explained, in part, by their seeing inclusive fitness as a "monist" alternative to a more "pluralist" multilevel selection that was not part of the Modern Synthesis. Here we clarify that multilevel selection and inclusive fitness are not competing explanations, but rather they address orthogonal issues, concerning the process of selection and the purpose of adaptation, respectively. We discuss the sense in which inclusive fitness is "monist" in providing the only generally correct adaptive maximand, but also "pluralist" in the sense of accommodating a diversity of adaptive agents. We also emphasise that multilevel selection was, in fact, part of the Modern Synthesis and, indeed, its inadequacies as a theory of organismal adaptation provided a crucial motivation for the concept of inclusive fitness.

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Keywords

Levels of biological organisation · Maximisation principle · Meta-science · Natural selection · Purpose of adaptation · Shifting balance

Dickins has made some thoughtful suggestions as to why the important contributions of inclusive fitness theory have not been more celebrated by the proponents of the Extended Evolutionary Synthesis, considering the extent to which inclusive fitness theory has accommodated and illuminated—and, indeed, been motivated by—their "laundry list" (Welch 2017) of supposedly neglected evolutionary factors. We agree that this oversight could be explained, in part, by their seeing inclusive fitness as a "monist" alternative to a more "pluralist" multilevel selection that was not part of the Modern Synthesis.

Here we clarify that multilevel selection and inclusive fitness are not competing explanations, but rather they address orthogonal issues, concerning the process of selection and the purpose of adaptation, respectively. We discuss the sense in which inclusive fitness is "monist" in providing the only generally correct adaptive maximand, but also "pluralist" in the sense of accommodating a diversity of adaptive agents. We also emphasize that multilevel selection was, in fact, part of the Modern Synthesis and, indeed, its inadequacies as a theory of organismal adaptation provided crucial motivation for the concept of inclusive fitness.

As discussed in our chapter, the kin selection and multilevel selection approaches concern different ways of separating the overall action of natural selection into its component parts (Hamilton 1975). The kin selection approach separates the total action of natural selection into its direct versus indirect components, and the multilevel selection approach separates the total action of natural selection into its within-group versus between-group components. These two approaches are empirically equivalent, in the sense that in principle they should always yield the same predictions; but they can be considered as competing explanations at a meta-scientific level, for example, in relation to how readily each approach facilitates the derivation of testable predictions (West et al. 2008).

In contrast, inclusive fitness concerns the design rationale for Darwinian adaptation (Hamilton 1964; West and Gardner 2013). As a consequence of natural selection—whether one chooses to frame this in terms of kin selection or multilevel selection—individual organisms are expected to appear designed to maximize their inclusive fitness. Only this maximand yields optima that correctly correspond to the action of natural selection. Alternative maximization principles, such as those in which individual organisms are viewed as maximizing the overall fitness of their social group, do not generally yield optima that correctly correspond to the action of natural selection; and in those scenarios in which they do yield the correct optima—for example, in the context of clonal groups in which all group mates are genetically identical—these exactly coincide with the inclusive fitness optima (Gardner and Grafen 2009; Rodrigues and Taylor 2018).

In this sense, inclusive fitness is a "monist" concept. However, it does allow "pluralism" in terms of permitting some flexibility as to the level of biological organization at which it is applied. One may view the immune system as an adaptation at the level of the individual organism, functioning to maximize inclusive fitness; or alternatively view the white blood cell as an adaptive agent in its own right, striving to maximize its own inclusive fitness; or alternatively view each of the genes that reside in the white blood cell as striving to maximize their own inclusive fitness (Gardner 2015). To the extent that these different viewpoints all yield exactly the same predictions they are all scientifically valid.

Does the dismissiveness of the proponents of the Extended Evolutionary Synthesis concerning inclusive fitness theory owe to a view that inclusive fitness is a natural entailment of the Modern Synthesis whilst multilevel selection represents a wholly different approach to understanding evolution? If so, then this view is historically unfounded. Sewall Wright, indisputably a core architect of the Modern Synthesis, placed multilevel selection firmly in the centre of his understanding of the evolutionary process, in the form of the "shifting balance" view of adaptive evolution (Wright 1932). This concerned the rate—rather than the purpose—of adaptation. Indeed, there appears to have been a quite pervasive belief among evolutionary biologists through the first half of the twentieth century that what is best for the individual is also necessarily best for the population, such that the issue of the purpose of adaptation would have seemed trivial and scientifically uninteresting.

To his credit, Wynne-Edwards (1962) understood that selection at the within-population and between-population levels would often be in opposition. However, believing that between-population selection must always trump within-population selection, his application of multilevel selection logic led him to the erroneous view that the action of natural selection will always result in individual organisms behaving for the good of the population. The explicitness with which Wynne-Edwards set out his faulty logic—in contrast to others, such as Lorenz (1963), who simply and unreflectively assumed that animal behaviour must be understood as being for the good of the species—ignited a controversy from which there fortunately emerged a correct understanding of the purpose of adaptation. Today we understand that, irrespective of the relative strengths of different levels of selection, the purpose of adaptation is to maximize inclusive fitness (Hamilton 1964; Hamilton 1975).

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Part VIII

Evolution of Bacteriophage Latent Period Length

23

Stephen T. Abedon

Abstract

The life cycles of bacterial viruses—known as bacteriophages or phages—alternate between the infection of individual cells, creating "virocells," and phage existence instead as free virions. The latter perform an extracellular search for new bacteria to infect. Virocells, by contrast, can display a variety of cellular manifestations. These include *latent* infections, also known for phages as lysogenic cycles, for which virion production is considerably delayed. Alternatively, virocells can be virion productive. With *lytic*-productive infections, virions are released from phage-infected bacteria rapidly, in association with both cell envelope destruction and an abrupt cessation of infection metabolic activity. For *chronic*-productive infections, release is ongoing, with no recognizable end point, and this release occurs from bacteria that remain both structurally intact and metabolically active. Among these various infection scenarios—lytic, chronic, or latent—virocells possess a diversity of lifespans, ranging from very short for lytic infections (as short as ~10 min) to very long for lysogenic cycles (up to effectively infinite). Lytic infections in particular are described as taking place over latent *periods*. The length of latent periods can vary with phage type as well as with infection conditions, but most strikingly can vary as a function of single nucleotide differences between phages. As a general hypothesis, we can predict that the fitter that virocells are vs. free virions, then the more natural selection should favor longer periods of phage infection, including longer latent periods. Explored in this chapter are the impacts that different ecological scenarios as well as tradeoffs may have on the evolutionary optimization of phage infection durations.

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Keywords

ΦX174 · Qβ · Adsorption · Adsorption rate constant · Arbitrium system · Arbitrium systems · Bacteria · Bacterium · Bacteriophage · Bacteriophages · Best of times · Binary fission · Biofilm · Biofilms · Burst size · Burst sizes · Chronic · Clumped dispersion · Clumped dispersions · Communication · Cooperation · Eclipse · Effective burst size · Effective burst sizes · Evolutionary optimization · Evolutionary stable strategy · Extended rise · Extracellular search · Extracellular searches · Fecundity · Fitness · Free phage · Free phages · Free virion · Free virions · Induction · Infection period · Intercellular communication · Iteroparous · Kinetics · Latent · Latent infection · Latent infections · Latent period · Latent periods · Latent period evolution · Latent period length · LLP · LLPs · Longer latent period · Longer latent periods · Lyse · Lysis · Lysis-lysogeny decision · Lysogen · Lysogenic · Lysogenic cycle · Lysogenic cycles · Lysogenizing the winner · Lytic · Lytic cycle · Lytic cycles · Marginal value theorem · Michaelis-Menten · Microcolonies · Microcolony · Motility · Optimal foragin · Phage · Phages · Plaque · Plaque-forming unit · Plaque-forming units · Post-eclipse · Post-reproductive · Pre-reproductive · Prophage · Prophages · Rise · Semelparous · Shorter latent period · Shorter latent periods · SLP · SLPs · Spatial structure · Superinfection exclusion · Superinfection immunity · Temperate · Virion · Virion accumulation · Virions · Virocell · Virocells · Virulence · Within-culture competition specialists · Worst of times

23.1 Introduction

There are two basic measures of organism evolutionary fitness, as determined over shorter time spans and in terms of population growth rates (Bull et al. 2011). These are (1) fecundity and (2) the rapidity with which fecundity is realized. Fecundity is the number of offspring produced, and in evolutionary terms this concept takes into account not just numbers produced but also the likelihood that these progeny survive to produce offspring of their own. Thus, offspring survival and subsequent offspring reproductive success—in combination with offspring number and how fast offspring are produced—together should play key roles in determining an organism's and its genotype's near-term evolutionary success.

I consider in this chapter various ecological factors impacting the near-term evolutionary success of viruses that infect bacteria, viruses typically described as bacteriophages or phages (Lehman 2018; Harper et al. 2021). Specifically, I consider the evolutionary ecology of phage generation times—how long it takes for newly generated phages to produce offspring of their own. My emphasis especially is on how natural selection may impact the *duration* of the infected-bacterium stage of phage life cycles. This duration is known, for phage lytic cycles, as a latent period (Fig. 23.1), but which I generalize here also as an "infection period." I provide especially qualitative predictions along with descriptions of experimental results rather than developing or explicitly presenting mathematical models. In addition, I

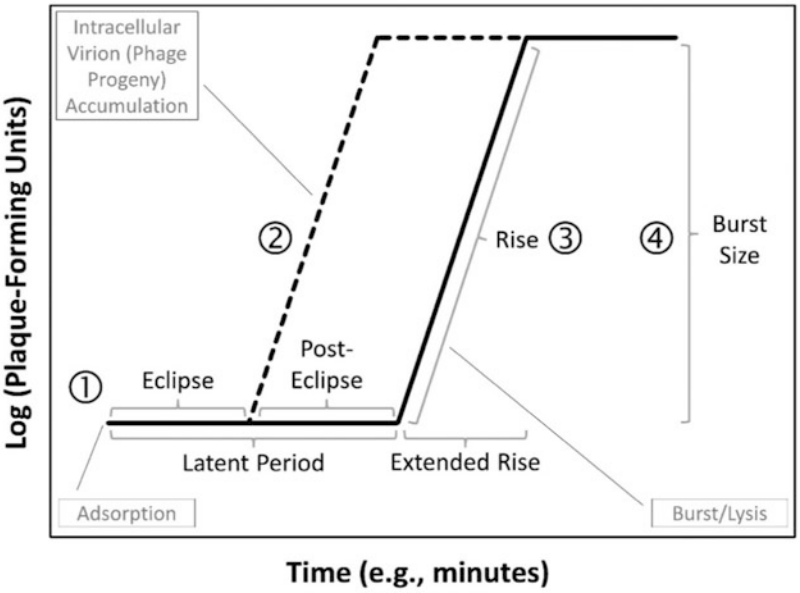

Fig. 23.1 A single-step (one-step) growth curve illustrating a phage lytic infection. Artificially synchronized, low-multiplicity virion adsorption is followed procedurally by dilution of phage-infected bacteria to prevent adsorption of virion progeny to new cells (Hyman and Abedon 2010; Kropinski 2018). The "Eclipse" (1) is initiated with phage adsorption, proceeds until the start of (2) intracellular virion accumulation (during the "Post-Eclipse"), and then (3) intracellular virion accumulation ends with phage-induced infected-cell lysis, resulting in a burst. The release of these virions, seen experimentally as a "Rise," does not usually occur simultaneously across an infected-bacterium population and hence is shown in the figure as "Extended." The result of all of these processes (4) is a "Burst size," which is the number of virions produced per phage-infected bacterium. The interval during which a bacterium remains phage infected is described as a "Latent period." Though not shown as such in the figure, across a phage population the latent periods of individual phage infections will end during the indicated "Extended rise," as considered in Sect. 23.5.6.1. Specifically, most of the individual phage infections—within a population of phage infections—will display durations that are longer than the minimum "Latent period" as labeled in the figure

mostly ignore underlying genetic and molecular details, as is a fairly standard approach to studies of life-history optimization (Bull et al. 2004).

23.2 Some Phage Biology and Ecology

Before turning to issues of phage infection-period evolution, I first consider and review relevant concepts of phage organismal biology and ecology. These include (1) latent periods, (2) latent period length variation, (3) eclipse periods and what here I describe as "post-eclipse" periods, (4) burst sizes and *effective* burst sizes, (5) pre-reproductive vs. post-reproductive periods, (6) chronic rather than lytic

virion release, (7) virocells, (8) extracellular searches, and (9) latent infections (Sects. 23.2.1 through 23.2.9). If you are already well versed in phage organismal ecology, however, then consider starting with Sect. 23.2.10, which begins the discussion of infection duration optimization. For recent reviews that I have co-authored on phage adsorption, infection, lysis, and ecology, as covered in Sects. 23.2.1 through 23.2.9, see Dennehy and Abedon (2021a, b, c).

23.2.1 Latent Periods Are Infection Periods

According to the *Oxford English Dictionary*, "latent" can mean ". . .present or existing, but not manifest, exhibited, or developed." Historically, prior especially to the late 1940s, whatever was going on inside of a bacterium during a phage latent infection was a mystery. Indeed, phage-infected bacteria were detectable—that is, as being "present" or "existing"—only as individual plaque-forming units (Anderson 1950; there also, "Phage-forming units"), which can also be described as "Infective centers" (Benzer et al. 1950). Soon, though, these infected bacteria would release, i.e., "manifest," free virions, though free virions, perhaps confusingly, also can be described as plaque-forming units or as infective centers (Fig. 23.1). Phage-infected bacteria therefore were seen as poised in some manner to release new phages. The infection portion of a phage lytic cycle thus is said to encompass a *latent* period (Ellis and Delbrück 1939; Hyman and Abedon 2009; Kropinski 2018) (Fig. 23.1).

Latent periods can be described more generally as the duration of a virocell stage of a phage life cycle (Forterre 2011, 2013). This, for strictly lytic phages, begins with the adsorption of free virions to not yet lytically infected bacteria. For temperate phages (Sect. 23.2.9), these latent periods as lytic infections can be initiated also via a process known as induction (Refardt and Rainey 2010). Latent periods persist for some length of time, such as between roughly 10 min to under one hour, though for some phages, including those of cyanobacteria, latent periods instead can last for much longer, e.g., Wilson et al. (1996). Latent periods then end in conjunction with the noted virion progeny release. For lytic cycles, the overall duration of the virocell stage and therefore of latent periods is determined explicitly by an infecting phage-caused "lysis from within" (Dennehy and Abedon 2021c). This lysis, of the phage-infected bacterium, can be mediated by a diversity of phage proteins, some of which also help to control lysis timing (Young 1992; Wang et al. 2000; Young et al. 2000; Young 2005; Young and Wang 2006; Cahill and Young 2019).

Note for the sake of avoiding confusion that "latent *period"* is never used synonymously with "latent *infection*," the latter which is also known for phages as a "lysogenic cycle." Indeed, latent periods by definition do not occur during latent infections nor *vice versa*. In addition, lysogenic cycles (which encompass latent infections), unlike lytic cycles (which include latent periods), are rarely described as occurring over some "period." Here, though, for convenience, I nevertheless describe both lytic and lysogenic cycles as occurring over some "infection period."

23.2.2 Latent Period Length Variation

The lengths of phage latent periods can vary as a function of both phage and bacterium properties and also can differ with infection conditions. For the latter, typically latent period lengths increase while phage burst sizes decrease as host growth rates decrease, i.e., such as due to reduced energy and nutrient availability (Delbrück 1940b; Webb et al. 1982; Hadas et al. 1997; You et al. 2002; Dennehy and Wang 2011; Choua and Bonachela 2019). Latent period lengths also can vary due to often subtle differences in phage genotypes such as single-nucleotide polymorphisms, e.g., Young and Wang (2006), Shao and Wang (2008), and Kannoly et al. (2020).

Phage latent periods additionally can vary as a consequence of phage-effected processes. An important example of the latter is lysis inhibition, which is an extension of the virion-production stage of a lytic infection (Abedon 2019). Latent periods can be extended in length as well due to a phenomenon that has been described as pseudolysogeny, which takes place prior to virion production, particularly as stemming from bacterial nutrient deprivation (Miller and Day 2008; Abedon 2009b; Łoś and Wegrzyn 2012). It is, however, especially differences in the duration of phage latent periods as occur *following* the start of intracellular virion production that are of interest here. Considered as well, though, are variations in the duration of phage infection periods more generally.

23.2.3 The Eclipse and Post-Eclipse Are Ecological Phenomena

The "eclipse," also known as an eclipse *period*, is a time during a phage productive cycle that immediately follows virion adsorption, or following lysogen induction, and then lasts for roughly the first half of a lytic infection (Doermann 1951, 1952, 1966; Hyman and Abedon 2009) (Fig. 23.1). During this time, fully assembled phage virion progeny do not yet exist, but they soon will. "Eclipse" as a term perhaps was intended to be synonymous with "obscuration," that is, of what is going on intracellularly during a lytic infection. Specifically, this was in terms of the detection of virions following the artificial lysis of phage-infected bacteria, and we now know that these virions are absent despite this lysis due to a combination of infecting virions having been dismantled during adsorption and progeny virions not yet having been fully assembled. Post-eclipse during a latent period, by contrast, is effectively the opposite of the eclipse, i.e., a time of detectable intracellular progeny virion accumulation (Fig. 23.1), or what Bull et al. (2004) refer to as an "Adult" period vs. "Juvenile" for the eclipse. The "post-eclipse" nevertheless, to the best of my knowledge, has no fully agreed upon name (Hyman and Abedon 2009), and some authors have incorrectly described the entire latent period as an eclipse.

Contrasting with eclipse vs. post-eclipse, modern virologists commonly differentiate virus infections instead into intervals that occur before vs. after the start of replication of the infecting virus genome. The eclipse, however, ends later than the production of the first progeny virus genome, as new phage genomes must be present

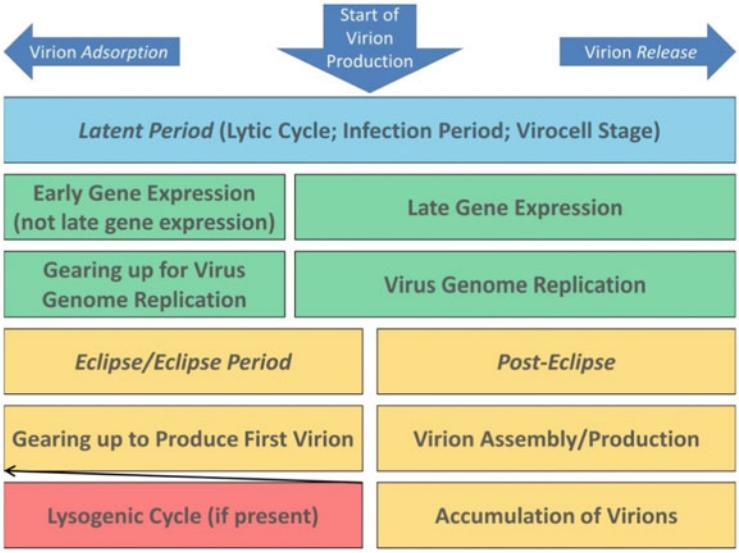

Fig. 23.2 Different views of the progression of phage infections. Light blue (upper rectangle): Lytic infection duration. Green (second two rows): Typical virological perspective of transitioning from early to late gene expression upon the start of virus-genome replication. Orange (most of the lower rectangles): Historically earlier perspective on the progression of a phage lytic infection, which is what I emphasize in this chapter and which is based on the timing of the start of new-virion accumulation. Red (lower-left): Lysogenic cycles as well as pseudolysogenic infections, both of which represent pauses delaying the gearing up of an infection toward virion production (the black arrow indicates that these take place prior to the eclipse). The "Accumulation of virions" box is provided for emphasis but is otherwise equivalent to intracellular "Virion assembly/production," which is to say that the post-eclipse begins with the successful assembly/production of the first mature virion to accumulate within a phage-infected bacterium

before new virions can be completed (middle rectangles, Fig. 23.2). The eclipse, moreover, is more relevant to phage ecology than the point of start of virus-genome replication, as ecology tends to be concerned with whole organisms, in this case functional virion particles, rather than solely with underlying molecular details. Furthermore, the intracellular assembly of new phage virions occurs over the full course of the post-eclipse and also, for lytic infections, there is an inability for assembled but not yet free virions to acquire new bacteria to infect. Thus, the most ecologically relevant breakpoints during phage latent periods are (i) the start of the eclipse (i.e., as occurring at adsorption, which is conversion of a phage virion into a phage infection), (ii) the end of the eclipse/start of the post-eclipse (when new-virion production begins), and (iii) the end of the post-eclipse, which for lytic cycles coincides with the conversion of intracellularly located virions into free virions. See Figs. 23.1 and 23.2 for illustration.

23.2.4 Burst Size and Absolute Fitness

The product of a successful latent period is commonly described in phage biology as a "burst" and bursts consist of released and thereby now *free* virion progeny. The *number* of virion offspring produced per infected bacterium is described, in turn, as a burst *size* (Fig. 23.1). Toward evolutionary success, those virion offspring must not only be released but also must go on to survive and reproduce themselves. From this can be derived the concept of an *effective* phage burst size, e.g., (Gadagkar and Gopinathan 1980; Abedon 2008b; Abedon and Thomas-Abedon 2010; Chan and Abedon 2012; Sieber and Gudelj 2014; Abedon 2017c; Eriksen et al. 2020; Doekes et al. 2021). An effective burst size simply is that number of virions produced per phage infection (burst size) that go on to find new bacterial hosts to successfully infect. Effective burst size, since it is a measure of numbers of progeny that survive per parental virus, therefore essentially is a phage's absolute Darwinian fitness, though with the rate at which progeny virions are both produced and acquire bacteria to infect being ignored. This concept of an effective burst size also is equivalent to what in epidemiology is described as a parasite's basic reproductive number (R_0) (Weitz et al. 2019).

The key take-home message regarding the meaning of effective burst size, or R_0, in any case is that if a phage infection produces, e.g., 100 virion offspring (burst size), then on average no more than 99 of these virions can fail to successful infect a new bacterial host (burst size minus effective burst size) for the parental phage infection to have replaced itself. If more than one virion survives to reproduce, then the phage infection would be more successful. As we will consider, a phage's effective burst size, along with the duration of its eclipse, are both important determinants of phage infection period-length optimization.

23.2.5 Pre-Reproductive vs. Reproductive Periods

The larger a virus' effective burst size, along with the faster new virions are produced and the faster that resulting new virocells are generated, then the faster should be a virus' population growth. Though more complicated for organisms that produce offspring over multiple episodes (iteroparous rather than semelparous), a common measure of the rapidity of microorganism reproduction is generation time. Specifically, for a semelparous organism, which by definition produce only a single "clutch" of offspring per life span, generations begin with birth or equivalent and end with birth of the next generation. Generations times furthermore can be differentiated into phases which are pre-reproductive vs. reproductive.

For phages, generations consist first (Fig. 23.3) of a pre-reproductive extracellular virion dissemination phase which can be called an extracellular search (Dennehy and Abedon 2021a) and then a pre-reproductive infection phase (the eclipse). A reproductive phase follows, during which new virions are produced (the post-eclipse). This for lytic phages is terminated by the free-phage generating virion release (lysis), with virion release for phages at least arguably equivalent to a birthing step. The

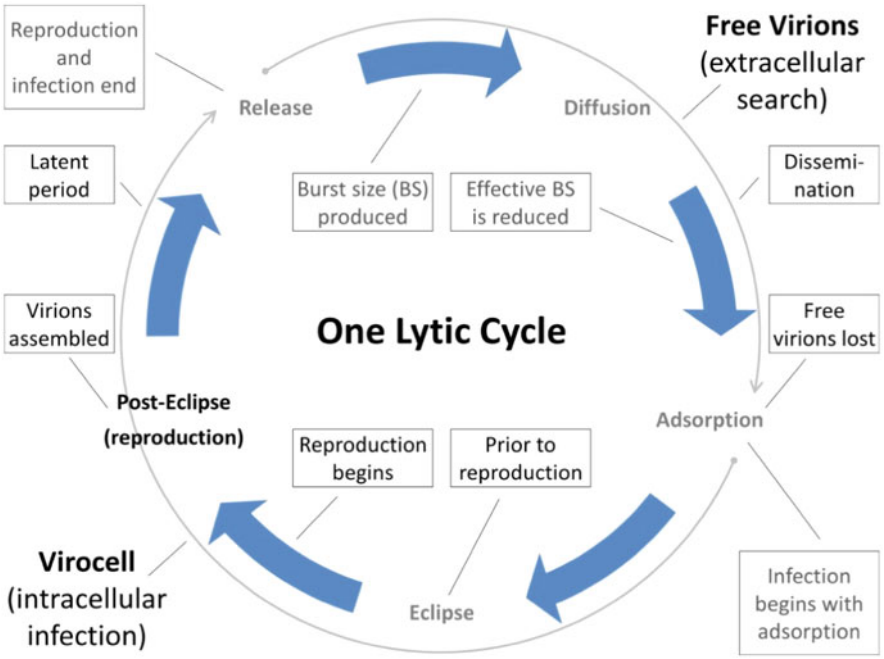

Fig. 23.3 Illustration of a phage life cycle, especially for lytic-productive infections. Dissemination is followed by a pre-reproductive infection phase ("Prior to reproduction") which is then followed by a reproductive phase ("Post-eclipse"). Burst size "reduced" refers to the diminishment of a phage's burst size toward its effective burst size, with the latter defined as the number of virions released, as a burst, that go on to produce successful new infections of their own. Though not shown in the figure, with chronic-productive infections (Sect. 23.2.6) the virocell stage continues despite virion release. With latent infections—also known for phages as lysogenic cycles and also not shown in the figure—infections instead pause at a point which, ecologically from the phage's perspective, is more or less equivalent to the start of the eclipse of a productive infection

shorter these phases, then the sooner that free virions—which more commonly are known as "Free phages" (Bronfenbrenner and Muckenfuss 1927; Benzer et al. 1950) or "Free phage particles" (Stent 1963)—are produced.

Optimizing the ratio of pre-reproduction—"Dispersal phase" and "Juvenile phase" as described by Bull et al. (2004)—to reproduction durations ("Adult phase") likely is a crucial aspect of infection period length evolution. That issue therefore will be returned to with some emphasis, starting with Sect. 23.2.10.

23.2.6 Chronic Release Extends the Post-Eclipse

Complicating the concepts of both latent period and resulting burst sizes are phages that release virion progeny chronically (Dennehy and Abedon 2021c). Prominent among these phages are members of family Inoviridae (Russel and Model 2006;

Mai-Prochnow et al. 2015; Hay and Lithgow 2019). These are the filamentous phages such as phage M13, though among chronically released phages, see also the pleiomorphic phage L2 (Maniloff 2006). For chronically released phages, much of the infection period involves virion release, with that release occurring in conjunction with both virion production and virion maturation. Thus, chronically releasing productive infections are not latent infections (since virions are produced) nor, strictly speaking, do these chronically releasing productive infections possess latent periods that encompass the reproductive period, since mature virions are released, that is, are "manifest" despite continued phage infection survival. Still, filamentous phages display both pre-reproductive and reproductive periods along with overall fecundities.

Phages that produce virions chronically also combine virion production with virocell reproduction. This occurs because bacteria continue to replicate via binary fission despite being phage infected. Furthermore, both infections and infected hosts can last effectively indefinitely rather than having predetermined end points. Indeed, phages infecting chronically are closer to being iteroparous in their reproductive strategies than the semelparity displayed by strictly lytic phages (Bull 2006). In addition, some filamentous phages can display both productive and latent infections (Yamada et al. 2007; Mai-Prochnow et al. 2015; Mai-Prochnow et al. 2015; Hay and Lithgow 2019), though not both at the same time. In considering the optimization of infection durations—with their (i) indeterminate post-eclipse reproductive periods, (ii) swift conversion of intracellularly produced virions into free virions, and (iii) effectively unlimited infection periods and "burst" sizes (Bull et al. 2004)—chronic infections are conceptually more complicated to model for infection-period length optimization than lytic infections. They nonetheless serve, along with latent infections, as examples of potentially very long phage infection (virocell) durations.

23.2.7 Virocells vs. the Extracellular Search

As noted, the term, "Virocell," has been used to describe the biological manifestation of the infected cell period of a virus' life cycle (Forterre 2011, 2013). This is the case whether or not an infection period leads immediately to the production of virions, i.e., so as to include latently infected cells as well. Virocells should be contrasted especially with free virions, that is, with the extracellular virion search stage of a phage life cycle (Trubl et al. 2020) (Fig. 23.3). It is upon free virion adsorption that the extracellular search ends, with those virions in combination with the bacterium they are now infecting converted to virocells (lower-left, Fig. 23.3) (Dennehy and Abedon 2020a). Virocells in turn are converted to free virions—or at least produce these virions in the case of chronic phage infections—in the course of the release step of virion-productive infections. Virion release from virocells thus initiates the phage extracellular search (upper-right, Fig. 23.3). For most phages, this release occurs over short intervals and involves destruction of both the host bacterium and the phage infection; that is, as consisting of an infecting phage-mediated bacterial lysis (Dennehy and Abedon 2020b) (upper-left, Fig. 23.3).

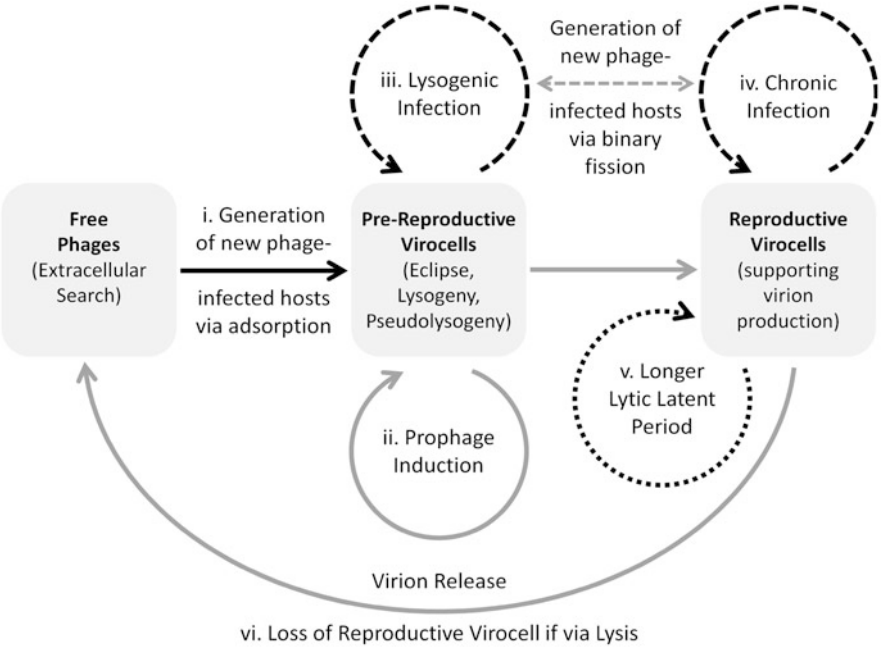

Fig. 23.4 Variations on phage life cycles, lytic vs. chronic vs. lysogenic. Virocells are phage-infected host bacteria and "reproduction" as a term is referring here to the production of fully functional phage progeny virions rather than the production of new virocells. Solid gray lines indicate progression toward conversion of virocells to free phages as following either (i) virion adsorption (if not leading to lysogeny) or (ii) prophage induction. Black lines show mechanisms leading to or sustaining greater numbers of virocells due to new virocell creation (i) via virion adsorption (solid line), due to infected-bacterium binary fission (dashed lines; lysogenic infections or chronic infections, iii and iv, respectively), or instead (v) as a consequence of delayed virocell loss (dotted line as referring to longer lytic-phage latent periods). Loss of virocells can be (vi) a consequence of phage-induced lysis or instead, but not shown, due to mechanisms of bacterial destruction that are independent of the action of the currently infecting phage

The number of virocells that are found within an environment, and their types, can be affected by a diversity of processes. Virion-producing virocells can be generated (Fig. 23.4) upon either (i) virion adsorption or (ii) prophage induction, with virion adsorption (i) potentially leading instead to the formation of latently infected virocells. The number of virocells that are found within an environment can be *increased* also as a consequence of the binary fission (iii) of lysogenically infected bacteria or (iv) of chronically infected bacteria. Loss of the virocell state can be delayed by lytically infecting phages by their (v) postponing lysis. Virocells will be *reduced* in number in environments—or at least not increase in number as rapidly—given (vi) phage-induced virocell lysis or due to phage-independent infected-virocell destruction, the latter not shown in the figure.

23.2.8 Extracellular Searches Can Be Costly

Individual virions during their extracellular search will vary both in whether they succeed in establishing new infections of bacteria and how long it will have taken these succeeding virions to have found those new bacteria to infect. The ability of phages to survive this free virion stage—thereby contributing to a phage's effective burst size (Sect. 23.2.4)—thus should generally be smaller (i) the longer the duration of the extracellular search or (ii) the lower a virion's inherent extracellular durability. Both criteria can be affected by circumstances, luck, and phage properties, with a search's duration varying especially with the availability of bacteria to adsorb: the more bacteria, the shorter the duration. Phage survival can also be affected *following* the phage extracellular search, that is, as a function of post-adsorption mechanisms of bacterial resistance to phages (Hyman and Abedon 2010; Labrie et al. 2010; Rostøl and Marraffini 2019), though those mostly are not our interest here.

Both the extracellular search duration and the potential for virion inactivation during that search can impact the benefits, to phages, of existing as virocells vs. lytically converting virocells into free virions. That is, if extracellular searches are sufficiently long in duration or result in a substantial lowering of a phage's effective burst size, then selection could favor phages continuing as virocells rather than transitioning to being free virions (Abedon et al. 2003). Another way of stating this idea is that the longer or more difficult it is for a phage virion to initiate a new, successful infection, then the more valuable a bacterium should be to an already infecting phage, potentially resulting in selection for a lytic phage to maintain its virocell for longer lengths of time. Related to these ideas is that phages themselves, by increasing numbers in the course of their population growth, can modify environments in such a way as to change the duration of phage extracellular searches, i.e., by giving rise to reductions in densities of possible new hosts to infect (Sect. 23.5.4). Thus, from Friedman and Gottesman (1983), p. 27: "Teleologically, a low ratio of phage to bacteria signifies a large supply of host for lytic growth, whereas a high ratio indicates an imminent loss of new hosts on which to propagate lytically."

23.2.9 Temperate Phages Can Vary Their Infection Period Durations

Phages that are able to latently infect are described as temperate (Bertani 1953) and during latent infections they are called prophages (Lwoff 1953). Prophage replication is tied to bacterial cell division, generating both new prophages and new lysogens per binary fission, but no new virions (Little 2005; Campbell 2006; Łoś et al. 2021). Latent infections can transition to producing virions in processes described as spontaneous production or, instead, as induction (McFall et al. 1958; Adams 1959; Stent 1963). Here I use "induction" or "induced" to describe either.

A prophage-infected bacterium is a "Lysogenic strain" (Benzer et al. 1950), or simply a "lysogen," with "lysogen" presumably a portmanteau word combining "lysis" and "generating." The term "lysogenic" (Hershey and Dove 1983), according

to the *Oxford English Dictionary*, was applied to bacteria at least by 1902 though does not seem to have appeared in a phage publication until 1921 (Gratia 1921a, b). "Lysogenicity" seems to have been first used in 1932 (Burnet 1932) and in a phage publication.

Life cycles of temperate phages typically are differentiated into lysogenic cycles (as latent infections) vs. lytic cycles (Bertani 1953). Immediately following adsorption, temperate phages must decide between these two strategies by making a lysis–lysogeny decision (Ptashne 2004; Brady et al. 2021), with lytic cycles rather than lysogenic cycles typically chosen upon the infection of log-phase cells by a single virion (Sinha et al. 2017); see also Refardt and Rainey (2010). As not all latently phage-infected bacteria are lysis generating (i.e., see "Chronic release," above), my preference when speaking generally is to distinguish between *latent* cycles and *productive* cycles (Hobbs and Abedon 2016), rather than lysogenic cycles and lytic cycles, though here I nonetheless use latent and lysogenic mostly interchangeably.

The likelihood of temperate phages latently infecting upon adsorption, or of prophages being induced, can vary with circumstances (Hershey and Dove 1983; St-Pierre and Endy 2008; Abedon 2017b; Igler and Abedon 2019). To the extent that a phage is able to display lysogenic cycles, then our expectation is that these latent infections may be selectively preferred over especially lytic cycles if environmental conditions result in extracellular searches being sufficiently costly (Sects. 23.2.8 and 23.3.1). Preference by a temperate phage for displaying a lysogenic cycle can occur either immediately following virion adsorption (lysogeny rather than lysis decision) or once lysogenic cycles have been established (avoidance of induction).

23.2.10 Infection Duration Optimization

All else held constant, organisms displaying shorter generation times, that is, time until their initial progeny production, should be evolutionarily fitter than organisms displaying longer generation times. Thus, for phages we expect that evolution by default should favor shorter infection periods, including shorter eclipses. That expectation can come with a number of qualifications, however. For phages these issues can include:

I. Shorter generation times are perhaps rarely achieved by phages genetically without creating costs in some other aspect of the phage life cycle, i.e., tradeoffs, such as in terms of the phage burst size (Sect. 23.4).
II. The utility to phages of releasing progeny virions sooner, thus shortening generation times, may be lower if free phages are less able to acquire new bacteria to infect (reduced effective burst sizes). For background, see "effective burst size" in Sect. 23.2.4, and then see Sect. 23.5.1.
III. The utility of shorter vs. longer phage reproductive periods (and thereby shorter vs. longer generation times) may vary depending on the degree that

phages are competing for the same pool of targeted bacteria. For background, see Sect. 23.2.5, and then see Sect. 23.5.2.

IV. Bacterial hosts are not necessarily homogeneously dispersed within phage-containing environments, thereby varying the duration of both a phage's extracellular search and generation time depending on a virion's location. For background, see Sect. 23.2.8, and then see Sect. 23.5.3.

V. Advantages of displaying shorter vs. longer infection periods may change both over the course of and due to phage population exploitation of bacterial populations found within a given environment. See Sect. 23.5.4.

VI. Not all phage life cycles are semelparous, thereby complicating explorations of infection period-length and generation time optimization. For background, see Sects. 23.2.6 and 23.2.9, and then Sect. 23.5.5.

VII. Even with semelparity (lytic cycles), phenotypic variation exists within populations in terms of both phage infection period durations and burst sizes. See Sect. 23.5.6.

VIII. While optimal generation times may be desirable, they may not be markedly more desirable under all circumstances, potentially interfering with the likelihood that a given phage populations will have succeeded in evolutionarily fine-tuning their latent periods to their current circumstances. See Sect. 23.5.7 as well as Sect. 23.4.2.

As a theme, I assume, as noted, that shorter generation times are desirable for phages, all else being equal, but I then emphasize how there can be opposing forces. First, though, I explore precedence for investigating the optimization of phage infection period duration.

23.3 Best of Times, Worst of Times

Of primary consideration in this chapter is the question of when shorter phage infection periods should be favored over longer phage infection periods, or *vice versa*. We can posit in particular that the favoring of shorter vs. longer infection periods likely has to do with the ease with which phages can find targeted bacteria vs. their having difficulties in doing so (for the latter, see Sect. 23.2.8). Metaphorically, a virion being able to easily find bacteria to infect I will describe in this section as the "Best of times" (Dickens 1859) for those virions (Fig. 23.5). Being easily found by lytically infecting virions we might describe for virocells instead as the "Worst of times." By contrast, the worst of times for virions—described below also as "Hard times"—is when bacteria are difficult to find, while the best of times for virocells, at least in part, should be when lytic phages to which virocells are susceptible are rare (Abedon 2011b). In this section, I consider especially the historical origins of this way of thinking.

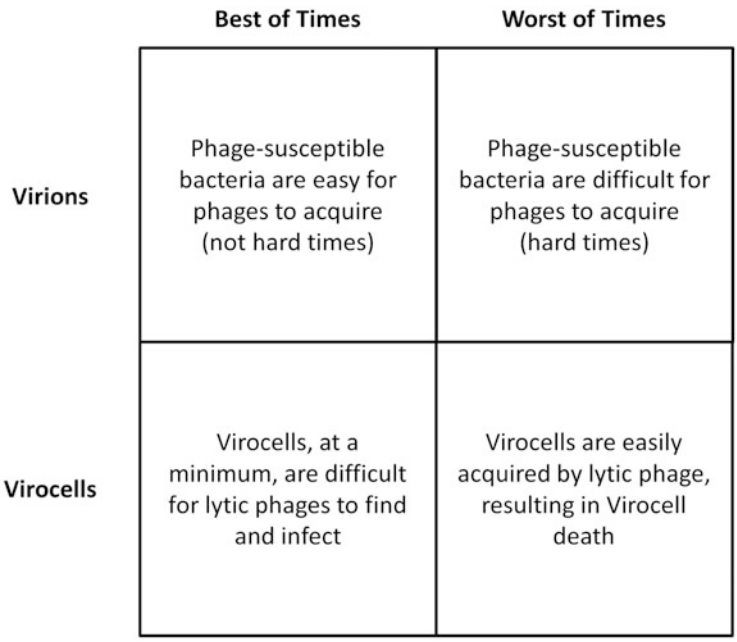

Fig. 23.5 Comparison of the best of times and worst of times for virions (as free phages) vs. virocells. The concept of "Hard times," as indicated in the figure, also is being used in this chapter. It is being used in the figure, however, solely to describe the free phage situation rather than also that of virocells and specifically refers to circumstances in which bacteria are less available to these free phages, such as due to low environmental densities of phage-susceptible bacteria and/or high rates of free virion inactivation

23.3.1 Virion Hard Times

The idea that phage virions might experience "Hard times" comes from Stewart and Levin (1984). Another way of saying this is that when resources are scarce and/or the danger associated with resource acquisition is high—"Bacteriophage with little chance for reproduction would be steadily lost for one reason or another" (p. 111)—then it is better to stay with an acquired resource than to search for a new one. For free phages, the most relevant resource is a bacterium to infect, and hard times for virions therefore would be when there exist "Great stretches of time and space where conditions permit only a sparse population of bacteria to persist" (p. 111). This thinking is equivalent to predictions stemming from optimal foraging theory's marginal value theorem (Wang et al. 1996), which is the idea that the duration over which an organism exploits a given unit of resource—a patch, such as an individual bacterium—should in part be a function of the availability of other, equivalent units of such resource. See also the "Cell-centric" viral fitness calculations of Weitz et al. (2019). The general idea is that when bacteria are less prevalent, then costs of extracellular searches will tend to be higher, and extracellular

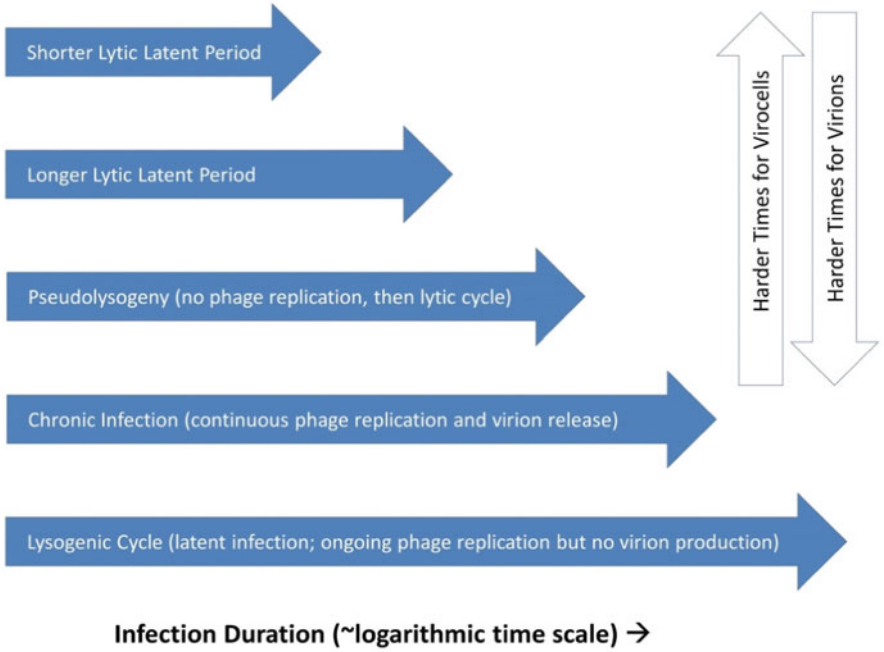

Fig. 23.6 General principles of selection for longer vs. shorter phage infection durations. Blue arrows all represent virocells while white arrows indicate selective forces. Chronic infections are drawn as shorter than lysogenic cycles on an assumption that greater costs to bacteria are imposed by the former than by the latter. In other words, and contrasting lysogenic cycles, can we really expect individual chronic phage infections, or even their virocell lineages, to last on the order of many years?

search times certainly will be longer. This circumstance in turn should enhance the utility to a phage of retaining a bacterium that it is already infecting, i.e., a virocell.

Stewart and Levin (1984) specifically hypothesized that such hard times for free virions should favor temperate phages that are displaying lysogenic cycles, as a form of longer or extended phage infection period (Abedon 1989), and this is rather than displaying lytic cycles. Thus, e.g., the fewer bacteria within an environment—particularly bacteria found within a phage's host range (Hyman and Abedon 2010; Chan and Abedon 2012; Abedon et al. 2021b)—then the harder the times for virions and the more likely that selection acting on phages, as according to Stewart and Levin, will be for longer periods of infection of individual cells (Fig. 23.6); see Wahl et al. (2019) for a recent updating of these ideas. Somewhat equivalently, I have described this balance as representing selection for "Bacteria-like vs. virion-like modes of existence" (Abedon 2008a). From a perspective of what thinking had already taken place on the evolution of phage infection duration, however, Stewart and Levin's idea of virion hard times should be viewed as having followed a relatively obscure—zero citations during its first 28 years of existence according to Google Scholar—1980 publication by Gadagkar and Gopinathan.

23.3.2 Gadagkar and Gopinathan (1980)

Gadagkar and Gopinathan (1980), p. 258, suggested that:

> . . .in nature it may be very important for the phage to lyse the bacterium as quickly as possible. This may be because of a certain finite probability of bacterial death which would increase with time. However, going through more generations rather than utilizing all the resources available in one bacterium may help leave more progeny behind. Thus the burst size may be adapted to adjust to such factors as availability of hosts, probability of death of hosts and the time and energy required to channel the host towards phage production. This would explain why phages could have evolved to utilize only a small proportion of whatever resources are available in a bacterium and also why maturation of the progeny is asynchronous leaving the actual time of lysis as the flexible control point.

My interpretations of this quotation are as follows:

1. "Lyse the bacterium as quickly as possible" suggests that, as a default assumption, shorter phage infection periods should be favored over longer infection periods.
2. "Certain finite probability of bacterial death" can be related to the worst of times for virocells, i.e., as due lysogen susceptibility to lytic infection by a different phage (Fig. 23.5), though also due to other causes. More generally this can be described as system- (virocell-) extrinsic forms of virocell mortality.
3. "Going through more generations" could imply a favoring of rapid lytic cycles rather than longer lytic cycles. This is presumably toward acquiring more bacteria and therefore more lytic infections, at least per unit time, when more phage-susceptible bacteria are present, that is, given virion best of times. See perhaps equivalently Levin and Lenski (1983), who note that, "The advantage of shortening the latent period lies in the earlier opportunity it provides for progeny phage to infect new host cells and further multiply" (p. 103).
4. "Burst size may be adapted to adjust to such factors as availability of hosts" could imply that there could be selection for greater infection fecundity when bacteria are less prevalent within environments, such as given virion worst of times/hard times scenarios (for how lysogenic infections may supply greater virion-producing fecundity, see Sect. 23.5.5).
5. "Time and energy required to channel the host towards phage production" might in part be reference to the phage eclipse, as I consider within this context in Sect. 23.4.3.
6. "Small proportion of whatever resources are available in a bacterium" could refer to displaying shorter latent periods at the expense of burst size, i.e., as implying a tradeoff (Sect. 23.3.3).

This publication by Gadagkar and Gopinathan (1980) thus seems to foretell some of the elements provided by Stewart and Levin (1984), at least in more general terms rather than Stewart and Levin's focus explicitly on "Why be temperate?"

Gadagkar and Gopinathan (1980) seem also to have anticipated in this passage many of the ideas subsequently presented by myself (Abedon 1989) regarding the evolution of phage infection duration within a context of lytic cycles (Sect. 23.3.3). The Gadagkar and Gopinathan publication therefore should be celebrated as perhaps the first to consider the evolutionary ecology of phage infection period duration. See, however, Abedon and Thomas-Abedon (2010) for discussion of how the main emphasis of the Gadagkar and Gopinathan study—that bacteriophage burst size may increase as a consequence of multiple phage infection of individual bacteria and as seemingly inspiring their writing of the above passage—actually may be mostly flawed.

23.3.3 Optimization of Latent Period Duration

More subtle than the distinction between the long infection durations of lysogenic cycles and the much shorter infection durations of lytic cycles are variations in the duration of lytic cycles themselves. Consistent with the above ideas, including those of Gadagkar and Gopinathan (1980), if "times" for free phages are "hard," then we might expect selection to favor longer lytic cycle latent periods. This should stem directly from the concept of a "bird in the hand is worth two in the bush," with an implicit assumption that there can exist significant barriers to obtaining the two in the bush (new bacteria to infect). Furthermore, lytically infecting phages in association with their progeny *cannot* possess all three "birds" at the same time, i.e., current as well as future bacterial infections. By contrast, if times for free phages are not hard, then selection for longer latent periods may be less strong due to there being plenty of new bacteria (birds in the bush!) to relatively easily infect. Thus, two birds in the bush could very well be worth more than one in the hand, and this should be particularly so if both in-the-bush birds are easily caught. Selection for shorter latent periods, particularly toward shorter phage generation times, therefore may be less constrained when new bacteria are more prevalent (free phage best of times; Fig. 23.5).

From the thinking of Levin and colleagues (Levin and Lenski 1983; Stewart and Levin 1984)—though at the time not yet from that of Gadagkar and Gopinathan (1980)—I developed the idea of a tradeoff between latent period length and explicitly that of phage burst sizes (Abedon 1989). Thus, phages further exploit bacterium-supplied resources in the course of displaying longer latent periods and they do so specifically to produce more virion progeny per infected bacterium, thereby resulting in larger burst sizes. When phage-susceptible bacteria are more abundant in environments, however, then longer latent periods—even with their larger burst sizes—may be selected against. This explicitly is because prior to lysis, phage virions are trapped in a non-free state, i.e., within infected bacteria. Longer latent periods, in other words, delay the ability of a phage to take advantage of free virion vs. virocell emphasizing modes of existence, and these delays can be costly especially when being a free virion is more advantageous (toward gaining "birds in the bush") than continuing being a virocell ("bird in the hand"). The result may be

selection for phages displaying shorter rather than longer latent periods, just as lysogenic cycles may be selected against during best of times for virions. Related to this is the concept of later-offspring discounting: earlier-produced offspring should be more valuable especially if they are able to contribute to population growth sooner, i.e., as should be the case especially when densities of phage-susceptible bacteria are higher (Abedon et al. 2003). Over the rest of this chapter, I build on these phage best of times or worst of times ideas.

23.4 Core Concept: Tradeoff Hypothesis

During lytic infections, as the eclipse ends, phage progeny virions begin to accumulate intracellularly (Sect. 23.2.5). Only upon lysis, however, are these virions able to begin their search for new bacteria to infect. Lysis, though, results in destruction of the phage infection, thus placing an upper limit on the number of phage progeny a single bacterium can lytically produce, i.e., its burst size (Sect. 23.2.4). There thereby exists an inherent and explicit conflict, a tradeoff, between shortening phage generation times by decreasing latent period lengths—should that shortening occur during the post-eclipse—and maintaining phage burst sizes. This (p. 76) "follows the classic life-history tradeoff between early and late reproduction" (Bull et al. 2004). Furthermore, a specific latent period may exist that serves to maximize phage population growth rates by balancing (i) tendencies to maximize rates of phage progeny acquisition of new bacteria to infect (shorter generation time) with (ii) competing tendencies to maximize the number of transmissible phage particles (larger burst size). In this section, I discuss this phage latent period tradeoff hypothesis and the associated concept of phage latent period optimization. For discussions of additional tradeoffs in phage evolution, see Goldhill and Turner (2014) and Keen (2014).

23.4.1 Shorter vs. Longer Latent Periods

Levin and Lenski (1983) suggested that there should be a utility for phages to lyse sooner and, as discussed above, see also Gadagkar and Gopinathan (1980). From p. 103 of Levin and Lenski, "The advantage of shortening the latent period lies in the earlier opportunity it provides for progeny phage to infect new host cells and further multiply." They also note (p. 103) that, "The growth rate of the phage population is directly related to the burst size. . . and hence any increase. . . should be favored by selection acting on the phage." I was then able to add, in what in fact was my first publication (Abedon 1989), that shorter post-eclipse periods, as resulting in shorter latent periods (SLPs), should also result in smaller burst sizes, thereby creating a tradeoff between generation time shortening and burst size increasing.

At the time I did not actually use the word, tradeoff. Instead, I emphasized the idea that the process of lysis halts ongoing intracellular virion accumulation. Nonetheless, phage infections in principle can display longer latent periods (LLPs) if they

can delay their lysis and this demonstrably can result in larger burst sizes, particularly holding infection conditions otherwise constant. Shorter latent periods, furthermore, are not necessarily a response to substantial declines over the course the post-eclipse in rates of intracellular virion production (Wang et al. 1996; Wang 2006), i.e., a scenario of burst size diminishing returns, but rather might represent an adaptive response to something else. In my 1989 publication, I suggested that this something else could be an abundance of new bacterial hosts to infect. That is, virion best of times (Fig. 23.5) may select for SLPs despite their shorter post-eclipse periods resulting in smaller burst sizes.

The model used in Abedon (1989) employed a fixed eclipse period duration of ten minutes and looked at the number of phage progeny sequentially produced over 100 minutes, given latent periods of 20 min, 30 min, 40 min, etc. That is, it was a pretty crude model. Densities of phage-targeted bacteria were varied in ten-fold intervals from 10^2 to 10^9 per ml. Down to 10^7 bacteria/ml, a latent period of 20 min produced more virions over those 100 minutes than one of 30 min, though the difference was not large. At 10^6 bacteria/ml, the advantage shifted to the 30 min latent period and by 10^5 bacteria/ml to 60 min or more. Overall, the implication was that a given bacterial density should select some phage latent period lengths over others, i.e., pitting SLPs against LLPs. Furthermore, higher bacterial densities should select for SLPs and conversely lower bacterial densities may select for LLPs.

23.4.2 Even Stronger Selection for Shorter Latent Periods?

The predictions from Abedon (1989) were not only that higher numbers (densities) of a phage's bacterial host should select for SLPs, but that selection for SLPs for various reasons could be stronger than as predicted by the core model. Specifically for the latter:

1. Any declines in rates of intracellular virion accumulation later in lytic infections, if such declines occur (Wang et al. 1996), should better favor SLPs over LLPs, since therefore the LLP burst size advantage would be smaller, i.e., the diminishing returns point made in the previous section.
2. When LLPs are advantageous, then total numbers of virions produced, per unit of environmental volume, should be smaller than when SLPs are advantageous, as due to there being lower numbers of bacterial hosts when LLPs are advantageous than when SLPs are advantageous, the latter being, i.e., explicitly when there are higher numbers of bacterial hosts present.
3. Phages should be less likely to even initiate population growth at lower bacterial densities, when LLPs would be favored, due to lower likelihoods of individual free virions even encountering bacteria before those phage virions become inactivated or are otherwise lost.

Consistent with this possibility of even stronger selection for shorter latent period, Bull (2006) suggested that selection becomes progressively weaker with lower densities of phage-targeted bacteria. I return to this latter issue in Sect. 23.5.7.

In Abedon (1989), a suggestion is also made that poorer bacterial growth conditions may help to select for SLPs vs. under more robust bacterial growth conditions; see also Wang et al. (1996). This latter proposition, however, may become less convincing given the inclusion in predictive models of additional phage parameters that can vary with bacterial growth conditions (Abedon et al. 2001), such as the phage adsorption rate constant (Delbrück 1940a; Hadas et al. 1997). In my opinion, the conclusions regarding the impact of changes in host *quality* on phage latent period evolution as presented by both Abedon (1989) and Wang et al. (1996) therefore should be ignored, at least in general terms, and particularly as more sophisticated approaches to exploring the impact of host quality on phage evolutionary ecology have been developed (Edwards and Steward 2018; Choua and Bonachela 2019). Nevertheless, the narrow conclusions from both studies (Abedon 1989; Wang et al. 1996) likely are correct: that shortening the eclipse *alone* (Bull et al. 2004 and the following section) or increasing rates of intracellular progeny accumulation *alone* both should result in shorter optimal phage latent period lengths. Indeed, so too should faster virion adsorption alone select for shorter infection periods (Levin and Lenski 1983; Bull et al. 2004; Shao and Wang 2008).

Returning to the second point from the above list (2), we may consider how especially lytic phages can modify environments particularly by reducing numbers of phage-susceptible bacteria. As a result, an environment could be modified from one that selects for SLPs (environments containing more phage-susceptible bacteria) to instead one of virion hard times that selects for LLPs (environments containing fewer phage-susceptible bacteria; see too Sect. 23.2.8). A result of this, contrary to the primary theme of this section, could be reduced selection for SLPs, i.e., so that phages also display some latent period-length adaption to phage-mediated subsequent dearths of hosts. The relative strength of that selection, for longer latent periods than may be optimal without taking subsequent host depletions into account, presumably would be stronger the fewer generations that the phage population goes through prior to this point, which should be inversely proportional to the volume containing the host bacteria, i.e., where smaller volumes should allow for fewer generations of phage infections (and therefore less selection for SLPs vs. LLPs) whereas larger volumes should allow for more generations of phage infections (and thereby greater selection for SLPs vs. LLPs; assuming in any case the same number of starting phages). This concept of greater environmental volumes within which phage population growth takes place potentially better favoring SLP phages over LLPs is returned to from a different though complementary perspective in Sect. 23.5.2.2. Alternatively is the concept of infection duration plasticity (Sect. 23.5.6), i.e., where longer phage infection periods are displayed at the point of phage-mediated depletion of phage-susceptible bacteria—especially as correlated with ratios of phages to bacteria coming to exceed 1—thus resulting in lysogenic cycles (for temperate phages) or instead extended lytic cycles, the latter called lysis

inhibition. Given such phenotypic responses to declines in numbers of bacterial hosts, then selection might be reduced or eliminated altogether for latent lengths that are longer than would be optimal prior to that point.

23.4.3 The Eclipse as a Juggernaut

There are limits to how short a successful productive phage infection can be. Prominent is the limit stemming from the eclipse period, which no matter what must be passed through for a phage to start producing virions. Still, the impact of the eclipse delay should be greater the shorter the associated latent period, i.e., as affecting per-unit-time rates of new-virion production if averaged over the whole of the latent period. The longer the extracellular search, however, then the less of a contribution the eclipse should make to determining latent period optima, since the smaller the relative contribution of the eclipse to phage generation times (Abedon et al. 2001; Bull 2006). A ten-min eclipse takes up a larger fraction of a 20-min overall generation time than it would of a 200-min overall generation, for example. In principle, then, the strength of selection for eclipse period shortening should vary with circumstances.

Not a great deal of information exists on the potential for phages to genetically modify the duration of their eclipses, though it is known that eclipse length can vary with the quality or availability of carbon and energy sources (Hadas et al. 1997; You et al. 2002). One example of a genetic shortening of the eclipse following selection for a shorter latent period, however, was observed with phage ST-1 (Chantranupong and Heineman 2012). This result is interesting also because phage ST-1 is a ssDNA phage (family *Microviridae*) that lyses its host by interfering with host cell-wall synthesis. This phage therefore might be more limited in its ability to shorten its post-eclipse (Sect. 23.2.3) vs. its eclipse.

Where other phage adaptations to exist that could result in shorter phage eclipses, then such adaptations would not necessarily be cost free. It is possible to speculate, for example, that a greater rush to gear up for phage progeny production might have a cost in terms of (i) the rapidity of subsequent phage assembly (Bull 2006), (ii) resulting virion functionality such as in terms of their durability or ability to adsorb new bacteria, or (iii) in terms of the infecting phage even surviving. For the latter, with phage T7 genome injection is delayed, presumably thereby extending the overall eclipse, and this occurs in a manner that allows the infecting phage to evade restriction endonucleases, thus enhancing infecting-phage survival (Molineux 2006). Toward the evolution of ever shorter latent periods, the duration of the eclipse thus likely represents a somewhat effective impediment.

23.4.4 Importance of Adsorption Kinetics

The duration of a phage's extracellular search is not fixed in length, even under constant conditions. This is because virion adsorption occurs as an exponential

decline in numbers of remaining free phages (Abedon et al. 2001; Hyman and Abedon 2009). Specifically, a constant fraction of free virions is lost to adsorption per unit time, e.g., 10% in the first ten minutes, with another 10% lost in the second ten minutes, and so on. Models that do not incorporate these exponential adsorption kinetics can exaggerate the impact of lower bacterial densities on phage latent period evolution. In practice, this means that selection for LLPs over SLPs at lower bacterial densities should be weaker than would be expected were adsorption rates instead fixed at some average value (Abedon et al. 2001).

What is going on is that a majority of phage population growth should be mediated by those virions that by chance happen to adsorb sooner. At increasingly lower bacterial densities, however, sooner-adsorbing virions adsorb progressively earlier than the majority of virions making up an extracellular phage population. As a consequence, for example, the optimal latent period for a bacterial density of 5×10^5/ml was predicted as 50 min given an assumption of virion adsorption as an exponential decline in numbers (Abedon et al. 2001) but roughly six times that value (~300 min) without this assumption (that is, instead using the so-called virion mean free time to describe adsorption kinetics). At 10^8 bacterial/ml, by contrast, the two predicted optima are nearly identical, at about 17 min; in all cases this is with an eclipse of 10 min in length. See Fig. 4 of that study for illustration.

Consideration of adsorption kinetics from a somewhat different perspective is found in a study by Shao and Wang (2008). They present experimental evidence that not only can bacterial density contribute substantially to phage latent period optimization but so too can intrinsic rates of virion adsorption contribute to this optimization. As those authors note, this should not be surprising since rates of phage adsorption to bacteria are direct, equivalent functions of both the phage adsorption rate constant (a measure of phage intrinsic rates of adsorption) and bacterial density.

23.5 Further Tradeoff Hypothesis Considerations

Though the evolution of phage latent period length seemingly is straightforward as based upon the output of simple models, real-world biology inevitably is more complicated than any model is capable of mimicking. Thus, the ideas of a tradeoff guiding phage latent period evolution as described above may be considered at best to point to possible trends, i.e., qualitative predictions of how phages might respond to specific ecological situations. By exploring presumed complications of real phage biology, however, it might be possible to refine models so as to gain greater understanding of the constraints impacting the evolution of phage infection period durations. I consider in this section some such complications. Parenthetical numbers in headings mirror the list presented in Sect. 23.2.10, with the first on that list (I) covered instead in Sect. 23.4.

23.5.1 Role of Effective Burst Size (II)

Burst size inherently plays a role in phage latent period evolution. This should be particularly given smaller burst sizes, or more specifically given low rates of post-eclipse intracellular virion accumulation. This is because, in absolute terms, low rates of virion accumulation can mean that the burst sizes associated with shorter latent period (SLP) phages can be exceptionally small. Phages with smaller burst sizes should be more vulnerable to having their effective burst sizes reduced to below 1, that is, as virion losses during extracellular searches are taken into account (Abedon et al. 2003). No matter what, a phage with an effective burst size of greater than 1—as should be more likely associated with a longer latent period (LLP) phage given their inherently larger burst sizes—is more fit than a phage with an effective burst size of less than 1. Thus, given greater rates of virion inactivation, then phages with very short latent periods may be selected against, and this is simply because those phages are not producing sufficient numbers of virions to even replace themselves.

This issue can have an interesting twist if effective burst sizes vary with densities of target bacteria. Specifically, the more rapidly that phage virions can reach bacteria to infect, then the lower should be absolute numbers of virion inactivations during the extracellular search, assuming that those inactivations occur with a constant likelihood over time. Thus, all else held constant, and given the presence of anti-free virion antagonists, then effective burst sizes should be higher at higher densities of phage-susceptible bacteria than at lower densities of those bacteria. That in turn means that the impact of free virion inactivation should be lower when SLP phages are already selected for, i.e., given higher densities of potential host bacteria. Conversely, the impact of free virion inactivation may be higher when LLP phages should be selected for, potentially exaggerating the LLP phage advantage at lower bacterial densities, and this should be particularly so the greater per-unit-time rates of virion inactivation. This effect, though, should countered to some degree by phages adsorbing with exponential kinetics (see Sect. 23.4.4), as those virions adsorbing sooner will both be driving rates of phage population growth and being subject more briefly to the possibility of inactivation as free virions.

Note that these various considerations could change if virion antagonists are not held constant in concentration. Heineman and Bull (2007), for example, used adsorbable but otherwise not phage-infection permissive bacteria as free-phage antagonists, with those bacteria serving as what can be described as sorptive scavengers (Hewson and Fuhrman 2003; Abedon 2017c, 2020b). There, as Heineman et al. report, 99% of bacteria present would kill phages upon adsorption. If that fraction were held constant but bacterial densities otherwise varied, then higher densities of permissive bacteria would not result in lower exposure to free-phage antagonists vs. starting with lower densities of permissive bacteria. That is, so long as these antagonists are allowed to increase in concentration as a direct function of numbers of susceptible bacteria, e.g., 99:1, then 99 out of every 100 phage adsorptions would still be inactivating.

Crucially, Heineman and Bull (2007) correctly did *not* vary densities of phage-permissive bacteria while holding the ratio of killer-to-permissive bacteria constant. Still, given propensities in phage biology to elevate the importance of relative densities over absolute densities (Abedon 2016, 2022), it is worth emphasizing that it is *absolute* concentrations of antagonists of virions that should control the extent of their negative impacts on virions. Therefore, differences between the mitigating influence of higher vs. lower densities of phage-permissible bacteria should be dependent on concentrations of virion antagonists present, and this is rather than just ratios of virion antagonists to bacterial, or virion, numbers.

23.5.2 Exploitative Competition (III)

Within a single bacterial culture, virions that, as a population, reach susceptible bacteria faster will outcompete virions that as a population reach susceptible bacteria more slowly, all else held constant. For example, phage adsorption rates can play important roles in phage fitness (Shao and Wang 2008), particularly at lower bacterial densities (Abedon 2009c; Bull et al. 2011), and this should be especially given head-to-head competition between phages for bacteria found within the same environment. Such head-to-head competition between phages for bacteria basically is exploitative competition, where organisms that are able to more rapidly obtain a given, limiting resource can outcompete other organisms competing to "exploit" that same resource. In the case of phage virions, this limiting resource consists of new bacteria to infect.

23.5.2.1 SLP Phage Invasion and Invasion Resistance

If SLP phages are able to obtain host bacteria to infect sooner, due to their shorter latent periods, i.e., rather than anything that has to do with virion adsorption rates, then this alone should allow SLP phages to outcompete LLP phages. Indeed, this can be the only advantage that SLP phages possess over LLP phages, meaning that even when bacterial densities are high, an SLP phage must be competing with an LLP phage within the same culture for the former to realize a fitness advantage (Abedon 1989; Abedon et al. 2003). This SLP advantage, however, should be realizable independent of the frequency of SLP phages within a phage population as it involves no more than individual phage lineages growing their populations faster and thereby reaching uninfected bacteria sooner (assuming, of course, that bacterial densities and other factors are sufficient to support this SLP over LLP growth-rate advantage).

Within a local population consisting of mostly LLP phages, this means that faster-growing SLP phages, even if consisting solely of a newly arising phage mutant, should be able to selectively increase their frequencies. Equivalently, within a local population of mostly faster-growing SLP phages, LLP phage frequencies should decline. Given especially the latter prediction, an optimized phage latent period therefore should represent an evolutionary stable strategy (Bonachela and Levin 2014), at least within the local environment within which that latent period length is optimal. That is, populations consisting of phages with optimal latent period lengths

should not be invadable by otherwise identical phages possessing sub-optimal latent period lengths.

23.5.2.2 Limitations on Invasion Avoidance

All else held constant, LLP phages growing without SLP phage competition should produce more virions overall than SLP phages, albeit with phage progeny produced more slowly (Abedon et al. 2003). This advantage is owing especially to the larger burst sizes of LLP phages, see Fig. 23.7 for illustration, as no directly antagonistic interactions with SLP phages would be possible under such circumstances, such as no superinfection exclusion or superinfection immunity (Sect. 23.5.6.2). This means that if an LLP phage can establish a clonal population such as due to genetic bottlenecking, e.g., starting the new population with a single LLP phage, then the LLP phage should be able to realize their advantage of producing more phages

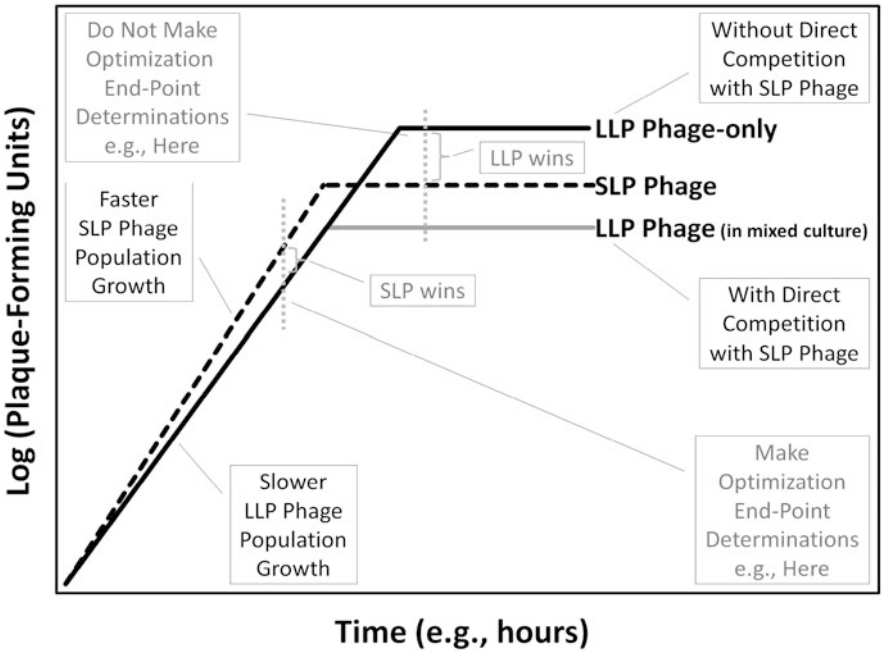

Fig. 23.7 SLP and LLP phage competition between and within cultures. A plaque-forming unit is either a free phage or a phage-infected bacterium (Sect. 23.2.1). Dashed black line: SLP phages both alone and in mixed culture with LLP phages. Solid black line: LLP phage culture without SLP phages present. Solid gray line: LLP phages in mixed culture with SLP phages. Gray, dotted, vertical lines: hypothetical timings of end-point titer determinations, indicating that LLP phages can be more fit than SLP phages following phage population growth even if SLP phages are more fit than LLP phages prior to the end of phage population growth. "LLP wins" refers to the between-culture success of LLP phages relative to SLP phages. "SLP wins" refers to within-culture success of SLP phages relative to LLP phages, which importantly could include SLP success against phages that come to display LLPs as a consequence of SLP phage mutation or SLP mutants that arise within otherwise LLP populations. This figure is based on an experiment presented in Abedon et al. (2003)

overall than SLP phages growing as a separate population under otherwise equivalent conditions (Abedon et al. 2003).

An advantage to LLP phages over SLP phages of existing within clonal populations nonetheless may be limited particularly to growth within relatively small-volume environments, given a potential for SLP phage mutants to invade these LLP-phage populations (Sect. 23.5.2.1). That is, frequencies of especially mutationally invading SLP phages are expected to increase as a function of the duration of competition experienced, which should be longer the greater the volume of an environment over which this competition can occur as well as the sooner an SLP phage invasion is initiated. Thus, while the SLP phage advantage at higher bacterial densities may be realized given within-culture competition between phages, that advantage may fail to be realized given between-culture competition between these phages, unless SLP phages have sufficient opportunity—mutationally or as a consequence of physical movement (a.k.a., migration)—to invade what otherwise could be isolated LLP populations.

Absent sufficient opportunity to invade otherwise clonal LLP populations, for SLP phages to realize a between-culture fitness advantage, then infecting more bacteria sooner would need to be more advantageous than producing greater numbers of virion progeny per infected bacterium, as achieved by LLP phages. Models supporting this hypothetical, solely between-culture SLP phage advantage have, to my knowledge, however, not been developed. I also personally find it difficult to imagine that this scenario would provide a robust utility to phages displaying SLPs unless the distinction between SLPs and LLPs in terms of latent period length and/or the advantage of the SLP phage's sooner release of new virions were substantial. That is, SLP phages given high bacterial densities may outcompete LLP phages during within-culture competition, and particularly given competition within larger local environments, but between cultures, without SLP phages invading LLP-phage populations, I speculate that an SLP advantage might be seen only if the LLP phages they are competing with possess *very* long latent periods, to the point that the rate of population growth by the latter is substantially delayed relative to the former, or instead the distance between these otherwise distinct bacterial populations is slight.

This speculation leads directly to a key question: Under what natural circumstances might within- vs. between-culture competition actually occur among phages? This is addressed as follows.

23.5.3 Bacterial Spatial Structure (IV)

Spatial structure consists of impediments to movement, which in the case considered here represents bacteria either being fully or partially inhibited from moving as well as instead avoiding such movement on their own. The most familiar artificial circumstance of bacterial growth within a spatially structured environment is in association with agar-based media, i.e., as also is associated with phage plaque formation (Abedon 2021). The familiar natural circumstance of bacterial growth within a spatially structured environment is in association with bacterial biofilms

(Abedon 2020a; Abedon et al. 2021a). In both cases, bacterial growth on microscopic scales can be found as microcolonies, i.e., clumps of especially clonal bacterial populations as often make up bacterial biofilms.

Note that spatial structure has been described as a route toward selecting temperate phages, such as phage λ with their ability to display lysogenic cycles, over virulent mutants of these phages that are unable to display lysogenic cycles (Berngruber et al. 2015), with a similar analysis but without spatial structure publisher earlier by the same group (Berngruber et al. 2013). The main mechanisms of this selection are virulent phage mutants locally exhausting supplies of phage-susceptible bacteria (as thereby resulting in virion hard times; Sect. 23.3.1) and lysogens serving as free-phage antagonists due to their display of superinfection immunity (though the authors use the term superinfection "exclusion" or "inhibition" instead of "immunity," with superinfection exclusion *sensu stricto* and its potential role in selecting from longer infection periods discussed here in Sect. 23.5.6.2).

The main premise underlying this Berngruber et al. (2015) analysis and conclusions is a somewhat extreme limitation not only on movement of bacteria but on free phage movement as well. In this section, however, while substantial limitations on bacterial movement are implied, i.e., as resulting in bacterial spatial structure, free phage movement via diffusion—and therefore potential virion access to new cells to infect—is assumed to be unimpeded except as stemming from bacterial spatial structure. Inactivation of adsorbing phages by superinfection immunity or superinfection exclusion is ignored, however, except implicitly to the extent that it can reduce effective burst sizes through their roles in free phage antagonism (Sect. 23.5.1). Thus, I ask how can competition between SLP and LLP phages be modified across environments given substantial limitations on bacterial movement but with much less limitation on the movement of phage virions, e.g., such as may be observed given phage infection of bacterial biofilms that are growing within otherwise fluid environments (Abedon 2020a, Abedon et al. 2021a)?

23.5.3.1 Well-Separated Microcolonies

An approximation of between-culture competition can occur outside of the laboratory if potential bacterial hosts are heterogeneously dispersed across environments. This could be if sub-environments can display relatively low levels of communication with other sub-environments, with each thereby approximating separate growth vessels as found in a laboratory. Bacteria, for example, could display clumped dispersions, with clonal microcolonies serving as the clumps and volumes between microcolonies representing regions of little or at least lower densities of these same bacteria. Microcolonies thereby could be viewed as regions hosting phage infections while passage through "extra-microcolony" regions could constitute the bulk of individual virion extracellular searches.

I have presented elsewhere scenarios of phage population dynamics both within and between bacterial microcolonies (Abedon 2011a, 2012b, 2015, 2020a). A general summary of those ideas is that phages must amplify their numbers while exploiting individual, clonal bacterial microcolonies in order to attain, despite

potentially harrowing extracellular searches between microcolonies, the equivalent of a basic reproductive number (R_0) of greater than or equal to 1. That is, here multicelled microcolonies would represent multicellular host-like entities to be "infected." Those phages that are able to produce the most virions per microcolony, i.e., per localized "focus of virion infection," should have the greatest potential to release enough virions that at least one of those virions survives to establish a new infection focus and ideally to achieve an R_0 of greater than one. Somewhat consistently, Levin (1996) describes burst size as an indication of virus transmissibility.

If microcolonies, ones consisting of bacteria that are susceptible to a given phage, are sufficiently distant from each other, and rates of extra-microcolony virion inactivation sufficiently high, then logic would suggest that phages producing more virions per infection, i.e., LLP phages, would be better able to supply at least one virion that survives to initiate the exploitation of a new microcolony. This may be especially so since virions not only may be inactivated during their extra-microcolony search but also might be able to enter regions that are less likely to contain susceptible microcolonies. For example, if virions diffuse away from submerged surfaces and into water columns, where susceptible bacteria are mostly lacking, then this also should increase the number of phages that would need to be produced just to maintain a phage population at equilibrium (more or less constant numbers) across these environments.

23.5.3.2 Clustered Microcolonies

If microcolonies are more spatially associated—such that they are clustered relatively closely together and thereby distances between them are both readily and rapidly spanned—then SLP phages might be selected over LLP ones even given between-microcolony (-clump, or -culture) competition (Abedon et al. 2003). This would be particularly so if virion inactivation and/or dilution during the extra-microcolony search is less likely due to those shorter distances between microcolonies and if SLP phages are able to start their extra-microcolony journeying sufficiently sooner than LLP phages (Fig. 23.8). An approximation of such a shorter extra-microcolony search might be seen during phage population growth as plaques.

Phage plaques form within bacterial lawns, with these lawns in many cases appearing to consist of numerous, closely spatially associated, clonal microcolonies of bacteria rather than homogeneously dispersed individual cells (Abedon and Yin 2008, 2009; Abedon and Thomas-Abedon 2010; Abedon 2011a, 2021). Though independent of microcolony presence, it is possible to consider the relative contributions of latent period and burst size to the kinetics of phage plaque development (Abedon and Culler 2007a), as well as to consider what latent period length may lead to a maximization of the number of progeny virions produced per localized, individual plaque (Abedon and Culler 2007b). In these circumstances, we have an expectation that SLP phages will produce larger plaques—as a measure of rates of phage population propagation (Yin 1991)—than will LLP phages (Yin and McCaskill 1992).

Larger phage plaques should allow for more opportunities for phages to infect bacteria, since more bacteria will have been found within the confines of the overall

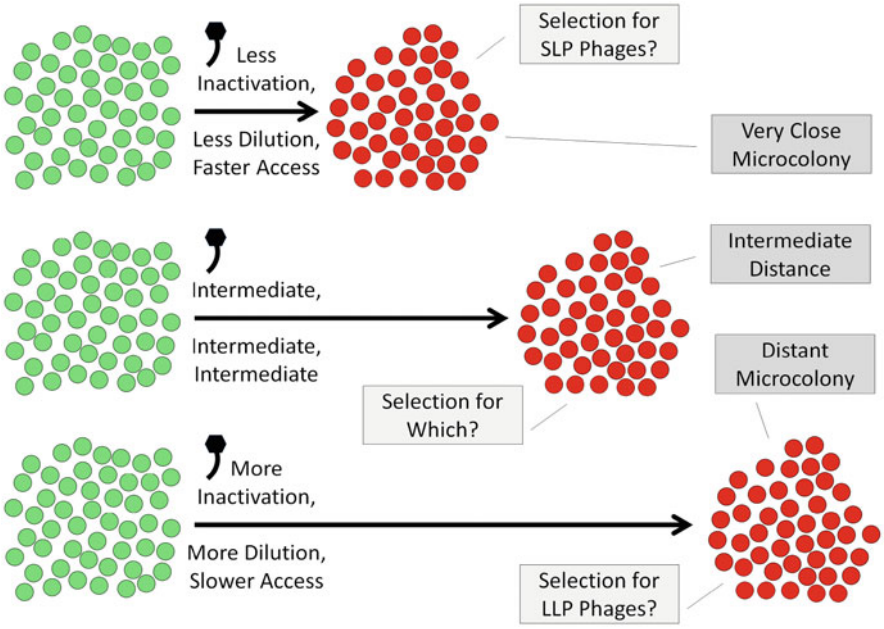

Fig. 23.8 Illustration of phage movement between bacterial microcolonies and potential for between-microcolony selection of SLP phages over LLP phages. After initiating the infection of one microcolony, an SLP phage should be able to generate new phages sooner, which then may be able to gain access to new, especially very close-by microcolonies sooner than LLP phages, all as owing to the shorter latent periods displayed by SLP phages. Over longer distances between microcolonies, the larger burst sizes of LLP phages may be more useful than the shorter latent periods of SLP phages given that a smaller fraction of a burst's virions may be able to reach new microcolonies to infect. Still, though not presented in the figure, if SLP and LLP phages should both infect the same microcolony—as a consequence of either co-invasion or phage mutational change—then owing to the high density of bacteria present within the microcolony, the SLP phage may be able to exploit that microcolony faster, i.e., reach new bacteria to infect sooner than the LLP phage is able to

plaque the more microcolonies the plaque has encompassed, and phage productivity overall is a function of both burst sizes (lower with SLP phages) and number of bacteria that are phage infected (higher with SLP phages during plaque growth as a result of producing larger plaques). Thus, at least in principle it should be possible to select for SLP phages over LLP phages in this context even if those phages are not simultaneously exploiting the exact same local environments. An important factor, however, will still be exactly how phage fitness is measured, with plaque size in and of itself not necessarily highly relevant unless competing plaques grow to the point of taking over entire bacterial lawns, which though rare, for some phages is indeed possible (Yin 1991). In that latter case, direct phage–phage exploitative competition again would be approximated, i.e., as occurring between plaques. Consistently, if microcolonies are close enough together, and SLP phages are reaching and then exploiting new microcolonies faster than LLP phages, then we can at least speculate

that SLP phages might possess a population growth advantage over LLP phages even if individual phages are not always competing for exactly the same bacteria.

Basically, here we would be replacing "individual bacterium" as a target for phage infection with "individual microcolony." Perhaps therefore the greater the number of microcolonies within an environment, then the greater the potential for SLP phages to more effectively reach and exploit those microcolonies vs. LLP phages competing within the same environment.

As a complication on these various ideas, we should expect that virions may move away from microcolonies, especially microcolonies present within fluid environments, more or less at the same time that those virions are released from lysing bacteria. That is, some fraction of virions produced per individual burst should diffuse outward (away from the parental microcolony) while some other fraction of virions from the same burst should diffuse inward, further into the microcolony (Abedon 2017c). Microcolonies that are experiencing exploitation by lytically infecting phages in effect therefore should be able to transmit virions into extra-microcolony volumes with little delay rather than going through a complex infection scenario such as of a multicellular organism where transmission to other individuals may be less directly linked to the infection of each individual cell.

This ongoing virion release means that phages collectively infecting bacterial microcolonies, particularly localized groups of infections that have been initiated by a single founding virion (i.e., a locus of phage infection), are less "semelparous" in terms of their virion production and release and instead more "iteroparous," with in this case this latter term referring to multiple rounds of virion production as a phage population exploits a bacterial microcolony. That is, loci of phage infections within natural environments may act more like chronically virion-releasing entities—though releasing from multiple cells making up bacterial clumps—rather than more like lytically releasing infections of individual bacteria. It therefore should be the number of virions that are produced earlier, across presumably multiple phage-infected bacteria, and then released earlier to start their search for new microcolonies, that could determine whether SLP vs. LLP strategies are preferable, with SLP phages potentially more able to produce those virions earlier in greater numbers and thereby being more fit the closer that microcolonies are clustered, i.e., the more useful that earlier virion production might be toward these progeny virions reaching these new microcolonies to exploit sooner.

The implication of these considerations, at least to me, is that it is not easy to intuit precisely under which circumstance, of bacterial spatial structure, each infection strategy may be preferable, SLP vs. LLP, and especially so if latent period-length differences are not large and microcolonies are spatially close to each other. Nevertheless, assuming that bacterial densities within microcolonies are high (Abedon 2012a, 2017a, c), there should be at least a potential for a favoring SLP phages over LLP phages in terms of exploitative competition occurring within individual microcolonies. These various ideas are summarized in Fig. 23.9.

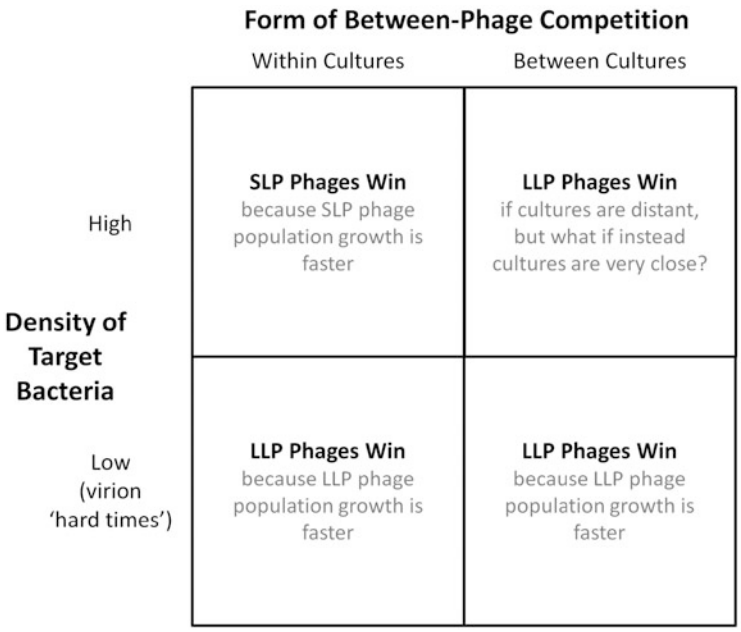

Fig. 23.9 Impact of spatial structure on optimization of phage latent periods. Here I use "culture," "clump," and "microcolony" synonymously. With within-culture competition, at higher bacterial densities, selection should favor SLPs while with lower bacterial densities within-culture competition instead should favor LLPs. Between-culture competition should favor LLP phages so long as burst sizes dominate competitive ability, and this will be particularly so given low bacterial densities within cultures. This latter claim may be less true, however, the more readily and faster that phages can move between cultures if those cultures contain higher bacterial densities, e.g., the plaque example from the main text

23.5.4 Environmental Change (V)

Environments can, of course, change over time. For our purposes here, those changes that can occur over relatively short time periods may be more relevant, e.g., such as over the course of phage population growth within a single bacterial culture. Besides phages increasing in number during that growth, there also are associated changes in numbers of phage-susceptible bacteria. It is the latter changes which are the primary emphasis of this section, and there are two scenarios of interest, though in my opinion one is less interesting than the other. The less interesting one is that within a growing bacterial population, phages displaying slower population growth would allow uninfected bacteria to replicate for longer, ultimately resulting in more bacteria, more phage infections, and more virions produced (Abedon et al. 2003). This effect in fact probably occurred during the experiments described by Fig. 23.7.

The more important situation, in my opinion, is simply that lytic phages can reduce numbers of phage-susceptible bacteria in the course of phage population

growth, e.g., (Edwards and Steward 2018; Doekes et al. 2021; Cheong et al. 2022; Shivam et al. 2022). Therefore, even if bacteria are initially present in numbers sufficient to select for SLP phages, nevertheless in the course of phage infection of new bacteria, phage-uninfected bacteria will decline in number. As we have thus far considered, lower densities of phage-susceptible bacteria may select for LLP phages over SLP phages. Thus, the population dynamics of interactions between phages and bacteria could result in changes in the fitness of SLP phages, measured in terms of rates of population growth, from higher than the fitness of LLP phages to lower than the fitness of LLP phages as phage populations grow in size (Abedon 1989; Levin 1996). In fact, certain phages seem as though they are able to respond phenotypically to such environmental changes such as may be differentiated in terms of ratios of phages to bacteria present (Sect. 23.2.8) along with absolute phage titers (Abedon 2022), and even as a consequence of quorum sensing (Sect. 23.6.3). These phenotypic changes are considered in the following two sections (23.5.5 and 23.5.6). Also considered in those two sections is the possible impact of increases in numbers of phage-infected bacteria as phage populations grow in size, as will occur as well in association with increases in phage numbers within environments.

23.5.5 Other than Lytic Phage Infections (VI)

Lysis–lysogeny decisions occur following temperate phage adsorption (Ptashne 2004). These decisions tend to be biased toward lysogenic cycles when phage numbers are higher, and particularly when phage-uninfected bacteria are less prevalent because then multiple phage adsorptions per bacterium are more likely (St-Pierre and Endy 2008); the resulting biases toward initiating lysogenic cycles we have dubbed, "High-multiplicity lysogeny decisions" (Abedon 2017b; Igler and Abedon 2019). As pointed out by Erez et al. (2017), p. 492, "In later stages of the infection dynamics, the number of bacterial cells is reduced to a point that progeny phages are at risk of no longer having a new host to infect. Then, it is logical for the phage to switch into lysogeny to preserve chances for viable reproduction." This represents a restatement of the "hard times" hypothesis of Stewart and Levin (1984) (Sect. 23.3.1) in combination with the dynamics discussed in the previous section (23.5.4).

There is, though, more to the utility of displaying lysogenic rather than lytic cycles than simply the presence of fewer new bacteria for virions to infect (Abedon 2009a; Abedon et al. 2009). For instance, (i) prophages can take advantage of bacterial survival strategies such as spore formation (i.e., by being more "bacteria-like"; Sect. 23.3.1), (ii) free virions but not prophages are susceptible to being killed by lysogen-effected superinfection immunity (Berngruber et al. 2015; Blasdel and Abedon 2017; Igler and Abedon 2019; Mavrich and Hatfull 2019), and also (iii) lysogens arguably can display higher fecundities than lytic infections, albeit with delay, and this is owing to lysogens possessing a more iteroparous phage reproductive strategy (Gill and Abedon 2003). Specifically for the latter, once lysogenic cycles have been established, they can then produce multiple new lysogens via

binary fission, with all of those resulting lysogenic bacteria possessing at least a potential to be induced to displaying productive cycles with attendant virion production (Abedon 2009d; Abedon et al. 2009). Furthermore, (iv) by lasting longer, lysogenic cycles may be able to take better advantage of the ability of bacteria to move about, via cell motility, thereby allowing for further or at least faster dissemination than can be achieved by free virions alone (Igler and Abedon 2019; Dennehy and Abedon 2020a). Motility of infected bacteria, however, also may come at a cost of increased susceptibility to infection by other phages (Dennehy and Abedon 2020a; Trubl et al. 2020), i.e., as potentially contributing to lysogens-as-virocells worst of times (Fig. 23.5).

23.5.5.1 Lysogenizing the Winner?

A further point is that bacteria which are able to grow faster when conditions are optimal appear to tend to carry more prophages (Touchon et al. 2016). This could represent there being a utility to temperate phages of existing as prophages particularly when host bacteria are especially fit and thereby potentially becoming more abundant (Touchon et al. 2017). This, that is, could be under circumstances when there might be an advantage to temperate phages to be bacteria-like, in this case due to better bacterial growth characteristics. Bacteria which have a potential to be either highly fit or highly abundant can also be more vulnerable as populations to phage infection, however, i.e., the "Killing the winner concept" (Jacquet et al. 2018). This could reasonably include infections by temperate phages, and the more infections initiated by temperate phages, then the greater the likelihood of establishment of lysogenic cycles, especially if those phages are able to reach higher multiplicities (Sect. 23.6.3 and above).

Thus, it seems reasonable that more lysogeny would be observed with those bacteria that are more fit (advantages of a bacteria-like existence) and this is *even though* those bacteria should be more vulnerable to infection by phages and also *because* those bacteria should be more vulnerable to infection by phages (greater opportunities for lysogen establishment). Or, in other words, I would suggest that these faster-growth bacterial characteristics are selecting for lytic population growth by those temperate phages which are able to infect them, i.e., selecting for shorter infection periods relative to lysogenic cycles, but a byproduct of the associated phage population growth is the longer infection periods of lysogenic cycles. The latter, however, is solely because a fraction of temperate phage infections tend to be lysogenic rather than productive (Sect. 23.2.9) and that is rather than because longer infection periods as lysogenic cycles are necessarily immediately selected for by higher bacterial growth rates. In addition, to the extent that there is an expectation of bacterial boom and bust dynamics, for which killing the winner can be an underlying mechanism, then that could select for lysogenic cycles, as bacterial bust times should correspond to hard times for free virions (Maslov and Sneppen 2015).

23.5.5.2 Also Lysogenizing Losers?

Pseudolysogeny is a pre-replicative delay in the progression of phage infections that is observed particularly though not exclusively under bacterial starvation conditions

(Miller and Day 2008; Abedon 2009b; Łoś and Wegrzyn 2012; Brady et al. 2021; Mäntynen et al. 2021). As pseudolysogenic infections fail to progress far into lytic cycles, they at least temporarily can be viewed as other than lytic. Like lysogenic cycles, pseudolysogeny too can be viewed as a tradeoff that can result in increases in phage fecundities at the expense of increases in phage generation times. Specifically, by essentially pausing lytic cycles until more nutrients are available to their bacterial hosts, pseudolysogeny may allow for the production of more virion progeny, or indeed production of any new virions at all, than might be the case were completion of lytic infections attempted while phage-infected bacteria are still starving (Breitbart et al. 2005; Abedon 2009d). That is, pseudolysogeny may allow infecting phages to delay progeny production until literally better times. See, equivalent to pseudolysogeny, the concept of phage "Hibernation mode" (Bryan et al. 2016). Note, though, that "pseudolysogeny" tends to be defined in different ways by different authors (Abedon 2009b; Mäntynen et al. 2021), with not all definitions describing phenomena that are compatible with the scenario I present in this paragraph.

An interesting parallel to pseudolysogeny is actual lysogeny as displayed by phage λ. Phage λ lysogenic cycles appear to be more likely, in terms of the outcome of lysis–lysogeny decisions, given infection of cells that are in stationary phase (hence, "Lysogenizing losers", in this case bacterial physiological "losers"). Perhaps equivalently, the phage λ lysis–lysogeny decisions tend to be biased toward lysogeny also when cell volumes are smaller (St-Pierre and Endy 2008). Indeed, as St-Pierre and Endy note (p. 20709), "…the lytic response may be preferred in infected cells capable of producing a large burst of progeny phage." Perhaps similarly, cyanophage S-PM2 possibly is more likely to display lysogenic rather than lytic cycles following infection of cells grown in reduced-phosphate media (Wilson et al. 1996). Again, "hard times" seem to be resulting in longer phage infection periods, though in the cases of pseudolysogeny or the initiation of infections by temperate phages, it is hard times being experienced by the phage-infected bacterium that seems to be driving the process, at least proximately, rather than explicitly hard times being experienced by free virions.

23.5.5.3 Chronic Release

Like prophages, phages that infect chronically also can take better advantage of bacterial adaptations than can phages during lytic cycles. At the same time, unlike with lysogenic cycles, chronically infecting phages do not have to choose between producing virions and allowing those new virions to find new bacteria to infect. Tradeoffs nevertheless do still exist (Breitbart et al. 2005; Abedon 2006). In particular, such phages can still form visible if somewhat cloudy plaques, with plaque formation in this case owing to the negative impact chronically infecting phages have on rates of binary fission of the bacteria they are infecting. The rates that chronically infecting phage virions adsorb to bacteria also can be somewhat lower than for other phage types (Kasman et al. 2002), suggesting a cost stemming from possessing virions with morphologies that allow for chronic release.

23.5.6 Infection Duration Plasticity (VII)

Plasticity in the duration of phage infections can be seen in the length of lytic infections, such as where poorer bacterial growth conditions typically will result in longer latent periods (Hadas et al. 1997). In this section, I consider instead variation in the duration of lytic infections as can occur within otherwise constant environments, i.e., as due to phage-infection stochastic effects or as can be a consequence of phenotypic variation existing between hosts such as in terms of the age of individual cells. I consider also inducible increases in the duration of lytic infections as associated with a phenomenon known as lysis inhibition. Of particular interest in this section is variation in the length of the post-eclipse as may or may not correlate with variation in burst size.

Lysogeny too is an inducible extension of the phage infection period (Abedon et al. 2001), one which also can be inducibly as well as stochastically terminated (the latter, i.e., via induction), and which in either case would imply a plasticity in infection period duration, i.e., with ongoing display of lysogenic cycles corresponding to the longer infection duration. The extension of the phage infection period with lysogeny is more equivalent to that of a lengthened eclipse rather than a modified post-eclipse duration, however, and lysogenic cycles also do not explicitly have associated burst sizes. For such plasticity in infection duration due to lysogeny, see especially the previous section (23.5.5) but see also Sect. 23.6.3.

23.5.6.1 Extended Rise

Seemingly random variation can exist in the duration of latent periods across a population of phage-infected bacteria, e.g., varying by perhaps 15 min for phage λ with lysis starting at about 50 min (Wang 2006), though see Dennehy and Wang (2011) and Kannoly et al. (2020) for more precise analyses using this same phage. The result may be described as an *extended* phage rise (Fig. 23.1), with "rise" a traditional description of the increase in phage titers that is seen as a population of otherwise synchronized phage infections lyse (Ellis and Delbrück 1939; Hyman and Abedon 2009; Kropinski 2018). Though this variation is fairly substantial for the tailed phage λ, the rise is especially extended for small, tailless, ssRNA or ssDNA, lytic phages such as Qβ or ΦX174 (Bull et al. 2004; Zheng et al. 2008). In either case, a single phage genotype under a single set of conditions will tend to display a range of latent period durations within a single environment rather than one unvarying latent period length.

Such variation in the timing of lysis across a single phage population might interfere with the precision of phage latent period optimization. This imprecision in lysis timing, rather than being a "bug," however, instead could be a "feature," perhaps allowing a single phage genotype to be effective in its population growth across multiple environments rather than being optimized for just one environmental condition. Perhaps most obviously, those individual phage infections that by chance display earlier lysis will inherently also display shorter generation times (Bull et al. 2011), as should be especially useful at higher bacterial densities where latent periods already make up substantial proportions of generation times. That an

extended rise might represent a feature rather than a bug is complicated, however, because rates of phage progeny accumulation, or even eclipse period duration, might also vary between infections, but not necessarily either in a manner that is useful to the phage or otherwise in a manner as might be anticipated by all students of phage biology.

For instance, those phages that by chance happen to display more delayed lysis times might also, during those same infections, display faster, slower, or even unpredictably varying rates of intracellular phage progeny accumulation, though see Bull et al. (2011) as well as Dennehy and Wang (2011) for modeling using an assumption of constancy in this rate. If instead there is a negative correlation between per-infection latent period lengths and rates of intracellular phage progeny accumulation (slower rates with longer lengths), then latent periods that happen to be longer than average could be associated with smaller than expected burst sizes. Such a negative correlation between latent period length and burst size can, for example, be attained mutationally (Nguyen and Kang 2014), albeit in that case as associated with a 32 base-pair deletion rather than a point mutation. The general trend nonetheless could be interpreted as "sicker" phage infections can potentially be simultaneously deficient in both latent period length (longer) and burst size (smaller than expected), i.e., as seen as well as in association with infection of bacteria displaying reduced bacterial growth rates (Sect. 23.2.2).

Alternatively, any phage infections that happen to display both shorter latent periods and faster rates of intracellular virion accumulation likely could dominate phage population growth. The latter may be the case with phage T4, where burst sizes that are three times the minimum are seen in combination with the shortest latent periods, with these maximally fit phage infections being of those bacteria that had reached their largest size just prior to dividing (Storms et al. 2014).

It is important to emphasize that intuitively one might make the opposite assumption—maximum burst size in association with maximum latent period length (positive correlation)—as this explicitly is the assumption that is made when studying the optimization of latent period length (Sect. 23.4). The distinction is that the observation of maximum burst size with minimum latent period (Storms et al. 2014) is a property of individual phage infections rather than of individual phage genotypes, where it generally is the latter (phage population characteristics) that is considered when studying lysis timing optimization. On the other hand, a positive correlation between the timing of lysis of individual cells and their burst sizes was inferred statistically by Baker et al. (2016), though this involved a ssDNA lytic phage (ΦX174) vs. the large, tailed, dsDNA phage that is T4; perhaps equivalently, see also Zheng et al. (2008). In any case, beware of making facile assumptions about the distribution of burst sizes as a function of latent periods across populations of phage-infected bacteria.

The answer to the question of whether an extended rise is a bug rather than a feature thus likely depends on the specifics of a particular phage's biology. Consistent though with the idea that it could be a bug, at least for non-single-stranded lytic phages, Kannoly et al. (2020) using an idealized system observed that plasticity in lysis timing may be minimal for wild-type phage λ in comparison to various mutants

of this phage possessing both longer and shorter times until lysis. They inferred from this that (p. 4) "buffering noise in lysis timing is ecologically relevant and is consistent with the existence of optima in lysis timing." In other words, at least in that system a more extended rise in some manner might be costly enough to the phage that it might in the past have been selected against.

23.5.6.2 Lysis Inhibition

Doermann (1948) found, for certain types of phages, that latent periods could vary as a function of environmental conditions, particularly in terms of whether or not phages of the same type were available to adsorb to already phage-infected bacteria. Description of this phenomenon, called lysis inhibition (Abedon 1990, 2019), was also the first indication, that I am aware of, that burst size can vary as a function of lysis timing (Hershey 1946a). More specifically, lysis inhibition represents an extension of the post-eclipse portion of the overall latent period. The lysis-inhibition phenotype also can be viewed as a transition from phage display of SLPs to phage display instead of LLPs that occurs explicitly within the context of lytic infections. Lysis inhibition furthermore has been of interest to me over the years for a variety of reasons. These include (i) its role in between-infection communication (Sect. 23.6.3) or "Sensory input" (Abedon 1990), (ii) its possible selective benefits including serving as a response to lower densities of uninfected bacteria in the environment, (iii) as an example of an anti-virion "Defection behavior" mediated by phage infections (Abedon 2009a), and (iv) as a possible adaptation to phage exploitation of clumps of bacteria in environments, e.g., such as bacterial microcolonies making up bacterial biofilms (Abedon 2012b).

Lysis-inhibited bacteria tend to also display a phenotype known as superinfection exclusion (Abedon 1994). Similar to superinfection immunity (see Sect. 23.5.5), superinfection exclusion results in the inactivation of phages adsorbing previously infected bacteria. Just as should be the case for lysogeny and superinfection immunity, the presence of these phage-infected bacteria, along with a dearth of phage-uninfected bacteria, should select for extended infection periods, i.e., such as those seen with lysis inhibition. Furthermore, the selective impact of superinfection exclusion should be exacerbated by the extended latent period that is lysis inhibition (Abedon 1990, 1992, 2008b, 2009a, 2019), as resulting in longer durations of expression of superinfection exclusion by individual phage-infected bacteria, just as the impact of superinfection immunity should be exacerbated by the extended infection periods of lysogenic cycles. Indeed, just as an increased likelihood that free virions will be inactivated should select for an extension of virocell states (Sects. 23.2.8 and 23.5.1), so too should phage-infected bacteria that can inactivate those same virions also select for an extended virocell state (Berngruber et al. 2015). Recently, similar arguments have been made by Aframian et al. (2022) regarding inhibition of induction of bacterial lysogens.

In the course of the delayed lysis of lysis inhibition, the burst sizes of these phage-infected bacteria can dramatically increase, i.e., as an SLP phenotype transitions to an LLP phenotype. This, in addition to avoiding superinfection exclusion, should also serve as a selective benefit of lysis inhibition. That is, ongoing phage

adsorptions should locally reduce numbers of phage-susceptible bacteria (i.e., see Sect. 23.5.4) at the same time that lysis inhibition should be induced by those same phage adsorptions. Within a typical broth culture growing in the laboratory, such as toward phage stock generation, the result can be higher phage population productivity than is the case without lysis inhibition. The same phage genotype, however, will not delay its initial population growth in the presence of higher bacterial densities in combination with a lack of excess free virions, since under those conditions the SLP phenotype will be displayed. Another way of looking at this process is that exploitative competition that favors SLP phages can transition to direct phage–phage antagonism, which can instead favor LLP phages (Abedon 2009a), i.e., as phage titers within environments increase. The latter perspective is also similar to the concept of ecological succession (Bull et al. 2006). As it does not involve transitions to new genotypes, however, it instead is more of a kin to "Phenotypic succession" (Pernthaler et al. 2001), i.e., SLP to LLP.

Lysis inhibition could similarly be of utility during phage exploitation of bacterial microcolonies (Abedon 2012b, 2019). It is possible, that is, that an absence of lysis inhibition during the initial exploitation of a single microcolony can allow for both rapid acquisition of multiple bacteria making up that microcolony and rapid diffusion of a subset of virions away from the microcolony, the latter just in case similarly phage-susceptible bacteria are found in nearby microcolonies (Sect. 23.5.3.2). This SLP-associated, more rapid phage population growth could then be followed by induction of lysis inhibition, resulting in an LLP-associated greater per-infection phage productivity, i.e., as could enhance acquisition of more distant microcolonies (Sect. 23.5.3.1). Phages that display lysis inhibition, in other words, could be both more effective within-culture (mixed culture) competitors and also more effective between-culture competitors, regardless of how close or distant cultures (bacterial clumps or microcolonies) may be from each other. It is important to recognize nonetheless that the lysis inhibition phenotype has been predominantly identified in only a minority of phage types, most notably the T-even phages (phages T2, T4, and T6) of *Escherichia coli*, though recently it has been found in a *Vibrio cholera* phage as well (Hays and Seed 2020).

23.5.7 Weak Selection? (VIII)

It is fairly well agreed upon that higher bacterial densities should select for SLPs, at least down to an optimum latent period length and under certain circumstances, while lower bacterial densities should select for LLPs, again at least up to an optimum latent period length. What is not necessarily agreed upon is whether this selection in either direction is in fact strong enough to be meaningful outside of the laboratory.

In one experiment addressing this question, at least in the laboratory, Shao and Wang (2008) competed λ phages with different latent period lengths, ranging from 30 min in length to about 70 min. What they found were relatively broad peaks over which differences in fitness were not exceptionally large. Wang (2006) similarly

observed nearly constant measured fitness values for phage λ latent period lengths ranging from 35 to 51 min, though with substantial drop offs at the next shorter (28.3 min) and next longer (53.7 min) latent period lengths. The former is not surprising as the eclipse length was measured at about 28 min, with the 28.3 min latent period's burst size reduced to about 10 phages/bacterium. The drop off associated with the 53.7 min latent period, however, is inexplicable, though it is possible that it is an indication that an unseen fitness peak exists somewhere between 43 and 51 min. The implication of these results is that while differences in population growth rates may be sufficient to distinguish between phages displaying substantially different lysis times, evolutionary fine-tuning of latent periods to perfectly match environmental conditions may not always be rapidly achieved (Bull 2006). It would be helpful, however, for experiments like these to include a greater variety of latent period lengths especially within the noted broad peaks. In addition, it is noteworthy that the experiments were done over four or five hours within replicating bacterial cultures, i.e., during which bacterial densities started at 10^6 bacteria/ml or less and then increased up to an indicated 10^9 bacteria/ml before declining due to phage infection. In other words, it is difficult to tell what bacterial density or densities the latent periods of these phages may or may not have been optimized to.

Suggestive instead of fairly fast directional evolutionary change, also in the laboratory, Heineman et al. (2005) were able to evolve a delayed-lysis deletion mutant of phage T7 to an approximation of the latent period length of wild-type phage T7—starting from about 28 min long and then down to about 12 min long—over an approximately 40-hour period of adaptation. Measurements of the fitness of the evolving phages provide little suggestion of either weak or weakening rates of increase in fitness with decreasing latent period length. In addition, wild-type phage T7 latent period length was found to evolve under equivalent conditions from about 14 min to about 10 min. Individual phage passages were initiated with greater cell densities (about 10^8/ml) and seem to have been run up to about an hour. Unfortunately, however, it is not certain—especially considering the deletion-mutant adaptation experiment—whether burst size also evolved and indeed it is possible that burst size actually increased rather than decreased over the course of adaptation of the deletion mutant down to the shorter latent period, though this was not determined.

An experiment addressing the question of whether selection, especially at lower bacterial densities, is weak is presented in Abedon et al. (2003), as illustrated here in Fig. 23.10. This was done in that study to address whether selection seen for SLP phages at higher bacterial densities would be reduced or even lost at lower bacterial densities, with cultures split 1:1 every 30 min to maintain an approximation of a constant cell density. The primary observation was that though SLP phages were found to outcompete LLP phages at bacterial densities of ~10^6/ml and above, at bacterial densities below 10^6/ml LLP phages appeared instead to outcompete SLP phages. This demonstrated that the SLP genotype under some circumstances could fail to outcompete LLP phages, but also showed that selection against SLP phages—even as measured over fairly short time frames (~2 hours) and at lower bacterial

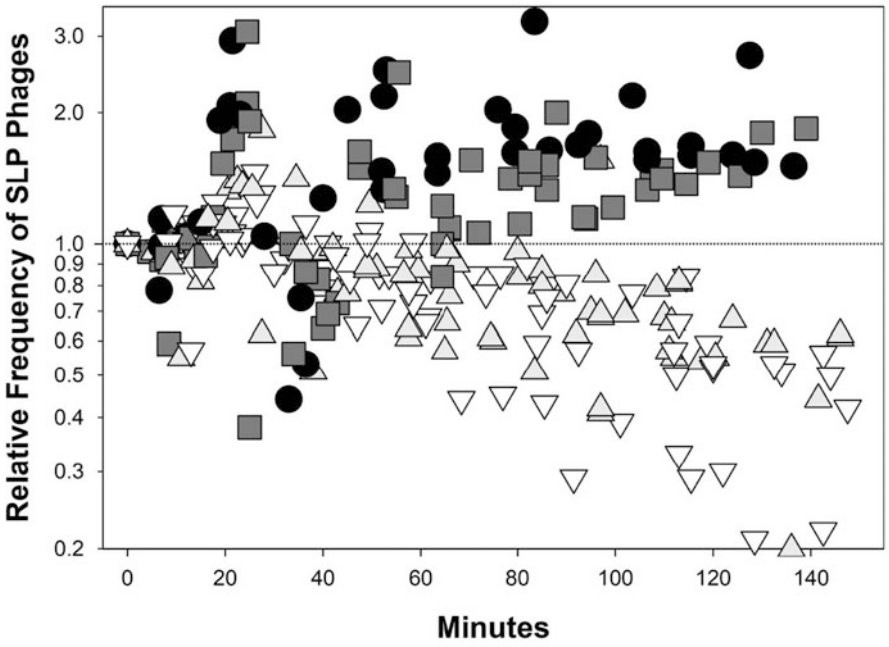

Fig. 23.10 Direct competition between SLP and LLP phages in well-mixed broth culture. A value of greater than 1.0 (dotted line) corresponds to SLP phages outcompeting LLP phages, and LLP phages outcompeting SLP phages corresponds to a frequency of less than 1.0. Black and darker gray symbols correspond to $\geq 10^{7.6}$ and $\geq 10^{6.6}$ bacteria/ml, respectively, i.e., higher bacterial concentrations. Lighter gray and white symbols correspond to $\geq 10^{5.6}$ and $\geq 10^{4.6}$ bacteria/ml, respectively, i.e., lower bacteria concentrations. The trend over time appears to be suggestive of higher bacterial densities selecting for SLP phages over LLP phages while lower bacterial densities are selecting for LLP phages over SLP phages. This figure otherwise is a re-rendering of a figure presented in Abedon et al. (2003). From that same publication, note that (i) the eclipse period for both phages was around 18 min, (ii) the latent period length defined as the start of the phage rise (Fig. 23.1) of the SLP phage (a phage RB69 mutant with a burst size of about 30% that of wild type) at best was only slightly longer than the eclipse, and (iii) the latent period length of the LLP phage (RB69 wild type) was about 21 min

densities, in this case especially lower than 10^5 bacteria/ml—is not necessarily inconsequential.

In light of that latter observation, it is worth noting that Heineman and Bull (2007) nevertheless struggled in their efforts to select for spontaneously occurring LLP phage mutants using a bacterial density of 1 to 2 × 10^6 bacteria/ml that was "progressively" reduced down to 10^5 bacteria/ml. From p. 1704 of that publication, "In a preliminary selection, there was little phenotypic evolution. . ." Overall, then, it is my opinion that the strength of selection for latent period optimization remains underexplored experimentally.

23.6 Related Phenomena

In this section, I consider some additional ideas stemming from the above thinking on the evolutionary ecology of phage infection-period duration.

23.6.1 Michaelis–Menten Kinetics Analogy

I have argued that phage latent period evolution can be viewed as analogous to the evolution of enzyme kinetics (Abedon 2009c). Increasing an enzyme's speed can involve either increasing its turnover rate (speed of a reaction once substrate is present in an enzyme's active site) or, alternatively, involve increasing substrate availability. The condition that should most favor an enzyme displaying a greater turnover rate, toward increasing its speed, is higher substrate availability. That is, the more readily that substrate can come to reach an enzyme's active site, then the greater the proportion of the duration of an enzymatic reaction that will be associated with the actual transition from substrate to product. Shortening an enzyme's turnover rate thereby will have a greater impact on the speed of a reaction when substrate is highly available than should increasing further an enzyme's affinity for that substrate. Analogously, greater bacterial densities (as a substrate or resource equivalent) along with greater affinity of phages for bacteria (as equivalent to an enzyme's affinity for a substrate, thereby increasing effective substrate densities) should favor the evolution of shorter phage latent periods (as analogous to reducing an enzyme's turnover rate), i.e., as has been explored experimentally by Shao and Wang (2008). Indeed, the Wang et al. (1996) model of phage population growth effectively *is* the Michaelis–Menten enzyme kinetics model from biochemistry (Abedon 2009c). (A yet alternative modeling approach that may be used to study the evolution of lysis timing is the so-called rate model where virions "are liberated at all times" (p. 33) at a defined lysis rate following virion adsorption (Bonachela and Levin 2014). Due to its lack of mechanistic realism, however, that approach is not considered here.)

With the Michaelis–Mention model, enzyme kinetics become more and more defined by turnover rate as substrate densities become higher, until changes in substrate density have little impact at all on reaction rates. So too, at ever higher bacterial densities—ignoring any changes in bacterial physiology that these higher densities might result in—a phage's generation time should more and more become defined solely by its latent period. Shorter latent periods thus are more readily selected when latent periods come to dominate generation times, but this increase in levels of selection for SLPs should occur only up to a point, as changes in bacterial densities to ever higher levels ultimately will have little absolute impact on the duration of extracellular searches. Thus, as bacterial densities become higher and higher, their impact on what latent periods are optimal tends to change less and less, e.g., above 10^8 bacteria/ml, whereas at lower bacterial densities (e.g., less than 10^6 bacteria/ml) there should be a linear relationship between cell densities and latent period optima (Abedon et al. 2001).

Table 23.1 Selection for longer phage infection periods

1	Lower bacterial densities (bacteria as phage resource or substrate equivalent)
2	Slower intrinsic phage adsorption rates (smaller adsorption rate constant; affinity equivalent)
3	Longer extracellular search (combines 1 and 2), an aspect of virion "Hard times"
4	Phage-antagonistic host bacteria, e.g., via superinfection immunity or superinfection exclusion
5	Greater rates of virion inactivation (combines 1 through 4), i.e., "Hard times"
6	Lower rates of intracellular virion accumulation during post-eclipse (see also 8)
7	Lack of declines in or even enhancements of rates of intracellular virion accumulation over time
8	Lower effective burst sizes, particularly if below replacement (that is, below 1)
9	Longer eclipse periods
10	Longer pre-reproductive periods generally (combining 1, 2, and 9)
11	Between- rather than within-culture competition among phages types
12	Substantial distances between exploitable bacterial populations, e.g., between microcolonies
13	Phage-mediated depletion of numbers of not-phage infected bacteria within an environment
14	Phage-mediated increases in numbers of phage-infected bacteria within an environment
15	Chronic rather than lytic virion release
16	Starving bacterial hosts (slowing eclipse, virion adsorption, or intracellular virion accumulation)
17	Starving bacterial hosts (resulting in increased likelihoods of pseudolysogeny or lysogeny)
18	Greater phage mobility during bacterial infections than can be achieved by virions alone
19	If phage-infected bacteria are not about to be killed, e.g., killed by unrelated phages
20	When bacterial fitness, in terms of survival or growth rates, is superior to virion fitness

Overall, the factors that can contribute to phage infection-period evolution, including those addressed in the previous paragraph, I have listed in Table 23.1. Note that though I present them from the perspective of selection for longer rather than selection for shorter phage infection periods, in many cases these issues can be reversed to result in selection instead for shorter latent period (SLP) phages over longer latent period (LLP) phages, keeping in mind as a partial exception that within-culture competition also requires that circumstances favor SLP phages over LLP phages in other ways (i.e., higher cell densities). In addition, not all of these listed circumstances are analogous to the evolution of enzyme kinetics.

23.6.2 Virulence Evolution

SLP phages might represent higher-virulence parasites in comparison with LLP phages, as equivalent to suggestions by May and Anderson (1983), as was cited (by reviewer suggestion!) in Abedon (1989). Similarities between phage latent period evolution and virulence evolution were subsequently suggested as well by Levin (1996). Additional thinking in this area was attributed to Abedon et al. (2003)

by Bull (2006), though this was inaccurate as it followed his reading of the Abedon et al. manuscript while that manuscript was in preparation, but the relevant passage was subsequently excised in response to anonymous reviewer concerns. Bull (2006) should be credited also with introduction of the idea that phage virulence might be measured in terms of loss of benefits to infecting phages, benefits that infecting phages otherwise would be gaining by maintaining the health of unlysed bacteria explicitly for the sake of producing additional phages. Thus, SLP phages, by that criterion, are more virulent than LLP phages.

As follows (two paragraphs), I supply an edited version of what we had intended to include in Abedon et al. (2003):

> Thinking on the evolution of parasite virulence—where virulence is a capacity to do harm to a host organism—has a muddled history. A modern view (Levin 1996) contends that virulence is a property of pathogens that either (i) is an unselected (i.e., "coincidental") byproduct of pathogen infection (e.g., Weiss 2002), (ii) is a consequence of "short-sighted" within-host competition between coinfecting pathogens (e.g., Frank 1996), or (iii) results from "direct selection," i.e., a positive association between harm to the host and greater rates of pathogen transmission to new hosts (Ewald 1994). For lytic phages, SLPs may be described from the phage perspective as displaying greater virulence, since SLPs result in sooner destruction via lysis of individual phage-infected bacteria (see Fig. 3B of Abedon et al. 2003). Selection for SLPs under high host density conditions, particularly due to faster phage acquisition of bacteria [as suggested here in Fig. 23.7, as equivalent to Fig. 3A of Abedon et al. 2003] therefore is supportive of the direct-selection hypothesis (Levin 1996). Similarly, selection for LLPs under low host density conditions [see Fig. 23.10, as based on Fig. 4 of Abedon et al. 2003] suggests, at least for that system, that lowering rates of pathogen transmission can select for reduced pathogen virulence. The latter result has been previously demonstrated given chronic phage replication within an environment that is completely lacking in phage-susceptible bacteria (Bull et al. 1991, Bull and Molineux 1992, see also Ebert and Bull 2003, for a broader perspective on this issue).

> We can alternatively emphasize the multicellularity of the host victims of pathogen virulence, describing, for example, an animal's body as a spatially limited, multi-celled environment within which pathogen replication takes place (Levin and Antia 2001). Phages similarly can be described as replicating within spatially limited environments, e.g., laboratory or colonic cultures that contain multiple, phage-susceptible bacteria [or in association with bacterial microcolonies; Sect. 23.5.3]. While from a modern standpoint it is perhaps a tortured view to consider phage "virulence" as occurring against whole bacterial cultures, particularly given common usage of the term "virulent" as the converse of "temperate" (Bertani 1953), in fact phages were originally described as entities that lysed bacterial cultures (d'Hérelle 1917). Those phages more capable of lysing these cultures, following growth from lower phage densities, therefore could reasonably be described as more virulent (d'Hérelle 1922, e.g., Smith et al. 1987, Summers 2001). Given such a scenario, we can describe SLP phages as possessing a growth advantage that is associated with greater virulence towards multi-celled environments, i.e., sooner lysis of whole bacterial cultures [see Fig. 3B of Abedon et al. 2003], or at least this greater virulence is the case when bacterial densities are higher. This greater virulence could be short sighted, however, if phage transmission to new bacteria-containing environments is more a function of phage burst size rather than within-culture phage-population growth rates [also as discussed in Sect. 23.5.3].

23.6.3 Intercellular Communication

The potential for phage infections to communicate with other phage infections, or to intercept bacteria-to-bacteria communications, has relatively recently become a hot topic (Erez et al. 2017; Silpe and Bassler 2019). Lysis inhibition, as discovered in 1946 (Hershey 1946a, b), and first described in intercellular communication terms in 1948 (Doermann 1948), likely however represents the first example of phage–phage intercellular communication (Abedon 2017b, 2019, 2020b). Also, Sklar and Joerger (2001), p. 25, had speculated that "...certain phage might have developed some quorum-sensing system that restricts lytic activity when the density of target bacteria is low and promotes lysis when the density of target bacteria is high. Such sensing has been proposed for temperate phage, such as lambda (Friedman and Gottesman 1983)."

Notwithstanding issues of precedence, it is of interest that these various proposed mechanisms of intercellular communication involving phages appear to have their primary impact on infection period durations. These include (i) the high multiplicity of phage adsorption-dependent lysis inhibition (Sect. 23.5.6.2), (ii) high-multiplicity lysogeny decisions (Sect. 23.5.5), and (iii) arbitrium systems (Erez et al. 2017; Aframian et al. 2022). The latter are phage-encoded quorum sensing-like systems that potentially are less dependent explicitly on high phage multiplicities of adsorption (Doekes et al. 2021) though nevertheless are still associated with higher rather than lower densities of the stimulating phage. Furthermore, each of these mechanisms results in extensions of phage infection periods. This is whether by lysis delay (lysis inhibition) or instead via biases toward display of lysogenic cycles rather than lytic cycles by temperate phages in the course of lysis–lysogeny decisions (Sect. 23.2.9). Arbitrium systems (iv) also appear to be able to stave off lysogen induction, thereby serving as well to extend durations of lysogeny (Bruce et al. 2021; Aframian et al. 2022).

On the other hand, (v) communication between phage-infected bacteria in association with higher multiplicities of phage adsorption can result in sooner lysis in the case of lysis-inhibited phage infections (Abedon 1992, 1999, 2009a, 2019). Similarly in terms of sooner lysis, (vi) phage interception of bacterial quorum-sensing communication has been found to promote prophage induction, thereby shortening the phage infection period (Silpe and Bassler 2019). It can be difficult, however, to appreciate the specific ecological utility of that latter system (Igler and Abedon 2019), and particularly so since (vii) the opposite response to bacterial quorums—repression of induction rather than increased induction—has been characterized with a different lysogen (Tan et al. 2020).

Though there can be other consequences of intercellular communication involving or impacted by phages (Bernard et al. 2021; Wang et al. 2022), so far there certainly seems to be a bias, at least in discovery, toward phage-associated intercellular communication systems supplying information correlating with environmental phage or bacterial densities that then impact phage infection duration (Stokar-Avihail et al. 2019; Duddy and Bassler 2021; León-Félix and Villicaña 2021).

23.7 Conclusions

The phenomenon of phage latent period evolution can be generalized as one of phage infection-period or even phage generation-time evolution. In any case, we have an expectation that environmental conditions favoring bacterial adaptations over phage virion adaptations, such as the so-called Hard times for virions (a.k.a., virion worst of times; Fig. 23.5)—which perhaps especially is when concentrations of target bacteria within an environment are low—should promote the evolution or display of longer infection periods. Favoring of longer infection periods should be particularly so given also virocell best of times, which at a minimum means virocells are experiencing low levels of phage predation pressure and which can be when concentrations of these bacteria also are low. Selection especially for longer latent periods could result as well from phages displaying inherently smaller adsorption rate constants (poor adsorption properties), longer eclipse periods, free virion exposure to phage-inactivating bacteria such as those displaying superinfection exclusion, or instead display by phage infections of superinfection immunity toward encouraging phage lysogenic cycles. See Table 23.1 for a summary of circumstances that could select for longer phage infection periods.

Alternatively, when phage-susceptible bacteria are highly prevalent, as representing virion best of times, then that should favor more rapid phage acquisition of those bacteria and therefore sooner production and release of new phage virions, which for lytic phages means shorter infection periods. In terms of bacterial densities alone, these concentrations need not be terribly high to select for shorter infection periods, e.g., about 5×10^6 bacteria/ml for a typical laboratory latent periods of 25 min to be optimal given an eclipse of 10 min (Abedon et al. 2001). So too lysogenic or chronic infection susceptibility to lytic phages could reduce the ability of these longer-term phage infections to remain viable under conditions that favor lytic population growth strategies by unrelated phages, i.e., as representing virocell worst of times. Virion best of times by contrast should favor, at least selectively, the initiation of virion production by latent infections, that is, induction.

These conclusions may be complicated by environmental spatial structure, with especially shorter latent periods likely fitter mostly given exploitative competition between phages replicating within the same local environment. As suggested in Abedon et al. (2003), those phages with shorter latent periods might thus be viewed as within-culture competition specialists, including in terms of competition with spontaneously occurring phage mutants displaying longer latent periods, while phages with longer latent periods may be more biased in their reproductive strategies toward optimizing between-culture or between-bacterial-clump transmission. It is perhaps the conflict between these two strategies, one optimizing within-culture competition and the other optimizing between-culture competition (Abedon et al. 2003)—as well as variability between the types of bacteria phages infect along with differences in especially the abiotic environmental conditions under which those infections occur (Bonachela and Levin 2014)—that result evolutionarily in the phage latent period lengths that we observe.

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Optimality and Idealisation in Models of Bacteriophage Evolution: A Commentary on Abedon

24

Benjamin J. A. Dickins

Abstract

Bacteriophages are ubiquitous bacterial predators that played a significant role in the development of molecular genetics and, later, in experimental evolution studies. I summarize this history to provide some context to Stephen Abedon's contribution to this volume. In his contribution Abedon identifies ecologically relevant timepoints and periods in the life cycles of phages and then considers the fitness consequences of variation in the timings of these from a perspective informed by *optimal foraging theory*. This is a fruitful approach and I discuss his use of analogies and *abstractions* to achieve this. Ecology also deals with interactions and feedback. A clonal phage lineage can deplete the bacterial population on which its growth depends. Abedon considers this from an optimality perspective also. Instances of *phenotypic plasticity* in the life cycle may be labelled adaptive if they maximize phage growth rate over a time period that integrates negative feedback from effects on prey populations. Here I note that this may overlap with the time frame for evolution (in response to a change in the biotic environment) by natural selection. Considering the relatedness of phages also invites us to relax assumptions made in models defined by competing phenotypes. These considerations suggest that phages may continue to be valuable model systems, especially for investigators interested in eco-evolutionary feedbacks and *reciprocal causation*.

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Keywords

Bacteriophages · Life cycles · Optimal foraging · Abstractions · Adaptive tracking · Phenotypic plasticity · Reciprocal causation

Bacteriophages (viruses that infect bacteria) are the most numerous organisms on the planet (Mushegian 2020) and, as predators of bacteria, they are highly ecologically significant (e.g. Puxty et al. 2016). Phages also have a historic role within biology. Although they were discovered in the early twentieth century (Duckworth 1976), initial research centred on the use of phages in treating bacterial infections (Summers 2001). The status of phages as viruses was confirmed only in the 1940s with developments in electron microscopy (Salmond and Fineran 2015). Unsurprisingly, developments in light microscopy had elucidated the unicellular world, including bacteria, earlier than this (Zewail 2010). What really brought phages to the fore was an appreciation by a group of biophysicists in the 1940s that the viral life cycle constituted a model system ideally suited to revealing the molecular nature of heredity (Stent et al. 1966). The phage group was remarkably successful with textbook discoveries including Chase and Hershey's confirmation of DNA as the heritable material in a famous radiolabelling experiment involving a kitchen blender (Hershey and Chase 1952). As well as giving birth to the field of molecular biology, phage-focussed research programmes also bequeathed practitioners with restriction enzymes and other tools for DNA manipulation (Salmond and Fineran 2015).

Work has continued in phage biology with biochemists revealing more details of the morphogenesis of capsids and other viral structures (e.g. for the Microviridae, see Doore and Fane 2016). The relatively late discovery of phages versus other organisms has, however, interesting implications. One of these may be that the diversity of phages has partly been revealed by culture-independent methods (via sequencing of environmental samples: Breitbart and Rohwer 2005; Dinsdale et al. 2008). For this volume we note how the study of phages has related to evolutionary biology. While early biochemical work on phages coincided with the development of the Modern Synthesis in the 1940s, it was only later that various phages became model organisms in experimental evolution research (Bull et al. 1997; Turner and Chao 1999; Messenger et al. 1999) reversing an early focus on animals (exceptis Dallinger 1878). The life cycles of phages were therefore first understood phenotypically (via one-step growth curves: Ellis and Delbrück 1939) and in terms of biochemistry and the emerging science of molecular biology (Clokie and Kropinski 2009). This chapter's author, Stephen Abedon, is a leading proponent and practitioner in the application of evolutionary theory to the life history traits of phages (Abedon 2008).

Abedon provides a description of diverse phage life cycles that emphasises parameters of ecological significance. For lytic phages, which form the bulk of the discussion, three key events in the life cycle are 1. attachment to a host cell, 2. the start of virion production within the cell, and 3. cell lysis with the release of progeny phages. Abedon labels the interval between 1 and 3 as the latent period, and divides

this into the eclipse (1–2) and post-eclipse (2–3) periods. Via qualitative models, and descriptions of more formal models, Abedon identifies a trade-off between the number of progeny produced at 3 and the duration of the post-eclipse period. In order to estimate the optimal length of the post-eclipse period, that is the duration that maximises population growth rate, the rest of the life cycle must be considered. Phages are trapped in the host cell during the latent period, so they are not able to search for new hosts to resume the cycle. This *opportunity cost* means that features of the system, such as the density of susceptible hosts or the vulnerability of free phage to inactivation, become relevant to this trade-off.

Much of what Abedon describes consists of the application of optimal foraging theory to phages qua predators/parasites. The way in which he partitions the life cycle is of interest. Changing the timings of the demarcation points identified above is expected to change the number of complete viruses that are produced in a time slice and Abedon acknowledges the role that whole organisms play in ecology (23.2.3). He is deploying abstractions, with molecular details, such as the timing of genome replication, omitted. For some models, assumptions are made about fixed features of the life cycle. For example, the eclipse period is assumed to be invariant, but as explained by Abedon this amounts to the claim that variation in this may not be possible without further side effects that bring the entire system further from optimality. In other areas, Abedon shows that details that may seem recondite are nonetheless significant. For example, he considers the exponential adsorption kinetics of phage (23.4.4) and notes that this causes predictions based on the mean adsorption to deviate from optimality: we must consider the lottery-like nature of phage attachment and the significance of the few lucky progeny that attach to a next host cell quickly. In another case, we are faced with a *brute* constraint imposed on latent period as a result of the passive lysis mechanism employed by phages in the Microviridae family (23.4.3) while most phage will actively achieve "lysis from within" via endolysins (Young et al. 2000). In the language of genetics, we can describe the post-eclipse period as subject to reaction norms that differ between genotypes. A general question that arises is how much utility a trade-off view has versus a constraint-based view (Goldhill and Turner 2014) especially considering the small genome sizes of many phages.

Another way in which Abedon makes use of abstractions is via the idea of effective burst size. While burst size describes the number of progeny phage produced by a single infection upon lysis (or bursting) of the host cell, the effective burst size reduces this number to retain only that fraction of progeny that are successful in going on to infect further host cells. The effective burst size therefore captures ecological details, such as the density of susceptible hosts, in a single parameter. This illustrates another way in which an abstraction may be deployed: in order to simplify the model building process.[1]

[1] For comparison note that a simple compartmental epidemiological model may contain a single transmissibility parameter that, for example, elides contact rate and behaviours that mitigate or increase risk.

Overall, we see that the omission of details is strategic and, if necessary, reversible.

An aim of this volume is to attend to phenomena of interest to advocates of the Extended Evolutionary Synthesis such as the details of development and the role of feedback between organisms and their environment. The inside-out history of phage biology described makes the effects of applying evolutionary models more transparent. We have seen that details of development may be neglected advisedly and according to the goals of explanation. This can also be revealed if we shift explanatory focus. For example, instead of parsing differences within lytic life cycles we may examine the different types of life cycle. A reasonable background assumption here may be that the different flavours of life cycle manifested by different phage species: lytic, lysogenic, and chronic-productive, represent approximately optimal solutions such that transitions between them are subject to heavy constraints (a complication here is the complexity of phage phylogeny), but what then should we pay attention to?

It is noteworthy that Abedon brings to bear the same verbal ("hard times", 9.3) model used to describe the duration of the latent period in lytic phages to the lysis/lysogeny switch in lysogenic life cycles. The latter is an epigenetic mechanism that includes a stochastic component, as well as a sensing of cell state, that allows some phages either to remain quiescent in the host (via integration of the viral genome into the host chromosome) or to switch to a lytic cycle.[2] What is apparent here is that the optimality view developed in the chapter helps us to understand this mechanism as an outcome of selection. To generalise, phages may provide an interesting model system for exploring the boundary between responding to environmental change through adaptation or via the evolution of adaptive physiological responses (adaptive tracking versus phenotypic plasticity: Botero et al. 2015).

In comparing life cycles, Abedon also makes use of analogies from life histories of the megafauna, characterising lytic and lysogenic phages as adopting a semelparous (or one-shot) reproductive strategy, while chronic-productive life cycles (exhibited by M13/fd phages) are iteroparous. This again shows the value of viewing mechanistic details in terms of their reproductive affordances and suggests that further work may be possible to characterise between-species differences.

A shift of focus suggested by niche constructionists is to consider feedback loops between organisms and their environments, also referred to as reciprocal causation (Oyama et al. 2001). From this chapter, it is apparent that this focus arises naturally from application of optimality models just as we saw in the example of dispersion given in Chap. 8. Abedon considers carefully the role of bacterial host cell density (including the role of inhomogeneous distributions such as microcolonies). The problem of phages depleting their own hosts is considered in this context, resulting in an expected departure from what would be optimal without this feedback, favouring longer latent periods and lower replicative rates. Abedon also considers competition between phages with different latent periods and we can see here an

[2] Described in loving and didactic detail by Ptashne (2004).

opportunity to look at this from an inclusive fitness perspective with different environments entailing different degrees of population viscosity (West et al. 2007). Usually the presence of competing phenotypes (that breed true) is an assumed feature of a particular model, but there would appear to be room for formal models that incorporate emergence of cheats by mutation as well as the dynamics of dispersal. In common with other mobile elements, phages are also significant in contributing to social traits (benefitting the host: Rankin et al. 2011), a trend expected to be modulated by *hard times* for virocells in Abedon's parlance. In general, the rapid generation times of phages invite questions regarding the time frames over which a particular life cycle may be optimal and the role of temporal fluctuations (Maslov and Sneppen 2015).

We began with an outline of developments in the study of phages, with the latter attracting the attention of researchers as putatively minimal systems, well-suited to the study of fundamental mechanisms such as heredity. Abedon likewise provides fertile ground by clarifying key concepts and describing common features. The power of this approach is that, as well as revealing potential adaptations, it also focusses attention on relevant features of the viral life cycle that serve as constraints on or modifiers of the identified dynamics. Many questions are opened and given focus by this approach.

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On *r-K* Selection in the Evolution of Bacteriophages: A Reply to Dickins

25

Stephen T. Abedon

Abstract

Bacteriophages, or simply phages, are viruses that infect only bacteria. Their life cycles consist of a dissemination or transmission phase between bacteria, the acquisition of new bacterial hosts via a process known as adsorption, an infection period, and then virion release from an infected bacterium, thereby re-initiating dissemination. The lengths of both dissemination and infection periods are impacted by genotypes which can vary between phages, thereby allowing for an evolution of their respective lengths. I consider in this reply to Dickins' commentary four areas which build upon considerations of evolution of bacteriophage latent period length, where a latent period is a phage infection period that ends in the lysis of the phage-infected bacterium. These considerations are, first and foremost, the concepts of *r* selection vs. *K* selection as relevant to the evolution of phage infection period lengths. I then touch upon related issues of molecular details (of phage infections), constraints (on phage infection-period evolution), phage adaptive physiological responses, and how the various ideas discussed might (or might not) be informed by what we know of megafauna evolutionary ecology along with the broader concept of cheating.

Keywords

Adsorption · Bacteria · Burst size · Burst sizes · Chronic · Comparative genomics · Cooperation · Defection · Hard times · Historical contingencies · Holin · Infection period length · Infection period lengths · Iteroparity · K selection · K-selected · K-type selection · Latent period · Latent period length · Latent periods · Life history ·

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Lotka-Volterra · Lysis-lysogeny decisions · Lysogenic · Lysogeny · Lytic cycle · Lytic cycles · Lytic infection · Lytic infections · Obligately lytic · Phage · Phages · Phenotype · Phenotypic plasticity · Phylogenetically · Population growth · Post-eclipse periods · Prisoner's dilemma · r selection · r-selected · r-type selection · Release · Semelparity · Temperate · Tradeoff hypothesis · Tradeoffs · Tragedy of the commons · Virulence · Viruses

The study of phage evolutionary biology can be distinguished between that which is especially phylogenetically based vs. that which is dominated more by considerations of phenotype. In modern times, the former approach is a product of nucleic acid sequencing, resulting, with phages, especially in comparative genomics. A standard approach toward phenotype-based evolutionary studies of phages can be described as experimental evolution. This involves defining conditions within which phages are propagated over many generations, within continuous cultures or via serial transfer (Bull 2008). A combination of mutation and selection, as well as genetic drift, results in various changes in what genotypes as well as phenotypes will come to dominate a phage population. Alternatively, one can start with existing genetic variants and compare these under some set of conditions. This consideration of how different phenotypes may fare within different environments, either in actuality or based on theory, is the study of evolutionary ecology. That, as applied to a specific-phage phenotypes such as duration of the phage infection period, is the emphasis of the chapter, "Evolution of Bacteriophage latent period length," with "latent period" just one of a few ways that phage infection period lengths can be distinguished.

A useful though somewhat overused perspective in comparative ecology is the concept of *r*-selected vs. *K*-selected organisms. Among the *r*-selected would be rapidly replicating organisms that produce large numbers of offspring and do so often despite longer-term negative consequences of the resulting population growth on that species' environment, and thereby on that species' future local population size (*r* referring to intrinsic rates of population growth). In contrast, *K*-selected organisms are by definition able to effectively maintain peak population sizes, once achieved, due to minimal local negative impacts by the species on the ability of environments to sustain their populations (*K* referring to a population's carrying capacity). The latter's peak population densities, however, generally are somewhat lower than what would be seen with an equivalent *r*-selected organism. This is a tradeoff between potentially environmentally destructive growth characteristics (*r* selected) vs. organisms that are somewhat less environmentally destructive but also less fecund (*K* selected).

In general terms, phages replicating using lytic cycles can be thought of as *r*-selected, meaning that it is likely that the primary component of the selection acting upon them, at least when their replication is possible, is for maximizing their population growth rates. Because of the nature of the phage lytic cycle, this

maximization of population growth rates seems to require a balancing of enhancing replication rates (shortening latent periods) and enhancing the numbers of offspring produced per phage-infected bacterium (increasing burst sizes). That it is not necessarily easy to achieve both simultaneously—shortening latent periods while increasing burst sizes, particularly for a phage which is already well adapted to infecting a given host under a given set of host physiological conditions—results in the tradeoff to which Dickins refers. Indeed, this is the basic premise of the tradeoff hypothesis, which the chapter extensively explores.

The chapter along with Dickins' commentary considers as well a number of complications on this tradeoff hypothesis. These are explored, in the chapter, primarily in terms of hypothetical competitions between otherwise identical phages that differ in their latent period lengths and thereby in their burst sizes. Though we have a firm theoretical understanding for why shorter phage latent periods may be evolutionarily successful based on the tradeoff hypothesis, [1] just how it is that specific phages have come to possess the latent periods that we observe is at best only poorly understood. In part, this is because whatever those phages were adapting to in the past is unknown to us, including what bacterial host strain (or strains) they were infecting as well as the environmental conditions under which this evolution would have occurred. Indeed, at this point all we really know for sure is that it is possible to select for shorter latent periods under the simplest of environmental conditions (well-mixed broth) while infecting high concentrations of laboratory strains of bacteria that the phages may or may not have encountered with any regularity in their evolutionary past. Thus, we have a simple system for which theory is to some degree predictive of experiment, though for which both experiment and theory nonetheless have been only minimally explored.

At least part of that exploration should, in my opinion, extend beyond the impact of phage latent period lengths, or more generally, phage infection period lengths, on *r*-type selection. Specifically, what about carrying capacity? Is that concept even applicable to phages which are able to display only lytic cycles, i.e., with their resulting obligate as well as relatively rapid destruction of their key resource, the host bacterium? That is, are strictly lytic phages unable to avoid standard Lotka–Volterra-type cycling of population sizes, at least so long as some degree of top-down rather than bottom-up control of phage population sizes (Bohannan and Lenski 1999) is possible? By contrast, one can imagine that these cycles could be damped via avoidance of lytic cycles altogether, with phage lysogenic cycles or chronic-productive infections thereby serving as possible phage answers to *K*-type selection.

Thus, phages which are stuck only with lytic cycles may be stuck as well with having to deal with current or future "Hard times" (23.3.1)—equivalent to dips during Lotka–Volterra cycling—perhaps by displaying somewhat longer latent period lengths by default even if longer latent periods otherwise would conflict

[1] Presumably this results in our perceiving that the observed latent periods in laboratory phages are relatively short.

with the utility of displaying shorter latent periods when times are not hard for these virions (23.4.2; the latter corresponding to Lotka–Volterra upswings). In contrast, some obligately lytic phages do not have to choose between displaying longer and shorter latent periods because they can display either, depending on circumstances (23.5.6.2). These two ideas—conflicts between the selective impacts of harder and not hard times vs. not having to choose a time-averaged optimum—are relevant to Dickins' consideration of "Adaptive tracking versus phenotypic plasticity."

In the alternative case of lysogeny, rapid production of virion progeny is forsaken, resulting in longer infection periods during which the bacterial resource is only minimally exploited (thus, "less environmentally destructive"). Display of such infections could be *for the sake of* sustaining phage population sizes nearer to carrying capacity than can be achieved via purely lytic infections, even given phenotypic plasticity in latent period length by the latter. Approximately the same utility could be the case for chronic-productive infections with population sizes sustained nearer to an environment's carrying capacity, though with seemingly greater levels of bacterial exploitation due to their ongoing production (and release) of virion progeny. These lysogenic or chronic-release infection types with their very extended infection periods equivalently may be described as better allowing these phages to avoid or at least minimize a tragedy of the commons (minimal impact on the common bacterial resource utilized by a phage population), which strictly lytic phages are somewhat less equipped to escape (Kerr et al. 2006, Abedon 2009). Chronic as well as lysogenic phage infections also arguably are examples of phage iteroparity vs. the much more weed-like semelparity of obligately lytic infections (23.2.6 and 23.5.5).

Presently we have ideas of how latent period evolution may respond to *r*-type selection, though this is only reasonably circumscribed for the simplest of ecological scenarios. Similarly, and under the same simple circumstances, we have an inkling as to why phages might find it useful to temporarily forego lytic cycles. What we lack, though, is a more nuanced perspective of how such things as shorter latent periods, lysogenic cycles, or chronic-productive bacterial infections might be beneficial particularly within the inhomogeneous environments in which phages presumably have done a majority of their evolving, and this is other than in terms of how lysogenic cycles in particular can contribute to the fitness of bacteria (Abedon 2022). Still, it is primarily the *r*-type selection scenario that most authors, including Dickins in his commentary and me in this chapter, have considered.

As follows, I address various additional ideas that Dickins brings to the table in his commentary.

25.1 Molecular Details

There actually is a large amount known of the molecular details of the timing of lysis. This has been studied—over the course of what coincidentally has been a majority of my scientific career—by Ry Young's group at Texas A&M. For example, see Cahill and Young (2019). A key take-home message, pointed out in the

chapter, is that especially latent period length often seems to be highly modifiable based upon seemingly minor mutational changes to individual genes, particularly what are known as phage holin genes. In other words, as the following paragraph considers, there appear to be relatively few historical contingencies regarding lytic phages mutationally modifying the length of especially their post-eclipse periods.

25.2 Constraints

Tradeoffs exist as a form of constraint on evolutionary change. These also can be described as antagonistic pleiotropies, and that is particularly so when those tradeoffs are associated with the alleles of individual genes. Another type of constraint can be described as the above-noted historical contingencies (Dickins uses the phrasing, "Heavy constraints," as equivalent). The difference is between whether the desirable aspect of the phenotype is easily achievable genetically (tradeoffs) or instead is not easily achievable genetically (historical contingencies). In terms of infection period length evolution, differences in latent period lengths seem to fall into the first category (constraints due to tradeoffs) whereas changes in lifestyle, particularly going from obligately lytic to either temperate (able to display lysogenic cycles) or chronically productive, or even display of inducibly longer latent or infection periods under certain conditions, seem to fall into the latter category (constraints due to historical contingencies). The chapter, though, deals entirely with antagonistic pleiotropies/tradeoffs rather than with historical contingencies. The existence of the different lifestyles in other words is taken as a given, with no consideration of constraints on how such lifestyles might genetically have evolved.

25.3 Adaptive Physiological Responses

This is certainly a currently active area of research, though in terms of lysis–lysogeny decisions that has been the case now for decades. Nonetheless, considerations of "Intercellular communication" (23.6.3) involving phages all represent adaptive physiological responses of one form or another. In addition is the variation in phage growth parameters such as latent period length as seen when phages infect bacteria differing in their physiological states (23.4.2 as well as 23.5.5.2 and 23.5.6.2). Though these various complications on the basic phage lifestyle all fall under the purview of phage evolutionary ecology, they are not nearly as simple either physiologically or conceptually as competitions between phages possessing different but nonetheless fixed latent period lengths within otherwise homogeneous environments.

25.4 Megafauna and Cheats

I'm a great believer in fitting phage life history characteristics into already well-established frameworks that have been developed for "macro-organisms." The trick, though, is to avoid achieving that fit by overly simplifying systems to a point where associations come closer to being fantastical rather than reality based. One such possible framework, though one which ultimately was cut from the chapter, is consideration of how infection-duration ecology or evolution can be restated in terms of the evolution of cooperation. This was alluded to above in terms of the tragedy of the commons. However, one can push those ideas further for phages, with either shorter or longer infection durations being either cooperation or defection behaviors depending upon circumstances. The trouble, though, is that these various ideas are difficult to fit succinctly or strictly into such popular constructs as the tragedy of the commons or the prisoner's dilemma. For discussion, as well as the related ideas of expediency vs. efficiency, see two chapters that I have published previously which to varying degrees emphasize those subjects (Abedon 2008, 2009). The allied discussion of phage virulence evolution (23.6.2), however, was retained in the chapter.

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Part IX

Plasticity and Information

26

Thomas E. Dickins

Abstract

In this chapter, I discuss developmental plasticity in relation to the concept of reaction norms. Standard treatments of reaction norms have been accused of gene-centrism by those seeking to extend the Modern Synthesis. I analyze this complaint with reference to uses and concepts of information. My central claim is that information concepts are both abstractions and idealizations, and as such have been designed for specific explanatory purposes. The purposes of evolutionary accounts at the population level demanded an instructional view of the information contained in the gene, but I argue that this was not how biologists ever thought of genes and in fact underpinning this view was a more detailed, cybernetic position that is entirely compatible with claims that emerged within developmental systems theory and that have been coopted by some in the Extended Evolutionary Synthesis. Finally, I use this analysis to briefly unravel some of the views allocated to West-Eberhard's magnum opus, *Developmental Plasticity and Evolution*.

Keywords

Plasticity · Development · Gene centrism · Developmental Systems Theory · Data · Context · Information

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26.1 Introduction

Ernst Mayr argued that a key innovation of Darwin was the introduction of population thinking to evolutionary theory (Mayr 1982, 1991). Prior to this evolution was understood in Lamarckian terms as a process of environmentally induced individual transformation that could be beneficial and inherited. It was fundamentally a developmental theory of evolution that saw cumulative individual change as the cause of phyletic change. Darwin saw evolution as the outcome of the natural selection of extant variation. The historical period of the Modern Synthesis after Darwin cemented the population level view and, with the onset of genetics, provided a theory of inheritance and a theory of populations as collections of genes (Dickins 2021).

Pigliucci has characterized the Modern Synthesis as a transition from a theoretical focus upon *form* to one upon *genes* (Pigliucci 2007). In making this point, Pigliucci is following Popper who claimed that evolutionary theory could not explain evolutionary novelties, the coming into being of something new, because it was strictly a theory of genes (Platnick and Rosen 1987). As Platnick and Rosen comment, to account for the transformation of form both developmental and epigenetic processes would be required. Pigliucci picks up this brief and argues that a mechanistic theory of form is required within the evolutionary theory of the Modern Synthesis, and he lists candidate phenomena that he hopes to see added to an Extended Evolutionary Synthesis. Among these he names phenotypic plasticity, the central topic of this chapter.

Recent advocacy for an extension of the Modern Synthesis has invoked what Love has termed developmental challenges to the population level, or gene-centric version of evolution (Pigliucci and Müller 2010; Love 2017). These challenges represent the project that both Popper and Pigliucci have raised. A crucial question, at this juncture, is whether this is a coherent or sensible project. One reason it might not be rests on another of Popper's contributions to philosophy of science, his discussion of definition in the context of a criticism of essentialism (Popper 1945). For Popper, definitions should be treated as scientific shorthand for coherent sets of phenomena. What this means is that definitions really should be read from right to left such that we choose to call the statistical outcome of trait variation, differential success, and inheritance *natural selection*, instead of saying "natural selection is *x*." We should not start our enquires by deciding that there is a phenomenon known as natural selection and then go looking for it to try and ascertain its fundamental properties. To do this is to commit to a form of methodological essentialism, to claim that natural selection is a thing with a fundamental essence that must be exposed. [1]

[1] Hull applied Popper's argument to typology (Hull 1965: 317): "The three essentialistic tenets of typology are (i) the ontological assertion that Forms exist, (2) the methodological assertion that the task of taxonomy as a science is to discern the essences of species, and (3) the logical assertion concerning definition." Mayr (1982) and Plotnick and Rosen (1987) also discuss the role of evolutionary theory in removing some vestiges of Aristotelian essentialism from biological science.

When biologists decided to term the fixation of traits in populations *evolution*, they were adopting a nominalist approach to definitions, in keeping with Popper's preference. Neo-Darwinism and the phase of restriction, that Gould (2002) emphasized, can be seen in this light as a decision by nominalist scientists to focus upon a set of phenomena and their dynamics. This meant that the processes of development, the ontogenetic emergence of form, were not added to the definition of evolution. Specifically, whilst this view of evolution relied upon trait variation, which is a matter of form, the causes of that variation are immaterial to the process captured by the term *evolution*. This does not imply that developmental processes cannot come under selection, and thus it does not imply that developmental mechanisms cannot be regarded as possible adaptations.

The emergence of form is an interesting and important scientific question, but it is not a question of evolution under the nominalist definition of evolution that emerged since Darwin. Given this it is possible that Pigliucci and all the other developmental challengers are not seeking to extend the Modern Synthesis, but rather to redefine *evolution*. Because the causes of transformation are important to these theorists, they understand the creative role of natural selection differently from those in the Modern Synthesis. The latter see selection as creating populations of a certain character, whilst the former see the creation of form during development as equally important. Nonetheless, those seeking a creative role for developmental processes also argue that these processes can provide source material for natural selection in a way that alters how we see the role of genes.

We shall now turn to discussion of phenotypic plasticity (§26.2) and its role in attempts to extend or redefine the Modern Synthesis. Following that, I will discuss the role of information in biology and introduce cybernetic information (§26.3) to clarify some of the muddle around gene-centric interpretations of the Modern Synthesis where many seeking extension have claimed that the Modern Synthesis positioned the gene as the instructional source of all. This view of information then leads to a discussion of levels of abstraction (§26.4) which helps to clarify the distinction between development and evolution, or rather helps to think about the distinction. I then conclude with some summary comments. The central conceit of what follows is that colloquial uses of the information concept have led to a very particular interpretation of the role of the gene in the Modern Synthesis that can make it appear as if it is all powerful, the total cause of all. My claim is that this was never the intention and that in the background a clear, modest view of information was always at work which places genes as necessary but not sufficient causes in development, but as essential for evolution. A proper engagement with information theory makes clear the separation of development and evolution, but also their complementarity, which is in opposition to the view that there is a similarity.

26.2 What Is Plasticity?

It was Johannsen who gave biology not only the term *gene* but also the distinction between *genotype* and *phenotype* (Johannsen 1911). In doing this he was replacing what he termed the (direct) transmission view of inheritance with the idea that what was inherited was the capacity to develop traits (Nicoglou 2018). The transmission view was the idea that direct, individual qualities were passed from parent to offspring. His view about a capacity, captured by the concept of the genotype, was influenced both by Weismann and his germline argument and Woltereck's views on reaction norms, where reaction norms are understood as the variation of the phenotype as a "continuous function of the environmental signal" (Stearns 1989: 436).

Whilst changes at the genotype level might be selected for this will take a long time and the effects will be felt further down the generational line. Wright was aware of this and argued that reaction norms enabled an uncoupling of the genotype from the phenotype, such that individual organisms could achieve a better fit with the environment (Stearns, 1989). In this claim we can see a focus upon form and transformation lingering within Wright's view of biology, but not necessarily of evolution. It was Dobzhansky who brought the reaction norm into the Modern Synthesis by arguing that the reaction norm was the focus of selection, leading to changes in relevant gene frequencies enabling particular developmental sensitivities and a range of response to become fixed in the population (Sarkar 1999). This view of reaction norms led to the more general concept of phenotypic plasticity which relies upon the genotype–phenotype distinction incorporating reaction norms and all kinds of "environmentally induced phenotypic variation" (Stearns 1989: 436).

In keeping with both Wright's and Dobzhansky's views, phenotypic plasticity has been characterized as a way of preserving the underlying genotype in the context of environmental change, a form of robustness solution (Meyers and Bull 2002) that can be partitioned into developmental plasticity and physiological plasticity. Whilst this interpretation suggests plasticity is an adaptation, Pigliucci and colleagues have cautioned against assuming all plastic responses are adapted as some may simply result from necessary developmental constraints (Pigliucci et al. 2006). Nettle and Bateson have provided clear guidance for determining adapted developmental plasticity (Nettle and Bateson 2015). Accordingly, we should expect developmental plasticity when cues in the current environment of the developing organism reliably predict future conditions, such that producing a relevant phenotypic response readies the organism for that future. To that end Nettle and Bateson note:

> (I)ndividuals who experience the developmental input and develop the phenotype must have higher expected fitness than those who experience the developmental input and do not develop the phenotype; whilst individuals who do not experience the developmental input and do not develop the phenotype must have higher expected fitness than those who do not experience the developmental input but do develop the phenotype. (Nettle and Bateson 2015: 4)

Physiological plasticity incorporates all day-to-day and moment-to-moment physiological responses to environmental inputs, and into this class we must incorporate

behavior as the product of neurophysiological mechanisms. For physiologically plastic responses to be adapted we would expect the same pattern of fitness effects as outlined by Nettle and Bateson, cashed out in terms of average lifetime inclusive fitness effects as not every behavioral token, for example, will provide a positive fitness gain.

26.2.1 Development and the Instructional Gene

Nettle and Bateson's (2015) view of plasticity is clearly a descendant of Dobzhansky's grounded as it is in inclusive fitness theory. [2] The heritable genotype has a causal role in the production of the phenotype, including in the production of an environmentally sensitive phenotype that can respond according to a conditional architecture. Thus phenotype *P1* will be produced under environment *E1*, and *P2* under *E2* such that $E1 \rightarrow P1$ and $\neg E1 \rightarrow \neg P1$, etc. (Dickins and Dickins 2008; Nettle and Bateson 2015). Such a view implies that there is a finite array of conditional responses, and that plastic response has parameters. A central question for many advocating for an extended evolutionary synthesis is just how much of a causal role should be ceded to the genotype: for example, are the parameters of plasticity encoded at the genotype level or the result of constraints imposed elsewhere? Thus, plasticity is regarded as a fact in want of an explanation. [3] It should be noted that there is nothing in the Modern Synthesis that specifically precludes extra-genetic constraints that have a causal role in the emergence of phenotypic response, but where that response is adapted then it must be genetically heritable.

A common complaint against the Modern Synthesis is that it privileges the gene with regard to form, as a consequence of its gene-centrism (for example (Jablonka and Lamb 2002; Brigandt 2016; Sultan 2019)). Pigliucci's (2007) argument is an example of this in that he seeks to reintroduce an account of form to evolutionary theory, understanding that the population concerns of the Modern Synthesis only had need of a mechanism for inheritance with the key properties of copying fidelity, fecundity, and longevity (Dawkins 1976; Williams 1996). Genes, understood in this way, are not sufficient to account for form and he looks to the proximate mechanisms of gene-regulation and development: other things must be added to the mix.

The developmental challenges to the Modern Synthesis, all of which are focused upon form, have their roots in developmental systems theory (Oyama et al. 2001). This framework emerged from a discontent with the pervasive dichotomy of nature versus nurture, a discontent that was first fully articulated as a criticism of Lorenz's concept of instinct in ethology (Lorenz 1950; Lehrman 1953; Johnston 2001).

[2] Indeed this view is commonly held among those adopting life history theory and working within evolutionary ecology traditions (Stearns 1989, 1992).

[3] This chapter will focus upon discussion of developmental plasticity, as this has been the core of discussion in recent debates. However, physiological plasticity is open to the same arguments. For example, we can see learning as the outcome of physiological processes and mechanisms that are themselves phenotypes with some relation to an underlying genotype.

Lehrman's view was that the concept of instinct was a preformationist one, and one not supported by the evidence from zoology that showed the emergence of form and behavior as the outcome of interaction between the organism and the environment. Where Lorenz laid claim to stereotyped behaviors, coming online during maturation, Lehrman saw reason to argue for the organism as a dynamical system whose development in one area may constrain the development of other aspects. For example, Lehrman felt that the pecking behavior of chicks may stabilize because of improved balance due to the development of the legs. This contrasted with Lorenz's view that pecking was instinctive and was simply revealed during development. For Lehrman, the use of instinct in ethology was a reification that ignored the subtlety of development, and in keeping with this he did not emphasize nurture either, but rather the complex interdependence of genes, learning and experience (Johnston 2001).

The developmental systems theory that has emerged from Oyama's work (Oyama 2000) claims that the Modern Synthesis embodied a preformationist theory of the gene. The gene is regarded as an instructional and representational unit that facilitates a developmental program leading to maturation in much the way that Lorenz claimed. Thus, Oyama's argument is that the Modern Synthetic view of development is gene-centric, placing all the causal efficacy within the gene. She, along with her colleagues, seeks to replace this with an interactionist view in which traits are built from developmental resources during ontogeny. These include non-genetic resources that are not inherited by transmission and nor is there any kind of representation of traits within those resources. Given that Oyama does not argue against the existence of a genotype this comes close to Johannsen's view of the inheritance of a capacity to construct a phenotype, but it is coupled with a view that biological systems are dynamical spaces with extra-genetic constraints that are of equal if not greater importance than the genes. Put simply, genes are one of many developmental resources involved in a developmental system with its own intrinsic properties, and these properties can explain the "transgenerational stability of form" (Griffiths and Gray 1994: 283).

We can see development systems theory at work in Sultan's recent discussion of the reaction norm (Sultan 2019). Her principal concern is that the Modern Synthesis has enabled a view of the reaction norm as determined by the genotype, that the norm of reaction is under genetic control, rather than seeing it as an outcome of interaction between developmental resources and the environment. She cites Chevin and colleagues as defining the reaction norm in terms of the phenotype expected of a genotype, as a function of the environment (Chevin et al. 2010). The intuition being primed by this citation is that the conditional architecture of the type $E1 \rightarrow P1$ is directly and deterministically coded for by the genotype, that there is no role for interaction, etc. This directly relates to one of two views about instructional information in biology, discussed by Griffiths and Gray (1994) in the context of developmental systems theory.

> The second, more practical way to make sense of the notion of information in development is to embed the information in one resource by holding the state of the other resources fixed as

> channel conditions under which that information is transmitted. But this move can be used to interpret any of the resources as the "seat" of the information guiding development, and so it, too, fails to generate the traditional asymmetry between genetic and other factors. (Griffiths and Gray 1994: 282–283)

As we can see Griffiths and Gray are suggesting that there is a problem with this view of information, as any privileging of one resource as the seat of information is entirely arbitrary. The suggestion is that a decision to privilege the gene would come from other considerations.

Some of the concerns of developmentally minded advocates of the Extended Evolutionary Synthesis can now be summarized. The Modern Synthesis has failed to account for the proximate emergence of form and has accounted for transgenerational stability of form in terms of genetic inheritance. In so doing the implication is that genes are instructional units that determine development, whilst bringing the properties of copying fidelity, fecundity, and longevity required for the Modern Synthetic view of evolution as population level changes in gene frequencies. This criticism has been made with reference to a particular view of information which is not without flaws. It is to information that we shall now turn.

26.3 The Role of Information

Central to the developmental criticism is the idea that the Modern Synthesis proposed a developmental program to account for the emergence of form. This idea variously arises in the literature as either a developmental program or a DNA program. This owes much to Mayr's discussion of DNA as containing programmed information, and his separation of jobs between those biologists interested in the mechanisms that decode this information during development and those focused upon the laws that change codes over generations (Mayr 1961: 1502). Mayr was clear that these DNA codes were open codes, and amenable to modification via learning, for example, permitting plasticity in the final phenotypic form. In other words, Mayr's DNA codes, whilst informational, were not wholly deterministic, but rather an antecedent condition in development.

Dawkins adopts Mayr's language when he tells us that the DNA program contains ancestral ideas about form and that this program is used to start development from scratch each and every time (Dawkins 1989: 261). The use of *ideas* clearly conveys an information concept. Dawkins sees genes as catalyzing development, and the genotype as conveying a capacity to build kinds of form, again in line with Johannsen's early speculations.

Both Mayr and Dawkins are laying claim to genes as among the sources of information during *development* and in so doing they are in keeping with the first of Griffiths and Gray's two views of information in biology:

> First, the entire set of developmental resources, plus its spatiotemporal structure, may be said to contain information about evolved developmental outcomes in the unproblematic, mathematical sense of systematic dependence. But as long as we confine ourselves to this notion

> of information, there is no causal asymmetry in the role of different resources which makes it legitimate to regard some of them as carrying the information and the others as merely providing conditions in which it can be read. (Griffiths and Gray 1994: 282)

However, if we focus upon the *evolutionary* commitment, both Mayr and Dawkins are arguing that DNA conveys information across generations to initiate development, and it is this information that is sifted and organized by natural selection. This appears to have more in common with the second view, a view of DNA as the seat of transmitted information. How that DNA is processed, the phenotypic variation arising from developmental process, might in turn appear to be merely noise where it is not regular and patterned in the way claimed for adapted developmental plasticity (Whitman and Agrawal 2009). Noise of this sort would be a property of the condition of the channels conveying information from the DNA to the next generation, and those channels are, of course, organisms. Whereas patterned plastic response is a property of the information contained within the genotype. Thus, Dawkins might be understood to be claiming that genes are catalysts in development, and thus a part of a developmental system, but genes are heritable catalysts that shift the equilibrium of a dynamic space in a direction selected for. When explaining evolution, it is the inheritance of those direction giving properties that is important; but when explaining development, the entire context of the system is relevant. These are methods of thinking to deliver specific epistemological goods.

The nominalist definition of evolution adopted by the authors of the Modern Synthesis was a population concept that saw natural selection as sifting genes, via selection over the phenotype. [4] Evolution was not a process of developmentally induced transformation at the individual level. But if one's ambition is to redefine evolution by incorporating a mechanistic theory of form, then it is easy to see how the two distinct interpretations of information, under two distinct task demands might be conflated into one project. Given this some scholars might come to assume that the Modern Synthesis regarded DNA as the seat of information and development as noise. That Mayr's meticulous response to those embryologists who claimed that evolution was a form of development, in which he clearly partitioned the phenomena, has been interpreted as his dismissal of development from evolutionary theory is indirect evidence for this conflation (Mayr 1984, 1992; Laland et al. 2011).

There is something more profoundly awry with these information-based accounts, and that is the concept of information itself. *Information*, as a concept, is in wide use within biology and it is most often used in a colloquial fashion (Maynard Smith 2000) and rarely scrutinized (Avery 2012; Dickins 2021).

Maynard Smith (2000) has defended the colloquial use of information. He notes that this usage is grounded in a communicative view of information, which is usually associated with Shannon's theory of communication (Shannon 1948) and he points out that this view is adopted as an analogy to facilitate explanation. However, a central point is that analogies work best when there is some level of isomorphism

[4] Mayr was very clear that selection was selection at the phenotypic level.

between systems, but more usually there is only qualitative similarity. The remainder of his paper discusses the degree of qualitative similarity between biological systems and communication systems. In doing this he establishes the idea that information is coded and decoded, transmitted and received along communication channels. Thus, Maynard Smith regards DNA as containing encoded information that is transmitted during protein synthesis and ultimately decoded in terms of a polypeptide chain. The information within the DNA is there because of natural selection, and this information gives the resulting protein a meaning, or a function (Chap. 14).

Shannon did not develop a theory of information, but rather a method quantifying the fidelity of transmission of a signal via a communication channel (Floridi 2010). His key insight was to understand the signal in terms of its components, and the available error as the possible permutations of those components. The more components there are, then the more possible errors, such that when the intended configuration arrives the outcome is more surprising, technically it has a higher surprisal value. Surprisal is the probability of a particular configuration arriving in a particular context. Communication channels can introduce noise, to be understood as random error in the signal. One way to deal with this is to introduce a certain amount of redundancy into the signal. Redundancy is the addition of more components to physically convey the signal than are needed to mathematically express the signal. For example, repetition of the signal is a form of redundancy that physically increases the components transmitted beyond the necessary mathematical limit for the whole message.

In communication we can regard the recipient of a signal as a system, and that system is in a state of uncertainty. Technically this means that the system can be in n states, and receipt of an unambiguous signal (i.e., with noise reduced) will determine which state the system is in. In this way, we can say that the signal, or input reduces the uncertainty of the system. The degree of uncertainty is a function of the number of states. From this we can draw an important lesson about what information is for Shannon. For him it was a functional relationship between an input and a system, such that the input will cause a state change from *S1* to *S2*. More formally put we can say that *information* = *data* + *context*, where data is the input and context is the system. If we apply this to Nettle and Bateson's (2015) account of adapted developmental plasticity we can immediately regard development as state changes in response to environmental inputs. In this way the developmental process is an informational one because of the functional relationship between the input (data) and the system (context).

This formulation is derived from Floridi's (2010) General Definition of Information in which he claims that *information* = *data* + *meaning*. Here meaning is the semantic context of a communication situation, and I take meaning to capture functionality. To this end, in biology, we are interested in systems that are designed to take specific inputs and respond in particular ways, as in the example of adapted

developmental plasticity. Functioning biological systems can be regarded as providing meaning if one prefers a semantic grounding for theory. [5]

Floridi (2010) expresses all this with a neat example. If we imagine a computer awaiting the outcome of a coin toss with a fair coin, there will be only two possible outcomes: <heads> or < tails>. Prior to the toss the computer is in a state of data uncertainty, but as soon as the toss has occurred, and the outcome is <tails> that data uncertainty is removed. The amount of uncertainty is measured as bits, a logarithmic function which in this case for two equiprobable outcomes is calculated as $\log_2 2 = 1$. Thus, the coin toss contains 1 bit of information, which we can understand as the number of questions required to resolve the data uncertainty: <did the coin land heads up?>, <no> provides complete certainty of the outcome. Thus, Shannon's quantification measured the reduction of uncertainty, and his theory is really one of data communication according to Floridi. By relating coin toss data directly to a query structure Floridi shows the relationship between data and context, in a semantic fashion. He argues that the query is semantic but can only be unlocked by the data, once unlocked the whole relationship is informational, or informative.

This view of information, [6] as a functional relationship between input and system, data and context, does not treat information as an object, as something to be transmitted. Colloquial uses of information reify it and treat information in an essentialist manner (Boisot and Canals 2004). Boisot and Canals drive this point home by noting the key distinction between data and information forced by encryption. We can access data from a data set, but it is of no value to us until we have the encryption key—only then does it play some semantic role for us, by which is meant it changes our uncertainty. For Boisot and Canals, data are discernible differences in the world, and I interpret this as *possibly* discernible where that possibility will be enacted by the emergence of a relevant system.

Maynard Smith's rendition of information in biology reifies it, and from my reading of the literature none of the authors of the Modern Synthesis have clarified their view of information. Instead, it has been a ready communicative concept to hand, put to work as an analogy on the assumption of a shared understanding. However, I think the cybernetic view of information outlined above is embedded within the Modern Synthetic perspective. Both Mayr's and Dawkins' view of DNA programs see DNA as an input at the beginning of a chain of developmental events, and indeed Maynard Smith does also when he describes the molecular transitions from DNA to RNA to protein. Each author understands that the initial input leads to a state change in a subsystem which in turns acts as an input for the next stage, etc. There is information here in the sense of a functional relation, but not in the sense of a core semantic truth being transmitted. The configuration of all the subsystems,

[5] The field of biosemiotics is taking on this semantic version of the task (Deacon 2017). And see Haig in this volume (Chap. 14).

[6] It is often referred to as a cybernetic or semiotic view (Avery 2012). I prefer the term cybernetic for its ready interpretation in conditional architectures of the sort used by Nettle and Bateson (2015).

which is a consequence of natural selection, delivers the meaning, understood in design terms.

The reified concept of information, as something out in the world to be grasped and transmitted is technically wrong, but it does allow fast work and the ready comprehension of various aspects of biology. In this way, it acts as an idealization and compresses much causal complexity in order to generate understanding (de Regt 2017). This is how idealizations work in science, and as Potochnik has noted idealizations often contain necessary untruths to do their work (Potochnik 2020). When the reified information idealization is deployed as Mayr and Dawkins did across both developmental and evolutionary considerations the readily available assumption is that the information bearing object, DNA, contains all that is needed for both development and evolutionary continuity, because DNA is the common factor in both accounts. To this end, it is easy to read such accounts of DNA as assuming DNA is both its own data and its own context. Without careful consideration the fact that DNA has only ever been modeled as a necessary but not sufficient condition in both domains will be missed.

Clearly, I am arguing that much of the gene-centric criticism, of the sort levelled by developmental systems theorists, has been a consequence of colloquial uses of information as an idealization of biological process. But I do not think the authors of the Modern Synthesis intended to sow confusion, as they consistently spoke under separate tasks demands, and indeed Mayr's (1961) clarification of proximate and ultimate causation provided a framework to clarify that. That this distinction has itself been criticized as non-interactionist in recent years is a reaction to the conflation I am highlighting (Laland et al. 2011; Dickins and Barton 2012). Nonetheless, genetic inputs are to be seen as not only necessary inputs to an overall developmental system, but as initial or early-stage inputs to that system. More precisely it is the replication dynamics and autocatalysis of DNA like molecules that initiated life and evolution, making use of available resource, and all subsequent innovations and their development have arisen from this (Deacon 2006; Root-Bernstein and Root-Bernstein 2015). This is the nature of the genetic necessity within ultimate accounts and that is qualitatively different from that of developmental processes.

The above points can be made more bluntly. When Mayr tells us that development is not evolution he is telling us that to produce an evolutionary account is to assume that development happens and that the detail of developmental process is not in and of itself relevant, it simply must happen. Evolution will continue just so long as genetic variation contributes to phenotypic variation and genes are heritable. He is not telling us that development is invisible to selection. This has been referred to as a method of black-boxing development and was a deliberate strategy of Mayr (1961) who adopted a distinction between explanation and prediction (Scriven 1959). Mayr makes a case for bracketing to deliver supervenient explanations, which will have greater utility due to the complex multidimensionality of reality. This is a case of idealization to handle complex causality and to draw out clear accounts about evolution, as population level change, without becoming bogged down in the minutiae of how traits develop, which will in fact differ hugely across cases. Indeed, there is reason to suppose development will never be amenable to a general

theoretical approach in the same way as evolution is (Brigandt 2016). Certainly developmental systems theory is less a theory and more a commitment to a negative thesis (anti-preformationism) and a holistic approach to explanation (Godfrey-Smith 2001).

An auxiliary support for my claim that the Modern Synthesis is in fact committed to a cybernetic view of information as a functional relationship between data and context is the use of statistical models. Mayr (1961) argued clearly that biological science was a probabilistic discipline and in so doing, along with his views on causation, was attempting both to unify biology and defend it from reduction to chemistry and then physics (Smocovitis 1996). Biology became increasingly statistical in its approach as a direct result of the innovations leading to population genetics and this extends to experimental work. In the previous section, I stated that Sultan (2019) cited Chevin and colleagues' view of reaction norms as the phenotype expected of a genotype as a *function* of the environment. This she took to be an example of the genotype as reified information, of genes as instructional units. But upon inspection, Chevin et al. lay out a clear statistical view of *function* that is grounded in the General Linear Model (GLM). To that end in reaction norm experiments the phenotype, Y, is predicted by an environmental exposure, x. In keeping with the GLM, $Y = f(x)$ where f is the function that packages the intercept and slope. [7] The assumption is that the environmental exposure is directed to the genotype, which is a constant, and the slope is a measure of plasticity as x is increased. Because the genotype is constant, Chevin et al. explicitly invoke non-genetic causes, such as physiological mechanisms, to account for the plasticity as well as the residual error. In this set up the relative roles of genetic and non-genetic components of the system can be assayed for their contribution to the phenotype. The genetic component is treated as an ancestral input that can produce a baseline phenotype (the intercept) which is modifiable by other mechanisms as environmental conditions change. This is a complex informational set up where information is the functional outcome of multiple *data* + *context* interactions. In this way, the Dobzhansky version of reaction norms conforms to the ambitions of developmental systems theory, effectively showing that the Modern Synthesis incorporated the concerns of Oyama and colleagues. This may well have been missed because Oyama's (2000) classic book, *The Ontogeny of Information*, whilst claiming that development is the process that creates information, at no point formally inspected the concept of information, but instead relied upon intuition and colloquial usage.

[7] A full treatment would add residual error to the equation.

26.4 Levels of Abstraction

The chapter has, to this point, inspected the concept of the instructional gene deployed by critics of the Modern Synthesis and found it to be based in a problematic view of information. I have attempted to show that the concept of the gene has been overinterpreted as instructional due to colloquial uses of information, and that a technically more correct and cybernetic view of information demonstrates a consilience between those who worked within the evolving framework of the Modern Synthesis and those interested in developmental systems. My argument was developed in the context of comments on information from Floridi (2010). In a broader philosophical treatment of information, Floridi introduces the concept of levels of abstraction (LoA), which is an epistemological, but avowedly not ontological method for gaining understanding (Floridi 2011). The following quotation will help to introduce the idea:

> An *agent* can be thought of . . . as a transition system (i.e. a system of states and transitions between them) that is *interactive* (i.e. responds to stimulus by change of state), *autonomous* (i.e. is able to change state without stimulus) and *adaptable* (i.e. is able to change the transition rules by which it changes state). However, each of those properties, and hence the definition of agenthood, makes sense only at a prescribed LoA. For example, whether a rock is deemed to be interactive depends on the length of time and level of detail of observation. Over a long period, it erodes and hence changes state. By day it absorbs solar radiation which it emits at night. But with observables resulting from scrutiny over a period of ten seconds by the naked eye from 10 metres, it can be deemed not to be interactive. (Floridi 2011: 60)

Floridi continues with similar examples, including machine learning software that can be seen as interactive, autonomous, and adaptable, and thus agent-like, but once its code is revealed we see that it is rule-following and not adaptive (in the strict sense of plastic). The LoA provides a definable perspective from which to make an interpretation. Put in informational terms we can see LoAs as contexts that will respond to specific data inputs. Floridi packages this in his query format again and argues that data are "answers waiting for the relevant questions" (2011: 77), and that once they enter a particular LoA they can be rendered informative. Or "alternatively: the relevant question is associated with the right answer at a given LoA" (2011:77). This brings Floridi to the following statement:

> Whether empirical or conceptual, data make possible only a certain range of information constructs at a given LoA for a particular purpose, and not all constructs are made possible equally easily. An analogy may help here. Suppose one has to build a shelter. The design and complexity of the shelter may vary, but there is a limited range of "realistic" possibilities, determined by the nature of the available resources and constraints (size, building materials, location, weather, physical and biological environment, working force, technical skills, purposes, security, time constraints, etc.). Not any shelter can be built. And the type of shelter that will be built more often will be the one that is more likely to take close-to-optimal advantage of the available resources and constraints, satisfying the given requirements and purposes. (Floridi 2011: 77–78)

Data are constraints and resources that enable information to be constructed within LoAs. This view pushes Floridi to a constructivist position on information, such that the world is not represented but designed by those systems that use data. In evolutionary terms this means that biological systems, as contexts for data, change states in response to that data and just so long as this is not detrimental to fitness (or more plainly put, the system remains alive and reproduces) they will have constructed a pragmatic response to the world but not a direct representation of it. Put in evolutionary terms it also means that biological systems can be seen as questions asked of the world, and those that are successful have found their appropriate data.

The idea of levels of abstraction (LoA) can be used to make sense of arguments about plasticity from those advocating for extension of the Modern Synthesis. Here is one use. Sultan's criticism of the reaction norm is that under the Modern Synthesis it was regarded as scripted—the genotype prescribed a phenotype, and that might be a one genotype to one phenotype mapping, or a one genotype to a finite set of phenotypes (Sultan 2019). And as I discussed above (§26.2), Sultan chose to analyze some key experiments by Chevin and colleagues as indicative of this belief (Chevin et al. 2010). Experiments rely upon specified LoAs to deliver description and explanation. Experiments are, by their very definition, queries in want of data, and their execution seeks the relevant data. My analysis of the experiments of Chevin et al. noted that they went onto hypothesize non-genetic causes of phenotypic variation. Those hypotheses were after the data collection. Given this Sultan would be within her *epistemological* rights to counter that the LoA used demanded environmental exposures to genes, and that the *exposure x genotype* was a form of data inputted into the experimental LoA query, and that this LoA is often sufficient for standard evolutionary biologists to claim they have understood the reaction norm. Sultan would not need to comment on the subsequent hypotheses about non-genetic causes of phenotype variation (the mediating causes of physiological mechanism, etc.) because at that point Chevin et al. were operating a new LoA. Sultan could claim that the initial, experimental LoA is the level at which the Modern Synthesis stopped.

From Floridi's perspective, a tight LoA that only focuses upon genetic data is not a problem just so long as it is formally neat and tidy. [8] It is a way of constructing a view of the world that has some leverage, and as a scientific strategy there is no commitment to this mapping the real world in some precisely veridical manner. Similarly, the LoA that seeks to incorporate physiology can work, but in working it does not negate the simpler gene only LoA. But of course, scientists hope to tell the truth about the world, and make discovery that closely approximates reality. This suggests that perhaps Sultan, and her many colleagues, feel they are producing a theory that is closer to the truth of nature, which has a finer level of granularity, that is less abstracted. Pigliucci's view that evolutionary theory requires a mechanistic

[8] Floridi (2011: 46–58) provides a set of formal criteria for LoAs and their components that I will not rehearse here as they do not impact on the broader point of this chapter.

account of form is perhaps another instance of this belief. But in doing this, Sultan, Pigliucci, and others are changing the LoA, shifting the questions and in turn demanding different data. The act of doing this is not in itself a refutation of a previous view, nor a method for determining the formal adequacy of an LoA.

Floridi discusses the idea of a gradient of abstraction (GoA) which is "a formalism defined to facilitate discussion of discrete systems over a range of LoAs. While an LoA formalizes the scope or granularity of a single model, a GoA provides a way of varying the LoA in order to make observations at differing levels of abstraction" (Floridi 2011: 54). This might seem like a hopeful route for those seeking extension of the Modern Synthesis. Perhaps there is some formalism that will allow the LoAs of the Modern Synthesis to relate to those of developmental biology in a newly unified science? Floridi later discusses Marr's three levels of analysis for cognitive devices and Dennett's three stances—each can be interpreted in LoA terms as explanations for complex systems, with each level independently useful but related by GoAs. For example, Dennett's stances are designed to capture accounts of organism functionality (Dennett 1987). Thus, one can discuss the systematic behavior of an organism in terms of hypothesized intentions, at one LoA, but then understand that systematicity in terms of design principles at the next LoA down. Beneath this is a physicalist LoA that gives the biomechanics of the situation. In the absence of data to fill this last LoA one can fruitfully operate at the other two levels to generate explanation with different kinds of data. The GoA is provided by tacit ontological commitments in each theory about how brains work as material systems, and the hierarchical relationship between each level within the system.

There is much to say about the GoA approach, but for the purposes of this chapter there is only one thing. The focal systems of development and evolution are different LoAs; they are the individual and the population. Dennett's model assumes that causality at the physical level can be broadly captured by causality at both the design and intentional levels. This does not necessarily mean a precise reductionism is assumed, but merely some kind of real relationship is place. So, even if the intentional stance is a form of idealization, it still captures real causality happening at the physical level. This is not the case when we contrast development with evolution—neither is an idealization of the other, and each has different causality that is not a translation or abstraction of the other. Evolution and development are quite simply different systems.

This has not prevented people from trying to think in GoA terms. West-Eberhard sees development as a source of variation, over which selection can operate:

> The dichotomy between selection and development, as if they are opposed factors in adaptive evolution, is misconceived. Adaptive evolution is a two-step process: first the generation of variation by development, then the screening of that variation by selection. (West-Eberhard 2003: 139)

She does not deny the dichotomy merely their opposition, and so she sees them as complementary to some extent. Developmental variation and natural selection are two aspects of the single process she calls adaptive evolution. I suspect she uses the

term *adaptive*, to force the plasticity intuition across developmental and evolutionary processes. All is about plasticity—within individuals and within populations. But this does not make West-Eberhard an unorthodox evolutionary theorist, and nor does it situate her as an advocate for the extended synthesis. She notes that:

> (I)f the phenotypic variation that causes selection has a genetic component, this causes evolution. . . Selection depends upon phenotypic variation and environmental contingencies only; it does not require genetic variation. But genetic variation is required for selection to have a cross-generational effect – an effect on evolution. From these causal relations, it is clear that development, not selection, is the first-order cause of design. Selection is a second-order cause that molds the distributions of traits in a population by screening the products of development and determining which ones persist and multiply across generations. (West-Eberhard 2003: 141)

West-Eberhard is giving development a creative role in design, and selection an organizing role: this is entirely in keeping with the argument given that questions of form are not questions of evolution. [9] In other papers (West-Eberhard 2007, 2008, 2009, 2019) she is most clear—the variation induced during development is a property of the underlying genotype but it does not rely upon mutation in the genotype. Evolution can happen due to available variation, such that the extant genetic variants that allow particular and more useful responses are retained. The genotype is clearly a product of the selection of novel mutations, historically, but selection can reveal more utility in that genotype under certain environmental conditions. Put more baldly, selection will remove certain extremes in extant reaction norms. This is a position straight from Dobzhansky's reformulation of the reaction norm. As West-Eberhard herself says, her view is not anti-Darwinian and nor is it Lamarckian.

Despite West-Eberhard's statements and clear genetic orthodoxy about evolution, her mantra that the phenotype leads in evolution has been taken to imply that developmental *processes* cause evolution. This can take the form of orthogenesis, in which development guides evolution along paths such that the trait is regarded as emerging before its genes (Laland et al. 2014). The idea that organisms' interactions with the world can cause latent development switches to be flipped has been attributed to West-Eberhard as an argument against standard evolutionary theory (Buskell 2019) because of a focus upon final form rather than upon West-Eberhard's distribution of those traits. And so on. All these arguments claim that developmental proximate processes can reveal trait variation and that this variation is different from genetic variation. This is trivially true—variants of genes are not variants of traits. But the argument is really about what has already been discussed, the causal role of genetic variation in trait variation. Advocates of the extended synthesis assume that the Modern Synthesis saw this as a one-to-one mapping in two levels of abstraction—those pertaining to evolution and those to development—because

[9] And it is also in keeping with Fisher's view that natural selection was not evolution (Dickins 2021).

these advocates think that evolution and development are about the same thing, the creation of form within the individual. In fact, the authors of the Modern Synthesis were well aware that developmental processes intervened between the gene and the phenotype, and as we have seen Dawkins who was gene-centric about evolution was catalytic in his view of genes, seeing them as development input. Indeed, it was the ability of core, catalytic data to be preserved across generations and then fed into developmental situations (governed by all sort of resource issues, as Oyama has told us) that enabled evolution to occur.

There is nothing in West-Eberhard's detailed view of plasticity that implies a GoA that can be applied across developmental and evolutionary LoAs. Instead, there is a clear view of interaction between individual variation and evolution via selection. That is not a gradient of abstraction but a complementarity, as West-Eberhard clearly states. Such complementarity can only be achieved by logically separable systems (Dickins and Barton 2012) and complementarity is not hierarchical.

26.5 Conclusion

The kind of plastic responses that we have discussed, and that feature in criticisms of the Modern Synthesis, may be revealed in some surprising way after a contextual (environmental) change, but they are of interest because the response is seen to be regular under that context and not chaotic. There is a pattern that can be described in conditional (and probabilistic) terms. The new response most certainly adds to the available account of a phenotype, and thus increases the number of possible variants of the phenotype within the population. To that end, we can talk about phenotypic variation at the population level. Within the individual we can discuss how the response is physiologically and mechanically mediated once the input has been achieved. And we can discuss how those individuals that so respond might find themselves distributed in the world, and how that response is distributed in the world.

The non-chaotic, systematic nature of plastic responses suggests that in fact there is a deep history of selection, but it only suggests it as a hypothesis. For it to be adapted it must conform to the strictures articulated by Nettle and Bateson (2015) about fitness maximizing. A currently non-adapted (i.e., not selected for) response that emerges as a new response to a change in context may fit those strictures due to that change and then selection will occur if that response is due to genetic variation. If it is not due to new genetic variation and is simply the outcome of developmental constraints that have changed because of the environmental change (so a shift in equilibrium in the underlying dynamic systems of growth) and causes no change in fitness it is of no relevance to evolution. All instances of regular response can be accounted for in cybernetic informational terms.

The use of cybernetic information makes clear the level of abstraction at play in any given account. In some cases, levels can be related, translationally, via a gradient, but those gradients make ontological assumptions. The Modern Synthesis made clear that development and evolution were different levels of abstraction, but

also that they were separate systems. Separate systems can interact, but that interaction does not imply a gradient between them but instead a definitive difference. This is straightforwardly because evolution is about populations, whereas development is about the individuals that belong to populations. As West-Eberhard has made clear, where individual development can lead to new variation, it is selection of variation that leads to the fixation of traits within a population. All the attempts to shoehorn plasticity, and other developmental processes, into evolution have amounted to attempts to redefine evolution and thus the level of abstraction. But all those attempts, in describing the phenomena at hand, have persistently relied upon standard levels of abstraction to explain the processes of plasticity and then subsequent selection. There is, so far, nothing new under the sun.

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Phenotypic Plasticity and Evolutionary Syntheses: A Commentary on Dickins, T.E.

27

Douglas J. Futuyma

Abstract

It has been suggestd that the evolutionary synthesis (ES) was mostly a thory of gene frequencies, neglecting developmental biology. I argue that the ES was far broader in scope, and has since been greatly extended. Among developmental phenomena, phenotypic plasticity has long been studied and its importance is widely appreciated.

Keywords

Evolutionary synthesis · Phenotypic plasticity

Massimo Pigliucci's (2007) essay "Do we need an extended evolutionary synthesis?" was among the early stirrings of the EES movement. Following Dickins, I will take it as a point of departure, although into less philosophical regions. Phenotypic plasticity is one of five topics that Pigliucci suggested would be necessary for an extended synthesis. (Among the others, evolvability and epigenetic inheritance have developed a significant literature; evolution on adaptive landscapes has received significant but slight attention, while complexity theory has not.)

As in much subsequent EES literature, Pigliucci's portrayal of the Evolutionary Synthesis (ES) differs considerably from the ES I think I know and that I have written about. Pigliucci describes "the classic textbook definition of evolution as a change in gene frequencies (Futuyma 1998)." That is not quite accurate, as the definition of evolution in that text is: "change in the properties of populations of

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organisms, or groups of populations, over the course of generations... Biological evolution ...embraces everything from slight changes in the proportions of different forms of a gene within a population...to the alterations that led from the earliest organisms to dinosaurs, bees, snapdragons, and humans" (Futuyma 1998, p. 4). In the most recent iteration of that book, biological evolution is defined in the text as "inherited change in the properties of groups of organisms over the course of generations" (Futuyma and Kirkpatrick 2017). (A considerably longer entry in the glossary notes that changes may occur by frequency changes within populations, among populations, or among species.)

In consciously avoiding the gene frequency definition of evolution, I adhered to my reading of the ES. The development of population genetic concepts and models was indispensable, but a concern that united the geneticists (Fisher, Wright, Haldane, Dobzhansky) and the natural historians (Mayr, Simpson, Rensch, Stebbins, and again Dobzhansky) was to affirm Darwin's central ideas of natural selection and gradualism. Mayr and Dobzhansky, moreover, aimed to elucidate the nature of species and speciation, Simpson to explain macroevolutionary patterns in the fossil record, and Rensch to explain patterns in comparative morphology. Rensch (1947, 1959) drew extensively on the descriptive developmental biology of his day. The ES was more than population genetics, as Mayr (1959) famously argued when he criticized "beanbag genetics" as an inadequate description of inheritance and evolution.

Nonetheless, the *core* theory of the ES concerned the elementary generation by generation process of the origin of inherited variation (all agree that noninherited change in individual organisms is not evolution) and its sorting by selection and genetic drift. This theory was wonderfully general, as it could apply to any character of any species, but it also lacks any account of specific classes of traits or sources of selection. Among proponents of an EES, the complaint is often expressed (by Pigliucci, among others) that the ES does not address the evolution of morphological form. Quite so, but neither does it address physiology, metabolic pathways, behavior, life history, or genomic properties such as chromosome number. Evolutionary models and understanding of these topics developed well after the ES, as biologists began to articulate questions and, in some areas, as technology was developed. Even evolutionary genetics made little empirical progress until Lewontin and Hubby (1966) used protein electrophoresis to show that diverse studies of allele frequencies were possible. Experimental embryology and comparative developmental studies revealed some of the developmental underpinnings of morphological evolution (see, for example, Futuyma 1986, 1998), but evolutionary developmental biology (EDB) could burst into flower only when, in the 1990s, molecular developmental genetics enabled study of gene expression and genetic networks. These approaches have led to substantial advance in understanding of morphological evolution, such as the origin of novel features (Wagner 2015; DiFrisco et al. 2020).

In EDB, the depth of evolutionary understanding is greatly enhanced by knowledge of biological mechanisms, such as the nature of gene regulation and transduction of environmental signals. Such information may not be part of evolutionary theory, but the theory—or at least its application to real organisms—is richer for

it. The same holds for other areas of evolutionary inquiry. In behavioral ecology, for example, predictions about social and other behaviors may be based on optimality theory, but in order to understand how crows and parrots can be apparently more intelligent than other birds, we would probably require information on specific brain functions. The distinction between the biology that is part of evolutionary theory and the biology that is not may be hard to make.

Phenotypic plasticity is a developmental phenomenon that has long been recognized and extensively studied (West-Eberhard 2003; Pfennig 2021a; Futuyma 2021). All organisms are phenotypically plastic in some respects, which can include not only plasticity of form, but also of biochemical function, physiology, and behavior, owing in part to changes in gene expression (Pfennig 2021b). Not all plastic changes are adaptive, but many are, and have been the focus of a large literature that includes questions about the costs and limits of plasticity. Pfennig (2021b) and Schlichting (2021), among others, have articulated important questions about plasticity. For example, Pfennig (2021b) poses five "key questions": does it provide a unique evolutionary advantage? What are its proximate mechanisms? When should it evolve? Can it influence evolution? Does it fit into existing evolutionary theory?

Both Pfennig and I (Futuyma 2021) answer the last question affirmatively. The only major claim about plasticity that might be controversial is the proposal, most extensively argued by West-Eberhard (2003), that initially plastic reactions to an environment may later become genetically accommodated, rather as Waddington suggested in proposing genetic assimilation. But, as Simpson (1953) affirmed in discussing the Baldwin effect (as he understood it), the process of genetic assimilation is entirely compatible with population genetic theory. The only question is whether or not this process has commonly occurred. Pigliucci (2010) rightly noted that "the difficult task. . .is to uncover convincing examples of the transition from phenotypic to genetic accommodation" and to show that it is common. A considerable number of demonstrated cases of "plasticity-led evolution," both in the laboratory and nature, now seems to show that it is not rare (Levis and Pfennig 2021; Scheiner and Levis 2021; Schlichting 2021).

How, then, do the perspectives on phenotypic plasticity differ between partisans of the ES and of an EES? My impression is that the difference is more rhetorical than real. Sultan (2021, p. 6) refers to "the simplified view of phenotypic causation that dominated both developmental and evolutionary biology," a "'gene-for-trait' model" to which "plasticity was considered an odd exception. . ." (See also Sultan 2017). But in the following pages, she notes that experimental studies of genotypic reaction norms go back more than 75 years, and she reviews a rich literature on genotype X environment interaction in quantitative and evolutionary genetics, much of it from the 1980s and before. Of course, the study of phenotypic plasticity, like development more broadly, has been greatly advanced by methods of assessing gene action and interaction, which provide deeper understanding and some surprises, such as epigenetic inheritance, that will reward study by evolutionary biologists. Schlichting (2021) describes "areas of evolutionary biology on which the SET [standard evolutionary theory] is silent," citing "the evolution of novelties; biases

in mutation or development; evolvability; evolution of the genotype to phenotype map, and the unfolding of development" (p. 382). Quite true—but are these evidence of an self-satisfied, reactionary stand, or evidence of normal growth of a science, based on new technology, new evidence, and new ideas? New theory and evidence on the evolution of social behaviors, or of sex and recombination, have greatly enlarged and enriched evolutionary biology in the last few decades, but were not portrayed as a challenge to a fixed and outworn theory. Discoveries in genomics, such as the astonishingly high fraction of noncoding DNA, were disconcerting at first, but soon enhanced our appreciation of different levels of selection.

So it is, I believe, with phenotypic plasticity. I cannot see that it demands greater revision of evolutionary thought now than it did 50 years ago. But like other topics in evolutionary developmental biology, phenotypic plasticity is important and its study has broadened our view of morphological evolution. It is indispensable in any view of the evolution of physiological and behavioral characteristics. It poses incompletely answered questions, the prospect of rewarding research, and greater understanding of organisms and their evolution.

> We must cultivate our garden.
> Voltaire, *Candide*

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On Rhetoric and Conceptual Frames: A Reply to Futuyma

28

Thomas E. Dickins

Abstract

In this reply to Futuyma I make clear that an interplay between rhetorical claims to error in the Modern Synthesis and colloquial views of information are principal factors structuring recent debates.

Keywords

Rhetoric · Information · Folk-theory

I am grateful for Futuyma's commentary on my chapter. He is right that different versions of the history of evolutionary biology are traded against one another in the to-ing and fro-ing between advocates of the Extended Evolutionary Synthesis and those who dissent. This is perhaps done for rhetorical purpose, but it certainly misses the subtlety of theory, and the complexity of its generation (Chap. 2). Futuyma is also right to point to the generality of theory emerging during the Evolutionary Synthesis, a generality that has enabled more recent developments in biology to be incorporated.

Rhetorical statements are designed for their effect. They allude to a meaning. When I ask my children <would you like to tidy up your toys?> my purpose is rhetorical, I am instructing them to put their things away.[1] Claims about the exclusion of development from evolutionary theory may appear rhetorical because

[1] This does not always lead to the toys being put away by my children.

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they seek to establish an error: they instruct us that an error has been made. Establishing an error then licenses a set of remedies from different sources.

A central element of my chapter was the claim that *information* is a concept that can be interpreted in several ways. Specifically, I used Maynard Smith's analysis of the colloquial uses of information in biology to ground a more formal, or analytic inspection of the term (Maynard Smith 2000). A key tension is between an instructional view of information (contained in the gene) and a cybernetic view, that I developed from Floridi's work (Floridi 2011). My claim was that the cybernetic interpretation makes better sense of the gene's-eye view biology developed in the mid-twentieth century. To that end, genes are to be seen as data that have systematic effects within specific contexts. The clear commitment from the gene's-eye view is that genetic data are necessary but not sufficient for producing an organism, and that genetic data have causal primacy in a long chain of *developmental events*. The key properties of longevity, fecundity, and copying fidelity attributed to genes in turn enable *evolutionary events*.

When thinking about genes in evolutionary contexts it is standard practice to assume development, to idealize by removing the causal details of development, and to assume selection on an endpoint phenotype. This can be seen as an epistemological outcome of Mayr's distinction between proximate and ultimate causation (Mayr 1961; Brown 2022). But Mayr's separation of types of causation was in fact an ontological statement. He did not rule out reaction norms, plasticity, nor even the concepts of genetic accommodation and assimilation. Nor did he make a simplistic commitment to all phenotypic variation being caused by genetic variation. Where he wrote about genetic programs being decoded by proximate processes, he was asserting that those programs were the outcome of selection, but none the less fed into a developmental context. Development may influence trait variation, as West-Eberhard noted (West-Eberhard 2003), and affect selection dynamics but evolution occurs only when the heritable genes are favored as a result of selection.

Information is a pervasive concept, and it is unlikely that scholars will come to evolutionary theory without at least a folk-theory of it. Those holding an instructional view, which is the more common colloquial usage, may more readily interpret the idealizations of optimality modeling, for example, as evidence of an instructional view of the gene. Genes, once interpreted in this way, will be seen as fully instructional in *developmental* and *evolutionary* events. It is only a short step from this to interpreting much of the history of evolutionary biology as an attempt to deliberately exclude development. Put more simply, if your belief is that information is instructional, then the idealization of development will not be understood as an idealization, but rather as neglect.

All of this can be summarized in a simple way. Development *was* excluded from some evolutionary explanations, where appropriate, to make the causal story easier to understand and use. A clear example of this is in adaptationist accounts of design, or of the purpose of phenotypic traits (Chap. 20). This exclusion was permitted due to the ontological distinction between development and evolution, that Mayr made clear, but also due to some less well articulated views about information and its relation to the concept of the gene. The lack of clarity around information concepts

has, I think, allowed strong historical claims to be made in good faith that nonetheless appear rhetorical to those who are clear about the cybernetic view of information at the center of modern evolutionary biology.

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Part X

The Curious Incident of the Wasp in the Fig Fruit: Sex Allocation and the Extended Evolutionary Synthesis

29

David M. Shuker

Abstract

How would we tell if we needed a new evolutionary synthesis? The rationale for the so-called Extended Evolutionary Synthesis (EES) is predicated on there being limitations, failings, or something missing from the current body of theory that evolutionary biologists use when seeking evolutionary explanations for the patterns of biodiversity we see around us. A number of topics have allegedly been neglected or obscured by evolutionary biologists, including the role of development in evolution ("evo-devo"), phenotypic plasticity, niche construction, behaviour, epigenetics, and trans-generational effects. These disparate topics more or less coalesce around two organising principles of EES thinking, that of organismal agency and non-genetic inheritance. In this chapter, I use the field of sex allocation to test the validity of the arguments that these topics have indeed been neglected. Sex allocation is a useful exemplar of evolutionary biology. Thanks to Fisher and Hamilton, it has a historically rich and well-understood theory base. Moreover, across more than five decades, there have been hundreds of empirical tests of components of that theory, across a huge diversity of organisms, such that sex allocation is one of the most successful and well-validated fields within evolutionary biology. If claims of the EES have credence, then the study of sex allocation should clearly highlight what we have missed or ignored. However, I show that all of the components put forward by proponents of the EES as needing to be added into evolutionary biology—with perhaps the exception of cultural evolution, as least outside of humans—have long been studied, implicitly and explicitly by those studying sex allocation. In many cases, the relevant concepts are there at the inception of the modern study of

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sex allocation, following Hamilton's ground-breaking paper in 1967. Having dispensed with the need for the EES, I finish by trying to understand why such a synthesis was ever called for in the first place.

Keywords

Evolution · Extended evolutionary synthesis · EES · Natural selection · Sex allocation · Sex ratios

29.1 Introduction

It is a truth, universally acknowledged, that an evolutionary biologist in possession of a new theory must be in want of a new evolutionary synthesis. Or at least, given the attempts to re-write evolutionary biology in recent years, it certainly sometimes feels like it (Welch 2017). Research fields and concepts such as evo-devo, niche construction, extended heredity, soft inheritance, epigenetics, phenotypic plasticity, and cultural evolution have been developed in the light of, or requisitioned in the hope of, providing a new over-arching framework for evolutionary biology, generally termed the Extended Evolutionary Synthesis (EES; Pigliucci 2007; Laland et al. 2014, 2015; Müller 2017; Uller and Laland 2019). Yet the proponents of the EES have so far failed to convince many evolutionary biologists that a major conceptual readjustment is necessary (Wray et al. 2014; Charlesworth et al. 2017; Futuyma 2017; Svensson 2020; other chapters in this volume). What is going on? Such a disagreement conveniently fits the narrative of an entrenched orthodoxy, gamely resisting attempts to undermine its intellectual integrity in the face of overwhelming and conflicting data, fighting off the oncoming paradigm shift for all it is worth. Moreover, as mortality has been suggested to be a primary driver of intellectual change (see Azoulay et al. 2019, following the famous quote by Max Planck), perhaps not enough ageing population geneticists are dead yet (see Charlesworth 2013 for a theoretical treatment of why they are not). Alternatively, perhaps the EES does not provide the novelty or newness needed for it to be a game-changing new paradigm after all?

As with any such emerging body of thought, however loosely defined, the EES no doubt means different things to different people. First, it is important to remember though that evolutionary biology—or any aspect of biology for that matter—has not stood still since the period, spanning the 1930s to the 1950s, when what we now term the "Modern Synthesis" (MS) emerged from the combining of Darwinian thinking and population genetics. The MS linked Mendelian genetics (however poorly understood and conceptualised "genes" themselves were during that period) with natural selection, adaptation, and ecological genetics at the population level, through to speciation, and patterns of macroevolution under the purview of palaeontologists (for a summary, see Mayr and Provine 1980). Importantly, the MS was not one "thing", but rather an emerging consensus, across sub-disciplines, that evolutionary change was fundamentally a population genetic process, even if one studied whole

organisms or fossils, and went nowhere near a molecular laboratory to look at proteins, or eventually DNA. Behavioural ecology is a good example of a field that emerged and flourished in the years following the MS, using evolutionary principles—or at least natural selection—to explain the origin and maintenance of behaviour, despite being rather agnostic about genetic details (Krebs and Davies 1978; Grafen 1984; Davies et al. 2012).

This lack of standing still has meant that any evolutionary textbook published since the 1930s–50s is necessarily "extended" in its treatment of phenomena within the scope of evolutionary biology (a point often made, e.g., Wray et al. 2014; see also Rose and Oakley 2007 for examples of how simpler thinking, especially about genes and selection, during the MS has been superseded). This is most obviously true if we think of the ground-breaking work of Bill Hamilton for instance, such as his conceptualisation of inclusive fitness (Hamilton 1964; for his full contribution, see Hamilton 1997, 2001, 2005; Segerstrale 2013; Gardner, this volume). Or if instead we consider the realisation of the importance of neutral genetic variation, and thus genetic drift, in evolution, especially in terms of understanding evolution at the molecular level (Kimura 1983; the debates over the importance of drift versus adaptive evolution, for instance, at the genomic level, continue, but no-one doubts the ubiquity of genetic drift). Inclusive fitness and the neutral theory of evolution have provided two enormous theoretical contributions to evolutionary biology, extending its scope dramatically, including the range of phenomena we can explain. However, both were developed as part of, and remain situated within, the framework inherited from the MS and did not require a paradigm shift from the view of evolution as a population genetic process (see also Futuyma, this volume).

Likewise, little has stood still empirically either, although perhaps many of our most important findings have been to confirm, from phenotype to genotype, the action of evolution (from bacteria in the lab, to finches in the wild: Bell 2007; Grant and Grant 2010), and the genetic underpinnings of that evolution (Charlesworth and Charlesworth 2010). That said, many empirical findings have also shaped and extended our conceptual space, from the discovery of the near ubiquity of mate choice and post-copulatory sexual selection (Andersson 1994; Parker 1970; Eberhard 1996; Simmons 2001), to the murky world of genomic conflict, with its transposable elements, endosymbionts, driving chromosomes, and truly selfish genes (Burt and Trivers 2006). Therefore, many if not all evolutionary biologists surely agree on some form of "Extended Modern Synthesis", in which the foundational tenet of *evolution as genetic change* is used both to interpret and inform our observations, be they of fossils, feathers, or foraminifera. For the rest of the chapter, I will view evolution as genetic change occurring within populations across generations and call the population genetic view of evolution that has developed from the original Modern Synthesis, with all its subsequent theory and experiment, Standard Evolutionary Theory (SET). SET has not stood still in terms of its content, but its over-arching conceptual framework would be familiar to the architects of the original Modern Synthesis.

However, others have argued that we need a truly new, truly extended evolutionary synthesis that does more than tinker with the Modern Synthesis and the resulting

SET (Laland et al. 2015; Müller 2017). Despite the previous discovery of groundbreaking new theories and empirical observations such as those alluded to above (of which I have barely scratched the surface), it is only comparatively recently that this EES has been called for, one that replaces SET. There will, of course, be those who find themselves in between these positions (e.g. one reading of Bonduriansky and Day 2018 is to place those authors there, as they state they are not challenging SET but rather showing how trans-generational effects fit into SET), but in many cases it seems that the end-point of EES thinking is the *replacement* of SET, rather than its continued burnishing with new theory or data (EES proponents vary on the scale of change required, but the changes apparently needed by evolutionary biology are generally non-trivial: see summary in Lewens 2019). And that is what I wish to critique and to understand. In particular, I wish to explore the EES by reviewing one of the best studied phenotypes in all of evolutionary biology: sex allocation. Sex allocation benefits from an especially rich theoretical background, with its origins with Darwin and then Fisher—himself one of the key architects of the Modern Synthesis—and its development by Hamilton and others in that period following the Modern Synthesis. As such, we can perhaps view sex allocation as an archetypal SET trait—born of Fisher then developed and tested in his shadow ever since.

In that case, what can sex allocation tell us about the EES? To do this, I will cast sex allocation in EES terms. By this I mean that I will take all the phenomena or conceptual spaces that EES proponents say are absent or poorly developed in SET and see how our study of sex allocation has fared under those terms. This will allow us to explore the overlap between the two explanatory frameworks, the SET and EES, in a *trait-focused* way. By doing so, I will critically scrutinise the claims of the EES with a well-studied phenotype in front of us. If SET has been unable to shed light on certain aspects of sex allocation, then bringing EES principles to sex allocation should make this obvious, consolidating the claims for such an extended view of evolution. However, if sex allocation theory and experiment has already pre-empted what is offered by the EES, then clearly the EES has less to offer than proponents might think. After all, why extend something that is already working, and already encompassing the apparent novelties of the EES? So, if the EES does represent something genuinely beyond the scope of SET, we might get a glimpse of just what that is when we have finished viewing sex allocation through the lens of the EES. My treatment of sex allocation will consider behaviour, phenotypic plasticity, niche construction, ontogeny, epigenetics, and trans-generational fitness effects and inheritance of multiple kinds, and so I will interrogate sex allocation fully in terms of what the EES claims to offer in terms of new evolutionary explanations and perspectives.

29.2 What Is Sex Allocation?

Sex allocation describes how sex and resources are allocated to offspring (Charnov 1982; West 2009). In terms of how sex is allocated, sex allocation is closely linked to, yet distinct from, sex determination (Bull 1983; Beukeboom and Perrin 2014).

For some systems, how sex is determined makes the mechanistic link to how sex is allocated (for instance, by the parent) fairly straightforward, at least at a phenomenological level. For example, in haplodiploid insects (such as bees and wasps), in which male offspring develop from unfertilised (haploid) eggs and female offspring develop from fertilised (diploid) eggs, mothers can determine the sex of their offspring by releasing sperm from their spermatheca to fertilise the passing eggs or not, immediately prior to oviposition (Cook 1993; Heimpel and De Boer 2008). Similarly, in species where the sex of offspring is determined by temperature during development (as in some reptiles), parents can determine sex of offspring by where eggs are placed or buried, or by the extent to which they bask prior to egg laying (Bull 1983; Janzen and Phillips 2006).

But sex allocation is also about how resources are allocated to the offspring of each sex, and indeed this is crucial for understanding the link between sex allocation and the sex ratio of offspring (either at birth/hatching, termed the primary sex ratio, or at some later ontogenetic point, such as offspring independence, termed the secondary sex ratio; West 2009). If each sex (i.e. male and female) costs different amounts to produce, then the fitness return per unit cost of producing sons or daughters may also differ (see below). Of course, each sex may cost the same in terms of energy and resources to produce (if that cost is just an egg, which either is or is not fertilised, for example). In these cases, the sex ratio of offspring will directly reflect sex allocation.

The theory of sex allocation has its origins with Darwin, Düsing, and Fisher, although the latter is usually credited with the key insights (Darwin 1871; Fisher 1930; Edwards 1998; West 2009). Fisher argued that selection on offspring sex ratios, and the sex allocation underlying sex ratios, will be shaped by frequency-dependent selection. This means that the selection acting on the parent (here we will consider the mother, for simplicity) in terms of her sex allocation depends in part on the sex allocation decisions made by other females in the population. The usual lecture-hall example is to assume that sons and daughters cost the same to produce, and then ask which sex is the best one for a mother to produce. In a male-biased population, clearly a mother would benefit from over-producing daughters, as they would find it easy to find mates, and the mother would maximise grand-offspring production. Producing sons would only add yet more males to the population, all competing for the limited number of females. Likewise, in a female-biased population, mothers would maximise their grand-offspring production by over-producing sons, who would again benefit from limited competition to find a mating partner. As a result, negative frequency-dependent selection, favouring production of the rarer sex, would lead to an equalisation of the population sex ratio, and unbiased sex ratios. (For completeness, note that at this population equilibrium, selection on sex ratios on a clutch-by-clutch basis is very weak; as the population sex ratio moves from equality, so the frequency dependence will again strengthen.)

Crucially though, Fisher realised that selection acted not on the sex ratio per se, but rather on sex allocation, including the energy required to produce a son or a daughter. So, all else being equal, at the sex allocation equilibrium the energy allocated to sons must equal the energy allocated to daughters. Here begins then

our theoretical understanding of biased, unequal sex ratios, through the realisation that all else is often not equal when it comes to sons and daughters.

The next major theoretical step-forward was made by Hamilton (1967). In an astonishing paper, Hamilton showed how the fitness benefits of producing male or female offspring can differ, and that this can lead to unequal sex ratios (from here on, we will consider sex ratio as the proportion of offspring that are male). Perhaps the most famous scenario from this paper is what happens when related offspring—usually males—compete for mates. Hamilton showed that if offspring of a single female developed and then mated together, the mother would maximise grand-offspring by producing a very female-biased sex ratio (Hamilton 1967, 1979). This would reduce competition amongst her sons for mates, and also maximise the number of possible mates each male obtained (Taylor 1981). However, as more females contribute offspring to each localised breeding patch, then the optimal sex ratio is less female-biased, as the competition among related males (e.g. the brothers from a given mother) is reduced, even though those males are now competing with unrelated males. This means that a mother could get more fitness through sons that are able to mate with both unrelated females as well as their sisters. As more and more mothers contribute offspring to a patch, so Hamilton arrived at the classic Fisherian solution, where the overall fitness return of producing a son equalled the fitness return of producing a daughter and—all else being equal—a sex ratio of 0.5 was favoured. Hamilton termed this competition among related individuals for mates Local Mate Competition (LMC).

Hamilton was not only a brilliant theoretician, but he was also a brilliant natural historian (and the two things were probably not unrelated). Hamilton knew that species such as fig wasps and various parasitoid wasps laid eggs in localised groups, and that kin interacted and mated before mated females dispersed. Unsurprisingly then, it has been amongst fig wasps and parasitoids that numerous tests of LMC theory have been undertaken (Godfray 1994; West 2009; Figs. 29.1 and 29.2), as we will consider in more detail below.

The insight that interactions between relatives of one sex may alter the reproductive value of offspring of that sex applies more generally. In fact, LMC is just one form of Local Resource Competition (LRC; Clark 1978). If offspring of one sex compete for any kind of resource (which could include mates, but also any other necessary resource, such as food, territories, or nest sites), then the same logic applies, with localised competition amongst kin leading to a reduction in the production of the competing sex. When the resources are not mates, competition for resources often occurs between females (such as in a variety of primates, Clark 1978), and so competition amongst daughters is predicted to lead to a male-biased offspring sex ratio, and indeed it does (for a review of the evidence, see West 2009). Finally, interactions need not be competitive; if interactions among kin of one sex generate fitness advantages instead of costs, for instance, through helping parents to provision subsequent broods of offspring, then Local Resource Enhancement (LRE) can occur. Under LRE, biased sex ratios are predicted in the direction of the cooperatively interacting sex, and again evidence for LRE has come from a variety of species (including cooperatively breeding vertebrates, such as the Seychelles

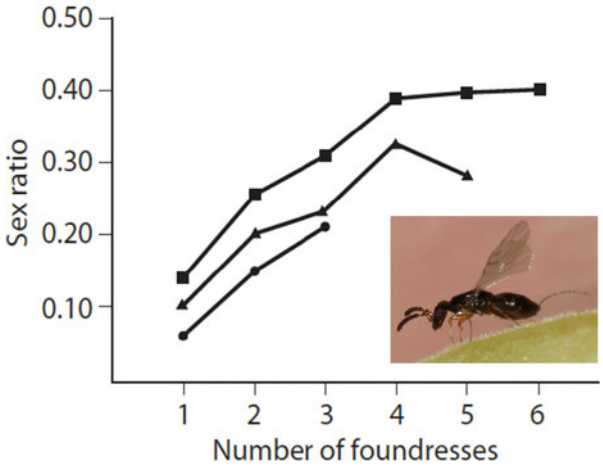

Fig. 29.1 Female fig wasps in the family Agaonidae allocate sex in line with predictions of Hamilton's theory of Local Mate Competition (LMC). Single foundresses lay extremely female-biased sex ratios, whilst as more foundresses lay eggs in the developing fig, so the number of males produced increases. Data re-drawn from Herre (1985). Sex ratio is the proportion of offspring that are male. Inset: a recently emerged female agaonid wasp, South Africa (Photo credit: Alan D Manson, CC-BY-4.0)

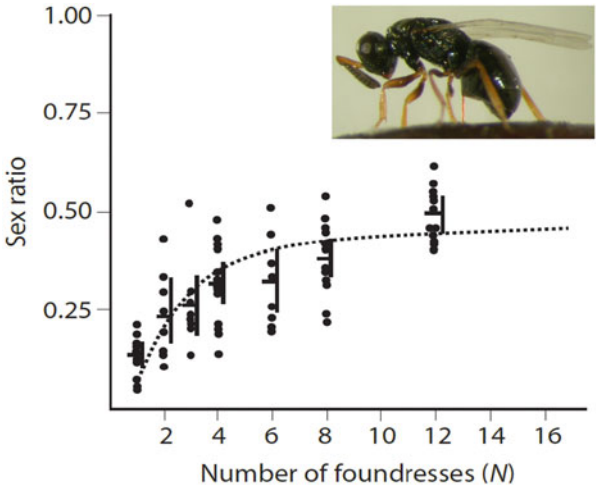

Fig. 29.2 Female *Nasonia vitripennis* parasitoid wasps facultatively allocate sex in line with LMC theory. Females respond plastically to the number of co-foundresses they lay eggs with. The dotted line is the predicted sex ratio under LMC for haplodiploids (Hamilton 1979). Data are re-drawn from Werren (1983). Insect: a female *Nasonia vitripennis* ovipositing on a blowfly pupa host (Photo credit: Dave Shuker and Stu West)

Warbler (*Acrocephalus sechellensis*), and primitively social bees: West 2009; Fig. 29.3). Importantly though, under LMC, LRC, and LRE, Fisherian negative frequency dependence is still acting, even if the optimum sex ratio is shifted from 0.5.

To go alongside the effects of interactions amongst kin influencing the reproductive value of offspring to parents, there is a second major strand of sex allocation

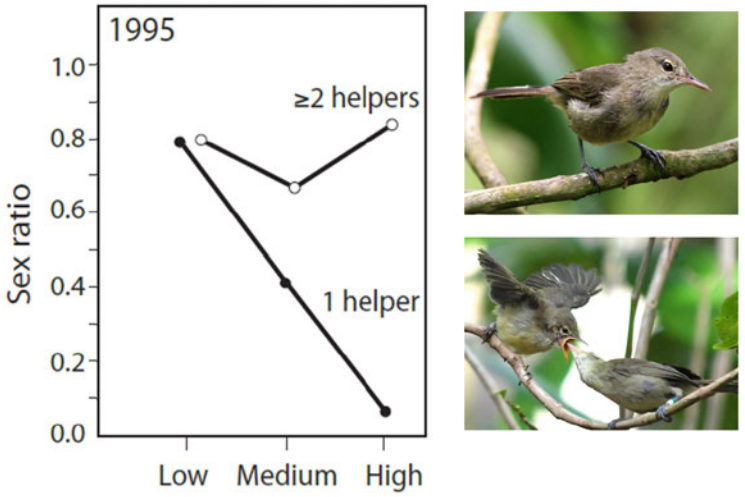

Fig. 29.3 The cooperatively breeding Seychelles Warbler (*Acrocephalus sechellensis*) allocates sex in line with predictions from Local Resource Enhancement theory. In this species, female young may remain at the next to help rear subsequent broods. In high quality territories, female offspring are favoured, in order to take advantage of the benefits of helpers at the nest. On low quality territories, there is insufficient food to support both new chicks and helpers, and so males are more likely to be produced. If more than two helpers are already present at the nest however, then the sex ratio is male-biased, regardless of territory quality. Data re-drawn from Komdeur et al. 1997. Insets: A Seychelles Warbler and feeding a fledgling (Photo credits: © Oscar Campbell; Charlie Davis). Sex ratio is proportion male

theory: condition-dependent sex allocation, also known as Trivers–Willard (TW) sex allocation (Trivers and Willard 1973; Charnov 1982; West 2009; for recent theoretical developments, see Veller et al. 2016). Under TW, the reproductive value of sons or daughters depends on the conditions under which those offspring develop. These conditions may be the extrinsic conditions of food availability, temperature, and so on, that brood experience from hatching or birth, through to adulthood. Alternatively, the conditions may be intrinsic to the parent (usually the mother), in terms of her condition and her ability to provide resources to the young (and, of course, both intrinsic and extrinsic factors may come into play).

The classic case of extrinsic condition-dependence involves sex allocation in solitary parasitoid wasps (Godfray 1994). Solitary parasitoids lay a single egg in or on an individual host (an egg, larva, or pupa of another insect, for example). The egg hatches, and the larva consumes the host, before pupating. Importantly, hosts are likely to vary in the resources they offer to a developing wasp larva. For instance, hosts may vary in size or stage of development, and thus vary in the amount or quality of food they represent. In insects, typically females benefit more in terms of fitness from a large body size than males do, as body size correlates more strongly with fecundity and hence fitness for females compared to how male body size influences fitness (for instance, through competition for mates). As such, a female

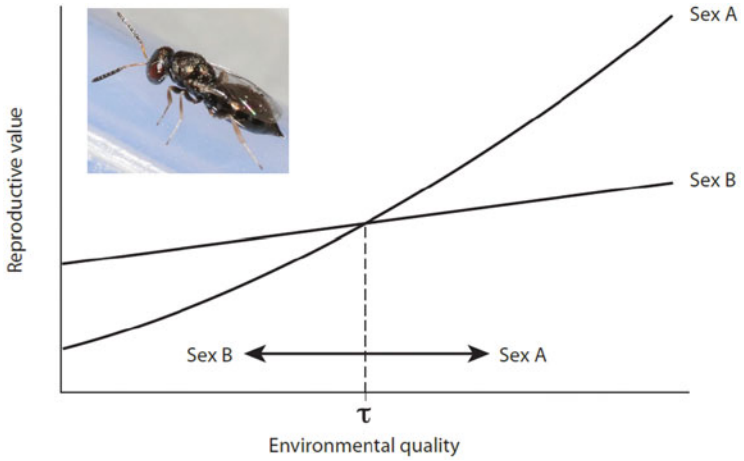

Fig. 29.4 Conditional sex allocation under the Trivers–Willard model. The reproductive value of an offspring of sex A or sex B varies with some component of environmental quality. At point τ, the fitness curves of the two sexes intersect. Below τ, production of sex B is favoured, and above τ the production of sex A is favoured. Inset: female *Anisopteromalus calandrae* places more male eggs on smaller hosts, and more female eggs on larger hosts, as predicted by T-W theory (Van Den Assem et al. 1983; Photo credit: ©James Bailey, BugGuide)

parasitoid is selected to place female offspring on large hosts, as large daughters offer the best route to fitness. However, sons still offer a route to fitness, and Fisherian frequency dependence still acts, so females are selected to produce sons when parasitizing smaller hosts. Put another way, male and female offspring have different fitness curves with relation to the size of host they develop in, and when those curves cross, offspring production should shift from sons to daughters (Fig. 29.4). This form of TW sex allocation was developed and tested by Charnov and colleagues back in the late 1970s (Charnov 1979; Charnov et al. 1981; Fig. 29.5), and since then the theory has been extensively tested and confirmed across dozens of species (Heinz 1998; West and Sheldon 2002; West 2009).

The classic case of Trivers–Willard sex allocation is associated with intrinsic condition however, in particular the condition of a mother during offspring development and the effect this has on the offspring's eventual fitness. If we consider a mammal such as a red deer, the condition of the mother influences embryonic development and size both at birth and as a yearling. Analogously to our solitary wasps, male and female deer benefit differentially from body size, only in this case large body size benefits males in terms of fitness disproportionately more than it does females. Large males are better competitors in the rut, better able to hold harems of females, leading to high reproductive skew amongst males. As such, a large son is an excellent way to produce a lot of grandchildren. Therefore, females in good condition should produce sons, whilst females in lesser condition should produce daughters (the evidence is broadly supportive in red deer, although appears ecologically context-dependent: Clutton-Brock et al. 1984, 1986; Kruuk et al.

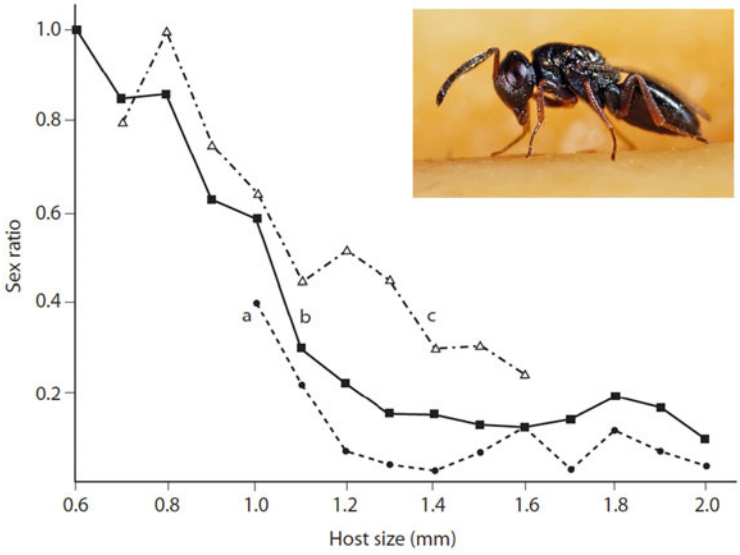

Fig. 29.5 Female *Lariophagus distinguendus* wasps vary their offspring sex ratios in line with Trivers–Willard conditional sex allocation. Males eggs are more likely to be placed on smaller hosts, and female eggs are more likely to be placed on larger hosts. The three lines (a, b, and c) represent three different protocols of presenting hosts of similar or different sizes to females and show that females take the relative size of hosts encountered into account (see Charnov et al. 1981 for details). Sex ratio is proportion male. Inset: a female *Lariophagus distinguendus* (© www.naturspaziergang.de/Andreas_Haselböck)

1999; Borowik and Jędrzejewska 2017; Fig. 29.6). Whilst more controversial in vertebrates (not least because of our poor understanding of how facultative sex allocation works in species with chromosomal sex determination, even though it clearly does: West et al. 2005), TW effects associated with maternal condition have been recorded in a number of species, and with intrinsic condition in other contexts in many more (Cameron 2004; Sheldon and West 2004). More generally, many aspects of sex allocation actually fall under the umbrella of condition-dependent sex allocation, including the evolution of sex-change in sequential hermaphrodites (Charnov 1982; West 2009), and it is probably only in a very few species that some form of condition-dependent effects are not present, however weakly, or however much they are dominated by Fisherian frequency dependence or interactions among kin. (It is worth noting that human primary sex ratios are not 0.5, but slightly male-biased, despite our chromosomal sex determination.)

All the above theory and experiment were done *within the framework of SET*. I will shortly frame such examples of adaptive sex allocation in terms of the ideas and framework put forward by the EES though, to see how sex allocation fits with the EES perspective. Beforehand, I will outline the EES in a little more detail.

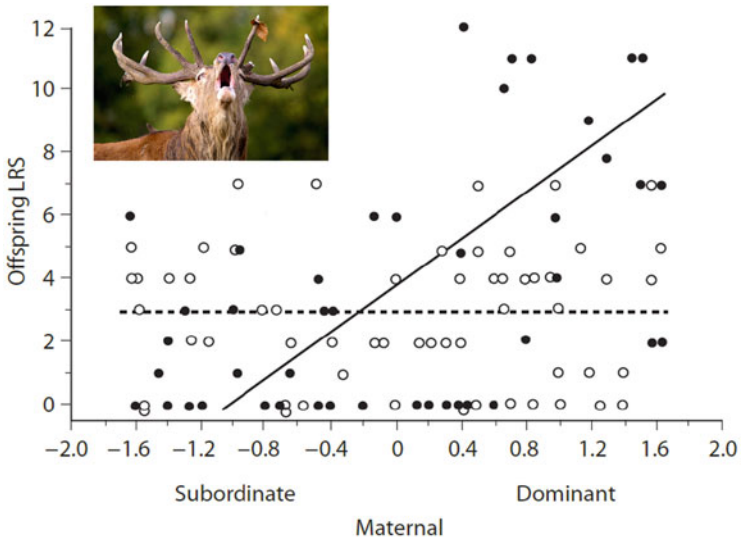

Fig. 29.6 Offspring lifetime reproductive success (LRS) varies for males (black circles, solid line) and females (white circles, dotted line) with respect to the social dominance of their mother. Dominant mothers in better condition produce the most successful sons. Data re-drawn from Clutton-Brock et al. (1984)

29.3 What Is the Extended Evolutionary Synthesis?

Briefly, the Extended Evolutionary Synthesis revolves around a set of ideas that have allegedly been (to a greater or lesser extent) excluded from, ignored by, or have only just become apparent to, the Standard Evolutionary Theory that we have inherited from the Modern Synthesis. In no particular order, these include developmental biology, phenotypic plasticity (both developmental, and at the organismal, behavioural, and/or life-history level), behaviour itself, niche construction, epigenetics, and trans-generational effects (which can be epigenetic in origin). In the interests of space, I will not pick apart the logic underlying these claims for each case, but rather I will try to distil out the essence of the claims made by the EES (e.g. Laland et al. 2014, 2015).

The EES seems to have two key components. First, there is an emphasis on *organismal agency*. This can be envisaged rather straightforwardly if we think of behaviour, as animals (and to some extent plants) can react moment-to-moment to facilitate necessary functions, such as feeding, copulating, or avoiding predators. More broadly, during development, the organism, at any or all levels of biological organisation, from the molecular through to the whole organism, can respond to internal and external stimuli, to release the next ontogenetic process or to maintain developmental homeostasis. The idea encapsulated by EES proponents when they mention phenotypic plasticity, niche construction or evo-devo say, is that organisms

are not passive players in their own lives. Instead, they get involved, they do stuff. And what they do at time point x influences what happens at time point $x + 1$, and so organisms are the architects of their own futures, including the evolutionary consequences of those futures. Taken to its fullest extent, this organismal agency might lead one to change how one views causation in evolutionary biology, introducing, for example, the idea of *reciprocal causation* (Laland et al. 2011; see also Svensson 2018).

Second, there is an emphasis on *non-genetic inheritance*. This can take several forms, some of which are clearer than others. On the one hand, many organisms—including plants—influence the environment their offspring will face, and so in some sense offspring inherit aspects of their environment due to the actions of their parents (and indeed other organisms in the environment). A slightly more concrete form of non-genetic inheritance can arise if females place their offspring in environments that they themselves experienced as juveniles, so that young inherit the juvenile environment of their parent. This has been long-appreciated by entomologists however, and there is some evidence that host-plant specificity can arise in this way in phytophagous insects, leading to the formation of host races (Jaenike 1990; Feder et al. 1994; Powell et al. 2006). The mechanisms underlying these effects are less clear however (Barron 2001). The contribution of the parents to the offspring reaches its climax in the phenomenon that is so obvious as to be almost banal: parental care (the study of which is itself an active, long-standing, and non-trivial subset of evolutionary biology: Clutton-Brock 1991; Royle et al. 2012).

Another form of non-genetic inheritance within the EES is the inheritance of knowledge about the world, either from parents or other individuals in the population. Social learning is a form of between-individual knowledge transfer, and animals can "inherit" information in this way, including across generations, either through some form of teaching (a controversial concept outside of humans) or some other mechanism of social learning, such as copying or stimulus enhancement (Heyes 1994; Whiten 2019). Under some conditions, what we may think of as *culture* may arise (see below).

Finally, perhaps the most genetic-like form of non-genetic inheritance is that which occurs when epigenetic modifications to DNA (including histone modifications and DNA methylation) are inherited through the germline (reviewed by Bonduriansky and Day 2018). These epigenetic modifications do not change the DNA sequences themselves, but are chemical modifications to the DNA, in terms of ligands and associated proteins. There is growing evidence that within-individual epigenetic changes can persist via transmission through the germline (although it seems as though they are typically only *recreated* rather than directly *replicated*, as DNA itself is: Bonduriansky and Day 2018).

We will return to issues raised by these two key attributes of the EES below, but now we turn to sex allocation, viewed through the paradigmatic spectacles of the EES.

29.4 Sex Allocation in EES Terms

29.4.1 Sex Allocation as Behaviour (Organismal Agency)

From the theory and examples sketched above, sex allocation is very clearly an example of organisms having agency, and not being passive in the face of environmental conditions that influence their evolutionary fitness. Whether we think of fig wasp females varying their offspring sex ratios in the light of how many other females contribute eggs to the fig fruit (Fig. 29.1), parasitoid wasps such as *Nasonia vitripennis* varying their offspring sex ratios in response to the number of other females laying eggs on a host in order to reduce LMC among their sons (Fig. 29.2; see below), or birds such as Seychelles Warblers varying the sex ratio offspring in line with predictions from LRE theory (Fig. 29.3), we see potential interactions among kin shaping how parents allocate sex. They are definitely not passive actors.

Likewise, examples of Trivers–Willard sex allocation—from solitary parasitoid wasps such as *Anisopteromalus calandrae* and *Lariophagus distinguendus*, to vertebrates such as red deer (Figs. 29.4, 29.5 and 29.6)—show how sex-allocating females respond to prevailing environmental conditions, or indeed their own physiological condition, to allocate sex so as to try and maximise fitness. Given the development of much of the key theory in the late 1960s and early to mid-1970s, and a raft of empirical tests of that theory since the 1970s (West 2009), it is clear that at least in terms of sex allocation, evolutionary biologists (in the guise primarily of behavioural ecologists: Davies et al. 2012) have been hugely appreciative of the agency of organisms in shaping their reproductive success, the reproductive success of their offspring, and hence their inclusive fitness. Indeed, the whole of behavioural ecology—the study of the adaptive significance of behaviour—is, of course, *wholly focused* on the evolutionary consequences of behaviour. Behavioural ecologists study the way in which organisms respond, moment-to-moment, to environmental conditions, both intrinsic and extrinsic, biotic and abiotic, in order to influence their ability to pass on their genes. Sex allocation is a wonderful example, not least because of our ability to test predictions from theory both qualitatively *and* quantitatively (West and Sheldon 2002; West et al. 2002; West 2009), an ability largely unparalleled in other parts of behavioural and evolutionary ecology, thanks to the underlying simplicity of the trait and nature of the fundamental trade-off (son versus daughter). However, sex allocation is only one part of behavioural ecology, and only one part of how the knowledge of the importance of behaviour in shaping fitness, and thus evolution, has been with us for *many decades*. In terms of the evolutionary causes and consequences of organismal agency, the EES is playing catch-up, more than five decades after key theoretical advances made via SET, and multiple journals devoted to the subject. And all this without the idea of "reciprocal causation" as well, albeit with a nonetheless deep-seated, historical understanding of feedback loops in evolutionary biology (Fisher 1930; Bailey 2012).

29.4.2 Sex Allocation as Phenotypic Plasticity

From the above examples, it is also abundantly clear that the ability to respond to the environment when allocating sex is a superb example of phenotypic plasticity. Indeed, it is *nothing but* phenotypic plasticity. To be more formal for a moment, phenotypic plasticity is the ability of a given genotype to produce multiple phenotypes across a range of environments (e.g. Schlichting and Pigliucci 1998). Classic lecture-hall examples of phenotypic plasticity include the variation in growth rates of clones of plants across a range of nutrient availabilities, ambient temperatures, or light regimes. Indeed, when we think of plants, the ubiquity of phenotypic plasticity is clear, even banal. Likewise, since all the different behaviours an animal exhibits can be considered different phenotypes (even if lumped into big classes, such as foraging, searching for mates, avoiding predators, finding shelter), then behaviour is itself avowedly phenotypically plastic. Indeed, it is the job of behaviour to be plastic, its whole point. Phenotypic plasticity is everywhere, and sex allocation is no exception, with Figs. 29.1, 29.2, 29.3, and 29.5 showing these plastic responses—or *reaction norms*—very clearly. Moreover, sequential hermaphroditism is sex allocation as phenotypic plasticity *par excellence*. Sequential hermaphrodites, including sex-changers such as *Amphiprion* clown fish, for example, produce different sexes across their lives, which is nothing if not developmental plasticity. Species can go from male to female (protandrous sex-changers) or female to male (protogynous sex-changers), or even—in a few extraordinary examples—make *two* changes during their lives (West 2009).

One of the benefits of our theoretical and empirical understanding of sex allocation is that we have been able in some cases to tease apart the basis of this phenotypic plasticity, for instance, in terms of the information females use when making more or less subtle sex ratio shifts. Take the gregarious parasitoid wasp *Nasonia vitripennis*. Females of this species parasitise blowfly pupae, laying multiple eggs per host. Males are short-winged (brachypterous), and so mating primarily takes place on the fly puparium when adult males and females emerge following eclosion inside. If only one female lays eggs on a given host, then her sons and daughters will mate with each other, which is exactly the scenario Hamilton envisaged for LMC to occur. *Nasonia* has not unsurprisingly been a well-studied model organism for how LMC shapes sex allocation, beginning with the pioneering work of Werren (1980, 1983). We know now that ovipositing female *Nasonia vitripennis* facultatively (i.e. plasticly) change their sex allocation in response to the presence and number of other females (termed *foundresses*) that also contribute eggs to a host or patch of hosts (Werren 1983; Shuker and West 2004; Burton-Chellew et al. 2008; Fig. 29.2), the size and hence fecundity of other foundresses (Flanagan et al. 1998), the number of eggs already on a host (Werren 1980; Cook et al. 2016), the presence of eggs on other hosts in the patch (Shuker et al. 2005, 2006), the number of other parasitized hosts on the patch (Shuker et al. 2006), the size of the overall patch (which influences how offspring from different broods interact: Shuker et al. 2007), as well as the timing of when other eggs were laid on the patch (Shuker et al. 2006). Just as importantly, we also know that female *N. vitripennis* do not respond to some

environmental conditions that theory would lead us to expect them to (i.e. they are not perfectly adapted). For instance, they do not take into account the relatedness of the male they mate with (Shuker et al. 2004a), the females they oviposit with (Reece et al. 2004; Shuker et al. 2004b), or even the difference between con- and hetero-specifics (Ivens et al. 1998). As such, we know quite a lot about what sorts of information female *Nasonia* use when facultatively allocating sex under LMC, i.e. how they are phenotypically plastic in the face of changing predictors of the level of LMC their sons will face.

Similarly, we have been able to address some questions as to the mechanistic basis of sex allocation and hence phenotypic plasticity in *Nasonia*. Briefly, in terms of genetics, data from Orzack and colleagues in the 1980s and 1990s showed that variation in sex allocation in *Nasonia* has a heritable component (albeit not a terribly large one; as might be expected for a trait so plastic and dependent on specific environmental conditions, the environmental component of sex ratio is large: e.g. Orzack and Parker 1986, 1990; Orzack 1990; Orzack and Gladstone 1994; Pannebakker et al. 2011). More recently, there have been studies of the mutational heritability of sex ratio (Pannebakker et al. 2008), and the first quantitative trait loci (QTL) for sex allocation in *Nasonia* have been identified (Pannebakker et al. 2011). The latest work suggests that sex allocation is highly polygenic however, with many genes influencing variation in sex allocation (Pannebakker et al. 2020).

The study of the mechanistic basis of phenotypic plasticity has been boosted in recent years by the growing availability of gene expression (transcriptomic) studies in non-model organisms, opening up the opportunity to study what genes and gene networks underlie the changes in phenotype visualised in reaction norms (e.g. Aubin-Horth and Renn 2009; Oppenheim et al. 2015; Lafuente and Beldade 2019). The study of sex allocation in *Nasonia* has been no different. However, interestingly, the extremely plastic and nuanced responses to LMC cues are *not* associated with changes in gene expression (*at all*: Cook et al. 2015a, 2018). Instead, the very impressive and rapidly responding patterns of sex allocation in this wasp seem to be products of the underlying neural architecture constructed prior to adulthood. Indeed, patterns of adaptive sex allocation have recently been experimentally disrupted by exposure to sub-lethal doses of neurotoxic neonicotinoid pesticides, which disrupt acetylcholinesterase receptors (Whitehorn et al. 2015; Cook et al. 2016). The phenotypic plasticity of *Nasonia* therefore seems to be largely hard-wired in.

In summary, sex allocation should be considered a canonical form of phenotypic plasticity, spanning behavioural and developmental plasticity. And so, for those interested in the genetics and mechanistic underpinnings of reaction norms or wanting to quantify the evolutionary consequences of plasticity—an avowed aim of the EES—then sex allocation should be a great place to start. And yet sex allocation is often absent from discussions of phenotypic plasticity. However, perhaps one of the problems for EES proponents, especially for those outside of the animal behaviour or behavioural ecology communities, is that the day-to-day, in-your-face obviousness of phenotypic plasticity means that it is not even mentioned in many cases. My own work on sex allocation summarised above has rarely if ever

included the term in the abstract or keywords of my publications; moreover, a Web of Science search reveals 2157 papers with the search term "sex allocation" (Web of Science Core Collection: 2/08/2021, search by DMS), but only 92 that include both "sex allocation" AND "phenotypic plasticity" (i.e. 4.3%). To truly understand how evolutionary biologists have appreciated and studied phenotypic plasticity requires more than just the easy scholarship of the search engine, but also a deeper understanding of the traits we study and how we study them.

29.4.3 Sex Allocation as Niche Construction

Niche construction is a concept that describes the effects organisms have on their environments (Odling-Smee et al. 2003). Taken at its broadest, as organisms continually interact with their environments and necessarily change them moment-to-moment, it just describes the fact that organisms interact with their abiotic and biotic environments; in other words, it is synonymous with many definitions of ecology. In these broad terms then, there is not really such a thing as niche construction theory (NCT), in the same way that there is not really a "theory of ecology". Rather, ecology is a "thing" about which we have many theories. A slightly looser, more manageable view of niche construction, and hence NCT, is to focus on the effects organisms have on themselves and other organisms via their behaviours and so forth, including their extended phenotypes such as nests. The overlap with the preceding discussion about behavioural agency will already be clear to many readers, but here I briefly put sex allocation in niche construction terms.

Put simply, sex allocation is a *perfect* example of niche construction. An organism does something—allocate sex—that constructs the niche for that offspring, i.e., the ecological consequences of being male or female, influencing the fitness of that offspring, and hence the inclusive fitness of the sex-allocating parent. More concretely, let us consider some examples. First, let us take fig wasps, such as the so-called pollinating fig wasps in the family Agaonidae. Adult females visit a developing fig (called the syconium), and lay eggs within the syconium, collecting pollen from the flowers within the fig as they do so. Females will visit multiple syconia, laying eggs and collecting and depositing pollen, and so providing pollinator services for the fig plant. The eggs are laid in the flowers and form gall-like structures, and male and female fig wasps develop by consuming the gall. Adult males are wingless and spend their whole lives inside the fig, competing for freshly-eclosed adult females to mate with. As such, fig wasps provide another case-study for local mate competition, with foundress females varying their offspring sex ratios depending on how many females contribute eggs to the fig, as predicted by LMC theory (Fig. 29.1; Frank 1985; Herre 1985; for further details Herre et al. 1997; for sex allocation in non-pollinating fig wasps, see Fellowes et al. 1999).

But there is also lots of niche construction here, from the females placing the eggs inside the developing fig (constructing the natal niche), the gall that forms around each egg and developing larva (constructing the larval niche), through to the mating dynamics of the males and females (constructing the mating system niche), which all

will be shaped by the oviposition and sex allocation decisions of the foundress females. Similarly, as we have already seen, gregarious parasitoids such as *Nasonia* likewise influence the interactions amongst their offspring—constructing their mating system niche—by influencing the sex ratio (and also, of course, clutch size, but we will focus on sex allocation). In both fig wasps and parasitoids then, mothers construct the mating niche of their offspring, taking into account the number of other foundresses laying eggs, plus a number of other more or less subtle determinants of local mate competition (see above).

Second, it is not only in terms of LMC that females construct the niches of their offspring. Under Trivers–Willard sex allocation, females may determine the niche of their offspring in a sex-dependent way. In the rather startling "zombie cockroach killer" wasp (better known as *Ampulex compressa*), females put a fertilised egg destined to develop into a female on a large cockroach host, and an unfertilised egg on a smaller cockroach host (Arvidson et al. 2018). Whilst this is niche construction—the food availability and hence the developmental trajectory of the male or female offspring is determined by the mother—*A. compressa* is rightly infamous for another bit of niche construction (well, invasive neuroscience really). Females inject venom containing neuropeptides into the brain of the cockroach host, which ablate its escape response, allowing the wasp to lead the "zombie" cockroach by the antenna to a burrow, where the wasp will then lay an egg on the immobilised, but not dead, cockroach (Haspel et al. 2003; Gal and Libersat 2008).

Third, a variety of organisms determine sex via experience of local environmental cues, for instance, during gestation or development within an egg. Perhaps the best-known form of environmental sex determination (ESD) is temperature-dependent sex determination (TSD), which occurs across a range of reptiles (see above). TSD does not preclude adaptive sex allocation however. Instead, mothers may manipulate the sex of their offspring by determining where the eggs develop, for example, how shallow or deep they are placed in a natal pit, dug in sand or soil. At different depths, the temperature will subtly change, influencing the resulting sex ratio of the brood. This is clearly niche construction: the mother *literally* digs a pit, of a given depth, and places eggs into it in such a way that the developmental trajectories are manipulated to give a certain sex ratio. As should be clear by now, the fitness of the resulting offspring will be influenced by the mother's decisions, and hence by her niche construction.

It is worth emphasising though that the theoretical and empirical studies reviewed in this section were conceived and conducted without the need for (nor indeed, without the formal conception of) niche construction as a concept; niche construction is merely one conceptualisation of things evolutionary ecologists have been doing for decades (a point also made by Keller 2003; Brodie 2005; Scott-Phillips et al. 2014; Gupta et al. 2017). Nonetheless, sex allocation is niche construction, and again we might think that it should be firmly under the aegis of the EES.

29.4.4 Sex Allocation as Ontogeny

Part of the EES manifesto is that development has been down-played or ignored by evolutionary biologists and was effectively absent from the Modern Synthesis (Laland et al. 2015). Whilst one may argue whether that is actually true, certainly the field of "evo-devo" has flourished over the last couple of decades, particularly on the back of our increased ability to scrutinise and manipulate DNA sequences. Indeed, in many ways evo-devo is largely the study of developmental genetics (see Diogo 2016, 2018 for a critique of evo-devo, and the absence of "evo"). However, here we are interested in evo-devo in terms of how ontogeny shapes, and is in turn shaped by, evolution. As will be increasingly apparent, the interconnectedness of aspects of the EES means that I begin to risk repeating myself. As such, I will be brief. Ontogenetic processes are clearly part and parcel of sex allocation in many cases. For instance, as we have seen above, the developmental consequences of a given environment or food resource for embryonic/larval development can differ between male and female offspring. Those sex-specific developmental trajectories influence the evolution of sex allocation—such as when to switch between producing male and female offspring—and are themselves subject to selection.

There are subtle interactions too between sex allocation and development. When the gregarious larvae of parasitoid wasps develop on the same host, the larvae will compete for resources, for instance, by scramble competition for food, with fast developing offspring perhaps benefiting. If development is sex-specific, and in many cases it is, with males being smaller and developing more rapidly than their larger sisters, then this competition may be asymmetric with respect to sex (asymmetric larval competition: Godfray 1986; Sykes et al. 2007). Again, for any given species, the developmental profiles of males and females will have been influenced by various aspects of natural and sexual selection (fecundity, mate finding, and so on) that influence how large and with what resources an offspring reaches adulthood. These developmental differences may shape how kin compete for resources, and thus again shape the reproductive value to a parent of producing a son or daughter. In the gregarious parasitoid wasp *Nasonia vitripennis* discussed above, the effect of asymmetric larval competition on sex allocation is small when compared to that caused by LMC (Sykes et al. 2007), but in other species competition between the sexes for resources arising from different patterns of development can be more striking (such as in the rather remarkable polyembryonic wasps: Grbić et al. 1992).

A lovely example of the sometimes very intimate relationship between development and sex allocation comes from sex-changing sequential hermaphrodites (Charnov 1982; West 2009). Sex-changing organisms switch between male and female sexual function over their lives, going in either direction (or even changing twice), depending on the shape and steepness of the male and female fitness functions with respect to age, size, or some other aspect of developmental state. This means that sex allocation—the decision to be male or female—is closely tied to the moment-to-moment development and growth of the organism, and what that means for its fitness. Thus, the important evo-devo links between (a) development

and the phenotype, and (b) phenotype and stage-specific fitness, such that fitness effects loop back to shape developmental trajectories and hence phenotypic trajectories, are clearly all present in these species, with the study of them decades old (Charnov 1982). In fact, one could argue that sequential hermaphrodites should be a research priority, or key set of model systems, for understanding how development, phenotypes, and fitness interact. What is more, as with the whole field of sex allocation, we have a robust theoretical framework which we can adorn with mechanistic details and empirical tests. And as all these examples show, sex allocation provides many clear exemplars of the ways in which organismal development influences phenotype and hence fitness. And again, sex allocation should therefore be a strong component of arguments promoting the EES. Unfortunately, SET got there first.

29.4.5 Sex Allocation as Epigenetics

When we turn to the role of epigenetics and sex allocation, here at last we come to a relatively newer component of the study of sex allocation, one which is more or less contemporaneous with the EES movement. First, I should clarify again that sex allocation is not synonymous with sex determination, and the role of epigenetics in sex determination, for instance, through DNA methylation being involved in the regulation of sex determination, is being increasingly well recognised (even if many mechanistic details are lacking). For instance, in sea bass (*Dicentrarchus labrax*) and the mixed-mating system Mangrove Rivulus fish *Kryptolebias marmoratus*, epigenetics have been implicated with sex determination (Ellison et al. 2015, see also Consuegra and Rodríguez López 2016 for discussion); there are also some associations between epigenetic modification and sex or mating type in plants (Harkess and Leebens-Mack 2017; and epigenetics may underlie *terroir* in our wine, but that is another story: Xie et al. 2017).

In *Nasonia vitripennis*, sex determination has been convincingly shown to involve some non-genetic trans-generational effect (i.e. there are neither sex chromosomes in this haplodiploid insect, nor single- nor multi-locus complementary sex determination: Verhulst et al. 2010; Beukeboom and Van De Zande 2010). Following extensive study, it is now clear that sex is determined by the presence (in diploid, fertilised embryos) or absence (in haploid, unfertilised embryos) of a paternally inherited copy of a gene (with the excellent name of *wasp over-ruler of masculinization*, or *wom*: Zou et al. 2020). The maternally inherited copy is epigenetically silenced, leading to male sex determination in unfertilised embryos. However, when it comes to sex allocation in *Nasonia*, there is now indirect evidence for a role of epigenetics too, and in particular DNA methylation, in terms of regulating sex allocation and thus sex ratio in this species (Cook et al. 2015b, 2019a, b).

The motivation for the study of Cook et al. (2015b) came from theory developed by Wild and West (2009), exploring the role of genomic conflict in sex allocation. In particular, they explored how genomic imprinting could influence sex allocation, across a range of scenarios, including facultative sex allocation under LMC.

Genomic imprinting, or the passing on of parent-of-origin information (i.e. whether a chromosome has been maternally or paternally inherited), is necessary for maternally or paternally inherited alleles to influence the phenotype—and hence the fitness consequences—of an offspring. The classic example is conflict between maternally and paternally inherited alleles in developing embryos in utero in mammals; theory suggests that paternally inherited alleles should try and extract more resources from the mother during development than maternally inherited alleles (Moore and Haig 1991; Haig 2000). Remarkably, data from a number of mammals suggest that such a conflict actually occurs. Importantly though, alleles need to "know" (very much in inverted commas) whether they have been maternally or paternally inherited, i.e. the parent-of-origin information is crucial, and so some form of genomic imprinting is necessary, to pass this non-genetic information across generations.

Cook et al. (2015b) tested the ideas of Wild and West (2009) in *Nasonia vitripennis*, using the demethylating chemical 5-aza-dC to disrupt patterns of DNA methylation across the genome (see Ellers et al. 2019 and Cook et al. 2019a, b for a discussion of the efficacy of this technique). The authors found that there was a small but significant shift in sex allocation in response to the DNA methylation manipulation, with sex ratios going up (i.e. becoming less female-biased). In terms of the predictions of Wild & West, this would suggest that unmanipulated sex ratios in *N. vitripennis* are closer to a maternally inherited allele optimum than a paternally inherited allele optimum, and disrupting DNA methylation shifts things upward (although again, we reiterate the effect is small, as predicted by Wild and West 2009). Importantly, Cook et al. (2015b) showed that the shape of the reaction norm—the pattern of facultative sex allocation with respect to foundress number—was unaffected by the chemical treatment. Thus, the facultative or phenotypically plastic aspect of sex allocation was not disrupted, rather sex ratios across the range of foundress numbers tested were higher for treated mothers than for controls.

Much remains to be explored here. More recently, Cook et al. (2019b) have shown that 5-aza-dC causes widespread changes in DNA methylation across the genome, and so candidate causal CpGs will be difficult to identify. Moreover, it has so far not been possible to show the parent-of-origin effects at the epigenomics level in *Nasonia vitripennis* required (Wang et al. 2016; Olney et al. 2021; but see Zou et al. 2020). Nonetheless, these data suggest a role for epigenetics in sex allocation, through an effect not just on sex determination mechanisms, but on the control of sex allocation itself. How widespread the role for epigenetic mechanisms will prove to be is as yet unknown, but theory such as that provided by Wild and West (2009) offer places to start to look.

Importantly for the overall message of this chapter though, it must be remembered that the theory of Wild and West (2009) was developed very much in the tradition of Standard Evolutionary Theory. In other words, they were interested in genetic evolution, and how genomic conflicts could evolve and influence *genetic transmission* across generations, through phenotypes influenced by non-genetic inheritance of epigenetic information. Likewise, one of the key originators of genomic imprinting and genomic conflict theory—David Haig—places his theory very much in the population genetic terms of SET (Haig 2000, 2007, 2014). This

again poses the same problem for proponents of the EES. If SET can foster and engender ideas and theory—in this case about the evolutionary development and consequentiality of genomic imprinting, and the non-genetic transmission inherent to genomic imprinting—then why are epigenetic effects put forward as evidence for the need of an extended synthesis? Hasn't SET again got there already?

29.4.6 Sex Allocation and Trans-Generational Effects

As should be obvious from the examples and discussion above, sex allocation is *all about* trans-generational effects, both in terms of how one generation (the parents) influences the phenotypes of another (the offspring), but also in terms of how those phenotypes then influence the fitness of the offspring, and hence the inclusive fitness of the parents. Indeed, we have basically been talking about nothing else apart from trans-generational effects, with parents shaping the environments, the phenotypes, and the fitness of their offspring. (As an aside, I note that this is of course true for other well-studied aspects of behavioural ecology, such as the evolution of parental care; the idea that the theory of the evolution of parental care was stunted under the SET is obviously a non-starter: Clutton-Brock 1991; Royle et al. 2012.) As such, it should be uncontroversial that sex allocation is all about trans-generational effects that go far beyond just the inheritance of genes, and so again it is hard to argue that the importance of trans-generational fitness effects has been absent from SET until very recently. Perhaps more importantly, nor do such effects over-turn or invalidate SET. Quite the opposite. We have been able to understand those effects for more than 50 years *because of* Standard Evolutionary Theory.

However, I will finish this section with just one further example, which again highlights theory developed multiple decades ago, which argues against a one-eyed view of the SET as comprising genes-only trans-generational effects, and within-generation only fitness effects. The so-called haystacks models of sex allocation are in effect extensions of Hamilton's LMC models. The name comes from an early model of Maynard Smith (1964), describing an idealised mouse species that colonises and lives within a haystack for several generations before dispersing. Already one can see that multiple generations living in the same localised resource will likely engender trans-generational effects, including the "inheritance" of a given habitat state, and of course niche construction. Bulmer and Taylor (1980) developed the first models of sex allocation in haystacks, with the main difference to predictions from LMC models coming from the extent to which mating can occur between individuals after dispersal, which can then interact with the number of generations spent in the haystack prior to dispersal to influence sex allocation (for a review of the theory, including later developments, see West 2009). The notion that SET comprises "unilinear" effects (for example, Müller 2017) belies the ecological realities, the *actual biology*, that the SET has been employed to explain, in many cases overwhelmingly successfully. Instead, sex allocation shows that SET not only can deal with interacting fitness effects, but it can also help *identify* and *predict* them through its predictive framework.

29.4.7 Sex Allocation as Culture

This last section addresses culture and the possible role of cultural evolution in patterns of sex allocation. To begin with, a sketch of relevant terminology will be useful, as definitions can vary. In terms of the biology of culture, I will consider that organisms may have traits that together provide the *capacity for culture*. These are more or less straightforward biological traits, with genetic underpinnings like any other biological trait, that allow organisms to be cultural. These traits include learning, and especially social learning, among-individual communication, the ability to perceive and to attend to relevant social cues and/or signals, and sufficient among-individual social interactions, including across generations, for cultural phenotypes to be transmitted. Many social organisms will have some or all of these traits to some extent, although culture is not a given outcome (for reviews of social learning and culture in animals, see, for instance, Whiten et al. 2011; Hoppitt and Laland 2013; Aplin 2018; Whiten 2019, 2021).

I will define *culture* itself as a set of one or more shared behavioural phenotypes, and any physical products thereof (such as artefacts), that are originated, learned, and transmitted within- and across-generations. Culture is thus an *emergent phenotypic property* of biological organisms, such that the nature and extent of the cultural phenotype is not itself genetic in origin, even though all the underlying machinery, and variation in expression of that machinery across individuals in the population, will have a genetic component. Cultural phenotypes may influence the fitness of individuals, and so we expect gene-culture coevolution to influence the evolution of underlying cultural capacities (Feldman and Laland 1996) and indeed what cultural products populations express. Cultural phenotypes, by their very nature, may be somewhat ephemeral and hard to pin down though. Whether instantiated only by behaviour or alongside physical artefacts, the cognitive basis of culture (as a learned "thing"), means that culture is neurally encoded, forming part of an organism's mental landscape, explicitly or otherwise.

What possible links are there between culture and sex allocation? First, for non-human animals, potentially rather few. This is not because of a dearth of culture: far from it. Over the last two decades or so, there has been a great flourishing of research into animal cultures, and we now know that cultural transmission of behaviours can occur not just in humans or other primates, but across cetaceans, birds, and indeed insects (Whiten 2021). It is likely that many more examples of culture and cultural transmission will be uncovered the more we look. However, it perhaps not immediately clear how, or under what circumstances, cultural preferences for sex allocation could evolve in non-human animals. One possible route would be if a cultural preference for a certain sex or sex ratio emerged by chance in a species in which there was selection for a different sex ratio, but no underlying genetic variation in sex allocation or sex determination. For example, if male offspring were rare, and that rarity led to greater attention by a parent due to an underlying attraction to novel stimuli, then through imitation or local enhancement, this could lead to greater attention being paid by other parents towards male offspring in other litters, and the greater likelihood of death of the increasingly

ignored female young. This would mean there was a culturally transmitted trait (parenting effort) that biased offspring survival towards males.

In humans, on the other hand, there is all too much evidence that post-conception sex ratios are manipulated due to culturally inherited preferences for one sex of offspring over another. In a number of human societies, female offspring have been or are valued less than males, leading to sex-specific infanticide or neglect (for a variety of perspectives across historical and contemporary cultures, see, for instance, Sen 1990; Hesketh and Xing 2006; Lynch 2011; Klasen and Wink 2002; Mitra 2014; Barman and Sahoo 2021; Beltrán Tapia and Raftakis 2021; for a theoretical gene-culture treatment, see Kumm et al. 1994). In these cases, the value of sons versus daughters may be associated with a whole raft of cultural traits that define the societal norms in which the individuals live, including how resources—such as land, money, and so on—are inherited within or among families across generations. These highly multi-layered cultural components of human societies are unequalled in other animals, and so the role for culture to play such a strong role in biological evolution, including in such fundamental traits such as sex allocation, is much more limited. That said, sex-specific infanticide is known in non-human animals, in organisms as diverse as ants, hamsters, and *Eclectus* parrots (Bourke and Franks 1995; Beery and Zucker 2012; Heinsohn et al. 2011). However, the role of cultural evolution in this infanticide is as yet unexplored.

29.5 Discussion

There are many aspects of sex allocation I have not been able to include here. This is true in terms of the sophisticated theoretical machinery that has already been developed, and the rich diversity of empirical examples across animals and plants (and of course protists: malaria parasites provide a wonderful example of sex allocation under LMC: Reece et al. 2008). Nonetheless, I have clearly shown that the study of sex allocation—very active for more than four decades, with a history dating back to the origins of the Modern Synthesis itself—has historically embraced, and continues to embrace, the aspects of biology emphasised by the Extended Evolutionary Synthesis, but that have supposedly been ignored by evolutionary biologists.

As such, sex allocation is the dog that did not bark in the night, curious by its absence from discussions of the EES. If the EES is predicated on the idea that evolutionary biologists working with the inherited Modern Synthesis framework—Standard Evolutionary Theory as I have framed it here—have missed, or ignored, or been unable to accommodate new findings in their SET paradigm, then sex allocation presents a considerable challenge to the EES. Why are not fig wasps a poster-child for an EES? Their extraordinary behaviour, its plasticity, and its evolutionary consequentiality born of trans-generational fitness effects, should all be grist to the mill of the EES. The key concepts of the EES—organismal agency through adaptive plasticity and trans-generational effects—are what sex allocation is fundamentally all about. The problem is though that the theory that underpins all these responses,

including the role of epigenetics in mediating genomic conflicts in a very EES sort of way, exists profoundly within the SET framework. Fundamentally, *no new way of doing theory was required*. Instead, sex allocation has been modelled in all sorts of way within SET, from very explicit population genetics models, through to the increasingly implicit genetics that lie at the heart of evolutionary game theory (West 2009). Sex allocation shows how SET can deal with all of the kinds of phenotypes the EES proponents say are problems or that require "a new perspective".

One might argue that sex allocation is small beer, only a fraction of the edifice that is evolutionary biology under SET; one could say that the EES is "bigger than sex allocation", for instance. Whilst it is true that sex allocation is only a part of modern evolutionary biology, it has an important standing because—as mentioned at the beginning—it has been a branch of evolutionary biology where some of the most explicit tests of SET have been attempted. But is sex allocation mainstream evolutionary biology? It is always open to debate as to how important or mainstream any one field is, but it would be hard to argue that sex allocation is just a niche interest for those who—like me—like little black wasps. Hamilton's foundational 1967 paper has been cited 2490 times (Web of Science Core Collection: 2/8/2021, search by DMS), and those papers themselves have been cited a total of 116,283 times (amusingly this means that Hamilton 1967 has its own *h*-score of 147!). Sex allocation theory is clearly mainstream evolutionary biology, and has been described as a touchstone for SET, as one of the most successful bodies of predictive theory in evolutionary biology (Frank 2002). Yet I hope that this review exhaustively shows that sex allocation also exhibits all of the main categories of "neglected" or "unappreciated" aspects of the world that the EES claims to bring to the party, including organismal agency, trans-generational effects, and the importance of developmental conditions shaping selection.

I suggest that arguments for the EES, and by extension for a rejection or a replacement of Standard Evolutionary Theory, have to emphasise or create the impression of a lack, a neglect, a "something missing", from SET and its outlook in order to look credible. Whilst it might be argued that all the EES is arguing for is an embellishment or two of SET, in line with the empirical and theoretical accretions discussed in the Introduction, many of the arguments made in favour of the EES seem to demand a more deep-seated change (see Introduction). However, the need for such change fits uneasily when one of the most successful areas of evolutionary biology, that has been developed and tested under the umbrella of the SET for decades, has so many of the apparent components of the EES deep in its warp and weft. Researchers have diligently explored how different evolutionary and ecological factors—including behavioural constraints—have shaped the evolution of sex allocation for decades, prefiguring most if not all of the "missing aspects" of evolutionary biology. Sex allocation is full to brimming of all the things the proponents of the EES say are missing or have been underplayed. Put another way, sex allocation is very much the offspring of SET, thanks to Hamilton, Trivers, and many others, and yet it fits remarkably well with what the EES claims to bring. Fundamentally, I suggest that the successful development of sex allocation theory demolishes arguments for the EES as having any necessary role to play and exposes

the fallacy of narratives of "neglect" in mainstream evolutionary biology (see also Gupta et al. 2017).

In conclusion, we have then the curious case of the wasp in the fig fruit. All along, the study of sex allocation has been utilising what EES proponents have argued has been ignored by evolutionary biologists, which should not be the case if SET presents this insurmountable barrier to properly understanding how phenotypes evolve. To emphasise this contradiction, I have presented a lot of biology in the preceding pages, but I make no excuses for that. Any successful call for some kind of new synthesis cannot just be in opinion pieces and philosophical reviews. It has to deal with the nuts and bolts of actual biology, and the biology that evolutionary biologists have done and continue to do. And in terms of that biology, if sex allocation has been hiding in plain sight, how many other sub-disciplines of evolutionary biology have also been studying their phenotypes of choice through a similar, multi-faceted kaleidoscope, even if they have not been avowedly "developmental", or have not used the term "niche construction", "phenotypic plasticity", or "trans-generational"? The truth is probably most, if not all of them. Which means that the EES has little to offer. So why the EES at all?

Philosophers of science have recently argued that maybe one issue is that the two frameworks are seeking different kinds of explanations for evolutionary phenomena, or have different explanatory "standards", even if it is not yet clear what those differences might be (Lewens 2019; Baedke et al. 2020). However, thanks to Tinbergen (1963), we have long known that there are many different kinds of questions that biologists ask about traits, spanning mechanism, ontogeny, function, and phylogeny. Yes, biologists can easily talk past each other, especially if careers are to be made, but I think the disconnect between the SET and EES (which is clearly real) is less about questions, and ironically not even necessarily that much about evolution at all. After all, why ignore adaptive sex allocation in pro-EES arguments? No, I suggest that the truth is that the EES is actually all about freeing organisms from evolution itself.

The focus of the EES is clearly the phenotype. Organismal agency and allowing organisms to change across generations independently of genetic inheritance is what the EES is all about. This breaking of the links between genotypes and phenotypes, to allow phenotypes to be free *from* evolution, or at least from free from evolution as genetic change, is what is at its core. The EES is much more about a theory of phenotype than it is a theory of evolution. And I think this key aspect of the EES needs to be understood. All of its components are about emphasising *how organisms are made*, with the bolt-on comment that we need to understand that phenotypic construction when studying evolution. The problem is that we already know very well that both organismal agency and trans-generational effects are both outcomes of, and contributors to, natural selection, as so clearly evidenced in the review of sex allocation presented here. These are not some new weird facts of biology either; we have known of examples of both (for example, for organismal agency read behaviour, for trans-generational effects read parental care), in terms of ecological context and evolutionary consequentiality, for many decades. As such, I do not think that the EES can contribute to evolutionary biology, because fundamentally it wants

to extend *away from* evolutionary biology. It wants organisms with some other kind of agency, phenotypes that are built and changed some other way, independently of natural selection. You might not read that many places, but how else to explain the implicit "something-else-ness" of the EES, and the slippery slope that maybe phenotypic change over time is evolution too, and that we can dispense with genetic change. Whether it is explicitly stated or not, clearly evolution as genetic change is the target of the EES. But phenotypic change without genetic change is not evolution as evolutionary biologists think of it and indeed is deeply problematic. This is perhaps Darwin's key insight, alongside the mechanism of natural selection itself (and not just in Darwin 1859, but even more so in Darwin 1868). Darwin ended up knowing full well that the biological diversity we see today cannot be explained by phenotypic variation alone: phenotypic variation is cheap, thanks to the ubiquity of phenotypic plasticity. Heredity was, and is, crucial. If you want to explain biological diversity, and traits such as sex allocation, then evolution needs to be framed in population genetic terms. And on these terms, the Extended Evolutionary Synthesis has no tenable argument to make, nothing *new* to offer, in the face of Standard Evolutionary Theory.

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The Nuances of Biological Syntheses: A Commentary on Shuker

30

Mitchell Ryan Distin

Abstract

In this chapter, David Shuker makes a forceful case against the EES by demonstrating its irrelevance within the mainstream subject of sex allocation. Many of the supposed "neglected" or "unappreciated" aspects of modern evolutionary theory, as claimed by proponents of the EES—such as organismal agency, transgenerational effects, and/or the salience of developmental factors driving selection—are not only well-accounted for within the research front on sex allocation today, but also have historically thrived under the dominion of SET. However, this is not to say that everything is fine and dandy with SET. The EES is right to call out the significant methodological and philosophical issues with respect to SET.

In this chapter, David Shuker makes a forceful case against the *EES* by demonstrating its irrelevance within the mainstream subject of *sex allocation*. Many of the supposed "neglected" or "unappreciated" aspects of modern evolutionary theory, as claimed by proponents of the EES—such as organismal agency, transgenerational effects, and/or the salience of developmental factors driving selection—are not only well-accounted for within the research front on *sex allocation* today, but also *have historically thrived under the dominion of SET*. Empirical examples such as the fig wasp should be a "poster-child for an EES" because of the well-documented cases of extraordinary behavior, plasticity, and transgenerational effects on fitness that are available in this literature. Yet importantly, theoretical

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progress on sex allocation has proceeded since the synthetic era without any need for the novel perspectives offered by the EES. On the contrary, sex allocation theory has been referred to as a "touchstone for SET" since "it has been a branch of evolutionary biology where some of the most explicit tests of SET have been attempted" and apparently has passed with flying colors, since sex allocation theory has been hailed as one of the most successful bodies of predictive theory in evolutionary biology (Frank 2002)—which is a major hallmark of a virtuous scientific theory since this demonstrates the external validity of our causal models (Anjum and Mumford 2018).

As echoed throughout this volume, Shuker points out the gaping historical issue with the EES, which is that SET is not an immutable structure throughout the history. Biologists have been building upon core theory since the foundations of modern biology in the synthetic era (Smocovitis 1996). Indeed, every evolutionary biologist worth their salt agrees on an "Extended Modern Synthesis" to some extent. But the motivations for the EES bypass any sort of "extension" of modern synthetic theory, as noted by Shuker, but rather seek to upend the genetic core of SET altogether (keeping in mind that, as a historian and philosopher of science, I question the fruitfulness of distilling the immense heterogeneity of viewpoints typical to the *synthetic era* and beyond as the product of only a few poignant tenets of "core theory"; see for more Gayon 1990). Shuker argues that the EES need to make plain their aims and intentions. What exactly is it about SET that has them so riled up and calling for such significant changes in our theoretical mainframe? Because as it stands today, the research front on sex allocation is doing perfectly fine within the bounds of SET.

What is brilliantly acknowledged by Shuker are the implicit or unspoken drivers of the EES that are demonstrable within the standing research on sex allocation. The EES wishes to place a causal explanatory emphasis on two related aspects within evolutionary thinking: (1) *the phenotype* and (2) *the organism*. Here I wish to add a third organizing principle of the EES that Shuker implicitly recognizes in his review, which is the issue of (3) *intrinsicality*.

As I make a similar reference to in Chap. 38, an organizing principle of the EES is the idea that the phenotype is causally and explanatorily more important than gene-centered explanations of similar evolutionary phenomena. It is clear as day that proponents of the EES wish to discard the foundational tenet of *evolution as genetic change.* Yet this presents a major issue for those left defending the EES. As demonstrated here by Shuker and elsewhere in this volume, many evolutionary explanations *require* both a phenotypic and a genotypic explanation. "Heredity was, and is, crucial" as Shuker says. Even some of the cases of sex allocation that were initially thought to be the result of non-genetic processes turned out, in the end, to require genomic imprinting to allow for the non-genetic information to be passed onto future generations. Thus, we cannot hope to have any sort of explanatory power bereft of gene-centered explanations in evolutionary biology.

The second organizing principle of the EES is *the organism*. Proponents of the EES wish to place a spotlight on the causal actions that organisms have in the evolutionary process. Yet importantly, individuals are not the organizational entity that does the evolving, although seminal causal events for the evolutionary process

indubitably occur at the individual organismal level. Populations are the entities that do the evolving. The salient aspect of any evolutionary explanation is the causal effect that it has on a population, not an individual. If we observe an allelic mutation occurring within an individual's life history, then this does not become *evolution* until it propagates through to the entire population, either by adaptive or non-adaptive processes. Evolutionary phenomena are typically defined by their causal effects or consequences, and the causal events that take place at an individual level are only important insofar as they produce some effect at the population level.

A third organizing principle that was implicitly recognized by Shuker is the emphasis put on *intrinsic causal factors*, precisely because the EES wants to discount the explanatory importance of selection in the evolutionary process. This is to say that EES explanations are not merely *anti-gene* but are also *anti-selection*, oftentimes due to the emphasis they place on *phenotypes* and *organisms*. Using an example from my research focal on *evolvability*, focusing on developmental mechanisms as the causal basis of evolvability *accents features internal to individual organisms* (Brigandt et al. (in press); e.g., Brown 2014): "The evolvability of an organism is its *intrinsic capacity* for evolutionary change... it is a function of the range of phenotypic variation the genetic and developmental architecture of *the organism* can generate" (emphasis added; Yang 2001, 59). As similarly recognized here by Shuker, sex allocation requires an appreciation for *the extrinsic* as well as *intrinsic factors* that bring about its manifestation. We cannot attempt to explain the many examples of *context-dependent sex allocation* without an extrinsic or selective-based explanation. Like most biological *kinds* or features, their evolutionary history was the result of a confluence of intrinsic and extrinsic causal factors playing out over time. Yet, the EES continues to emphasize the intrinsic factors all the while excluding extrinsic factors, and from my experience researching the novel concept of evolvability, this all boils down to their want to curtail the favored epistemic status of selective explanations in evolutionary biology. Well, good luck with that, I say, because the empirical evidence coming out of evolutionary ecology over the past 40 years is telling a different story of evolution, one that gives selection a far greater explanatory role because of the nearly constant and continuous causal influence that biotic and abiotic ecological factors have on natural populations (Kingsolver et al. 2001; Bell [1997] 2009; Hendry 2017).

However, I diverge from Shuker on several key points, and one of these is that the EES deserves a molecule of credit, where credit is due. Although extremely ambiguous and disorganized, their movement was founded on an accurate premise, even if many of their own soldiers fail to recognize the underlying reason why they disagree so heartedly with the SET. The ensuing battle over evolutionary territory is rarely the product of evidential or theoretical disputations, yet rather the result of questions on the structure and maintenance of our knowledge content in evolutionary biology (being aware of the important interrelations between *content* and *structure*; see for more Love 2019). Should we give precedence to some kinds of explanations over others? How should we integrate and organize our standing knowledge?

We see these questions arising because of the significant issues with how science is conducted today. Theoretical construction in the sciences, particularly in the

biological sciences, is hampered by the immense heterogeneity of viewpoints that propagate without any foundational basis to build out and up from. Even proponents of SET are not immune to these struggles. We continue to talk past each other because we have no unified basis for theory, no coherence amongst disciples of oftentimes "competing" disciplines. Like evolution, we will not see any progress until we have a consensual and heritable theoretical core that can be continually updated and improved. Today, the way scientific knowledge is communicated and maintained is not conducive to its success. Therefore, we are at a timepoint that calls for methodological and theoretical progress, yet not of the sort that has been proposed by the EES.

This is why I vehemently disagree with the claim that everything is *A-OK* with SET. The underlying philosophical principles of SET have steered us in a direction that precludes conceptual integration and unification. So, while it may be true that "no new way of doing theory was required" in the history of sex allocation, as noted by Shuker, I do think there are prudent reasons to retire many of the methodological and philosophical underpinnings of SET. For one, SET was conceived within the broader scientific zeitgeist of logical positivism that favored the philosophical principles of *reductionism* and/or *monism* (Smocovitis 1996). Complex phenomena such as sexual reproduction cannot be explained under these philosophies, since past population genetic models have abstracted away from the innate complexity of sexual processes, leading sexperts to switch their theoretical strategies toward pluralistic, multilevel, and multicausal explanations (Bell [1982] 2019; Otto 2009; Neiman et al. 2017). New and improved theoretical strategies are unquestionably necessary for most domains of evolutionary research.

To reiterate, *evolution cannot be framed only in population genetic terms* precisely because recent ecological evidence implies that we need new theoretical strategies to effectively map the complex causal fields of evolutionary phenomena. Historically speaking, we have been thinking about evolution too greatly in statistical terms and too little within natural and ecological contexts. A significant divide between theory (e.g., population genetics) and empiricism (e.g., ecology) has always existed in evolutionary biology until the past 40 years, when we have seen evolutionary biology beginning to incorporate ecological findings into core theory. As a result, evolutionary theory was initially constructed in an abstract vacuum that was not particularly informative of the evolutionary dynamics found in nature *in space and time*. Thus, in some instances, synthesis and integration are indeed warranted efforts to improve upon the epistemic core of SET, while staying cognizant of the underlying issues with SET—however, not from an EES.

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On Genetics, Ecology, and the Role of Philosophy in Evolutionary Biology: A Reply to Distin

31

David M. Shuker

Abstract

In this reply, I consider the valuable points raised by Distin in the light of the actuality of evolution as a population genetic process, and how philosophers of science may come to help evolutionary biologists tackle the problems that matter to them.

Keywords

Darwin · Evolution · Natural Selection · Philosophy · Population Genetics

Your philosophy,
Is totally lost on me.

Impatience, *We Are Scientists.*

Alongside the welcome support for many of the points raised in my chapter, Distin provides important food for thought for evolutionary biologists. However, he also raises issues that need to be considered by the philosophers of evolutionary biology as well.

Distin and I are in strong agreement that the current body of evolutionary thinking, encapsulated by our rich and diverse body of evolutionary theory, tested and challenged every day by a richer and more diverse body of empirical work, remains a work in progress. As stressed in my chapter, what I have characterised as Standard Evolutionary Theory (SET; other characterisations are possible) is not

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some conceptual monolith of finished thinking, but rather a theoretical framework, one that is based around the notion of biological evolution as genetic change in populations over time. Here, genetic change refers to changes in frequencies of DNA sequences ("alleles"; or RNA sequences, as in annoying entities such as coronaviruses). Within that framework, new theories explaining the evolution of phenotypes, old and new, are continually being developed and tested. However, whether the framework itself needs tinkering with, as hinted at by Distin, is perhaps more contentious. In part this is perhaps down to the historical development of evolutionary biology from Darwin onwards.

I will make two brief comments in response to the insightful discussion by Distin. First, what is the role of population genetics in evolution? To address this, we need to be clear about what we are talking about in terms of population genetics. On the one hand, population genetics is a field that describes the genetic structure of populations and how the genetics of populations change over time (synonymising itself with evolutionary genetics as a field of study). On the other hand, population genetics can also be a shorthand for population genetic theory, the often-formidable body of theoretical work that has been developed to help empirical population geneticists go about their business (Charlesworth and Charlesworth 2010). This association between data and theory has been reflected in the development of population genetics since the very beginning however, with the ecological genetics of Dobzhansky (1951) and Ford (Ford 1964; see also Birch 1960) developing alongside the population genetic theory of Wright, Fisher, Haldane and others. We can therefore see that understanding the genetics of populations within a real-world ecological context ("putting ecology into population genetics") has always sat within some parts of population genetics, even if it is more typical to equate population genetics with equations. Now, it might be true that the birth of molecular population genetics (from the 1960s onwards, first of proteins, then of DNA) initially focused more on population genetics statistics (F_{ST} and the like) than the ecology of the populations being studied, but Distin is correct in saying that in recent decades evolutionary biology has been rather rarely undertaken naïve to the underlying ecology of the study organisms (the importance of model study organisms such as *Drosophila melanogaster* notwithstanding). Population genetics texts are quite maths-heavy, but that should not be over-interpreted as a reflection of what those equations are then used to explore. This is clear from a cursory reading of any of the major evolutionary biology journals today, and indeed the journal *Molecular Ecology*, which very much epitomises modern empirical population genetics.

More generally, there have been repeated calls for a greater synthesis of ecology and evolution, some of which are almost as old as the Modern Synthesis itself (see above). A longer view, however, shows that that the extent to which evolutionary biology and ecology have been bound together has varied over time, at times stronger, at times weaker. Darwin (1859, 1871) began the study of evolution with a deep-seated understanding of what we now call ecology, especially in terms of within- and among-species interactions, driving natural and sexual selection (what else is the "law of battle" for instance?). The natural history that provided Darwin with so much ecological insight was perhaps eclipsed to some extent by the rise of

modern professional biology and the re-discovery of Mendelian genetics, generating the field of genetics and other aspects of molecular biology. However, the emergence of ecology as its own professional discipline, while obviously developing its own rich collection of ecological theory, also saw the more-or-less contemporaneous emergence of evolutionary ecology, for instance in terms of figures such as David Lack (e.g., Lack 1965). Alongside the ecological genetics of Ford, Dobzhansky, and others, the stage was set for what we now call molecular ecology. As such, calls for further synthesis can be frustrating in terms of this already rich history of the interaction between ecology and evolution, if only very lightly sketched here.

In addition, there is also something of an irony in the structure of population genetic theory itself. Whilst many of the models within the canon of population genetics theory might lack ecologically explicit assumptions or context, that lack can be viewed in two ways. On the one hand, it could be argued that population geneticists are patently oblivious to the ecological truths their theory wishes to speak to. On the other, it could be that the abstractions inherent in population genetics models in fact allow a vast array of ecological circumstances *into* population genetics models. Let us take the humble selection coefficient, *s*. A simple population genetics model that specifies selection on a given allele, as denoted by *s*, is in fact allowing that any plausible component of fitness, across any plausible ecological circumstance that influences that component of fitness, can be modelled and insight therefore gained, all through the abstraction of *s*. Therefore, how population genetics models deal with ecology, either explicitly or implicitly, is perhaps not a very good way to assess how well integrated ecology and evolution actually are.

Second, Distin touches on the philosophy of the theory of evolution, making the case – albeit briefly due to space constraints – that work remains to be done here. Indeed, more philosophical aspects of evolutionary theory (including how we do evolutionary biology, and what assumptions, explicit or otherwise, we make when we do it) enrich many of the chapters of this volume. But there is also a clear disconnect between the philosophy of evolutionary theory and what evolutionary biologists do – and care about, to be frank – day-to-day. For instance, debates about statisticalist versus causalist views of evolution continue well away from the mainstream evolutionary journals (see Otsuka 2016 for a review of the debate). For better or worse, philosophers of biology need to make a much stronger case that there are issues that need addressing by evolutionary biologists on the ground, for example something that is genuinely missing from their thinking, couched in language and with *empirical examples* that ground that absence in tangible terms. This will require a substantial appreciation of the corpus of modern evolutionary biology, as the thoughtful comments on evolvability by Distin clearly show. After all, as my chapter highlights, this failure to seem aware of, or accept the reality of, modern evolutionary biology is a crucial failing of the Extended Evolutionary Synthesis. And as with the EES, opinion pieces and self-citations will not sway many evolutionary biologists, but data and new phenotypes to theorise about and puzzle over, just might.

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Part XI

The Evolving Evolutionary Synthesis

32

Douglas J. Futuyma

Abstract

I review the controversy advanced by advocates of an "extended evolutionary synthesis" (EES) in the light of other controversies in evolutionary biology since the Evolutionary Synthesis, including the neutralist–selectionist controversy, punctuated equilibria, and adaptation vs. constraint. These episodes were productive in that they stimulated research that expanded understanding of evolutionary processes. The main elements of the EES include some that arise from relatively recent discoveries, such as the prevalence of epigenetic inheritance. Other elements describe topics that are well known (such as niche construction) or are already the subjects of mainstream research (such as evolutionary developmental biology and phenotypic plasticity). Knowledge is increasing on many of the topics included in the EES, but it is not clear that proclaiming an extension is substantially changing the course of research or understanding.

Keywords

Adaptation · Controversies · Evolutionary developmental biology · Evolutionary synthesis · Extended evolutionary synthesis · Neutral theory · Punctuated equilibria · Sympatric speciation

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32.1 Evolutionary Biology and the Evolutionary Synthesis

Every area of science undergoes an evolution, as new information amplifies the known, corrects some misunderstandings, and reshapes explanations of the field's *explananda*. Most of the content of the genetics textbook I used as an undergraduate (Srb and Owen 1952) is correct, but a genetics textbook today will convey greatly expanded knowledge and a quite different conception of much of heredity. Practitioners of a science often favor different hypotheses (is the genetic material protein or DNA?) and sometimes are presented with radical or outlandish ideas (such as Goldschmidt's (1940) mass reorganization of the genome during speciation). But at least in the twentieth-century biology, these episodes are resolved, with greater or lesser increments in knowledge and understanding. As far as I know, the history of biology provides little support for the notion that science progresses by revolutions that replace one major paradigm with another (Kuhn 1962). Watson and Crick's elucidation of DNA structure surely had a greater impact than any other event in the twentieth-century biology, but this was a matter of discovery and data analysis, not of overturning a former verity. In what follows, I will not venture further into the nature of theory, explanation, causation, and how sufficient or insufficient standard evolutionary theory is in these respects, because I cannot claim much command of the philosophy of science. I will focus mostly on historical and current understanding of evolution, based on the interplay between theory and empirical evidence.

Evolutionary biology seems to be peculiarly subject to claims that its fundamental theory needs rethinking, and propositions to replace or extend it. I will superficially review some of the major controversies since the 1960s, to provide a context for the current debate about the claimed need for an "extended evolutionary synthesis." Most of these controversies revolved around certain tenets of the Modern Synthesis (MS), or Evolutionary Synthesis (ES), which I consider to have transpired between 1930 (R. A. Fisher's *The Genetical Theory of Natural Selection*) and 1950 (G. L. Stebbins's *Variation and Evolution in Plants*). To a large extent, the ES affirmed Darwin's major propositions about the causes and course of evolution, especially that organisms' features evolve chiefly by the action of natural selection on hereditary variation among individuals in species populations; that this process can produce great changes in organisms' features over vast periods of time; that *natura non facit saltum*, but great changes transpire instead by relatively slight increments ("gradualism," but see below); that ancestral species split into multiple descendant species that diverge as these processes transpire independently in each; and that all living beings probably have descended from one original form of life. Many of the assertions developed in the ES were explicit refutations of rampant anti-Darwinian views, including variants of Lamarckism, orthogenesis, and mutationism (the origin of major new organisms, e.g., higher taxa, in a single mutational step); but they were also affirmations of natural selection, which most biologists had abandoned as a significant agent of evolution (Bowler 1989).

Among the major architects of the ES, Fisher, Wright, and Haldane created the synthesis between Darwinism and genetics, mathematically formulating the fundamental processes by which features, and the genes that underlie them, evolve within

and among populations. Genes are the necessary focus of this theory, because evolution requires inheritance. Although the nature of the gene was completely unknown, two empirical observations were central to the development of a mechanistic theory of evolutionary change: that inheritance is based on particles (not blending) and that the environmental and other experiences of parents do not affect inheritance (following on Weismann's refutation of Lamarckian inheritance of acquired characters).

I think it is important to recognize that although population genetic theory describes mathematically necessary features of evolutionary change (e.g., the impact of drift versus selection as a function of effective population size), most of the population genetic theory developed during the ES says nothing about the characteristics of real organisms, or even their genes. The alleles in the equations could have large or small effects on a character, and a "mutation" could be any alteration of genetic material, from what we now recognize as a single base pair change to a chromosome reconfiguration or even a whole genome duplication. The affected character could be any feature whatever, in any sexually reproducing organism (and, with minor adjustments, any asexual organism as well). An important corollary is that the theory says nothing about the origin of variation, whether in biochemical processes, modifications of morphological traits, or the appearance of novel characters. Likewise, the source and nature of selection could be external, such as predators, or internal, such as metabolic or developmental functionality. This generality is both the great strength of the theory—it is a truly encompassing description of how evolution works—and its great limitation, for in its general form it tells us nothing about particular features or taxa. As the theory describes the generation-to-generation dynamics of change within populations, it can make short-term predictions, about, say, the time to allele fixation, but not long-term, "macroevolutionary" events such as trends or rates of diversification. Even Darwin's proposed gradualism was rationalized in the ES not by selection equations, but by a geometrical metaphor that Fisher used to argue that large changes are less likely to be advantageous than smaller ones. The biology of real organisms—their structure, development, physiology, behavior, ecology—is lacking from the core population genetical theory. Pigliucci (2007) argued that the ES lacks and needs a theory of the origin of variation in form, based on developmental biology. By this argument, an EES should also include the origin of variation in metabolic pathways, behaviors, and the rest (all of which vary greatly among the major clades of life): it should not only apply to and explain, but also should assimilate, all of functional biology.

To some extent, this has occurred. Since the ES, population genetic theory has been greatly amplified to describe the particulars of sexual selection, the evolution of cooperation, life histories, speciation, genome evolution, and much more. And the biology has been supplied by field and lab studies across the full realm of biology, from ecology to genomics and development.

During the Synthesis, the biology was supplied by world leaders in experimental and ecological genetics (Theodosius Dobzhansky, E. B. Ford), zoology (Ernst Mayr, Bernhard Rensch, Julian Huxley), botany (G. Ledyard Stebbins), and paleontology

(especially George Gaylord Simpson, also Bernhard Rensch).[1] These individuals marshaled evidence for abundant genetic variation in many features and in fitness, natural selection, adaptation, the nature of species and the gradual nature of speciation (but abrupt speciation by polyploidy), and the evolution of certain major morphological changes by alterations of development, such as allometric growth. The evidence for gradual evolution came not from geneticists, but from paleontology and comparative morphology. Drawing on cases like the origin of mammals, Simpson (1944) emphasized that the multiple distinctive features of higher taxa evolve more or less independently (mosaic evolution) and that each character changes incrementally (as in the lineages of horses). Mayr (1942) described a continuum of phenotypic differences among geographic populations of single species, through "semispecies" to reproductively isolated, sympatric species with various degrees of phenotypic difference.

It is widely recognized that physiology, developmental biology, ecology, and (surprisingly) phylogeny were largely absent from the ES. In the case of ecology, which I know best (Futuyma 1986, 2013), there was a long, complex history of union with evolutionary biology (Collins 1986), but also conscious separation as some leading ecologists sought to establish ecology as an independent discipline, distinct from taxonomy, genetics, and evolutionary biology (Kingsland 1986, Kimler 1986).

A recurring charge today is that developmental biologists were excluded from the ES, but this claim has been contested by Smocovitis (1996), Amundson (2005), and Love (2009), inter alia. The developmental biologist Viktor Hamburger (1980, p. 98) described how comparative embryology went into decline after Wilhelm Roux founded the study of *Entwicklungsmechanik* by experimental methods, "as a deliberate countermove against Haeckel's categorical verdict that phylogeny is the sufficient cause of ontogeny" (p. 99), and how, by the 1930s, experimental embryology and genetics had grown apart. Hamburger wrote that the leading books on experimental embryology in the 1930s did not treat evolution and that "the modern synthesis did not receive assistance from contemporary embryologists" (p. 98). However, several "architects" built on the massive literature in comparative embryology, as in Huxley's analysis of allometry. The zoologist Bernhard Rensch, the great unsung hero of the ES, is relatively unknown today because his book (*Evolution Above the Species Level*) was not published in English until 1959. In addition to penetrating analyses of the fossil record, he drew extensively on both anatomy and embryology. For example, he showed that Dollo's "law" of irreversibility has many exceptions, but seldom is there complete reversion to the original morphology because, he proposed, "the whole organism [functional organization] of the animal has undergone change," so that reversal has to be functionally integrated with an altered system. In a 27-page passage, he drew on both comparative and experimental

[1] These are the generally acknowledged leaders, but many others made important contributions. For example, Clausen, Keck, and Hiesey (1940) showed how morphological variation among plant populations could be ascribed to both genetic and environmental influences.

embryology to show that ontogenies can be altered in so many ways that the direction of evolution cannot be determined by internal autonomous factors. Nevertheless, developmental pathways can shape evolution; for example, Rensch argued that parallel evolution can arise from similar natural selection or from similar hereditary factors and developmental organization, as in the wing patterns of diverse Lepidoptera.

Among experimental embryologists, Waddington was the first to address evolutionary processes, during the latter part of the ES—although his contributions did not draw on the experimental manipulations that had demonstrated processes such as induction. Simpson (1953) judged Waddington's proposal of genetic assimilation to be fully compatible with ES theory, but rightfully noted that there was no evidence that it occurred commonly in natural populations. Dobzhansky (1951, 1970) embraced Waddington's and Schmalhausen's (1949) ideas about internal selection and canalization. Mayr (1963) made repeated references to little understood consequences of developmental integration. For example, "Every group of animals is 'predisposed' to vary in certain of its structures, and to be amazingly stable in others... Only part of these differences can be explained by...selection pressures..; the remainder are due to the developmental and evolutionary limitations set by the organisms' genotype and its epigenetic [developmental] system...[which] sets severe limits to the phenotypic expression of mutations; it restricts the phenotypic potential" (pp. 607–610). Like Rensch, Mayr considered development a likely cause of some cases of parallel evolution among related species, in which development sets bounds to the direction of evolution.

32.2 Controversies

The major controversy today, and the subject of this book, is the self-described Extended Evolutionary Synthesis (EES), which has been perhaps most fully articulated by Laland et al. (2015) and by several authors, including Müller (2017), Sultan (2017), Laland et al. (2017), and Jablonka (2017), in a theme issue of *Interface Focus* (vol. 2, issue 5, 6 Nov. 2017). It might be useful to place this in context, by describing what I take to be the main features and consequences of some other controversies in evolutionary biology.

32.2.1 Neutral Theory

There is no question that by the 1960s, selection was thought to be by far the most important of the factors that influence allele frequencies, owing to diverse studies in ecological genetics (especially by Dobzhansky, Ford, and their associates), functionally interpreted clines in various species, early demographic evidence of selection on morphological traits within populations, and other sources of evidence. The neutral theory of molecular variation, proposed by Kimura (1968, also King and Jukes 1969), contrasted strikingly with the current thinking, and resulted in the

neutralist–selectionist controversy that engendered a large, diverse range of research programs in evolutionary genetics for the next decade or more. The advent of abundant DNA sequencing greatly amplified both the abundance and kind of data—such as synonymous versus nonsynonymous variation—that could be analyzed in these terms. The outcome, we now know, is that sequence variation within and among species includes both selected and neutral (or nearly neutral) sites, with immense consequences for understanding genome variation and evolution (Charlesworth and Charlesworth 2010). I take this as an example of a controversy that was based on a new kind of information and which initiated productive and far-reaching research that has greatly enhanced our understanding of evolution. Of all the events in evolutionary biology that I have witnessed, this is the single one that has most changed our understanding revolutionary.

32.2.2 Levels of Selection

Partly in reaction to a group-selectionist interpretation of flocking behavior by Wynne-Edwards (1962), the concept of group selection was crystallized in the 1960s, with analyses and interpretations of group-beneficial or seemingly altruistic traits being reinterpreted in terms of individual benefit or kin selection (e.g., Hamilton 1964; Maynard Smith 1964; Williams 1966). A mild controversy ensued, partly due to a "trait group" model of group selection by Wilson (1975, 1982). The controversy sharpened thinking about levels of selection, has led to interesting philosophical treatments (e.g., Sober 1984; Okasha 2006), and has greatly reduced naïve group-selectionist thinking. West et al. (2010; also, Kay et al. (2020) argue that all the models based on spatial structure that have been represented as alternatives to kin selection are actually models of kin selection.

32.2.3 Sympatric Speciation

In two highly authoritative books, drawing on an enormous taxonomic literature, Mayr (1942, 1963) provided a definition of animal species and the thesis that animal speciation requires strong spatial separation (allopatry) of populations that evolve into sister species. Maynard Smith (1966) was the first to counter with a population genetic model of sympatric speciation, and Bush (1969) was among the first to provide evidence from a case study (not without challenge [Futuyma and Mayer 1980; Coyne and Orr 2004]). Since then, growing theory and evidence have established that although speciation is largely allopatric in many taxa, "speciation with gene flow" occurs, even if its prevalence, taxonomic distribution, and actual levels of gene flow are still unclear (Nosil 2012; Bolnick and Fitzpatrick 2007). In this case, controversy has led to deeper understanding of evolutionary processes, due to the theoretical and empirical research it stimulated.

32.2.4 Punctuated Equilibria

This may have been the most turbulent and broadly divisive controversy in the recent past. Eldredge and Gould (1972) applied the term "punctuated equilibria" to an empirical pattern that they said was prevalent in the fossil record: long periods of relative morphological constancy ("stasis") interspersed with abrupt shifts in one or more characters. (Their chief example was the number of rows of eye lenses in a lineage of trilobites.) They also applied this term to their hypothesized process: that stasis results from developmental or genetic constraints, and the rapid shifts represent the incursion of new species that originated elsewhere, expanded their range, and replaced the previous species. They explicitly described this as a paleontological representation of Mayr's (1954) verbal model of founder-effect (peripatric) speciation, in which Mayr postulated that a population founded by a few individuals would have allele frequencies altered by chance (genetic drift), and that interactions (epistasis) among genes would drive population evolution to different, new genetic equilibria, and possibly reproductive isolation from the source population. In Eldredge and Gould's interpretation, established populations are static because of complex epistatic interactions, and only the founder event frees them to evolve by selection and drift.

All parties to this controversy accepted that lineages in the fossil record generally vary in the rate of character change and agreed that although the pattern is sometimes a result of a discontinuous record, rates of evolution are expected to vary, and do. But the proposal that evolutionary change is prevented except in concert with speciation flew in the face of abundant evidence and population genetic theory and evidence (Levinton 1988; Charlesworth et al. 1982). Most population geneticists vigorously rejected the mechanism of punctuated equilibria that Eldredge and Gould postulated, and some modeled and found wanting Mayr's scenario of peripatric speciation, as well (e.g., Charlesworth et al. 1982; Charlesworth and Rouhani 1988). At this time, there appear to be few established examples of the fossil pattern that Eldredge and Gould claimed, and there is no evidence that large populations are unable to change under selection.

According to Eldredge and Gould (also Stanley 1979; Gould 2002), an important corollary of the association between phenotypic evolution and speciation is that most anagenetic change in form occurs by species selection: differences among species in rates of speciation and extinction. This is very dubious, although there is some phylogenetic evidence that phenotypic evolution can be associated with speciation (Bokma 2008; Mattila and Bokma 2008, Pagel et al. 2006), surely for reasons other than epistatically enforced stasis (Futuyma 1987, 2010). The punctuated equilibria debate brought attention to species selection, and it may have helped to bring into focus differences among clades in rates of diversification, an active area of research today (Jablonski 2008; McPeek 2008, and many others).

32.2.5 Adaptation and Constraint

The punctuated equilibrium paradigm was wrong in minimizing the role of natural selection, but useful inasmuch as it focused attention on constraints on evolution (Futuyma 2010). Lewontin (1974, p. 92) after reviewing literature on the subject, famously wrote that "[t]here appears to be no character – morphogenetic, behavioral, physiological, or cytogenetic – that cannot be selected in *Drosophila*," and that "there is good reason to suppose that any outbred population or cross between unrelated lines will contain enough variation with respect to almost any character to allow effective selection." This widely held view, supported also by data from natural populations of many species, suggests that traits can evolve nearly to their optima. Nevertheless, population geneticists and other adherents to the ES readily recognize "developmental constraints" and "genetic constraints." The authors of a seminal overview of developmental constraints included population geneticists (Maynard Smith et al. 1985), and "evolution along lines of least genetic resistance" (Schluter 1996) is a prominent research theme today.

From constraints, Gould went on to a critique of the "adaptationist program" in a famous paper on "the spandrels of San Marco," joined by Lewontin, who adopted a rather different stand than his 1974 conclusion suggested (Gould and Lewontin 1979). Although strongly criticized (e.g., Borgia 1994; Queller 1995), this paper certainly was influential. I think their charge of naïve adaptationism was largely unfounded then, and certainly seldom applies to recent research, in which pleiotropy, developmental constraint, and genetic drift are commonly considered. Adaptive interpretations usually depend on detailed analyses of trait function, evidence of adaptation from the comparative method, or fit to predictions from theory, often cast in terms of optimal models (ably defended by Maynard Smith 1978). Numerous studies of fitness and quantitative traits in natural populations have provided evidence for the ongoing process of natural selection on many, if not most, characters (Kingsolver et al. 2001). Gould and Lewontin illustrated what they dismissively called adaptive story-telling with horns and tusks, which, they sarcastically wrote, "once viewed as responses against predators, become symbols of intraspecific competition among males" (p. 486). There is now abundant evidence, from diverse animal taxa, that although such weapons sometimes do serve for predator defense, they are overwhelmingly and indubitably the outcome of sexual selection (Emlen 2008; Metz et al. 2018). There is good reason to think that most organismal features have been at least partly shaped by natural selection. In contrast, little support has accrued for some of Gould and Lewontin's alternatives to adaptation. Their paper has been cited in at least 3500 publications, but almost no examples of biological "spandrels"—traits that are necessary, nonadaptive consequences of organismal construction—have been described since their essay (Olson 2019).

I infer from this history of post-Synthesis controversies that they have usually helped to enlarge our knowledge and understanding, even if the original claims and propositions were wrong (as in the case of punctuated equilibria) or excessive (as in developmental/genetic constraint). In some cases, as with sympatric speciation, the view held during the ES was shown to be too restrictive. Other cases brought

attention to neglected aspects of evolution, and punctuated equilibria highlighted the possible importance of species selection and constraints on character evolution. In the case of the neutral theory, evolutionary biologists had to adjust their adherence to pervasive selection because they were confronted with utterly unprecedented information. Whether or not these advances would have occurred anyway, in the absence of controversy, is hard to know (as with counterfactual histories generally), but I suspect they would have. Variation at the molecular level would eventually have been explained by both selection and genetic drift, whether researchers had divided into camps or not. With the great growth in phylogenetics, differences in rates of clade diversification—species selection, writ large—would surely have become a subject of interest, without reference to punctuated equilibria and stasis. Of course, much or most of the great growth of evolutionary biology has been based on ideas and data that are unrelated to these controversies, but they are nevertheless germane to some aspects of the proposed extended evolutionary synthesis.

32.3 The Extended Evolutionary Synthesis

No one could possibly claim that our understanding of evolution is complete, or that there will be no significant changes or additions to evolutionary theory. I contributed to a short position piece in *Nature*, under the title "Does evolutionary theory need a rethink? No, all is well" (Wray et al. 2014), but I doubt any of the article's authors would have written such a title (provided by journal editors), with its presumptuous self-satisfaction. The major points of our response to the advocates of the EES (Laland et al. 2014) were that the topics highlighted in the EES are already studied by evolutionary biologists, that they are only four among many topics that may well deserve greater attention, and that studies of these topics may well extend evolutionary theory, because "this is how our field has always advanced." The four topics highlighted in this exchange were niche construction, developmental biology, phenotypic plasticity, and inclusive inheritance. I will comment on each of these rather briefly and then address the complaint (as expressed by Müller 2017, p. 4) that past discussions "seem not to have altered the preponderant stance to hold on to. . .gradualism, adaptationism, selectionism and gene-centrism."

32.3.1 Niche Construction

"Organisms modify and choose components of their local environments, a phenomenon known as 'niche construction'" (Laland et al. 2017). Beavers and web-building spiders literally construct parts of their environment, but Laland et al. (also Odling-Smee et al., 2003) include habitat choice under this term, as in the case of desert insects that live in shaded grass clumps with very different temperatures than they would experience on the open desert floor. This same theme had been developed earlier by Lewontin (1985) in an essay on "the organism as subject and object in evolution." Both Lewontin and Laland et al. noted that organisms' activities can

affect the nature of natural selection on the "niche-constructing" species (and sometimes on other species as well). The general point is that the evolutionary history of the species determines its effective environment, and therefore the selection it experiences.

I confess that having been a naturalist from early boyhood, I found Lewontin's insight glaringly obvious. *Of course* the effective environment differs between a swallow and a grebe, or between a canopy-dwelling oriole and a ground-living quail—or, for that matter, a forest-floor herb and an epiphytic bromeliad. Evolutionary biologists have long recognized that animals' behavior often determines the context of selection on other phenotypic traits (e.g., Mayr 1960; Wcislo 1989; Futuyma and Moreno 1988; Duckworth 2009). For example, models and empirical studies of herbivorous insects treat the evolution of both their host-plant preference and adaptation to the plants' chemical defenses (Futuyma 1983; Castillo-Chávez et al. 1988; Feder and Forbes 2007). The literal construction of a burrow or other physical element of an organism's environment does not differ conceptually from the evolution of habitat or feeding preference, and its influence on natural selection.

Laland et al. (2017, p. 5) offer ten predictions from niche construction. In most of them, substituting "niche[s]" for "niche construction" would not change the meaning and would state an already accepted understanding. For example, "niche construction will frequently generate parallel patterns in selective responses among independent lineages" seems to say that species with similar niches evolve similar adaptations (as in the independent evolution of webbed or lobed feet by several lineages of water-living birds, and countless other well-known examples). Or, "novel. . .niche construction activities will initially. . .generate unusually strong selection. . .typically followed by a weakening in the directional response. . .followed by stabilizing selection once the species becomes adapted." How does this differ from the standard view of adaptation to a novel environment? Or, "innovations in niche construction will commonly lead to the rapid evolution of functionally coordinated and eventually genetically correlated suites of traits." How genetic correlations might evolve under selection is an interesting question (e.g., Cheverud 1982; Lande 1984; Wagner 1986), but otherwise, this prediction states that a population exposed to a novel environment—which may well stem from its use of habitat or certain food items—is likely to adapt to it.

The term "niche construction" has been in use since the 2003 book by Odling-Smee et al. Has it helped to generate new programs or directions of research? As of this writing (July 2021), the search term "niche construction" calls up more than 1200 articles in Web of Science. The vast majority concern anthropological or sociological contexts. Fewer than 50 references are in major journals of evolutionary biology, and of those, a considerable fraction is by authors of the 2003 book. Among more biological articles, the phrase usually seems to be a gratuitous name for a well-known phenomenon. For instance, a study by Bittleston et al. (2020), showing that the species composition of bacteria in pitcher plant leaves is affected by the composition of earlier assemblages, concludes that "niche construction through interspecific interactions can condition future community states on past ones"—a statement that would be unchanged if the first three words were deleted. Several years ago, I

wrote, "Will 'niche construction' be merely a label or 'brand' that advertises its advocates' research, or will it be uniquely productive of insight and understanding? So far, no new, general theoretical principles that promise to guide novel empirical research have been articulated by proponents of niche construction" (Futuyma 2017). Perhaps it is still too early to judge.

32.3.2 Evolutionary Developmental Biology

"Development is the greatest mystery in biology, but we may need to understand its complexity in biochemical detail before we can understand the alterations of ontogeny that are the history of evolution" (Futuyma 1979, pp. 182–183). This sentence, from the first edition of my textbook *Evolutionary Biology*, ended the chapter on "The origins of evolutionary novelties," which I cast in largely in the terms of the emerging field that would be named "evolutionary developmental biology" (EDB) or "evo-devo."

Development was largely neglected in the aftermath of the Synthesis. Only about 20 articles in the first 20 years of the journal *Evolution* (1947–1967) emphasized development—although these included important papers by Waddington (1953) on genetic assimilation, by R. L. Berg (1960) on adaptive character integration in plants (the same theme as the influential book *Morphological Integration* by Olson and Miller [1958]), and by Rendel (1959) and Sondhi (1962, 1967) on threshold traits, and the release of cryptic variation in *Drosophila* bristle patterns when canalization is broken by major mutations. All of these papers invoked concepts in developmental biology, such as canalization, thresholds, prepatterns, and morphogen gradients. In the 1970s and 1980s, a resurgence of interest was spurred by some paleontologists (e.g., Raup 1962; Gould 1977), developmental biologists (e.g., Raff and Kaufman 1983; Alberch and Gale 1985), and the systems biologist Riedl (1977, 1978). The evolutionary geneticist Russell Lande (1978) comprehensively reviewed patterns of limb reduction and loss in lizards and other tetrapods, interpreted consistent patterns in terms of developmental processes, and developed a quantitative-genetic model for the evolution of threshold traits such as digits. He was among the authors of an exposition on developmental constraints that integrated the viewpoints of a diverse group of evolutionary and developmental biologists (Maynard Smith et al. 1985), a paper that impelled discipline-wide recognition of developmental constraints.

The enormous growth of molecular and developmental genetics in the 1980s and 1990s then transformed EDB, as mechanisms and patterns of gene regulation were discovered and experiments showed how changes in regulation and regulatory networks accounted for many changes in phenotypic traits (Carroll et al. 2005; Carroll 2008; Stern 2011). Together with ideas developed in the 1970s and earlier, this led to further insights and models of phenotypic evolution, including modularity, evolvability (e.g., Wagner and Altenberg 1996), and a concept of character identity and homology based on distinct gene regulatory networks that seems to have substantial support (Wagner 1989; Müller and Wagner 1991; Davidson and Erwin 2006; Shubin et al. 2009; Lynch et al. 2011). This is a major

step toward defining and understanding the origin of novel characters (Moczek 2008; Wagner 2015; Peterson and Müller 2016). Many or all of these topics are treated, often in a dedicated chapter on evolution and development, in all the major current textbooks of evolutionary biology.

Genetic studies in EDB have provided wonderful insights and surprises, as well as new questions. Among the first surprises was that *Hox* genes play a role in establishing the body plans of all animal phyla, followed by the unexpected revelation that these same genes could be important in specifying utterly different localized features, such as the pattern of hairs on a fly's leg. Such discoveries continue to enhance our understanding of the origin and diversification of characters; for example, critical genes in the development of insect wings and legs also specify other evaginations of the body wall, such as the horns of scarabaeid beetles (Hu and Moczek 2021). This is an example of "deep homology," in which much the same gene regulatory network underlies seemingly different, sometimes novel structures (Davidson and Erwin 2006; Shubin et al. 2009; Wagner 2014). Conversely, corresponding to embryologists' discoveries long before, indubitably homologous structures in diverse taxa sometimes prove to have quite different genetic foundations. For example, Müllerian mimic *Heliconius* butterflies in two different clades have almost identical wing patterns, but these are determined by different gene regulatory networks, in which the same patterning genes are deployed differently (MacMillan et al. 2020). This phenomenon, called "developmental system drift" (True and Haag 2001), might be explained by a recent model of "*ch*aracter *i*dentity *m*echanisms" that are postulated to organize any of a variety of possible inputs (gene regulatory networks) into one or more phenotypic outcomes (DiFrisco et al. 2020). For example, a candidate "ChIM" for a tissue might consist of the cell types that mutually maintain each other via cell–cell signaling. The tone of most of this literature, as I read it, is not a challenge to mainstream evolutionary theory, but celebration of the discoveries and understanding that the newly possible research brings to light.

Studies of evolutionary changes in gene regulation have greatly helped to explain morphological evolution, but they are not a complete explanation, because they do not describe how the genes' products result in a phenotype. This is a matter of physical chemistry that evolutionary biologists will often make do without, just as they often do not concern themselves with how genetically encoded proteins assume three-dimensional form, or how that form results in the protein's function. Nevertheless, a complete account of development includes physical processes that may be imposed from without or be generated by the activities and properties of cells and tissues. The charge by some adherents to the EES that standard evolutionary theory fails to explain the evolution of form, and does not do justice to inherent properties of organisms' development, seems largely to concern these physical events (Müller 2017, Peterson and Müller 2016). Love et al. (2017) suggest that physical principles describe the immediate, proximate causes of developmental events, whereas genetics—and evolutionary biology—aim to explain differences. I would add that evolutionary biology provides a historical perspective. For example, neurulation in vertebrates involves mechanical events that stem from cell elongation, cell adhesion,

and other events that are based on specific gene products. But the expression of these genes in those cells evolved in the ancestors of vertebrates and is lacking in other deuterostomes. The physicochemical developmental event of neurulation came into existence by an evolutionary process that included mutation (sensu *lato*) certainly, natural selection probably, and genetic drift possibly. Developmental phenomena such as diffusion-reaction dynamics that may generate patterns (Turing 1952; Kondo and Miura 2010), and thresholds that affect the expression of a trait or even the emergence of novel phenotypes (Peterson and Müller 2016), are certainly "internal" factors that can affect the possible paths of evolution, but their expression in a clade is a historical consequence of the fundamental process of genetic evolution.

32.3.3 Phenotypic Plasticity

Ever since Woltereck introduced the concept of a "norm of reaction" in 1909, biologists have recognized that a single genotype often expresses different phenotypes under different environmental conditions. Numerous studies in the early twentieth century, especially by botanists, showed how variation could be partitioned into genetic and environmental, often plastic, components, and that genetic and environmental effects often can generate similar phenotypic effects, resulting from similar perturbations of developmental pathways. The emerging understanding was welcomed by leaders of the ES, such as Dobzhansky, who, in the first edition of *Genetics and the Origin of Species* (1937), referred to the work of Turesson, Clausen et al., and Goldschmidt, and emphasized that "what is inherited. . .is not this or that morphological character, but a definite norm of reaction to environmental stimuli." Attention to phenotypic plasticity increased in the 1960s, marked by Bradshaw's (1965) magisterial review, and the topic attracted increased empirical and theoretical study in the 1980s. Research was directed largely to the quantitative genetics of reaction norms and to plasticity as a common adaptation to temporal or spatial variation in environment (reviewed by Scheiner 1993), and therefore as a component of ecological niche breadth (Futuyma and Moreno 1988; van Tienderen 1991). Plasticity can therefore often be viewed as an adaptive trait, with benefits and costs (e.g., Van Buskirk and Steiner 2009, Snell-Rood et al. 2018).

I am therefore puzzled by the argument of some EES supporters that plasticity is not appreciated by mainstream evolutionary biologists. Sultan (2017), for example, portrays a one-genotype → one phenotype relation as a "deterministic model of phenotypic expression" that is "inherent to the Modern Synthesis," so that "the organism's adaptation to its environment is set by its genotype" and "fitness differences originate in genetic differences." This characterization is all the more baffling because Sultan cites much of the extensive literature I have referred to. It is obvious that the phenotype and fitness of an *individual* organism can be a consequence of its individual manifestation of its genotype's reaction norm; but there will be no *evolution* of adaptation unless some genotypes are more likely to generate the advantageous phenotype than others, or to generate it at lower cost. A genotype's fitness coefficient in population genetic theory is not the reproductive success of an

individual, but the mean reproductive success of multiple members of a population that have that genotype. My reproductive fitness (zero) or Johann Sebastian Bach's (considerable) has evolutionary significance only inasmuch as they are included in the mean fitness of all population members who share an allele of interest. Sultan writes that biologists must "replace the 'genetic programme' model of internal developmental control with one in which each genotype may express different phenotypes depending on its environment." That "replacement" has been part of mainstream evolutionary biology since at least the 1930s.

At this time, the most debated question about phenotypic plasticity is whether or not it is often an early step in the evolution of a fixed, or canalized, adaptive trait, a proposition that has gained attention especially because of West-Eberhard's (2003) *magnum opus*. (My comments here are based on a longer treatment elsewhere [Futuyma 2021].) Such "plasticity-led evolution" (Levis et al. 2018) is based on Waddington's "genetic assimilation," whereby a phenotype that is initially part of a broader reaction norm becomes less plastic, expressing a relatively fixed phenotype, as a consequence of selection on genetic variation in the reaction norm (Waddington 1953). This is closely related to the earlier recognition that genetic and environmental factors can evoke similar reactions from developmental processes (phenocopies). Simpson (1953) included genetic assimilation in what he termed the "Baldwin effect," and wrote that all of the processes that underlie the Baldwin effect are known to occur, that there is no reason to doubt that they could co-occur, that "there is even some probability that they must have produced that effect sometimes," and that "the Baldwin effect is fully plausible under current theories of evolution." He wrote that he knew of no cases that seemed to require this scenario and questioned whether it has been common. I have been impressed by how little it seems to have bothered adherents to the traditional view; the question was simply whether or not it has commonly occurred during evolution. That was still the case until a few years ago. (As I can attest, having searched for convincing cases that I could include in my textbook.) Recently, a number of examples of "plasticity-led evolution" have been established (Schlichting and Wund 2014; Schlichting 2021), such as the loss of ultraviolet-induced melanization in *Daphnia* populations, due to natural selection imposed by introduced predatory trout (Scoville and Pfrender 2010). In this and quite a few other cases, the evolution has been an abridgement of a broad ancestral reaction norm.

"Plasticity-first evolution" is most likely if the ancestral reaction norm happens to be directed more or less toward a new phenotypic optimum (Ghalambor et al. 2007). In some instances, the plastic response is nonadaptive (as in developmental instability) or maladaptive (as in countergradient variation (Conover and Schultz 1995; Storz and Scott 2019; Grether 2005), and in such cases is less likely to enhance genetic adaptation. The ancestral reaction norm is likely to be directed toward the optimal phenotype if the new environment is not entirely novel, but is more or less an extension of the ancestral environment (Pigliucci 2010; Snell-Rood et al. 2018), and if the phenotypic states expressed in ancestral and new environments are genetically correlated (Chevin and Hoffmann 2016).

It is not at all surprising that selection could narrow or abbreviate an advantageous ancestral reaction norm, as in the case of unmelanized *Daphnia*. More novel and interesting are cases in which the ancestral state is a seemingly nonadaptive reaction norm. Juvenile tiger snakes (*Notechis scutatus*) with larger heads have an advantage in island populations, where prey are large. In populations on recently colonized islands, juveniles develop larger heads if they are fed larger prey, but this plasticity is lower in older island populations. Aubret et al. (2004) take this difference as evidence of genetic assimilation. Oddly, however, the mainland source population does not exhibit plasticity in head size, which appears to have evolved de novo in the island populations, within which there was then canalization of enlarged heads. Another interesting example is afforded by larval spadefoot toads in the genus *Spea* (Ledón-Rettig et al. 2010; Levis et al. 2018). Larvae that eat animal prey early in life develop into a "carnivore morph" with larger jaw muscles, a shorter gut, and several other differences from larvae that feed only on detritus—which is the ancestral habit that is the norm in the sister genus, *Scaphiopus*, and more distantly related genera. Surprisingly, *Scaphiopus* larvae that were fed shrimp developed some features of the carnivore morph, such as a shorter gut, although there is no reason to think here has been any history of selection for this developmental response. There are no species in which the carnivore morph has been fixed, as we might envision if plasticity were the evolutionary prelude to an obligate derived phenotype.

Several other questions about plasticity are currently subjects of research. One is the possibility that it might hide genetic variation that can be exposed by environmental stress (Paaby and Rockman 2014). In a population genetic model, Draghi and Whitlock (2012) found that a plastic trait can accumulate genetic variance that can enhance response to selection. In a meta-analysis of relevant studies, Noble et al. (2019) concluded that plastic responses to different environments are, indeed, fairly well aligned with phenotypic dimensions that are highly genetically variable. A major topic is the role of phenotypic plasticity in rescuing populations endangered by environmental change. Russell Lande, a longtime contributor to genetic models in the ES mode, has addressed this question in a number of papers, including one entitled "Adaptation to an extraordinary environment by evolution of phenotypic plasticity and genetic assimilation" (Lande 2009; see also Chevin et al. 2010). These are among many studies by authors who do not claim to be expanding the evolutionary synthesis.

32.3.4 Inclusive Inheritance

EES proponents complain that the standard theory that grew out of the ES privileges genes to the exclusion of other forms of inheritance. It does, and for very good reason: until very recently, inheritance has been understood to reside overwhelmingly in DNA sequences. Cultural inheritance is interesting and is central to studying human diversity, but is phylogenetically very restricted (although somewhat more broadly distributed than usually thought; Whiten 2019). Maternal effects, sometimes

transmitting the consequences of the mother's environment, have been recognized and modeled (Wolf et al. 1998), but again are far less general than genetic inheritance.

Only very recently have the nature and prevalence of epigenetic inheritance been recognized and its importance appreciated. This has rightly become a topic of interest in evolutionary biology, and the subject of a large literature that owes nothing to EES argumentation. Just as in the case of evolutionary genomics, a largely unrecognized, fairly ubiquitous phenomenon has come to light, methods to study it have matured, and it irresistibly cries out for study and for integration into our ever-growing knowledge of evolution.

This topic has been extensively reviewed by other authors (e.g., Turck and Coupland 2014; Bonduriansky and Day 2009, 2018; Bonduriansky 2021), and I do not know it well enough to have anything to add. Processes such as methylation and demethylation of genes are often responsive to environmental conditions and stresses of many kinds, and in some cases enhance performance and fitness under stress; in such cases, epigenetic alteration of gene expression may often be viewed as a mechanism of adaptive phenotypic plasticity, which can evolve under selection. Such evolution requires that there be genetic variation in the propensity of a gene to be epigenetically modified, and this is indeed the case (e.g., Dickins and Rahman 2012; Herman et al. 2013). Especially in plants, field samples often show population differences in methylation, although often it is not known if these reflect immediate changes during the development of those individuals, or inherited epigenetic states. There is certainly evidence that some of these differences enhance fitness in the local environment, that they may be inherited for a variable number of generations, and that the methylation state of particular genes can be influenced both by environment and by cis- and trans-acting genes (e.g., Schmid et al. 2018, De Kort et al. 2020). Some evidence that levels and patterns of methylation show phylogenetic signal is intriguing—and again implies that the propensity for methylation is heritable, i.e., is affected by genes (Alonso et al. 2015).

There is little to be gained from calling the inheritance of environmentally induced transgenerational epigenesis Lamarckian, and good reason not to (Haig 2007). There is plenty to be gained from posing and testing hypotheses about its importance and evolutionary consequences. What is the genomic distribution of changes in epigenetic state in response to particular environmental factors? Temperature, for example, seems to evoke epigenetic change in a great many genes (McCaw et al. 2020); how many of these are functionally relevant to the stress? This bears on the extent to which epigenetic changes can be considered adaptations. Are there any generalizations about which epigenetic marks are inherited, and which not, and if so, do these bear the stamp of adaptation? How do epigenetic states change in response to selection for or against expression of particular traits or genes? How is the inherited basis of adaptive phenotypes reflected in changes in DNA sequence? Is the origin of epigenetically inherited adaptive modifications ever *not* grounded in sequence change? Does epigenetic state, and its inheritance, affect certain classes of traits more than others? For example, it is more likely to contribute to population or species differences in physiological tolerances than in morphological traits? Are any

features of species or larger clades based on epigenetic inheritance? Or is the transition rate between epigenetic states too high to sustain population- or species-wide characters?

32.3.5 Overview

As I described in the first part of this essay, previous controversies in evolutionary biology have probably expanded or added to our understanding of evolution, if only by stimulating research and drawing attention to neglected phenomena, such as stasis, species selection, and speciation with gene flow. How instrumental the controversies were in these expansions is hard to know. Likewise, several of the topics at issue today seem to be stimulating research that will almost certainly expand our knowledge and perhaps alter or advance our understanding. This is surely the case with phenotypic plasticity. Evolutionary developmental biology has been flourishing for about three decades, but the EES controversy will have done good service if it helps to spur integration of genetic and physicalist approaches, in an evolutionary context. In contrast, the discovery of the mechanisms and ubiquity of epigenetic inheritance, a phenomenon that was hardly known a decade or two ago, has sparked abundant, diverse research that will greatly add to our understanding of inheritance and evolution without any need for the EES debate.

Achieving greater breadth and depth of knowledge and understanding does not necessarily mean that current orthodoxy needs to be discarded. Müller (2017, p. 4) complains that "All the extensive discussions, led over decades, seem not to have altered the preponderant stance to hold on to the classical prerequisites of gradualism, adaptationism, selectionism and gene-centrism. . .Hence, the claim of continuous incorporation of new conceptual components by the MS theory is misleading." I think this statement is not entirely accurate, but that insofar as modern evolutionary biology adheres to these four points, it has good reason.

On gradualism, the architects of the MS, almost entirely on empirical grounds, argued against saltationist claims that higher taxa, with their discontinuous differences from related taxa, in multiple traits, arose in discrete jumps. That does not and did not mean that the evolution of traits and trait complexes evolved through infinitesimally small steps, as in simple polygenic models. Threshold traits have been known since the 1930s, and polymorphisms and crosses between species showed that some trait differences are based on single gene differences with substantial effects (Gottlieb 1984; Orr and Coyne 1992). Whether or not an *Ambystoma* salamander is neotenic depends largely, although not entirely, on a single gene (Page et al. 2013). Genes with large, discontinuous effects account for striking differences in color patterns among *Heliconius* butterflies (Merrill et al. 2015). These cases might have shocked Ernst Mayr in the 1940s, but most or all evolutionary biologists today recognize that evolutionary change in certain morphological characters can be very rapid and sometimes discontinuous. And, of course, they highlight the role of development.

On adaptationism, the critique by Gould and Lewontin (1979) may have had a significant impact; today's biologists are perhaps more conscious than their predecessors that claims of adaptive function need supportive evidence. But the "adaptationist program" remains valuable because it generates hypotheses (Mayr 1983). In my view, moreover, adaptation proves to be the rule, not the exception, in studies of phenotypic traits. "Form and function" are indissoluble in much of biology; functional morphology presents countless cases of conformity to principles of engineering or design. Color patterns in birds, insects, and other animals are demonstrably favored by ecological selection (e.g., predation), sexual selection, or social selection. For example, the convergent evolution in Müllerian mimetic *Heliconius* butterflies extends to fine details of color pattern (MacMillan et al. 2020), and the survival advantage of the mimicry has been experimentally demonstrated. Innumerable studies in behavioral ecology find a match between species' traits and predictions of adaptive theory, including kin selection. Traits that cannot be experimentally manipulated can often be analyzed by the comparative method, which has found abundant evidence of adaptation in convergent evolution (e.g., Harvey et al. 1978 on primate teeth, Edwards et al. 2016 on leaf form). Some features of genomes may well have evolved by nonadaptive processes (Lynch 2007), but that would be an implausible interpretation of most phenotypic traits.

If, indeed, many or most traits are adaptations, then they are in large part the result of natural selection. (I and many others would actually define an adaptation as a feature that evolved by selection.) Even in cases of "plasticity-led evolution," the ancestral expression of plasticity is likely to have been shaped by natural selection, acting on genetic variation (as in the differences between sun-grown and shade-grown plants of many species). Cases such as the carnivore morph in *Spea* toad larvae are interesting precisely because the plastic response seems to antedate any selective value. I suspect such cases will be few, but they will repay study. Is "selectionism" justified? Quite aside from evidence that traits are adaptive, selection in natural populations has been measured on hundreds of traits. This is not to deny that the arena for selection is set—or constrained—by the species' development, form, behavior, physiology, ecological niche: its evolutionary inheritance.

Evolutionary biology is "gene-centric" inasmuch as evolution consists of changes in organisms' inherited features: we do not count as evolution the differences between members of a single genotype that developed in different environments. Consequently, an essential part of evolutionary theory—the part that Darwin painfully lacked—concerns how changes in the genetic basis of characters occur over time. Can anyone, however dedicated to the importance of development or niche construction, imagine a theory of evolutionary change without genetics? I don't think so, and so I interpret Müller's complaint to mean that other aspects of evolution aren't given their due. And so, back to niche construction, behavior, plasticity, development.

32.4 Summary

Evolutionary biology has been the stage of several controversies during the last six decades. Most appear to have led to clearer ideas and to have stimulated research, and in some cases they have amended tenets of the Evolutionary Synthesis. The only controversy that led to what might be called a substantial "paradigm shift" was the neutral theory of molecular evolution. Otherwise, ES theory has largely survived controversy and been expanded by a great range of research programs, including phylogenetics, evolutionary ecology, behavioral ecology, sexual and social selection, evolutionary genomics, and an evolutionary developmental biology made possible by molecular methods and thriving quite independently of conceptual controversy.

In reviewing four major themes of the proposed extended evolutionary synthesis, I find few, if any, new insights offered by "niche construction." The emergence of evolutionary developmental biology (or "evo-devo"), largely enabled by molecular genetics and genomics, is a major, much needed addition to evolutionary biology, as it provides crucial information on the origin of variation in form, including the origin of novel characters. It is complementary to mutation and natural selection in explaining evolution in multicellular organisms and is not at all in conflict with population genetic explanation of evolutionary change. Much or most of the advance in this area seems to have been achieved by normal science, independent of any complaint that development is insufficiently appreciated. An important aspect of development is phenotypic plasticity, the subject of extensive evolutionary research for decades. The old proposition that plastically generated phenotypes may become canalized, species-typical characters has found some new empirical support, and might prove to be more common than previously thought. Among the four EES topics I consider, the evolutionary role of inherited epigenetic states is the most novel and exciting. Fitness-enhancing epigenetic modifications can be viewed largely as mechanisms of adaptive phenotypic plasticity that are genetically variable and subject to natural selection. There is no reason to interpret them in Lamarckian terms, but much reason to study the mechanisms by which they have evolved, their role in population adaptation, and their impact, if any, on the features of species and clades. But as in the case of evolutionary developmental biology, the active research in this area seems to result simply from the revelation of little known biological processes, and a need to know how they fit into evolution. All in all, the considerable, often exciting research on these various subjects is proceeding as it has in the past, propelled largely by unanswered questions, methodological advances, and biological discoveries that call for evolutionary understanding—not by concerns about the philosophical sufficiency of current evolutionary theory, or its claimed flaws.

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Inclusive Fitness Theory as a Scientific Revolution: A Commentary on Futuyma

33

António M. M. Rodrigues and Andy Gardner

Abstract

Futuyma reviews what he suggests are the main controversies to have gripped the field of evolutionary biology since the time of the Modern Synthesis. He argues that although some of these developments have led to significant and lasting insights (and he names the neutral theory as having been the most important in this respect), none have amounted to an actual revolution in the sense of the "overturning of a former verity". Futuyma then considers the call for an Extended Evolutionary Synthesis, and he argues that this is, in effect, already underway, though only as part of the normal evolution of our field, which proceeds by building upon—rather than rejecting—that which has come before. We fully agree with Futuyma's assessment of the Extended Evolutionary Synthesis. However, we disagree with his suggestion that there have been no revolutions within evolutionary biology since the time of the Modern Synthesis. We contend that the explosive and far-reaching growth of inclusive-fitness theory represents a major revolution in evolutionary understanding. Indeed, the inclusive-fitness revolution has involved the only revision to the core logic of Darwinism since the 1850s—let alone the 1950s. And its ramifications certainly far surpass those of the neutral theory.

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Keywords

Adaptationism · Appearance of design · Darwinism · Natural selection · Research programs · Scientific revolutions

In his chapter, Futuyma reviews what he suggests are the main controversies to have gripped the field of evolutionary biology since the time of the Modern Synthesis. He argues that although some of these developments have led to significant and lasting insights (and he names the neutral theory as having been the most important in this respect), none have amounted to an actual revolution in the sense of the "overturning of a former verity". Futuyma then considers the call for an Extended Evolutionary Synthesis, and he argues that this is, in effect, already underway, though only as part of the normal evolution of our field, which proceeds by building upon—rather than rejecting—that which has come before.

We fully agree with Futuyma's assessment of the Extended Evolutionary Synthesis. In our own chapter, we similarly detailed how the "laundry list" (Welch 2017) of supposedly neglected factors highlighted by proponents of the Extended Evolutionary Synthesis (Laland et al. 2015) have readily been integrated into evolutionary biology, with a particular focus on the inclusive fitness research programme. We have shown that these factors have not merely been accommodated by the theory of inclusive fitness, but indeed that some (such as the whole-organism view of adaptation and the role of organisms in modifying their own selective environment) have provided the very motivation for the concept of inclusive fitness and others (such as epigenetics and macro-evolutionary patterns) have themselves been illuminated and explained by application of inclusive fitness logic.

We do, however, disagree with Futuyma's suggestion that there have been no revolutions within evolutionary biology since the time of the Modern Synthesis. We contend that the explosive and far-reaching growth of inclusive fitness theory represents a major revolution in evolutionary understanding. Indeed, the inclusive fitness revolution has involved the only revision to the core logic of Darwinism since the 1850s—let alone the 1950s. And its ramifications certainly far surpass those of the neutral theory.

At its core, Darwinism is a theory of design. The logic of natural selection not only explains the process by which adaptive design emerges through purely mechanical means—and thereby destroys the "Argument from Design" for the existence of a supernatural creator—but it also reveals what this design is for (Gardner 2009). Those heritable variations that are associated with higher reproductive success have a tendency to accumulate in natural populations and accordingly—Darwin (1859) argued—each organism will appear designed to maximize its own reproductive success, i.e., Darwinian fitness.

However, Darwin's argument confuses correlation with causation (Gardner and West 2014). A heritable variation that causes a decrease in its bearer's reproductive success whilst also increasing the reproductive success of its bearer's relatives can, on account of the tendency for relatives to share heritable tendencies in common,

enjoy an overall positive correlation with reproductive success, and hence be favoured by natural selection. Accordingly, the organism will not generally appear designed to maximize its own reproductive success. Instead, it will appear designed to maximize the total reproductive success of all of its relatives, each being weighted according to their degree of relatedness, i.e., inclusive fitness (Hamilton 1964).

It is difficult to think of a stronger example of the "overturning of a former verity" within evolutionary biology than the discovery of inclusive fitness. And the implications of this fundamental revision to the core logic of Darwinism are immense. Of perhaps most immediate consequence are the ramifications for the adaptationist programme—the scientific value of which Futuyma has underlined in his chapter—as optimality modelling can only deliver accurate predictions of organismal phenotypes insofar as we have correctly understood the criterion according to which they are optimized. Outwith evolutionary biology, the concept of inclusive fitness has found useful application right across the life sciences and beyond.

The wide reach of inclusive fitness theory is illustrated by considering the twenty-five Web of Science research areas for which Hamilton (1964) is most cited (Fig. 33.1). The number of citations of Hamilton (1964) for each of these research areas exceeds—often greatly—those accumulated by Kimura's (1968) work on the neutral theory, with the sole exception of the category "Biochemistry Molecular Biology". And the influence of Hamilton (1964) is evident across a wider span of

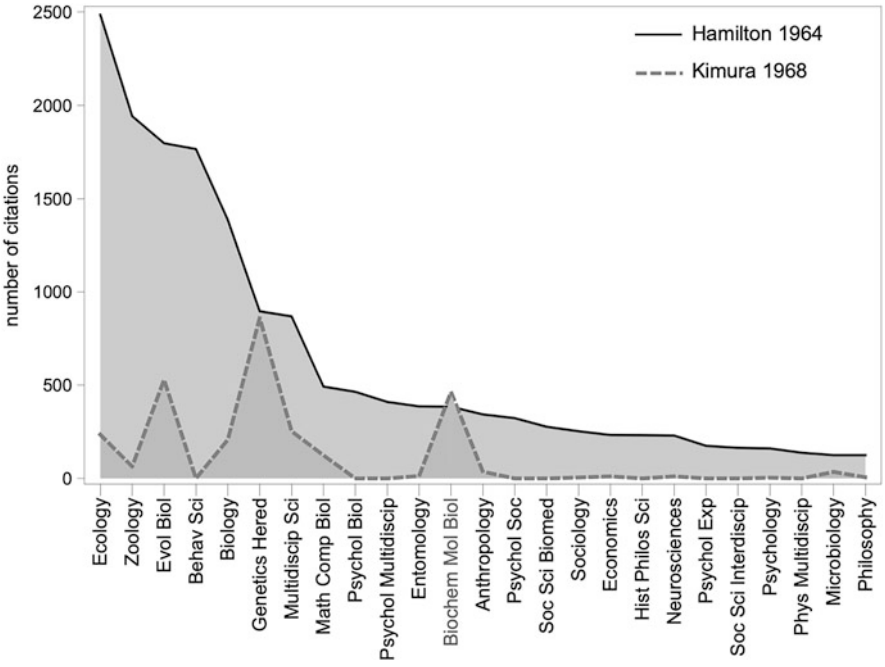

Fig. 33.1 Citations of Hamilton (1964) and Kimura (1968) for the top-25 Web of Science research areas citing Hamilton (1964), as of the time of writing

categories than is the influence of Kimura (1968)—for example, Hamilton (1964) is cited ten times or more in ninety-six different research categories (i10 = 96), whereas Kimura (1968) is cited ten times or more in only forty-two different categories (i10 = 42), as of the time of writing.

Whilst we disagree with Futuyma's assessment of the major advances in evolutionary understanding since the Modern Synthesis—in particular, feeling that he has overlooked the inclusive fitness revolution—we are in close agreement with his proposition that successful advances, such as the neutral theory and inclusive fitness theory, have been driven by consideration of new (or at least relatively neglected) sources of data, whereby empirical observations are made that cannot readily be explained by pre-existing evolutionary theory. In the case of the neutral theory, the new sources of data were molecular and revealed the existence of nucleotide sequence and amino acid variation to a degree that was difficult to account for in terms of selective advantage. In the case of inclusive fitness theory, the observations came from the study of altruistic behaviours which—although lightly touched upon by Fisher (1930), Haldane (1932) and Wright (1945)—had been largely excluded from the Modern Synthesis and which, clearly not being compatible with individual advantage, led many—such as Lorenz (1963)—to frame them in woolly "for the good of the species" terms. This contrasts sharply with the major motivation for the Extended Evolutionary Synthesis, which seeks to complexify evolutionary models apparently for realism's own sake rather than because pre-existing models cannot adequately explain empirical observations.

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Inclusive Fitness Theory Prefigured: A Reply to Rodrigues and Gardner

34

Douglas J. Futuyma

I am gratified that Rodrigues and Gardner largely agree with my assessment of the "extended evolutionary synthesis," although one point could well warrant further discussion. That is their characterization of the EES as seeking "to complexify models apparently for realism's own sake rather than because models cannot adequately explain observations." Whether or not a model adequately explains observations is a judgment on which individuals might differ, perhaps depending on the details they might want explained. For example, inclusive fitness theory may explain epigenetic inheritance, but an evolutionary geneticist who studies epigenetics and a behavioral biologist who studies sibling cooperation might look for different elaborations of the theory. Similarly, evolutionary developmental biologists who study flower development would want to know if principles that govern limb evolution in vertebrates also apply to plants. As a naturalist who hopes to understand not only features broadly shared among living things, but also the immense diversity of their features, I appreciate elaborations of general models that explain why some species are ecologically more versatile than others, or are more fecund, or have differently modified morphology. Perhaps I misunderstand what is meant by "realism's own sake."

Rodrigues and Gardner are more concerned to question my suggestion that the development of the neutral theory of molecular evolution is the closest to a "revolution" in evolutionary thought since the Evolutionary Synthesis (ES), noting that I equated "revolution" with "overturning a former verity." They would propose inclusive fitness (IF) theory for that title.

My remark about the revolutionary nature of the neutral theory was not at all a reasoned argument, but instead a throwaway comment in the context of discussing

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major controversies since the ES. The neutral theory was unquestionably controversial, as it called into question the supreme, ubiquitous role of natural selection that had developed since the ES. IF theory was not controversial. It was, instead, a critically important contribution to a debate on the explanation of socially beneficial behaviors, in particular the role of group selection. Wright (1945) envisioned small populations as the units of selection. Sturtevant (1938) thought that social insects must have evolved by selection "on at least three levels: on the individuals, on the colonies, and on the populations within an area." Haldane (1932) and Williams and Williams (1957) described families or sibships as the units.

Hamilton's (1964) formulation of inclusive fitness was certainly a major event in the recent history of evolutionary biology, moving beyond the notion of selection among kin groups to the population genetics of shared genes. It has served as entry to broader theory and diverse research. The key concept is the probability that individuals share genes identical by descent. But this has antecedents in earlier, nonquantitative discussions. Haldane did not only remark (apocryphally) on how many relatives he would sacrifice himself for; in describing the origin of worker sterility in social insects, he noted that queens and workers are samples of the same set of genotypes, so any even suicidal behavior of workers, if beneficial to the hive, will benefit the reproductives, and so spread (Haldane 1932, p. 208). Darwin (1859, chapter 8) devoted six pages of *The Origin of Species* to explaining how the distinctive features of the workers in social insects could evolve, even though they do not reproduce. The difficulty "disappears, when it is remembered that selection may be applied to the family," as when cattle breeders select for features that are seen only when they are slaughtered, by further breeding from the parents of individuals with the desired trait. Plant breeders do the same, he wrote, in developing varieties with sterile flowers. Later, quantitative geneticists recognized that family selection is more efficient than individual selection for characters with low heritability (Falconer 1981).

I would not diminish the importance of Hamilton's formulation of inclusive fitness theory, but is it, as Rodrigues and Gardner propose, "hard to think of a stronger example of 'overturning a former verity'"? Darwin's argument by natural selection, they say, explains design by individual reproductive success, but for traits that evolve by inclusive fitness, "the organism will not appear designed to maximize its own reproductive success." Evidently, Darwin (with no knowledge of heredity beyond the observable fact of its existence) thought his theory could explain those very organisms. I am not convinced that IF theory overturned a former verity, but I would not claim to know a philosophically defensible definition of "scientific revolution." Revolutionary or not, the neutral theory and inclusive fitness theory both initiated major research efforts and deepened our understanding.

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Part XII

Genes and Organisms in the Legacy of the Modern Synthesis

35

J. Arvid Ågren

Abstract

The gene's-eye view of evolution is an prominent but controversial perspective on biology. It emerged in the aftermath of the Modern Synthesis and both proponents and detractors have stressed the link between the two. In particular, both the Modern Synthesis and the gene's-eye view have been criticized for overemphasizing the role of genes at the expense of organisms in evolutionary explanations. In this chapter, I discuss the connection between the Modern Synthesis and the gene's-eye view and evaluate the status of genes and organisms in contemporary biology. I show that while the gene's-eye view traces its origin back to the Modern Synthesis, it can most accurately be said to represent a specific—adaptationist and gene-centric—version of it. To assess the role of genes and organisms, I examine the intimate relationship between the gene's-eye view and another post-Synthesis development, the concept of inclusive fitness. I argue that the popularity and influence of inclusive fitness theory demonstrate that the individual organism remains safe at the heart of modern evolutionary biology.

Keywords

The gene's-eye view of evolution · Adaptationism · Inclusive fitness

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35.1 Introduction

Following a public poll in 2017 to celebrate the 30th anniversary of the Royal Society book prizes, Richard Dawkins's *The Selfish Gene* (Dawkins 1976) was named the most influential science book of all time. (*The Origin of Species* came in third place.) Regardless of one's views on the poll's results—or the book's argument—the far reaching sway of *The Selfish Gene* means that anyone interested in the history and future of evolutionary theory has no choice but to grapple with its ideas. Chief among these is the so-called gene's-eye view of evolution. This is the approach to biology originally introduced by George Williams in *Adaptation and Natural Selection* (Williams 1966) and elaborated and popularized by Dawkins, that it is genes, and not organisms as Darwin originally envisioned, that deserve the status as the unit of selection in evolution. Emerging in the decades succeeding the Modern Synthesis, the gene's-eye view of evolution has become an emblem of orthodoxy in biology. That symbolism has been especially prominent in the minds of those who criticize current evolutionary thought for being too focused on genes at the expense of organisms.

Depending on who you ask, and when, the Modern Synthesis has meant different things (Huxley 1942; Provine 1971; Mayr and Provine 1980; Gould 1983; Smocovitis 1996; Pigliucci and Müller 2010; Huneman and Walsh 2017; Dickins 2021). That is perhaps not too surprising, given that it involved contributions from fields ranging from palaeontology to plant ecology, physiology to fly genetics. One answer was provided in a 1951 letter from one Modern Synthesis architect to another. In it, Julian Huxley (author of *Evolution: The Modern Synthesis*; Huxley 1942) wrote to Ernst Mayr (editor with W.B. Provine of *The Evolutionary Synthesis*; Mayr and Provine 1980) to say that he considered the central claim of the synthesis to be that

> Natural selection, acting on the heritable variation provided by the mutations and recombination of a Mendelian genetic constitution, is the main agency of biological evolution. (quoted in Huneman 2017)

From the very beginning, the gene's-eye view of evolution has emphasized its place in the Modern Synthesis. And in Huxley's letter emerges a picture of a framework committed to adaptationism and gene-centric explanations (Huneman 2014a; Huneman 2017), much like the gene's-eye view. Indeed, in *Adaptation and Natural Selection*, Williams argued that

> genic selection should be assumed to imply the current conception of natural selection often termed neo-Darwinism. (Williams 1966, p. 96)

And when Dawkins in his autobiography reflected upon how he came to the concept, he noted that

> I should point out that neither in my lectures of the 1960s nor in *The Selfish Gene* did I see as very novel the idea of the gene as the fundamental unit of natural selection. I thought of it –

> and clearly said so – as implicit in the orthodox neo-Darwinian theory of evolution. (Dawkins 2013, p. 268)

Leaving aside the issue of conflating neo-Darwinism and the Modern Synthesis (both Smocovitis and Svensson this volume), the sentiment that the gene's-eye view represents the essence of the Modern Synthesis has been shared by both supporters (e.g., Dickins 2021) and critics (e.g., Noble 2011) of the two. However, the connection is not straightforward. Take Mayr, one of the few active participants in the Modern Synthesis that lived long enough to comment on the value of the gene's-eye view, who suggested that

> the funny thing is if in England, you ask a man in the street who the greatest living Darwinian is, he will say Richard Dawkins. And indeed, Dawkins has done a marvellous job of popularizing Darwinism. But Dawkins' basic theory of the gene being the object of evolution is totally non-Darwinian. (Mayr 1999)

Another long-living Modern Synthesis architect and gene's-eye view detractor, Sewall Wright, was equally critical in one of his last publications when he described the gene's-eye view as a "false statement, backed by great prestige" (Wright 1980).

In general, the gene's-eye view has divided biologists, philosophers, and laypeople since its formation half a century ago. In *The Gene's-Eye View of Evolution* (Ågren 2021a), I traced its origin and development and considered its position in contemporary evolutionary theory. In this chapter, I am concerned with two specific issues:

1. The relationship between the Modern Synthesis and the gene's-eye view.
2. The criticism that the two have caused the field of evolutionary biology to lose sight of what ought to be its primary object of study, organisms.

To that end, I start by outlining the core argument of the gene's-eye view. I show how it stems from a particular version of the Modern Synthesis that is committed to the centrality of adaptations and the form of population genetics spear-headed by R.A. Fisher. Next, I discuss the relationship between the gene's-eye view and Hamilton's inclusive fitness theory, another major post-synthesis development. The concept of inclusive fitness was instrumental in the rise of the gene's-eye view, and most of the time the two are equivalent and complementary approaches. When they do diverge, it is over whether organisms should be abandoned in evolutionary explanations, which the gene's-eye view favours and inclusive fitness theory rejects. Examining the gene's-eye view's debt to Fisherian population genetics and Hamiltonian social evolution theory helps clarify the role of organisms and genes in the legacy of the Modern Synthesis.

35.2 The Core Argument of the Gene's-Eye View

Though the term itself would come later—most likely in Barash (1980)—the concept of the gene's-eye view came onto the scene in the 1960s and 1970s. Whereas *The Selfish Gene* (Dawkins 1976) has enjoyed enormous sales, in multiple languages, *Adaptation and Natural Selection* (Williams 1966) has had a more limited, academic, readership. Among professional evolutionary biologists, however, its influence might well exceed that of *The Selfish Gene* (Cronin 2005; Sober and Wilson 2011; Boomsma 2016).

In both *Adaptation and Natural Selection* and *The Selfish Gene*, the overarching argument is that evolutionary biologists should shift their explanatory focus away from the level of individuals and groups—the way Darwin had originally introduced the theory—to the level of genes. To see why such shift is necessary, it is important to appreciate that advocates of the gene's-eye view have a clear opinion not only what the most important question in evolutionary biology is, but also about how to answer it. According to the gene's-eye view, the problem of design (that is, the existence adaptations) is the most significant issue in biology. Understanding adaptations requires figuring out what they are good for (Williams 1997; Dawkins 1998), what Elizabeth Lloyd called the beneficiary question (Lloyd 2017): what is the thing that ultimately benefits from natural selection?

According to Williams and Dawkins, only genes possess the necessary qualities to answer the beneficiary question. Only they have the required evolutionary longevity; organisms (and groups) are too salient to work (Williams 1966, pp. 23–24; Dawkins 1976, p. 34). In *Adaptation and Natural Selection,* Williams illustrates this point using the life and death of Socrates:

> Socrates consisted of the genes his parents gave him, the experiences they and his environment later provided, and a growth and development mediated by numerous meals. For all I know, he may have been very successful in the evolutionary sense of leaving numerous offspring. His phenotype, nevertheless, was utterly destroyed by the hemlock and has never since been duplicated. If the hemlock had not killed him, something else soon would have. So however natural selection may have been acting on Greek phenotypes in the fourth century B.C., it did not of itself produce any cumulative effect. The same argument also holds for genotypes. With Socrates' death, not only did his phenotype disappear, but also his genotype. (. . .) Socrates' genes may be with us yet, but not his genotype, because meiosis and recombination destroy genotypes as surely as death. (Williams 1966, pp. 23–24)

As hinted at by Williams's last sentence, the gene's-eye view defines "genes" in a rather special way, and this definition provides the key to why only genes can be the beneficiary of natural selection.

The term "gene" has undergone many changes over the years (Griffiths and Stotz 2013; Kampourakis 2017). If some biologists have advanced an empirically informed concept, revised, and refined in light of new discoveries, the gene's-eye view has relied on a more old-fashioned notion, agnostic about the precise material basis (Lu and Bourrat 2018). For example, molecular biologists have typically meant something like a sequence of DNA that encodes a product with a specific function. In

contrast, Williams and Dawkins used a gene definition whereby a gene simply is any chromosome part that is not broken up by recombination and crossing-over during sex. As long as the same structure is transmitted intact long enough, the sequence can in principle be arbitrarily long. As Dawkins, building on Williams (1966, p. 24), put it: a gene is

> any portion of chromosomal material that potentially lasts for enough generations to serve as a unit of natural selection. (Dawkins 1976, p. 28).

Following this definition to its logical conclusion, it means that, for example, the whole mitochondrial genome counts as one gene. Ultimately, both Williams and Dawkins favoured a notion whereby genes should be thought of not in terms of molecules, but in terms of the information encoded in those molecules (Williams 1985, 1992, 1996; Dawkins 1986, p. 111).

With this gene definition in place, the central tenet of the gene's-eye view then emerges. Evolution by natural selection requires two entities: replicators and vehicles (Hull 1980, 1981; Dawkins 1982). As Dawkins put it in one of the most quoted parts *The Selfish Gene*:

> What was to be the fate of the ancient replicators? They did not die out, for they are past masters of the survival arts. But do not look for them floating loose in the sea; they gave up that cavalier freedom long ago. Now they swarm in huge colonies, safe inside gigantic lumbering robots, sealed off from the outside world, communicating with it by tortuous indirect routes, manipulating it by remote control. They are in you and in me; they created us, body and mind; and their preservation is the ultimate rationale for our existence. They have come a long way, those replicators. Now they go by the name of genes, and we are their survival machines. (Dawkins 1976, p. 20).

Replicators are whatever entities whose information is copied and passed on from generation to generation. In practice, this usually means genes, though the concept can in principle apply to any entity that satisfies the key properties of longevity, fecundity, and copy-fidelity (Dawkins 1978). These are the very properties that give replicators their unique role in evolutionary explanations. Because only replicators meet all criteria and form lineages of information across generations, they are the only entities that are the beneficiary of natural selection.

Vehicles (survival machines in Dawkins' nomenclature above) are where replicators are bundled together and housed in. They are the entities that interact with the surrounding environment, a responsibility usually taken on by individual organisms. Crucially, vehicles live and die, whereas replicators are immortal. The gene's-eye view is therefore also known as selfish-gene thinking, as vehicles provide the battle ground for selfish replicators competing for transmission to the next generation. Most of the time, the fitness interests of replicators and vehicles align—the higher the fitness of the organism, the higher the chance of transmission for a specific allele—but sometimes they diverge, such as in the case of genetic conflicts (Ågren 2016a). In general, the replicator-vehicle distinction is a way to articulate the principles of evolution by natural selection in the abstract. There are

others, the most serious rivals being those inspired by Richard Lewontin's recipe approach (Lewontin 1970; see, for example, Godfrey-Smith 2009 for a Lewontin inspired critique of the gene's-eye view).

In sum, the gene's-eye view takes adaptation as its central problem and argues that the way to approach the problem is to recognize the unique properties of genes (replicators) relative to organisms (vehicles). To locate this view of life in the legacy of the Modern Synthesis, I now turn to examine its historical origins.

35.3 The Genesis of the Gene's-Eye View

I have argued that the intellectual core of the gene's-eye view is built on three areas (Ågren 2021a), which I will summarize here.

The first is the above-mentioned focus on adaptations. Accompanying this focus is an argument that the cardinal problem in evolutionary biology is to provide an account for the appearance of design among living organisms. This tradition in biology has been called "neo-Paleyan", in reference to the clergyman and Christian apologist William Paley, the author of *Natural Theology or, Evidences of the Existence and Attributes of the Deity, Collected from the Appearances of Nature* (Paley 1802). *Natural Theology* was Paley's last book, but the one that left a significant impression on biology. In particular, he popularized a version of the "argument from design" for the existence of God. Paley opens the book with an account of the so-called watchmaker analogy—just as the intricate design of a pocket watch implies the work of a watchmaker, so do the remarkable adaptations of animals (Paley was not too impressed by plants; Paley 1802, Chap. 20) imply the presence of a creator. Paley's writings had a strong influence on several generations of especially English biologists (Kohn 2004; McGrath 2011; Lewens 2019). While people like Darwin were convinced by the actual arguments, others have used Paley more for rhetorical purposes (e.g. Maynard Smith 1969, p. 82; Gardner 2009). Dawkins devoted a whole book to the topic where he compared natural selection to a blind watchmaker (Dawkins 1986) and noted that

> I suppose people like me might be labelled neo-Paleyists, or perhaps 'transformed Paleyists'. We concur with Paley that adaptive complexity demands a very special kind of explanation: either a Designer, as Paley taught, or something such as natural selection that does the job of a designer. (Dawkins 1998, p. 16)

Williams also paid tribute to Paley. To him, there was a strong link between the gene's-eye view and adaptationism (Williams 1985). Paley makes an appearance in *Adaptation and Natural Selection* and even more so in *Natural Selection: Domains, Levels and Challenges* (Williams 1992) where Williams included excerpts from Paley in the book's appendix. Thus, whereas the neo-Paleyan adaptationist tradition appears to have been especially strong in British biology (Lewens 2019), the American Williams highlights that putting too much emphasis on that aspect of history is too simplistic.

The second core area of the gene's-eye view is a Fisherian version of population genetics. R.A. Fisher, J.B.S. Haldane, and Sewall Wright, the triumvirate who showed how evolution can be mathematically described as changes in allele frequencies over time, played a central part in the general synthetic project (Provine 1971). Although the three had several spirited disagreements, including over the relative importance of selection, drift, epistasis, and dominance (Provine 1971, Chap. 5), their work helped put genes at the heart of the synthesis. This move was criticized by fellow architects, especially by Mayr who felt their mathematical models contributed little beyond the obvious (see the exchange between him and Haldane; Mayr 1959 and Haldane 1964).

Similarly, Wright also had reservations about too much focus on individual genes, rather than organisms. The gene's-eye view grew out of Fisher's worldview (Sarkar 1994; Okasha 2008; Edwards 2014) and several lingering differences in opinion over the gene's-eye view can be traced back to disagreements between Fisher and Wright (Ågren 2021b). In particular, Wright emphasized that he, in contrast with Fisher, was modelling "organismic, rather than genic selection" (Wright 1980) and that "selection relates to the organism as a whole and its environment and not to genes as such" (Wright 1931). Furthermore, Fisher had a commitment to adaptationism, which Wright lacked. Fisher's views were manifested both through his scepticism of genetic drift and his collaboration with the empiricist E.B. Ford (Turner 1985), whose hyper-adaptationist outlook left a long-lasting footprint on Dawkins's Department of Zoology at Oxford (Dawkins 2015a, pp. 342–345).

More technically, Fisher's importance for the gene's-eye view is revealed by examining his 1918 paper "The correlation between relatives on the supposition of Mendelian inheritance" (Fisher 1918). It was here that Fisher first introduced the concept of variance and with that an expanded version of the environment foundational for the gene's-eye view. He gets to this point through his method to distinguish between genetic and environmental effects. Fisher's move may upon first reading not seem like much, but it has the consequence that from the perspective of an allele, the rest of the genome, as well as the whole segregating gene pool, are now part of the environment in the same way as the surrounding pH, rainfall, or temperature. "Genotypes have dual significance as genetic environments in which a gene temporarily resides and as sets of instructions for producing phenotypes", as Williams (1985) put it. This way of thinking about the environment only makes sense under a gene's-eye view.

The final area contributing to the origin of the gene's-eye view was the rejection of group selection. Group selection has a tumultuous past that has been reviewed numerous times (Cronin 1991; Sober and Wilson 1998; Borello 2010; Wilson 2015). In its most basic form, it is the idea that selection acts not just on individuals but also on groups. The concept has featured most prominently in explanations for social behaviours that are harmful to the individual performing it, but that increase the fitness of other individuals. In the lead up to writing their own books, both Williams and Dawkins were frustrated with the popularity of certain kind of group selection, a form "for the good of the species"-arguments popular at the time. Williams was once

so exasperated with the state of things that he told his wife, Doris Williams (also a distinguished biologist), that if such arguments were considered sound, he would rather quit biology altogether than stay in a field with such poor standards. It was also the frustration with naïve group selection that led Dawkins to be so taken by the many advances in social evolution that centred on individual level selection, including Maynard Smith's game theory models (Maynard Smith and Price 1973), Trivers's idea of reciprocal altruism (Trivers 1971), and, especially, Hamilton's concept of inclusive fitness (Hamilton 1963; Hamilton 1964). As will become clear, however, Hamilton's insistence of keeping the individual as the central unit of explanation would cause some frustration for Dawkins and other proponents of the gene's-eye view.

The emergence of the gene's-eye view takes place after the completion of the Modern Synthesis. While claiming to represent the Modern Synthesis as a whole, even to offer a "truer and clearer expression" of it (Dawkins 1982, p. 239), it can more accurately be traced back to the Fisherian version of the theory. As Michael Wade concluded in his review of Dawkins's attempt to summarize the state of evolutionary theory in *The Selfish Gene*: "[if] evolution in natural populations followed the paradigm developed by R. A. Fisher, he might have succeeded" (Wade 1977). Whether we live in a Fisherian world or not is still a source of debate (Ågren 2021b).

35.4 Has Evolutionary Biology Forgotten About Organisms?

The influence of the gene's-eye view has contributed to a worry that contemporary biologists pay too little attention to organisms (this worry has been expressed in, for example, West-Eberhard 2003; Bateson 2005; Walsh 2006; Walsh 2015; and evaluated by Huneman 2010 and Huneman 2014b). Furthermore, the concern is a key part in the call to update the conceptual framework of the Modern Synthesis, resulting in a so-called Extended Evolutionary Synthesis (Pigliucci and Müller 2010; Laland et al. 2014; Laland et al. 2015). In a recent paper, a chief proponent of the Extended Evolutionary Synthesis described the key dividing line between the Modern and the Extended Synthesis as being that the former was genotype-centric and the latter phenotype-centric (Uller and Helanterä 2017).

However, the tensions over genic and organismic approaches to evolution is not just a matter of Modern vs. Extended Synthesis, but very much existed within the Modern Synthesis itself. This tension was manifested in the differences in modelling preferences between Fisher and Wright as well as in the Mayr-Haldane exchange. In both debates, the interlocutors probably agreed on more than they disagreed and all four were instrumental in their own ways in the construction of the Modern Synthesis. Another example of where the gene-organism tension arises among erstwhile close allies is the relationship between the gene's-eye view and inclusive fitness.

Inclusive fitness represents one of the most significant post-synthesis developments in evolutionary theory (Rodrigues and Gardner this volume). Importantly, it is also a phenotype-centric approach, focusing on the individual organism

as a fitness maximizing agent. The models of inclusive fitness are a way to account for how an individual may causally affect her genetic contribution to the next generation through either her own reproduction (direct fitness) or through that of her relatives (indirect fitness). A key distinction between inclusive fitness and other related approaches (such as neighbour modulated fitness) is that inclusive fitness is under the full control of the individual organism. It is this unique control that formally justifies treating inclusive fitness as the goal that organisms should appear designed to try to maximize (Hamilton 1964; Grafen 2006; West and Gardner 2013; Grafen 2014a).

Hamilton's insight can also be captured from a gene's-eye view. In his very first paper, Hamilton provides an early and eloquent expression of this approach

> Despite the principle of 'survival of the fittest' the ultimate criterion that determines whether [a gene for altruism] will spread is not whether the behavior is to the benefit of the behaver but whether it is of benefit to the gene [itself]. (Hamilton 1963)

Inclusive fitness and the gene's-eye view emerged around the same time and played mutually supportive roles. Hamilton himself used both approaches in his work, with the best example of the gene's-eye view in action is his 1967 paper on extra-ordinary sex ratios (Hamilton 1967). In Hamilton (1972) he explains altruism in social insects from both a gene's-eye view perspective and an organism-centric inclusive fitness perspective. Hamilton plays a dominant role in *The Selfish Gene* and Williams was quick to recognize the importance of his ideas in *Adaptation and Natural Selection*. Both Williams and Dawkins viewed their gene-centric models as providing a more lucid account of Hamilton's insight. Dawkins described inclusive fitness as "that property of an individual organism which will appear to be maximized when what is really being maximized is gene survival" (Dawkins 1978) and notes that Hamilton approved of this definition (Dawkins 2015a, p. 318).

The consensus view that the gene's-eye view and inclusive fitness are equivalent is well articulated in the influential textbook on animal behaviour by Krebs and Davies

> the field biologist sees *individuals* dying, surviving and reproducing; but the evolutionary consequence is that the frequencies of *genes* change. Therefore the field biologists tend to think in terms of individual selection whilst the theorists thinks in terms of selfish genes. (Krebs and Davies 1993, p. 375; original emphasis)

Yet, there have also been frustrations. Dawkins once described inclusive fitness as a "brilliant last-ditch rescue attempt to save the individual organism as the level at which we think about natural selection" (Dawkins 1982, p. 187). More recently, Dawkins re-iterated the complaint:

> I think it was unfortunate that Hamilton, having realized this very important insight, chose to stick with the individual organism as the entity of action. He therefore coined the phrase "inclusive fitness", as the mathematical function which an individual organism will maximize if what it's really doing is maximizing its gene survival. It's a rather complicated thing to calculate. It's difficult to calculate in practice and this has led to a certain amount of, not

> hostility, but a certain amount of skepticism about inclusive fitness as a measure, skepticism which I share. But for me the remedy of that skepticism is to say, well, forget about the organism and concentrate on the gene itself. (Dawkins 2015b)

Similarly, Maynard Smith described inclusive fitness as an "absolute swine to calculate", noting that he much preferred the gene-centric approaches of Hamilton's 1963 paper (Maynard Smith 1997; see also Maynard Smith 2002). To Dawkins and Maynard Smith, it seems, Hamilton was a revolutionary thinker who never completed his own revolution.

Contemporary inclusive fitness theorists clearly see value in the gene's-eye view. At times, however, they have afforded it a more limited role, such as in the study of genetic conflicts (West and Gardner 2013; Levin and Grafen 2019). How to handle the biology of genetic conflicts is an area where the gene's-eye view and inclusive fitness may come into tension. In *The Extended Phenotype*, Dawkins noted that

> there is a sense in which a "vehicle" is worthy of the name in inverse proportion to the number of outlaw replicators that it contains. (Dawkins 1982, p. 134).

In other words, the presence of genetic conflicts may erode the necessary unity of purpose required for the individual organism to act as the sole fitness maximizing agent (Gardner and Grafen 2009; Okasha 2018, p. 29). It is not that the mathematical tools of inclusive fitness theory cannot be used to study genetic conflicts. They very much can, as conflicts can be modelled as a situation where the inclusive fitness of genes—here defined as a scrap of nucleic acid—diverges (Gardner and Welch 2011; Gardner and Úbeda 2017). Instead, the importance of genetic conflicts are downplayed in the name of understanding organismal phenotypes (West and Gardner 2013; Grafen 2014b). From the perspective of the gene's-eye view, this phenotypic gambit is awkward. A major strength of the gene's-eye view is that it forces us to reckon with why organisms are unified wholes to begin with—to re-discover the organism (Dawkins 1982, Chap. 14). Under the gene's-eye view, the best way to conceptualize organisms is not as cohesive fitness maximizers, but as "adaptive compromises" (Haig 2006; Haig 2014) of multiple agents whose fitness interest mostly align but far from always.

Today, most biologists are happy to ignore genetic conflicts in their work on phenotypic evolution. Genetic conflicts have often been viewed as the best evidence of the utility of the gene's-eye view, but still represent a minor part of the field of evolutionary biology. Take, for example, the largest evolution meeting in history, the 2018 Second Joint Congress on Evolutionary Biology in Montpellier, France. The meeting brought together some 2700 attendees from almost 60 countries, presenting 800 talks and around 1200 posters across 78 thematic symposia. Only six symposia had fewer submissions than the one dedicated to genetic conflicts. My impression is that most of my colleagues in evolutionary biology do not think of genes first and as organisms as adaptive compromises, as Dawkins (1982), Haig (2014), Maynard Smith (1985), and I (Ågren 2014; Ågren 2016b) tend to do. The

dominance and success of inclusive fitness theory show that evolutionary biology has not forgotten about organisms.

35.5 Conclusion

The gene's-eye view has been at the centre of evolutionary debates for the past half-century. While claiming to represent the whole of the Modern Synthesis, it can more accurately be traced back to a certain version of the synthetic project. It views adaptation as the most important problem of our field and uses a Fisherian approach to population genetics to conceptualize the answer. Critics of adaptationism (such as Wright) or of gene-centric explanations (like Mayr) are as much part of the Modern Synthesis as Fisher.

The gene's-eye view rose to prominence thanks to and alongside inclusive fitness theory. The two frameworks are equivalent in most situations but do differ in their emphasis on genes and organisms. While the gene's-eye view would prefer to talk about genes rather than organisms, the success of inclusive fitness means that there is a strong post-synthesis movement, and consensus, to keep organisms at the centre of evolutionary explanations.

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The Parallax View: A Commentary on Ågren

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From one point of view, the passions aroused by the gene's-eye view are difficult to explain. As Ågren (2021; this volume) shows clearly, the methods of Williams (1966) and Dawkins (1976) continue to be useful for many problems (e.g., Boomsma 2016: R1254)—even Dawkins' throwaway remarks about green beards (1976: 96; Hamilton 1964: 25) still generate valuable science (e.g., Gruenheit et al. 2017; Gardner and West 2010); and yet the methods were never more than a part of the biologist's toolkit (Dawkins 1982: Ch. 1; Williams 1992: 31; Grafen 1992; Kitcher 2001: 407; Haig 2012; Boomsma 2016). In any case, as Ågren also shows, the gene's-eye view differs only subtly from one sort of organism-centered approach (e.g., Hamilton 1964; Brockmann et al. 1979; Grafen 1999). Many of the initial disputes about the gene's-eye view were cleared up long ago by Williams (1985) and especially Lloyd (1988, 2001), who showed how different biologists, interested in different questions, had been using terms like "unit of selection" to mean different things (Maynard Smith 2001). But consider the following, from a review of Ågren (2021) in the journal *Evolution*:

> Without wishing to reify this as a dualistic battle, and certainly not as a Manichean one between "good" and "evil," there is a powerful tradition opposing the gene's-eye view. (Winther 2022: 685)

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Why would disclaimers like this seem necessary? Winther's framing, so puzzling from one point of view, does I think make better sense if we consider the contributions of two giants of evolutionary genetics: R. A. Fisher and R. C. Lewontin.

36.1 Fisher

Ågren argues that "The gene's-eye view grew out of Fisher's worldview", and with a few qualifications, [1] I agree. Most relevant to gene's-eye view is Fisher's Fundamental Theorem of Natural Selection—a project whose meaning and importance remain highly disputed (Edwards 2014; Grafen 2015, 2021; Lessard and Ewens 2019). Fisher's technical goals, it is now generally agreed, were (1) to identify the partial change in mean fitness that is attributable to the direct effects of allele frequency change, as caused by natural selection (excluding changes due to statistical associations between alleles, and changes to the environment, including those caused by the allele frequency changes themselves), and (2) to show that this partial change was equal to the additive genetic variance in fitness—i.e. the variance calculated one-gene-at-a-time. [2] Because variances are non-negative, the partial change in mean fitness (once correctly defined) is thus revealed to be a source of "improvement" (Fisher 1930: 37; Grafen 2003, 2018; Kokko 2021). Fisher's project, therefore, resembles a statistician's attempt to identify the effect of an experimental treatment (adding fertilizer to crops, say), but with the treatment, and the measure of success both *defined* so that one leads reliably to the other. Moreover, nothing in the theorem implies that variance will be mostly additive, nor that the partial change in mean fitness will be a good approximation for the total change.

This strange project might have been designed to provoke a pair of criticisms: that the theorem is empirically empty, and that it invokes goal-directedness by spurious means; the claim of constant improvement is achieved with contrived definitions, which simply ignore parts of what happens. These features do make sense, I think, if we accept that Fisher's goal was to understand biological adaptation (Fisher 1930: 37–38; Grafen 1988). Not all evolutionary change is adaptive (Fisher 1930: vii), and so any attempt to understand adaptation will have to identify and isolate the relevant part of the change. Moreover, adaptations appear designed but weren't (Fisher 1930: 38), and so any attempt to understand adaptation will have to invoke *some* notion of goal-directedness. But even if this helps us make sense of Fisher's project, it does not

[1] Fisher insisted that "natural selection [. . .] in reality acts upon individuals" (Fisher 1941) and his grandest summaries of evolution invoked agential organisms, whose enterprises, conscious and unconscious, interact with their whole ecological situation (Fisher 1934, 1950; Turner 1985). When Fisher wrote that "each gene is constantly tending to create genetic situations favourable to its own survival" (1930: 95) his topic was epistatic modifiers and coadapted gene complexes, and he was, in any case, rephrasing a point he had made in other terms. Fisher's major comment on agential language was to disown Darwin's "struggle for existence" (1930: 43–44).

[2] Without social effects, or different modes of transmission, (1) and (2) capture the only ways in which the theory is "gene centered."

mean that the criticisms were merely false; and nor does it tell us what sort of follow-up science might be warranted by Fisher's result (e.g., Williams 1966: 20; Maynard Smith 1978; Edwards 2014; Grafen 2018; Lessard and Ewens 2019; Grafen 2021).

The reception of the fundamental theorem was further complicated by two of the contexts in which it appeared. The first context was Fisher's ill-tempered debate with Sewall Wright about the latter's Shifting Balance Theory (Wright 1932; Fisher 1941, 1953; Frank 2013). Wright's aim was to establish conditions for ongoing adaptive evolution, asking how populations might reach the highest fitness peak in a multi-peaked landscape. But while Fisher had strong views on this topic (apparently believing that Wright was solving a non-problem; Fisher and Ford 1950; Frank 2013) his fundamental theorem was not obviously relevant to this debate. Nevertheless, Wright often wrote as if Fisher's theorem was a meaningful *alternative* to the Shifting Balance Theory and an alternative that relied on frankly implausible assumptions, such as random mating and a simple genotype-phenotype map. Fisher, moreover, did little to correct this view (e.g., Fisher 1953: 515–7; Frank 2013). The remarkable outcome was that Wright "produced more commentary on the fundamental theorem than Fisher" (Frank 2013: 42), with most of this commentary misrepresenting both the theorem's assumptions and its purpose.

The second relevant context was entirely of Fisher's own making. The fundamental theorem appears toward the start of a book that culminates in Fisher's views on "the decline of civilizations"—and a book whose two halves were, Fisher insisted, "inseparable" (Fisher 1930: x). It is natural, therefore, that the phrase "Fisher's world view" should invoke far more than a view of adaptation; its associations would have to include Fisher's conservative Anglican Christianity, with its emphasis on husbandry, and his vaguely Nietzschean concern with "great men" (Fisher 1950; Box 1978: 11; Turner 1985; Kohn 2004: Chaps. 4–5)—both of course congealing in his enthusiasm for the eugenic "improvement" of humankind (Fisher 1930: 29; Rutherford 2020b, 2022). Already in 1915 with C. S. Stock, Fisher had insisted that "no apology is needed [...] for introducing a subject so apparently remote from Eugenics as a particular theory of adaptation" (Fisher and Stock 1915: 46), because "it is not easy to exaggerate the importance to Eugenists of the broad principles outlined in the *Origin*" (1915: 60).

The result is that Fisher's fundamental theorem—a result which underpins the gene's-eye view—is subject to continued uncertainty—both about its aims, and about the assumptions on which it relies; this uncertainly combines with the impression that Fisher believed his result to have clear (and to us, deeply unpleasant) political implications.

36.2 Lewontin

Winther identifies Lewontin as the central figure in "the powerful tradition opposing the gene's-eye view" and, again with qualifications, [3] I agree. Like Fisher, Lewontin could write with a "sharp pen" (Angier 2021), and like Fisher too, he wrote as a scientist on political issues, acknowledging a substantial connection between his science and his politics (Levins and Lewontin 1985; Maynard Smith 1986; Singh et al. 2001: 3). However, Lewontin's best-known interventions were broadly influential, and a force for good (see, e.g., Ruvolo and Seielstad 2001; Graves 2019). So, when Lewontin dismissed the gene's-eye view—as he did repeatedly, and sometimes with apparent contempt (e.g., Lewontin 1977, 1990, 2000, 2001: 8; Sober and Lewontin 1982; Levins and Lewontin 1985)—his views were buttressed not only by his great technical authority but by his moral authority too. Lewontin's critiques, moreover, combined technical and moral strands.

Consider, for example, a pair of technical assumptions that Lewontin convincingly attacked: that there exists some quantity which evolution reliably maximizes (Franklin and Lewontin 1970; Felsenstein 2000; Edwards 2014; Grodwohl 2017); and that trait heritabilities are reliable guides to norms of reaction (Lewontin 1970; Lewontin 1974a; Feldman and Lewontin 1975; Kitcher 2001: 397–9). Both assumptions, as Lewontin showed, hold only with a very simple genotype-phenotype map. As Lewontin also showed, the second assumption has a pernicious history in human genetics; the heritability of IQ, for example, has been used time and time again to obscure or defend social injustice [4] (Lewontin 1970, 1974a; see also Glymour 2001; Coop and Przeworski 2022).

But does the gene's-eye view rely on these faulty assumptions? At first glance, a link is plausible. The gene's-eye view is strongly associated with optimization-based methods, including some kinds of fitness maximization (Maynard Smith 1978; Williams 1985: 12; Birch 2016; Grodwohl 2017), and is grounded in Fisherian variance partitioning; it often uses the rhetoric of "a gene for trait X" (Dawkins 1979: 189–190; Kitcher 2001: 405–6, 409–11; Kohn 2004: 272–3); and, of course, Fisher *was* led by hereditarian assumptions to catastrophic errors. [5] On the other hand, advocates of the gene's-eye view have never denied that genotype-phenotype maps

[3] For all its great breadth, Lewontin's technical work was usually focused on tracking genotype frequencies. The scientific work of Dawkins and Williams, by contrast, is much closer to whole-organism biology (e.g., Boomsma 2016).

[4] Lewontin noted "the terrible mischief that has been done by confusing the spatiotemporally local analysis of variance with the global analysis of causes," when only the latter could "provide us with the basic knowledge we require for correct schemes of environmental modification and intervention" (Lewontin 1974a).

[5] See, e.g., his industry-funded denials of the link between cigarettes and cancer (Proctor 2012); and his explicitly racist dissenting opinion to UNESCO (1952). It is arguable whether similar assumptions underlay his belief in the potential efficacy of eugenics (Dawkins 1999: 19–20; Paul and Spencer 2001; Rutherford 2022).

are complex (e.g., Dawkins 2004: 392; Queller 2020); total fitness maximization was explicitly rejected by Fisher (1941), Williams (1966) and Dawkins (1976), as part of their well-known attack on "good-of-the-species" thinking (Frank 2013; Kokko 2021); and the causal claims that the gene's-eye view requires (Fisher 1941; Haig 2012; Lee and Chow 2013) [6] are not those criticized by Lewontin (1974a). In linking Dawkins (1976) to genetic determinism, Lewontin came close to flat-out misrepresentation. [7]

Of course, it makes no sense to criticize methods unless we are clear what the methods are for (Fisher 1953: 516; Lewontin 1974a; Williams 1985; Grafen 1988; Glymour 2001; Kitcher 2001: 407). Another characteristic of Lewontin's critique is his relative lack of interest in, and occasional suspicion of, the *questions* that motivated Williams and Dawkins. While this point should not be exaggerated (see e.g. Lewontin 1978) I think there were "basic differences in perspective on what would constitute productive research" (Williams 1985: 3). Consider, for example, Lewontin's overview of his field:

> The problematic of evolutionary biology is the explanation of changes and diversity in the characters of organisms [. . .] The theoretical apparatus is a form of a dynamical theory. (2001: 9, 2002; see Maynard Smith 1986)

This clearly describes an important part of evolutionary biology, and from a certain perspective, its vision is generous and expansive. The vision encompasses dynamical models, like Wright's, where lots of factors are included; and more broadly, lots of things might explain change and diversity, and so all might be equally interesting and important. For the same reason, however, the vision can be subtly limiting [8]—especially as regards the study of adaptation. For example, the

[6] A source of confusion here is the unusual sort of averaging employed by the fundamental theorem (see, e.g., Winther et al. 2013; Allen et al. 2013; Okasha and Martens 2016).

[7] Lewontin, for example, denied that people are 'in the mistaken phrase of Richard Dawkins, author of The Selfish Gene, "lumbering robots," who are ruled by our genes "body and mind"' (Lewontin 1982: 18); but the part about being "ruled by our genes" was added by Lewontin. Elsewhere Lewontin simply misquoted Dawkins as claiming that genes "control us, body and mind" (Lewontin et al. 1984: 287; Lewontin 1990). Dawkins in fact wrote that genes "created us, body and mind" (1976: 9), adding that "we have the power to turn against our creators. We alone on earth can rebel against the tyranny of the selfish replicators" (1976: 201; see also Dawkins 1999: 21–2; Kohn 1996: 176; 2004: 323–9). Dawkins (1985) is a tart response to Lewontin et al. (1984).

[8] Claims of overreach, or undervaluing alternative research programs, are also recurrent criticisms of the gene's-eye view (e.g., Winther 2022: 1–2). And some instances can be found—e.g., when Dawkins wrote "I expect the ESS concept to revolutionize the science of ecology" (1976: 84) or insisted that "the unit of selection" *should* be defined in a certain way (Lloyd 1988, 2001). Nonetheless, Dawkins never denied the legitimacy of any of Tinbergen's (1963) four questions, nor did he suggest that they be collapsed into one question ("What has happened": Ghiselin 1983; Dennett 1983: 386–7). When Dennett (2011) claims that the question *cui bono*? Is "central to all evolutionary thinking," we can read this uncharitably—as implying, e.g., that Kimura or Lewontin (1974b) were not engaged in evolutionary thinking—or charitably, perhaps as insisting that the language of purpose is essential to some evolutionary thinking, and not just a picturesque add-on. It

overview recognizes the study of natural selection as it appears in the "neutralist-selectionist" debates (Lewontin 1983), which were central to Lewontin's own scientific work (and in which Fisher had also participated—on both sides: Fisher 1929: 556; Fisher and Ford 1950). However, in these debates, the idea of "adaptation" is either marginal or dispensable (Krimbas 1984). Conspicuously absent from the overview are methods of studying adaptation in which the action of natural selection is not, itself, tested, but is used instead as a posit from which further testable predictions are made, or as a way of viewing the outcomes of evolution, from which explanatory patterns emerge (Maynard Smith 1978; Ross 2002: 277–8, 288–9). If we focus exclusively on the goal of predicting evolutionary dynamics, we will inevitably misread Fisher's fundamental theorem (Franklin and Lewontin 1970; Lewontin 2001: 19), and the work of Williams and Dawkins. Those authors were not doing population genetics badly, they were doing something else (Williams 1985; Grafen 1988; Hammerstein 1996; Edwards 2014; Birch 2016).

In his more philosophical pieces (e.g., Levins and Lewontin 1985; Lewontin 2000), Lewontin suggested a more radical position, casting doubt on "both the idea that we can think of organisms adapting to environments that are independent of them and the idea that we can think of the phenotype as dependent on causal interactions between genotype and environment" (Kitcher 2001: 400). Kitcher argued that these positions stemmed directly from Lewontin's opposition to genetic determinism and its pernicious consequences, and a corresponding wish for a "stake-in-the-heart move" (Oyama 1985: 26–7; Kitcher 2001). The ideas that Lewontin challenged are, however, central to the study of adaptation; without them, the *problem* of adaptation becomes very difficult to coherently state, let alone solve (Rosenberg 2000: Ch. 5; Rosenberg and Bouchard 2005; Fromhage and Houston 2022). At times, Lewontin did come close to dismissing the study of adaptation per se—and did so using versions of the claims that are leveled at all theories of adaptation: empirical emptiness (Gould and Lewontin 1979) and inappropriate use of goal-directedness (Lewontin 2010).

36.3 Discussion

The gene's-eye-view is sometimes viewed as "bad science" (Ågren 2021: 3; Winther 2022), and in more ways than one. The writings of Fisher and Lewontin help us to see how such a view might have arisen. Nevertheless, the tensions cannot be fully explained by the views of these two singular writers, nor even by the unique properties of the gene's-eye view. Similar tensions have been aroused by all attempts to explain biological adaptation—at least since Agassiz accused Darwin of

is also notable that many of the topics placed by Winther in the "powerful tradition opposing the gene's-eye view" were major topics of research for prominent advocates of the gene's-eye view; these include Fisher on epistasis (1930: Chaps. 3 and 7); Williams on species selection (1992; see also Fisher 1930; Grafen 1992; Haig 2015: 868); and Maynard Smith on development and evolution (Maynard Smith 1960; Maynard Smith and Sondhi 1960).

> a scientific mistake, untrue in its facts, unscientific in its method, and mischievous in its tendency. (Agassiz 1860: 154)

The gene's-eye view, like Fisher's fundamental theorem and Hamilton's Rule, joins natural selection on the list of ideas that strike some researchers as deep and important, and others as false or vacuous (Maynard Smith 1969; Edwards 2014; Nowak et al. 2017). And there have always been writers, firmly "on the side of the angels" (Monypenny, Buckle 1929: 108) who view natural selection primarily as a source of akrasia, like Sin in the Epistle to the Romans (Gaventa 2004), a threat to freedom (Fisher 1934, 1950: 16–17; Lewontin et al. 1984: 283; Dupré 1998: 168–9; Dawkins 1999: 21–2; Barker 2002: 87–94), and an excuse for vice (Whitehead 1954: 45; Dupré 1998: 167; Clark 2000: 119; Midgley 2006: 271; see Ross 2002; Haig 2011; Radick 2017: 49). [9]

Part of me is glad that Ågren's discussion of the gene's-eye view avoids these topics, and glad too that he "downplays many of the arguments and framing devices by the Lewontinians" (Winther 2022), since I think these framings often miss the point of the methods, preventing us from seeing them clearly—including their limitations. By the same token, however, Ågren's treatment makes it difficult to see why the methods remain controversial. Criticisms of the gene's-eye view can be historically important, even if they lack an objective correlative in the methods themselves.

On a second point, my argument is more with myself than with Ågren. While the gene's-eye view has no necessary link to any political position—as is clear from the diverse views of its advocates (e.g. Maynard Smith 1986; Dawkins 1999: 19; Haig 2003; Kohn 2004: esp. 23, 323, 329; Trivers 2015) [10]—I think Lewontin was right to note that hereditarian and adaptationist theories have been, and continue to be, misinterpreted and misused in characteristic ways. And he was right to fight against this; while most errors in biological theory are only ridiculous, these errors are dangerous. And while such errors and their legacies persist, those of us who

[9] This line of criticism, of course, fails to pick out what is ugly about eugenics, with its assertion of "our" freedom to direct evolution toward "higher types"; whatever is meant by this, it cannot be defined in terms of current reproductive success.

[10] It is also notable that, in their best-known statements on human racial differences, both Fisher and Lewontin strayed from their most characteristic science. Lewontin, for whom "complexity and interaction are of the essence" (Lewontin 1974b: 318), summarized human genetic variation using the simplest "one-gene-at-a-time" approach (Lewontin 1972), obscuring the information about ancestry in multi-locus associations (Edwards 2003; Leslie et al. 2015)—a fact which, let us be clear, provides no support to racist pseudoscience (Hardimon 2017; Rutherford 2020a). More grievously, Fisher, who urged us to let the data speak without prior assumptions, and who argued for the necessity of randomization in experimental design, was nonetheless happy to opine that "available scientific knowledge provides a firm basis for believing that the groups of mankind differ in their innate capacity for intellectual and emotional development" (UNESCO 1952: 56); and yet failed to mention the fatal absence of randomization-over-environment in human data (Lewontin 1970; Novembre and Barton 2018), and failed in fact, to mention any data at all.

would dearly love to separate the science from its murky political origins will not, and should not, get our wish.

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Why We Disagree About Selfish Genes: A Reply to Welch

37

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Abstract

In his insightful commentary, Welch notes that a key reason for why the disagreement over the gene's-eye view have proved to viable is that it is made up of both scientific and political dimensions. Here, I address both issues.

Keywords

Gene's-eye view · Lewontin · Science and politics

The reviews of *The Selfish Gene* (Dawkins 1976) penned by Richard Lewontin and W.D. Hamilton could not have been more different. In *Science*, Hamilton (1977a) lauded the book as an excellent summary of the cutting edge of evolutionary theory and said it "should be read, can be read, by almost everyone." Over at *Nature*, in contrast, Lewontin described the approach to evolution outlined by Dawkins as "vulgar" and titled his review of the book "Caricature of Darwinism" (Lewontin 1977a). Lewontin's rhetoric greatly upset Hamilton, who called it a "disgrace" in a letter to the editor (Hamilton 1977b). He also compared Lewontin's criticism to Bishop Wilberforce's dishonest tactics at the 1860 meeting of the British Association where he debated T.H. Huxley on the status of the then-new theory of evolution. The letter ping-pong ended with Lewontin mocking Hamilton for thinking too highly of himself; he was no Darwin and Dawkins no Huxley (Lewontin 1977b).

The high-profile exchange between these Darwinian heavyweights would set the tone of the debate over *The Selfish Gene* and the gene's-eye view of evolution for the

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next half-century. But why has the debate persisted for so long? And why has it often been so ill-tempered? As Welch notes in his insightful commentary, the answer likely lies in the fact that the disagreements combine both scientific and political issues. To Lewontin in particular, the two were usually inseparable, and in his disapproval of the gene's-eye view, he took aim at both.

First, science. Welch highlights two aspects of the gene's-eye view of which Lewontin was especially critical: its intimate relationship with optimality-based approaches (especially of the fitness maximization variety) and its relaxed relationship with the genotype-phenotype map. To this, we can add that Lewontin formulated what would become the main rival to the gene's-eye view's replicator-vehicle framework in his 1970 *Annual Reviews* paper (Lewontin 1970): as long as there is phenotypic variation that is correlated with heritable fitness differences evolution by natural selection will occur. There is no need to invoke separation between replicators and vehicles. Furthermore, together with Elliott Sober, Lewontin argued that just because you can calculate selection coefficients for individual alleles it does not mean that they are causally real (Sober and Lewontin 1982). Rather, they are statistical artifacts. In the case of heterozygote advantage, for example, it is possible to determine allelic selection coefficients by taking the average across all genotypes. While these can be used to predict evolutionary change, Lewontin and Sober did not consider them causally appropriate in the way that the diploid genotypic selection coefficients are.

These technical issues are all important. But as Welch argues, and I agree, they cannot sustain a dispute as persistent as the one over the gene's-eye view. After all, the role of optimization models in evolutionary theory has been extensively defended (Maynard Smith 1987; Grafen 2006; Davies et al. 2012); Dawkins's chapter on genetic determinism in *The Extended Phenotype* (Chap. 2, Dawkins 1982) was praised by erstwhile critics like Mary Midgley and Patrick Bateson (Midgley 1983; Bateson 1986); and the heterozygote advantage argument of Lewontin and Sober has been addressed numerous times (Rosenberg 1983; Sterelny and Kitcher 1988; Weinberger 2011).

Instead, much fuel for the debate has come from the political and moral views (perceived to be) associated with the gene's-eye view. As Welch points out, in my contribution to this volume—as well as in *The Gene's-Eye View of Evolution* (Ågren 2021)—this aspect of the debate takes a backseat to the scientific issues. Given that several key people featuring in the debate—such as Fisher and Lewontin—saw their science and politics as one whole, this can be a limitation. I would like to think that the scientific treatment of the topic in Ågren (2021) provides the first half of the puzzle and that historians of biology more versed in social issues than myself take up the challenge of completing the second half. A topic as important as the gene's-eye view deserves as complete of a picture that it can get.

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Part XIII

Genetic Evolvability: Using a Restricted Pluralism to Tidy up the Evolvability Concept

38

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Abstract

Advances in the empirical sectors of biology are beginning to reveal evolvability as a major evolutionary process. Yet evolvability's theoretical role is still intensely debated. Since its inception nearly 30 years ago, the evolvability research front has put a strong emphasis on the non-genetic mechanisms that influence the short-term evolvability of individuals within populations by causing phenotypic heterogeneity, such as developmental trait plasticity, phenotypic plasticity, modularity, the G-P map, robustness, and/or epigenetic variation. However, genetic evolvability mechanisms such as mutation or recombination have a deeper history in evolutionary thought that is often overlooked by those in the evolvability research front, with recent evidence suggesting that species switch to genetic evolvability mechanisms when short-term evolvability strategies fail to relieve selective pressures. For this reason, a causal distinction must be made between *genetic evolvability* and the more recently emphasized *non-genetic (or evo-devo) evolvability* to allow for its maturation as a central explanatory concept. I conclude by arguing that the anachronisms of the scientific process are the main culprit behind recent divisions in biology and likely beyond. To streamline theoretical progress, we need to build a new science with new underlying philosophies like *restricted pluralism*.

Keywords

Evolvability · Adaptive genetic variation · Theoretical progress · Pluralism · Scientific concepts · Evo-devo · Scientific causation

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38.1 Genetic Evolvability: Using a *Restricted Pluralism* to Tidy up the Evolvability Concept

Evolvability is an unusual concept. Its *unusualness* stems from its *long past but short history* in evolutionary thought, despite its unambiguous role in the evolutionary process. Darwin assumed that all extant species hold some capacity for evolution, going so far as to suggest that some species may be better at evolving than others (Sansom 2008). In the synthetic era (circa 1916–1950), theorists formalized the latter notion that species vary in their response to natural selection dependent on the production and conservation of *genetic variation* (e.g., Fisher's [1930] *Fundamental Theorem*; Dobzhansky 1937), therefore establishing the conceptual foundations of *genetic evolvability*.

Yet for a variety of reasons, evolvability was initially "taken for granted" and built within evolutionary theory as "a given premise" (Hansen 2016, p. 83). For one, the causal mechanisms behind the production and conservation of novel genetic variation—i.e., from mutation or recombination—were assumed to be the result of "random" or stochastic processes separate from selective influence. [1] A causal boundary was drawn between (1) *the random or stochastic mechanisms that produce heritable variation* and (2) *the process of natural selection acting on this variation to cause adaptation* (Mayr 1982). These became independent and sequential causal events in the adaptation process, implying that most species were continuously replenished with sufficient variation for natural selection to subsequently act upon "without any need for special mechanisms generating new variability" (Charlesworth et al. 2017, p. 8).

However, the assumption that the production of novel genetic variation is *random* and independent of selection has been subtly overturned in the last 60 years, despite the curious reluctance of some theorists to accept these novel findings. New evidence arising from microbiology, ecology, and experimental biology has established that mutation and/or recombination modifier genes are not only *exposed to the direct influence of selection* (Otto 2013), but most species exhibit greater flexibility to cause adaptive genetic changes in response to selective pressures than previously supposed (Swings et al. 2017; Fitzgerald and Rosenberg 2019; Bonnett et al. 2022), often with no observable benefit to individual organisms.

This leads us to the main reason why evolvability was initially overlooked. Evolvability is an emergent dispositional property whose manifestation is causally relevant at higher levels of biological organization over longer stretches of time and frequent spatial changes—with several biologists viewing evolvability as *the best example* of an emergent biological adaptation (Lloyd and Gould 1993; Maynard Smith 1998; Folse and Roughgarden 2010). Yet empirical limitations and methodological constraints have naturally obscured the causal complexity of biological systems for the majority of evolutionary research. Early genetics research was

[1] Although mutations were known to be sometimes caused by external but non-selective forces such as UV radiation or the application of other environmentally induced lethal mutagens.

often limited to within-population analyses (Nei 2013) and performed within restrictive spatiotemporal parameters (Ford 1964; Levins 1968; Endler 1986), therefore concealing the complex evolutionary and selective dynamics of natural populations. As a result, evolutionary theory was initially constructed in an abstract vacuum that was not particularly representative of evolution in nature (Otto 2009; Hendry 2017).

The divisions between theory and empiricism were further exacerbated within the twentieth-century scientific zeitgeist of *logical positivism,* which favored the mathematical reductionism of theoretical population genetics (Smocovitis 1996). Evolvability was therefore *imperceptible* or largely ignored by evolutionary theorists who placed a premium on reducing biological causation to one privileged level or lower levels of biological organization. Biologists clinging to these theoretical traditions still doubt the empirical realism and/or theoretical significance of evolvability for precisely the same reasons (e.g., Barton and Partridge 2000; Partridge and Barton 2000; Chicurel 2001; Poole et al. 2003 [2]; Sniegowski and Murphy 2006; Lynch 2007; Charlesworth et al. 2017).

Yet now in the age of evolutionary ecology where we can readily observe how evolutionary and selective dynamics unfold in the space and time of capricious ecosystems, and/or construct real-world experimental parameters that simulate these natural contexts, evolvability is beginning to be revealed as a major evolutionary process. Evolvability explanations are essential to explain why some species survive when others go extinct within *the evolutionary rescue* research front (Gomulkiewicz and Holt 1995; Carlson et al. 2014; Bell 2017). A clear causal link has also been established between genetic evolvability mechanisms and the evolutionary survival of species or lineages—e.g., from **meiotic recombination** (Bell [1982] 2019), **stress-induced mutagenesis** (Ram and Hadany 2012, 2014, 2019), **hypermutation** (Swings et al. 2017), **horizontal gene transfer** (Soucy et al. 2015), **transposable element domestication** (Brunet and Doolittle 2015), and **gene duplications/whole-genome duplication events** (Van de Peer et al. 2017). The observed ubiquity and conservation of these genetic evolvability mechanisms across biological domains point to higher-level selective processes such as *species or lineage selection* as the underlying causal reasons why these mechanisms are maintained in the long-term, thereby facilitating adaptive evolution through the production, conservation, or domestication of novel genetic variation when environments change, often with no observable benefits to individual organisms or *selfish genes*.

Thus, the *unusualness* of evolvability in the history of evolutionary thought is precisely what makes it an interesting concept. The reasons why a central process such as evolvability can go relatively unnoticed in theory shed specific light on the philosophical anachronisms that have been stalling theoretical progress for over a

[2] Poole et al. (2003, p. 163) made the claim that "The concept of evolvability covers a broad spectrum of, often contradictory, ideas. At one end of the spectrum it is equivalent to the statement that evolution is possible, at the other end are untestable post hoc explanations, such as the suggestion that current evolutionary theory cannot explain the evolution of evolvability." Evolutionary theory, and in particular natural selection theory, is not equipped to explain the existence of higher-level selective features such as evolvability.

century. Today, philosophers of biology generally agree that complex biological phenomena such as evolvability—which are only just being revealed by superior empirical methodologies—justify a switch in theoretical tactics away from explanatory reductionism, monism, and monocausal modeling toward a theory that embraces pluralistic, multilevel, and multicausal explanations (Dupre 1993; Mitchell 2003, 2009; Potochnik 2017; Anjum and Mumford 2018).

However, the ensuing chapter has little to do with *the realism of evolvability* or what can be accomplished by integrating evolvability into modern evolutionary theory. For those evolutionary biologists who derive from empirically rich traditions, the central role of evolvability in the evolutionary process is self-evident. Yet I also take issue with how evolvability is presented by progressives on the other side of the spectrum, which will be the focus of this chapter.

Evolvability has been referred to as "a cornerstone of the EES" (Pigliucci 2008, p. 75; Pigliucci and Müller 2010) because (1) development was ostensibly missing from the modern synthesis, and (2) evolvability is largely construed as a developmental phenomenon by most in the evolvability research front (Ibid; Hansen 2016; Hansen et al. in press; Nuño de la Rosa 2017). However, the historicity of (1) is in question (see for more Chap. 12; Futuyma 2017), and here I reject (2) that evolvability is largely a developmental phenomenon. Evolvability may very well be "the proper focus of evo-devo" (Hendritske et al. 2007), but *evo-devo is not the proper focus of evolvability*. Genetic evolvability has always been, and shall remain, the central focus of evolvability thought.

Hansen et al. (in press) continue in the tradition of placing a strong emphasis on development and broadly argue for *an unrestrictive or "anything goes" type of pluralism* for the evolvability concept, following similar philosophical prescriptions by Nuño de la Rosa (2017), Brown (2014), and Pigliucci (2008). But a budding concept such as evolvability does not benefit from an overly broad type of pluralism, explanatory or methodological. New concepts benefit from a *restricted pluralism*, where we can still accept the many different viewpoints of evolvability, but leave space for further conceptual refinements and causal distinctions made between these (oftentimes competing) viewpoints.

Here I suggest the utility of maintaining the ultimate/proximate causal distinction (i.e., *Weismann's barrier*) of modern genetic theory to build a more accurate causal picture of adaptation by *evolvability* (c.f. Uller and Laland 2019; Laland et al. 2011). *Why?* Because drawing a causal distinction between *genetic evolvability* and the more recently emphasized *non-genetic* (or *evo-devo*) *evolvability* grants us taxonomic clarity. [3] It organizes similar phenomena while also maintaining a concreteness in conceptual parameters that should be preferable to broad conceptualizations of evolvability that categorize all evolvability-related explanandum under the same

[3] Like most things in biology, there is some phenomological overlap between non-genetic and genetic evolvability mechanisms. Genetic variation is not only maintained and conserved by sex, but also by developmental mechanisms which sometimes releases cryptic genetic variation. In another sense, sexual processes could even be considered developmental mechanisms.

conceptual umbrella—i.e., phenotypic plasticity, developmental plasticity, epigenetic variation, the genotype-phenotype map, modularity, robustness, evolutionary capacitance, and adaptive genetic variation. Re-organizing the evolvability concept by making further causal refinements is thus a must if evolvability is to progress into a mature concept within the background of modern evolutionary theory.

38.2 The Neglected *Long Past* of Genetic Evolvability

> Concepts such as evolvability, for instance, did not exist in the literature before the early 1990s... [T]he majority of the new work concerns problems of evolution that had been sidelined in the (*Modern Synthesis*) and are now coming to the fore ever more strongly, such as the specific mechanisms responsible for major changes of organismal form (Pigliucci and Müller 2010, p. 4 and 12).

But evolvability was never "sidelined," at least in the same way as the other proposed novel concepts of the *EES*, nor was it non-existent in the literature before the 1990s. Early theorists and empiricists—such as Weismann, Fisher, Ford, Wright, and Dobzhansky—granted evolvability a central role in their investigations when they were attempting to model how populations respond to selection. However, given the limitations imposed by the methods and instruments contemporary to their time, they were never able to effectively reveal evolvability dynamics, so they instead built the evolvability concept implicitly within their theoretical models (e.g., Fisher's *Fundamental Theorem* or Wright's *Shifting Balance Theory*). We also need to take into account the broader scientific zeitgeist, i.e., *logical positivism*, that favored reductionistic interpretations of biological phenomena (Smocovitis 1996), thus concealing the emergent nature of evolvability.

This is why evolvability can be said to have *a short history but a long past*. Many recent historiographies on evolvability have suffered from historical revisionism and presentism by neglecting its *long past*. These historiographies—often briefly mentioned at the start of reviews—are subjectively directed toward the authors' *present* conceptualization of evolvability. For example, the quantitative geneticist Thomas Hansen (2016) gave a brief historical account of evolvability, but only as it is conceived in developmental biology today, claiming that evolvability is a relative newcomer to evolutionary biology because development was "black-boxed" during the modern synthesis (cf. Futuyma 2017; Chap. 32). He goes on to note that this all began to change in the 1970s and 1980s due to the renewed interest in evolutionary constraints, setting the stage for evolvability to become an official research front. Hansen and Pèlabon (2021), Porto (2021), Minelli (2017), and Brigandt (2015) made similar historical assertions, seeing evolvability as a relative newcomer because of their evo-devo lens.

The issue with these historical accounts is that they fall under the fallacy of *presentism*. They regard evolvability and its history as it is most commonly presented today, as a developmental phenomenon within the context of modern

biology. They entirely disregard the intellectual contexts that incubated evolvability, thus excluding a significant portion of the history of evolvability thought.

This is a misuse of history because it falsely promotes certain conceptualizations of evolvability over others. Today, this has manifested into the precedence given to an evo-devo approach of evolvability over its *long past as genetic evolvability*. How scientific ideas are conceptually constructed influences their perceived history, yet good history and science rely on the opposite to be true. *History should influence how scientific ideas are conceptually constructed*. Thus, we need an accurate philosophical history of evolvability to help inform its conceptual construction today.

Evolvability *as an idea* has a much deeper history that is indeed quite relevant to its modern conceptual construction. The philosopher Massimo Pigliucci (2008) was the first to give a historical account of evolvability *as an idea*, no matter what the biologists back then were calling it. Surprisingly, few authors have since followed in his footsteps. Here I provide a brief historiography of evolvability *as an idea* that is not currently acknowledged by those in the evolvability research front.

Many years before the coinage of the term "evolvability," the main aspects of evolvability loomed in the thoughts of early biologists when they were reasoning on the functionality of genetic variation. For example, August Weismann (1889, p. 272; 1904, p. 223) was the first to explicitly note the ontological connection between (a) the production of novel variation (from recombination), (b) variation in the ability to adapt between species, and (c) the subsequent beneficial effect this would have on a biological entity higher than the individual. This was the first instance that the mechanisms behind heritable variation were considered to hold some adaptive value.

After the neo-Darwinian era, many biologists retreated into a developmental viewpoint of evolution (an era called the "eclipse of Darwinism" [Bowler 1983]). In this era, developmental theories of adaptation, such as Lamarckian or Orthogenetic theory, superseded natural selection theory. "Adaptability"—a progenitor term for evolvability—was gained from organismal plasticity responses that tended toward Lamarckian inheritance (e.g., Baldwin 1896; Osborn 1896). However, Lloyd Morgan (1896) presented an eerily modern account of the evolvability process that did not suffer from any Lamarckian connotations, in which organismal plasticity or developmental mechanisms were seen as transitory responses that allow time for genetic mechanisms to cause adaptive evolution.

The modern synthesis (circa 1916–1950) delivered a decisive blow to the developmental perspectives of evolution. In this era, the population geneticist R.A. Fisher (1930) formalized Weismann's ideas with his *fundamental theorem of natural selection.* This theorem (in the vein of physical reductionism [Smocovitis 1996]) mathematically demonstrated that the rate of change of mean fitness is equal to the genetic variance of a species. It follows from Fisher's theorem that the potential of a species to respond to selective pressures is contingent on the amount of genetic variation (or more precisely, additive genetic variation), which became a crude measurement of evolvability that is still in practice today by quantitative geneticists.

Like Weismann, the evolutionary geneticist Theodosius Dobzhansky was an early empirical investigator into the origins and nature of genetic variation in natural

populations. Due to his empirical work, Dobzhansky claimed that populations with increased variation would eventually outcompete populations with lower variation because of the fitness advantage it would eventually confer, despite the short-term fitness costs to individuals within a population (Borrello 2010). "A species perfectly adapted to its environment may be destroyed by a change in the latter if no hereditary variability is available in this hour of need. Evolutionary plasticity can be purchased only at the ruthlessly dear price of continuously sacrificing some individuals to death from unfavorable mutations" (Dobzhansky 1937, pp. 126–127).

Evolutionary ecologists built off the observational work started by Dobzhansky in the latter half of the twentieth century. The concepts of "adaptability" or "environmental flexibility" captured the general idea of evolvability when ecologists would discuss how a population survives and adapts in multiple or changing environments, and how this was tied to populational properties of standing genetic variation (e.g., Levins 1968; Lewontin 1974; Endler 1986, p. 48).

Yet the original notion of evolvability as adaptive genetic variation would eventually become superseded by a far more general and broad view of evolvability that instead focused on the phenotypic consequences of transient "evolvability" mechanisms. This transition was likely due to the concomitant expansion of developmental evolutionary biology in the 1990s, around the same time that evolvability was becoming popularized (Nuño de la Rosa 2017).

Therefore, the evolvability research front began to mature within an intellectual milieu that placed a greater emphasis on development and phenotype, moving away from what many saw as the outdated reductionism of gene-centered perspective. I think the initial motivations for this movement were worthy and have inspired much progress in our thinking about evolution, as evidenced by the large compendiums now devoted to developmental thinking within evolutionary biology (e.g., Nuño de la Rosa and Müller 2018).

Evolvability is not merely a function of generating genetic variation (Burch and Chao 2000). Explaining the evolution of complex traits requires knowledge of the organization, growth, and development of *organisms* (Kirschner and Gerhart 2005). No sensible student of evolvability could ignore the causal importance that development plays in the adaptation process, especially when we start to distinguish the evo-devo concepts whose causal explanatory importance is likely greater (e.g., the G-P map and/or developmental modularity) than the non-genetic concepts whose evolutionary consequences are perhaps more transient or less consequential (e.g., epigenetic variation, protein promiscuity).

However, by focusing too greatly on development, those in the evolvability research front have continually overshadowed the causal explanatory import of long-term (or higher-level) genetic evolvability. Massive and pertinent literatures exist outside of the traditional bounds of evolvability research, above (e.g., ecology) and below (e.g., microbiology) the typical scope of developmental research on organisms.

Evolvability cannot be *only* couched in terms potent to evo-devo, precisely because 30 years of research have made it abundantly clear that evolutionary biologists and developmental biologists *generally* investigate phenomena that

operate at different timescales, at different levels of biological organization, or that differ in their downstream evolutionary consequences. Evo-devo approaches can readily explain short-term adaptation to novel environmental stimuli, but they run into difficulties when they try to explain longer-term evolutionary trends and adaptation at higher levels. By disregarding the successes of modern genetics and genetic theory, those in the evo-devo camp have routinely overrepresented the theoretical significance of development in the evolutionary process, with evolvability being the perfect example of this trend. This is why we need to reemphasize the importance of the G side of the G-P map, to aid in the explanation of long-term or *macroevolvability* trends.

38.3 Evolvability Theory Today: The Issue(s) with Evolvability

Within the past 30 years, understanding why biological entities vary in their capacity or propensity for evolution—i.e., *evolvability*—has bloomed into a central research front within evolutionary biology, catching the attention of biologists from every major sub-discipline (Nuño de la Rosa 2017; e.g., Houle 1992; Wagner and Altenberg 1996; Kirschner and Gerhart 1998; Gerhart and Kirschner 1997, 2007; Earl and Deem 2004; Pigliucci 2008; Brookfield 2001, 2009; Wagner and Draghi 2010; Arenas and Cooper 2013; Brown 2014; Minelli 2017; Payne and Wagner 2018; Porto 2021; Riederer et al. 2022; Hansen et al. in press). Despite the influx of new work that describes or mentions evolvability, it remains more conceptually fuzzy now than it did when it was first popularized over 30 years ago, evidenced by the diversity or "plurality" of conceptions of evolvability, or by the large volumes dedicated to explaining such diversity (e.g., Hansen et al. in press).

We are no closer to agreeing on *what evolvability is*; that is, *what are the bearers of evolvability* (the entity possessing the capacity to evolve, e.g., traits, individuals, populations), *what biological features make up the causal basis or causally contribute to evolvability* (e.g., developmental systems, genetic systems) and how do they differ in their *causal attributes,* such as *causal influence* (Lewis 2000) or *causal specificity* (Woodward 2010), and finally, *what phenomena should evolvability be conceptualized to explain?*

Brookfield (2001, 2009), Love (2003), Pigliucci (2008), and Brigandt et al. (in press-a, in press-b) mark the conceptual confusion surrounding evolvability as likely the result of the term being used to refer to multiple distinct, but overlapping, phenomena related to the production or storage of novel variation (both genetic and phenotypic) and its consequent effects on adaptation. It is for this reason that most in the evolvability research front agree on the prescription of a broad and *unrestrictive pluralism* for evolvability to solve its conceptual issues (e.g., Hansen et al. in press; Nuño de la Rosa 2017; Brown 2014). These broad models of evolvability encompass all evolvability-like phenomena under the same conceptual umbrella—i.e., phenotypic plasticity, developmental trait plasticity, epigenetic variation, G-P map, modularity, robustness, evolutionary capacitance, or genetic evolvability.

While it is true that the eclectic assemblage of definitions and associated concepts certainly speaks to the need for a broad-type pluralism of "evolvability" or something similar across multiple domains, taking such a broad and inclusive approach exacerbates the issue(s) with evolvability. What these broad conceptions of evolvability often gain in generality (e.g., are easily understood, increased explanatory breadth) they lose in specificity (e.g., explanatory/predictive power, causal adequacy, theoretical coherency). Much of the conceptual confusion surrounding evolvability is caused by this lack of specificity, which in turn hinders the capacity of evolvability to exist within the theoretical background of modern evolutionary theory, like an oversized puzzle piece.

38.4 Non-Genetic (or *Evo-Devo*) Evolvability

To expand on these issues, let us consider the most popular cluster of evolvability conceptions, *the evolutionary developmental biology (evo-devo) concept of evolvability* (Nuño de la Rosa 2017). Richard Dawkins (1988) and Peter Alberch (1991) jumpstarted the evo-devo concept of evolvability with their initial focus on development, effectively defining evolvability as a "property of embryological systems, i.e., certain types of developmental systems are better at evolving" (Alberch 1991, p. 9).

With their two publications in top journals, Wagner and Altenberg (1996) and Kirschner and Gerhart (1998) propelled the evo-devo approach into the mainstream and made development the proper focus of evolvability theory (Nuño de la Rosa 2017). The authors broadly emphasized the role that development plays in the production or structure of phenotypic variation, defining evolvability as *the capacity to generate heritable adaptive phenotypic variation* (influencing others to do the same: e.g., Payne and Wagner 2018; Minelli 2017; Porto 2021). They argue that properties of developmental systems—such as the G-P map, protein versatility, weak linkage, compartmentalization or modularity, developmental trait plasticity, exploratory behavior, or the epigenome—were related to evolvability since they bias the amount and kind of phenotypic variation expressed in evolutionary systems so that more favorable and nonlethal kinds of variation are made available to natural selection in times of need.

These approaches have been referred to as *non-genetic evolvability* since they go beyond the mechanisms of genetic change, from mutation or recombination, to focus on the organizational and structural mechanisms of organisms that influence and optimize variation production in complex systems (Wagner and Laubichler 2004). Of course, many "non-genetic" mechanisms may be underpinned by genetic processes, as rightly recognized by many in the evolvability research front. However, the organizing theme of the evo-devo concept of evolvability is the special emphasis that it places on the production or structure of phenotypic variation since

"phenotypic variation is the selectable material of natural selection" [4] (e.g., Brookfield 2001; Payne and Wagner 2018).

For example, significant research attention within the evolvability research front has been directed toward the modularity of the G-P map and how phenotypic robustness promotes evolvability (Wagner and Altenberg 1996; Kirschner and Gerhart 1998; Wagner 2005; Masel and Trotter 2010; Wilder and Stanley 2015; Pavlicev et al. in press). This work has convincingly shown that most species have an innate "evolvability" to (a) buffer lethal mutations and (b) reduce the number of mutations needed to produce phenotypically novel traits. Both observations correspond to the way that genetic variation is modulated (compartmentalized) and turned into phenotypic variation by the G-P map.

However, despite its success to discover new and exciting phenomena related to evolvability, the evo-devo approach has perpetuated and, in some cases, exacerbated the conceptual issues with evolvability. Several have argued that the broadness of the evo-devo concept is a virtue, thus focusing on the explanatory breadth of evolvability to capture multiple overlapping phenomena (e.g., Pigliucci 2008; Nuño de la Rosa 2017; Brown 2014; Payne and Wagner 2018; Brigandt et al. in press-a, in press-b). Taking such a broad approach to evolvability has in turn distracted us from the complete causal field of evolvability, including upstream causal events like genetic evolvability.

38.5 Drawing a Causal Distinction Between Non-genetic and Genetic Evolvability

Because of its emphasis on development and phenotypic variation, the evo-devo concept often fails to delineate between short- and long-term evolvability phenomena. This is to say that the evo-devo concept ***does not delineate*** between the mechanisms that generate genetic, long-term, and heritable change from the mechanisms that generate non-genetic and non-heritable (or *transiently heritable*) change, such as epigenetic variation or stochastic gene expression. Both types of mechanisms contribute to the evolvability of populations, but *they contribute in different and significant ways*. Non-genetic evolvability mechanisms generate phenotypic heterogeneity without creating genetic variation, making these changes more transient in the evolutionary process.

For example, in a recent review of evolvability published in *Nature Review Genetics*, Payne and Wagner (2018) considered four non-genetic mechanisms that create phenotypic heterogeneity as "evolvability mechanisms"—i.e., stochastic gene expression, errors in protein synthesis, epigenetic variation, and protein

[4]The assumption "phenotypic variation is the selectable material of natural selection" underdetermines the causation of natural selection, and in many considerations, runs parallel to the *random variation assumption* mentioned at the start, since it assumes that genetic mechanisms are stochastic and selectively unimportant.

promiscuity.[5] According to the authors, the phenotypes created by these non-genetic mechanisms "may themselves be heritable, eventually made permanent by mutation or epigenetic modification, or they may simply 'buy time' for a population to adapt in other ways to an environmental change" (Payne and Wagner 2018, p. 25). [6]

The authors go on to demonstrate this point by arguing that epigenetic modifications can create phenotypic heterogeneity from the changes in the protein conformations of prions. For example, the prion [PSI^{+}] in *S. cerevisiae* is an aggregated conformation of the translational suppressor Sup35 protein, which causes reduced translational fidelity. Some of these errors reveal cryptic genetic variation, producing adaptive phenotypes that are transiently heritable for several generations in response to pressures. The authors suggest that these epigenetic modifications of prions "buy time" for mutation and recombination mechanisms to catch up and cause an adaptive, long-term, heritable change.

In the causal story of evolvability extrapolated by the authors, the non-genetic mechanisms that cause phenotypic heterogeneity are the salient causal aspects that lead to the ensuing *evolvability*. The *causal emphasis* is put on the phenotypic variation generated, even though the authors confusingly recognize the secondary or "conditional" causal role that the non-genetic mechanisms play by "buying time" for mutation and/or recombination mechanisms to catch up and cause adaptive and long-term change. Alas, this is an example of how most causally conceive of the evolvability process today. Their attention is put on the biological mechanisms *downstream* from genetic evolvability mechanisms.

What is being neglected by the evolvability research front is the upstream disparity in causation, or a disparity in the mechanisms that cause genetic evolvability *between species*, rather than all species having the same capacity to produce, conserve, or domesticate genetic variation. Indeed, if every species had the same capacity for genetic evolution, [7] and most of the differences of variation existed at the organismal-developmental level, then the most salient aspect of evolvability, as well as the direct causal element of evolvability, would be non-genetic evolvability mechanisms that modulate invariable or stochastic genetic variation that subsequently turns into phenotypic heterogeneity.

[5] Most of the non-genetic mechanisms mentioned by Payne and Wagner (2018) may be better served under the conceptual umbrella of phenotypic plasticity—or the ability of an organism to change its phenotype in response to changes in the environment (Pigliucci et al. 2006, p. 2363). Phenotypic plasticity mechanisms are genetically ingrained mechanisms that reflect non-genetic adaptive changes. Every mechanism that these authors have thus classified as non-genetic "evolvability mechanisms" functions better under the concept of phenotypic plasticity since their evolvability-related effects are rather transient in comparison.

[6] The underlying causal mechanisms governing these processes are not well understood, and they may as well be the result of genetic contributions (Merilä and Hendry 2014; Birney et al. 2016; Lappalainen and Greally 2017). We are thus left to assuming some amount of epistemic risk when we claim to know the causal basis for the observed phenotypic heterogeneity.

[7] Constant, Invariable, or stochastic genetic evolution is often a presupposition for other failed notions such as the random variation assumption or the molecular clock.

However, species do not exhibit the same capacity for genetic evolution due to non-random selective reasons. Mutation and/or recombination rates are incredibly variable throughout taxa and levels of biology, often dependent on numerous causal factors, including selection (Lobkovsky et al. 2016; Swings et al. 2017). It has been widely appreciated in microbiology that natural selection *can causally intervene and influence the mechanisms of genetic variation, with some species being more genetically evolvable* since at least the 1960s (Fitzgerald and Rosenberg 2019).

This seems to be a point that is strangely absent in most considerations of evolvability (e.g., Brown 2014; Brigandt et al. in press-a, in press-b). For example, Nuño de la Rosa and Villegas (2019) note that the G-P map governs how "random genetic mutation" translates into non-random, structured, and possibly adaptive phenotypic variation for characters exhibited by particular types of organismal systems. What is missing from these considerations is the fact that there is *non-random* discriminate sampling in the processes of mutation/recombination themselves, with the variation in these processes being best attributed to *between populations* because of the interlevel conflicts that typically accompany the emergent benefits (i.e., the individual-level costs of recombination or mutational load).

Such a disparity strongly suggests that higher-level natural selection acting on mutation/recombination modifiers is an upstream causal event from the generation of phenotypic heterogeneity that needs to be distinguished from other downstream causal events of evolvability, such as those pertaining to *non-genetic evolvability*. It also suggests that causal distinctions can be made between the amount of causal influence (Lewis 2000) or specificity (Woodward 2010) [8] that these two types of evolvability exhibit, with early indicators suggesting that genetic evolvability is more causally influential and causally specific than non-genetic evolvability mechanisms.

When we draw parallels between other literatures with similar explanatory goals, such as the evolutionary rescue research front, we see similar observations arising. Developmental mechanisms (and dispersal methods) might be initially sufficient to relieve minor pressures and allow for population persistence. Yet when too great of pressures are applied, populations generally shift their strategies to facilitate adaptive evolution through genetic changes (Carlson et al. 2014; Merilä and Hendry 2014; Bonnett et al. 2022). Indeed, such a distinction is important to make in the evolutionary rescue research front, exhibited by the work that attempts to establish the time frames over which genetic change versus existing phenotypic plasticity will be

[8] Relating to causal specificity, most students of evolvability are the first to recognize how little is still known about the underlying mechanisms of the G-P map, or how genetic variation is turned into phenotypic variation. There seems to be an element of epistemic and/or aleatory risk involved with accepting evo-devo conceptions of evolvability (in a similar but less-dire case as that presented by Biddle 2016). The ontological causal relationship between non-genetic evolvability mechanisms and their ensuing evolvability-related effects remains a major question mark. This suggests that non-genetic mechanisms may be less causally specific than genetic evolvability mechanisms since we have a clear causal relationship between genetic evolvability and its effects on adaptation.

most important for population persistence (Chevin et al. 2013; Kovach-Orr and Fussmann 2013; sources drawn from Hendry et al. 2018).

The available evidence thus implies an ontological and causal distinction between the direct causal elements of genetic evolvability (e.g., evolvability mechanisms like HGT, stress-induced mutation, or meiotic recombination) that generate genotypic heterogeneity, and the non-genetic causes that influence how effective genetic evolvability mechanisms are at producing adaptive phenotypic changes (structural causes) or the developmental mechanisms that produce phenotypic heterogeneity and "buy time" for genetic evolvability mechanisms to cause adaptive evolution. This is because non-genetic evolvability mechanisms appear to be *conditional elements* of genetic change (i.e., structural causes), rather than the direct causal elements (or what is called a *triggering cause* in the causal literature) of evolvability.[9] Non-genetic mechanisms certainly aid in the facilitation of adaptive evolution, but their role is better cast as *conditional* rather than *causal*. Like a silencer to a pistol, conditional non-genetic mechanisms likely evolved to augment and modulate the mechanisms of genetic variation.

Other examples include the influence of the G-P map on evolvability since it promotes greater evolvability *but also greater robustness* (Pavlicev et al. in press). The G-P map is best described as a structural cause that eases the selective constraints of genetic mechanisms, which in turn allows for the direct causation of genetic mechanisms to generate more adaptive mutations in the future (Masel and Trotter 2010). Likewise, Brown (2014) noted that weak constraints on developmental traits afford a greater probability that traits can evolve in response to environmental demands. Such constraints only make the probability of a beneficial mutation of a trait more likely, whereas the direct causal action of evolvability is contingent on the genetic mechanisms (that are also probabilistically dependent but upstream causal events).

The causal distinctions that abound between genetic and non-genetic "evolvability" mechanisms—such as differences in causal influence and downstream effects, causal specificity, and/or spatial location—strongly suggest that we must maintain something like the ultimate/proximate causal distinction between genes/development (i.e., *Weismann's barrier)* in the evolvability concept, contrary to what several progressives have argued (c.f. Uller and Laland 2019; Laland et al. 2011). For precisely the same reasons why we drew a distinction between development and genetics in evolutionary biology over a century ago (i.e., *Weismann's barrier*), and why we still view the explanatory utility of Lamarckian or soft inheritance as inferior to hard-inheritance structures, are precisely the same reasons why we should draw a similar distinction between *shorter-term phenotypic*

[9] The distinction made here is similar to what Mackie [1965] refers to as the predisposing causes (causal conditions that set the stage for an event to occur) from triggering causes (causes that trigger the event's occurrence), which is a common distinction made in the medical literature (e.g., smoking increasing the probability of causing cancer).

evolvability from developmental mechanisms and *longer-term higher-level genetic evolvability*.

If evolvability is to become a mature causal explanatory model, we must have a good grasp of its causal relations. When we are constructing a causal explanatory model, especially one that attempts to capture the complete causal field of a complex phenomenon such as evolvability, we must make further causal distinctions so that we can accurately and precisely model and replicate this process in the future. Maintaining such a causal distinction between non-genetic versus genetic evolvability thus organizes similar phenomena while also achieving a concreteness in conceptual parameters, which should be preferable from the typically broad conceptual model of evolvability that hastily categorizes all the evolvability-related explanandum under one conceptual umbrella. We need to reemphasize the *evolvability gained from genetic changes* rather than the nondescript evolutionary potential gained from non-genetic, non-adaptive developmental processes.

38.6 Prescribing a *Restrictive Pluralism* to Solve Evolvability's Conceptual Issues

Many argue for a broad or *unrestricted "anything goes" type of pluralism* to resolve the conceptual issues with evolvability, which is often overly inclusive of overlapping phenomena since they include developmental evolvability mechanisms alongside genetic evolvability mechanisms without drawing any major causal distinctions between the two types or other typologies (Hansen et al., in press; Nuño de la Rosa 2017; Brown 2014). Yet rather than taking such an inclusive approach, comparing and contrasting the various merits of competing accounts of evolvability may prove useful to enhance its conceptual clarity and allow for its successful integration into modern evolutionary theory.

This rationalist strategy has been referred to as *restricted pluralism*, more commonly recognized in the economics literature when strictly contrasted with an "*anything goes*" *type of pluralism* (Marques and Weisman 2008), because it tolerates a heterogeneity of viewpoints within some sort of homogenous cluster, while simultaneously calling for the discrimination of the heterogeneity within such a cluster. This is to say that *restricted pluralism* allows for the comparison of competing models, ideas, or hypotheses based on any sort of demarcationist criteria, thus allowing space for the construction of more nuanced and complex theoretical models. Restricted pluralism is thus a rational reaction to an ever-increasing ontological complexity found in most sciences today. And a complex concept such as evolvability likely necessitates taking such a nuanced pluralistic approach.

Restricted pluralism goes hand in hand with building philosophical literature on *scientific theoretical virtues,* which acts as the objective criteria to reliably sort through similar yet competing ideas. Philosophers of science have recently devoted much attention to systematizing the scientific theoretical virtues (Kuhn 1977; Brock and Durlauf 1999; Keas 2017; Schindler 2018). Indeed, this is an excitingly novel approach to theoretical argumentation in the sciences. Instead of arguing for a

scientific theory (or a promising hypothesis) by demonstrating its underlying *empirical adequacy* or other strictly epistemic virtues (e.g., Popper 1959), which is the most common practice in science today and throughout history, we can now compare and contrast the various scientific virtues—those of an epistemic, non-epistemic, or pragmatic nature—of a theoretical model within a neat and orderly standardized framework (e.g., Baedke et al. 2020).

Thanks to ecology and other empirically rich traditions that have allowed for the investigation of causally complex evolutionary dynamics over longer stretches of time and frequent spatial changes, *genetic evolvability* is now an epistemically virtuous concept. However, what also sets *genetic evolvability* apart from *non-genetic evolvability* is the non-epistemic virtue of *theoretical coherency* (or what Keas [2018] calls *universally coherent). Theoretical coherency* is a non-epistemic virtue because it relates to how our knowledge is structured and how new knowledge can be best integrated within a prevailing scientific paradigm. When constructing a novel concept, we must pay heed to the existing theoretical structure; to how well the novel concept sits with most of our modern theoretical structure of evolution. [10] Yet the novelty of a promising concept often blinds us to thinking about how it might fit and integrate within an existing theoretical structure.

It is important to remember that scientific concepts are social constructs, subjectively framed to integrate homogenous phenomena or data under a common, normalized representational model that is externally valid. The "goalposts" of our models (or *conceptual parameters*) can always be moved following new observations and evidence. Often the more rigid the parameters of a concept are drawn, the easier it is to understand its causal workings in a specific context, which in turn generally enhances its understandability within the larger causal picture of putative theory. When concepts lose their rigidity, they become subject to ad hoc reasoning (Schindler 2018), and they also tend to lose their meaning and procure confusion. [11] This is precisely what is happening in the evolvability literature today.

Genetic evolvability maintains *Weismann's barrier* and thus keeps with the causal criteria of modern genetic theory better than its alternatives. [12] Non-genetic evolvability, in contrast, places too great of an emphasis on development and not enough emphasis on genetic evolvability. And while development clearly plays an

[10] This is also a likely reason why evolvability was initially built within evolutionary theory and neglected for so long.

[11] In no other science, I think, is this better appreciated than in biology due to the immense conceptual and phenomenological overlap.

[12] There are, however, several aspects of genetic evolvability that are *incoherent* with our existing theory, and excitingly, this is where theoretical progress should happen. The reductive atmosphere that born the modern synthesis and modern evolutionary theory has been proven by ecology to be too abstract and unrealistic of natural parameters. We must move toward a pluralistic, multilevel, multicausal model of natural selection if we wish to explain complex adaptations, like those surrounding genetic evolvability (i.e., sex and adaptive mutation; see for more Distin, in press). For these reasons, we must conserve several remnants of genetic theory, such as *Weismann's barrier*, while calling for the general theoretical progress away from the reductionistic causal modeling of biology old.

integral role in the evolvability process, there is not enough evidence to discharge *Weismann's barrier*. Until we find more evidence that demonstrates how developmental or non-genetic evolvability mechanisms influence the evolutionary process in the long term, *Weismann's barrier* is here to stay.

It is thus problematic to encompass all the mechanisms of evolvability under one conceptual umbrella, without making any further distinctions. In the first place, the generality of these definitions makes it difficult for biologists to form a proper quantification of evolvability; one that can be used in theory by quantitative and population geneticists (Hansen et al. 2011) or as a standard for experimental practice and comparison. These are important for the prediction of evolutionary outcomes in natural populations (Pigliucci 2008; Palmer and Feldman 2012). Often in the history of science, we have seen a tradeoff between the explanatory breadth (how many phenomena a concept can explain) and the predictive power of scientific theories. When too wide of an explanatory net is cast, predictive power becomes more difficult.

Yet generality of explanation (i.e., explanatory breadth, explanatory consistency) is still considered by many to be a hallmark of good science. However, the history of science indicates that as scientific disciplines grow and mature, they evolve to form more specific explanations that better explain the causal field of complex phenomena—*they often tend toward explanatory pluralism at a discipline-wide level* (Dupre 1993; Mitchell 2003, 2009). Yet scientists still instinctively lean toward this generality when constructing new conceptual models, which is a significant deterrent to forming better, more accurate, and more predictive causal explanatory models, especially in a discipline with a casually complex explanandum like biology. For these reasons, some sort of broad pluralism is indeed warranted at the discipline-wide level of biology. But pluralism should not be overly prescribed, which is often the case under an *unrestrictive or "anything goes" type of pluralism*.

This is why we must first reach a consensus within the evolutionary biology community as to what constitutes *evolvability* and set our sights away from any broad or unrestricted type of pluralism for the time being. Scientific concepts benefit from a restrictive pluralism while in their infantile stages, to first construct a sturdy conceptual parameter around one or a few readily observable phenomena and then build out from this foundational point. Explanatory pluralism generally follows once a concept is established and advanced, as standardized methods become further refined enough to investigate more peripheral phenomena related to the concept.

One needs to look no further than natural selection theory as the perfect example of this trend. Natural selection *was not ready to be pluralized* until the recent synthesis between evolutionary biology and ecology. We simply did not know enough about selective dynamics until we began to incorporate ecological analyses in evolutionary biology, which is why natural selection theory was initially best served by the philosophies of *explanatory monism* and/or *reductionism*. Early progenitors of evolutionary theory were therefore not doing a disservice by reducing or *monizing* natural selection to lower causal levels; rather, their *modus operandi* was aligned with the best interest of biology during their time, within their

intellectual zeitgeist (i.e., logical positivism), and with their technological limitations and scarce epistemic reservoir.

For these reasons, taking a more narrowed or *restrictive pluralistic* approach is not only in the best interest of the evolvability concept given the available evidence but also in the best interest of evolutionary theory more generally. This calls for the further refinement of the evolvability concept, cutting up its conceptual parameters to arrive at a more accurate causal picture of evolvability within the broader scope of evolutionary dynamics.

Evolvability is thus not ready to be a "foundational block" of the *EES* (e.g., Pigliucci 2008; Wagner and Draghi 2010), especially when it is conceived as a developmental phenomenon. Progressives that make such calls are clearly lacking (a) sufficient empirical evidence to support their recommendations, and (b) the foresight necessary to neatly construct and integrate a novel concept into the existing theoretical structure—similar to what I have recognized in the rest of the EES as well.

38.7 Theoretical Progress in Biology and the Failures of Modern Science

Biology is unlike any other scientific discipline, hence why we need *scientific disunity* (Dupre 1993; Cartwright et al. 1996). We have never undergone a paradigmatic revolution as described by the philosopher of science Thomas Kuhn in his famous *Structure of Scientific Revolutions* (1962). Biological theories are not *incommensurable* between competing or parent-offspring "paradigms," as theory is generally built cumulatively and progressively. Biologists have been building upon the same theoretical core of adaptation, inheritance, and variation since the formalization of modern biology in the synthetic era (Smocovitis 1996).

To echo what was said by D.J. Futuyma in Chap. 12, new conceptual ideas or theoretical additions have been consistently uploaded into evolutionary theory through the "synthesis" with other biological subdisciplines. Population biology was added in the 1960s, microbiology in the 1970s, ecology in the 1980s, evo-devo and conservational biology in the 1990s, systems biology in the 2000s, synthetic biology in the 2010s—so on and so forth. Each synthesis has brought with it a new understanding of the evolutionary process, all revolving around the same theoretical core ideas as before (with a rotating yet refined emphasis put on one concept over another, e.g., selection vs. variation vs. neutral evolution). Biology is thus not a discipline of *scientific revolutions*, but of *syntheses*, with *The Modern Synthesis* receiving the most attention (and equal *misunderstanding*) because it was our founding synthesis—but we have progressed theory a lot since then.

One reason why we have seen so many syntheses in the history of biology, and why we see the rising tensions today, is because of the subject matter we investigate. Biological systems are singular in their causal complexity. Biological causation is context-dependent because the phenomena we explore are extremely variable in space and time (why there are no such things as *laws* in biology). Our causation also

tends to be multifactorial, multilevel with upward and downward causation between biological levels of organization, with a myriad of evolutionary variables (e.g., life-history, population dynamics) that affect biological features in the present (see for more Mitchell 2003). Add onto this the notion of *reciprocal causation*, that organisms influence their environment and vice versa. Biological causation is indeed very *messy* (see for more Anjum and Mumford 2018).

Such causal complexity invites the divergence of various subdisciplines. Causal ideas arrive from many places in biology, precisely because we investigate the same causal phenomena from various perspectives (e.g., *adaptation*) using different methodologies (e.g., ecology vs. microbiology). In her brilliant book *Idealization* (2017), philosopher of science Angela Potochnik convincingly demonstrates how and why scientists selectively attenuate their research agendas to a particular cause or causal pattern of interest. Their investigations result in the construction of over-simplified and idealized causal models, that disregard other important causal information that lie outside of their chosen periphery. Perhaps no other science is this better appreciated than biology. Biologists routinely over-emphasize and/or neglect key causal information that lie outside the traditional scope of their discipline, due to the messy causation inherent to biological systems, which results in continuous calls for theoretical progress and new syntheses (whether they are founded or unfounded).

In the case presented here, I believe there is a significant place for evo-devo to make great insights where population genetics has overstepped and limited their research focal. But on the other hand, evo-devo has, in many ways, overstepped its boundary and neglected important causal information deriving from modern genetics and ecology. To explain such a causally complex world as we have in biology, we likely need an integrative pluralism (Mitchell 2003, 2009). But in the instance of evolvability, before we can reach any such integrative point, we first need *a restrictive pluralism* to identify the core causes of evolvability.

This is why proponents of the EES are right to call out the fact that biological theory is having a hard time keeping up with the waves of new evidence coming in from all walks of biology (e.g., Uller and Laland 2019). There is no question that we do need new explanatory and theoretical strategies to explain the causal complexities that are only just being revealed by our superior empirical methodologies.

Yet I see this as a symptom of the scientific process in general, and not something that is terribly specific to biology. Technological progress over the past half-century has led to a dramatic increase in knowledge in every scientific discipline, which is consequently having a hard time being translated back into theory. For this reason alone, I have a growing suspicion that the rising dissension between biologists has less to do with metaphysical or epistemological concerns, and more to do with the inadequacies of how the scientific process is structured itself.

For example, we still communicate and verify science using nearly the same journal system as we did 350 years ago. Such methods were suitable for knowledge production and dissemination back in the time of snail-mail and when "horsepower" actually pertained to horses. But in our modern context, these methods are due to reformation. With the amount of new information constantly being turned out by the scientific machine, theoretical progress is stalled by having such a slow uptake

process and no unified set or standard for theory to become concretized and/or upended.

Scientific fact and theory are based on consensus, yet we have no practical means for surveying the opinions of scientists, despite living in a technologically capable world. Scientists must resort to reading between the lines of esoteric and extensive literatures, quite literally guessing where theory currently stands. Until this issue is resolved then every scientific theory can expect repeated and unwarranted attacks, especially in evolutionary biology.

The abstractness of evolutionary theory lends itself to more attacks than perhaps any other scientific theoretical structure (as implicitly noted by Welch [2017], Futuyma [Chap. 32]). Evolutionary theory is too esoteric for any scientist working outside the traditional scope of evolutionary biology to pick up our literature and understand where our knowledge currently stands and how it maps back into theory (perfectly exemplified by those esteemed biologists who found success in their respective discipline and mistook their success for knowledge of evolution, i.e., "*The Third Way of Evolution"*). Students of evolution do not know where evolutionary theory currently stands because *we as a discipline do not know where our theory currently stands.*

Indeed, I think that these intellectual battles are to the benefit of science and scientific theory. Still, it would be helpful to have a standardized representation of theory that scientists could continually argue and update, which happens outside of the traditional bounds of "normal science" or empirical efforts.

The main issue, then, is not the fault of progressives or conservatives, but the scientific process in general. Biological theory mimics the phenomena we study; we have an ever-changing, amorphous theory. Slight theoretical modifications are constantly "being added" or advocated, but are they being understood or integrated? Because we have no good means of surveying the opinions of scientists, scientists are constantly shooting at an ever-changing and imperceptible theory, literally guessing where the edges of theory lie. If we had a set or standard for forming and maintaining theory, then this would allow for more accurate critiques and streamline theoretical progress. Therefore, modern science needs a massive makeover, or else the history of science will continue to be one of slow, ineffectual theoretical progress, as demonstrable by the history and present of *evolvability*.

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Pluralism and Progress in Evolutionary Biology: A Commentary on Distin

39

J. Arvid Ågren

Abstract

Distin offers a fascinating account of evolvability and its vexed place in evolutionary theory. I draw on the history of social evolution to offer some suggestions how to move the debate forward.

Keywords

Evolvability · Kin selection · Pluralism

When I was in the third year of my doctorate at the University of Toronto, I became co-president of the department's graduate student association. The tasks of the job ranged from organizing social events like Darwin Day, to representing PhD students at the monthly faculty meetings. Upon learning the news, my father—a long-time chair of a university department—had one piece of advice: if you want to get anything done in such a role, the best approach is to present whatever changes you want to implement as the natural continuation of how things have always been done. If you can do that, you can implement rather drastic reforms.

I was reminded of this advice when reading Distin's discussion of evolvability and the tension between conservative and progressive science. I have previously discussed the sentiment in reference to the emergence of the gene's-eye view of evolution (Ågren 2021, pp. 183–184) and Distin's chapter offers another data point. The fate of scientific ideas depends not only on their substance but also on how it is introduced in the context of the history of the field. That is especially true for ideas

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that are more like conceptual frameworks, rather than straightforward empirical hypotheses. For example, by demoting the organism—in many ways the *raison d'être* of biology—from the center of evolutionary explanations, the gene's-eye view is really quite a bold proposal. It deviates from how many people were used to thinking about evolution and natural selection. At the same time, it was always presented as the natural extension of Modern Synthesis. The conservative framing of George Williams and Richard Dawkins was in contrast to contemporaries, such as Stephen Jay Gould, who never missed a moment to emphasize the radical nature of their proposals (e.g., Gould 1980).

The history of evolvability shows similar features. The concept has come in many guises over the years, sometimes presented with radical implications, sometimes not. It has been associated with both defenders of the classical version of evolutionary theory, like Dawkins (who made a big deal of the variety of evolutionary trajectories of the biomorphs he simulated for *The Blind Watchmaker*; Dawkins 1986), as well as proponents of more far-reaching change, like Massimo Pigliucci (Pigliucci 2008; Pigliucci and Müller 2010).

Evolvability's untidy history, combined with its theoretical implications, is a source of both fascination and frustration for Distin. As he notes, evolvability on one level simply means the ability of a system to evolve. Problems arise when we try to nail down exactly what that is supposed to be mean. Under some definitions, it refers to little more than how the amount of genetic variation (measured by, say, heritability or the G-matrix) causes a response to selection. Other definitions are more concerned with biases in evolutionary trajectories that may stem from developmental constraints, and some center on how evolutionary innovations may lead to novel kinds of phenotypes. Running through all definitions is the question of who is the bearer of evolvability. A population, a species, a lineage? Or all of them?

With these issues in mind, Distin makes a number of recommendations for how to move the study of evolvability forward. I will pick up on one theme that runs through several of them: the interplay between history, semantic confusion, and scientific progress through pluralism.

Whereas the term itself is relatively young (credit for coining it seems to belong to Dawkins 1988), the ideas of evolvability are much older. Evolvability has a short history but a long past, as Distin nicely puts it. Once this history is appropriately acknowledged, the radicalness of the concept is somewhat deflated. Evolvability was never ignored, but instead biologists were wrestling with its implications before, during, and after the Modern Synthesis. Scientific progress requires an appreciation of history. Both so that we avoid re-inventing the wheel, but also so that we can properly acknowledge genuinely new findings and ideas.

I agree with Distin that biology is a special science. As a historical discipline with messy and context-dependent causality, the kind of elegant unification by mathematics that characterizes the history of physics—such as when Newton showed that what makes the apple fall on Earth is the same force that keeps the moon orbiting around it—is typically out of reach. At the same time, biology is also characterized by plenty of unity. Molecular mechanisms are often strikingly similar in widely diverged species, from *E. coli* to elephants. Furthermore, in contrast with physics,

the theory of evolution by natural selection means that biology already has its grand unifying theory.

Distin's reflections on the role of pluralism in scientific explanations are therefore interesting. In biology, we are quite used to the idea that there may different ways to describe a specific phenomenon, whether in the form of Mayr's ultimate-proximate distinction (Mayr 1961) or Tinbergen's four questions (Tinbergen 1963). A question that follows, however, is how much effort should be spent on integrating across explanations or models, as opposed to just getting on with it? I am of two minds on this, and it reminds me of that joke about the difference between scientists and philosophers. Scientists are puzzled by why philosophers spend so much time worrying about the exact meaning of words, whereas philosophers are befuddled about how scientists can get anything done without properly defining their terms. So on the one hand, all work we do as evolutionary biologists is ultimately in service of empirical understanding of living organisms in natural populations. And I say whatever helps you get there is good. On the other hand, when approaches are based on incompatible starting assumptions, I cannot help but worry.

This worry comes to me when reading Distin's historical sketch of the field of evolvability. The review reveals that the field is in desperate need of a unified taxonomy. Development of a common vocabulary will need to serve both descriptive (how the term is and has been used) and normative (how ought the term be used) ends. Semantic confusion takes us nowhere. Empirical progress and evaluation of what role evolvability should have in evolutionary theory can only happen if people are talking about the same thing. Distin makes the case for focusing on genetic variance well, but all three groups of definitions highlight some aspect of the concept worthy of study.

Given the issues facing the study of evolvability, I wonder if lessons from the group selection debate may offer a way forward. Despite (or perhaps thanks to) its cantankerous nature, the disagreements over group selection has a lot to teach us about how biology is done. Of particular relevance here, is what it tells us about the way we use terms and what consequences that may have for scientific progress. The distinction between group selection and kin selection goes back to John Maynard Smith, who coined the latter drawing on the work of W.D. Hamilton (Maynard Smith 1964). When reading Maynard Smith's paper, group selection—which here means his own haystack model—and kin selection seem very different. Half a century and a lot of debate later, most social evolution researchers hold the opposite view; the two are formally equivalent (e.g., Lehmann et al. 2007; Wilson and Wilson 2007). They are (merely) separate ways of doing the sums and conceptualizing the causality of natural selection.

Key to the contemporary equivalence claim is that both group selection and kin selection models have become more general over time. This generality, however, comes at a price. Mathematically the two can be related through the Price equation. The resulting statistical formalism underlying their connection is so abstract as to almost lose the biological meaning of the basic terms (Birch 2019). As a consequence, tensions between the two frameworks still linger. Part of the problem is semantic confusion. For example, take something as basic as what is a group. In the

group selection critic Maynard Smith's haystack model, the groups are well-bounded and reproductively isolated. In contrast, in the group selection advocate David Sloan Wilson's influential trait-group model, the 'group' has no discrete boundary (Wilson 1975). Instead, it can encapsulate something as fleeting as two individuals interacting once. These days, we can mathematically link the two kinds of models. However, if you are interested in the question of whether group selection is a major evolutionary force in natural populations, or in the history of life, the answer will depend on what kind of group you have in mind.

The notion of relatedness (r) has undergone a similar transformation. It initially corresponded roughly to a folk notion of genealogical kinship, but it is now often a regression-based estimate (Queller 1992). The cost (c) and benefit (b) components in Hamilton's Rule (that selection can favor costly social behavior if $rb > c$) have seen parallel generalizations. The generality of the terms has been incredibly helpful, as it allows disparate models to be conceptualized in the $rb > c$ inequality. But a problem similar to that of the meaning of groups noted above arises. The terms relatedness, cost, and benefit are now population measurements, rather than being in reference to specific social interactions, as our intuition would have it. Again, the risk for semantic confusion is real.

The evolvability debate also has a generality problem. As noted above, the term is used to refer to such a wide range of processes as to hamper progress in studying its importance. The solution offered by Birch (2019) for the group and kin selection debate may therefore have something to offer the evolvability one as well. Birch proposes a two-dimensional scheme that he calls K-G space, with a measure of kinship or genome-wide relatedness on one axis (K) and how "groupy" the collectives are on the other (G). Such a separation is helpful as both parameters are important to characterize social life. In the high K—high G corner, you find clearly defined groups with high relatedness, such as eusocial insects or the early stages of multicellularity. Next to that, in the low K—high G corner you have well-bounded groups of unrelated entities. A significant evolutionary moment in this space will have been the endosymbiotic origin of the eukaryotic cell. In the low G half of the space, social interactions are more fleeting. When combined with low K it may result in greenbeard-driven social behaviors and when combined with high K a possible scenario is that of the loose aggregations of early humans. Placing social behaviors on this K-G space can help us assess the relative importance of different processes in evolution and retain a meaningful pluralism of approaches.

Can a similar scheme be developed for evolvability? Systems may have a high propensity to evolve for different reasons, whether caused by genetic variation or developmental constraints, but it may still be meaningful to study them within the same conceptual framework.

In sum, Distin's historical approach to assessing the biological salience and theoretical status of evolvability is welcomed. Evolvability is an important topic and it deserves to be properly incorporated into contemporary theory. How radical the implications of such assimilation are will depend on how it is conceptualized and on what lessons we draw from history. The field of social evolution has a long

history of comparable disputes about vocabulary and integration. Learning from those may allow the study of evolvability to make faster progress.

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Genetic Evolvability: A Reply to Ågren

40

Mitchell Ryan Distin

Abstract

Genetic evolvability is an important concept because it demonstrates the need for progress that progressives are calling for, but within the traditional viewpoint and vernacular that conservatives can appreciate.

Keywords

Genetic evolvability · Gene's-eye view of evolution · Richard Dawkins · Reductionism · Emergence

Thank you for your insightful commentary, Arvid. Given the discrepancies between the two subject matters we both investigate—myself focused on an emergent viewpoint and yourself on a reductive viewpoint of evolution—it was to my surprise that I found myself agreeing with a lot of what you said. As you importantly noted, the *gene's eye view* was "always presented as the natural extension of the Modern Synthesis" because it worked within the reductive scientific atmosphere that born Modern Synthetic Theory.

I am glad you brought up Richard Dawkins. The most surprising finding from my historical inquiries was the nearly ubiquitous agreement between champions of a reductive viewpoint of evolution on the realism of *evolvability*, as an emergent property of *populations* or *species*. This is why I often refer to evolvability as "the best example of an emergent adaptation" because even those "dyed-in-the-wool, radical Darwinian(s) like me" (Dawkins, 1988, 201) would come to accept

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T. E. Dickins, B. J. A. Dickins (eds.), *Evolutionary Biology: Contemporary and Historical Reflections Upon Core Theory*, Evolutionary Biology – New Perspectives on Its Development 6, https://doi.org/10.1007/978-3-031-22028-9_40

evolvability as "a kind of higher-level selection, a selection not for survivability but or evolvability" (Ibid, 218). (Interesting side note: Dawkins was not the *coiner* of the term *evolvability*, as has been wrongly claimed by several in the evolvability research front, but he did appear to be the initial popularizer of the term, which in many respects is more historically salient; see Nuno de la Rosa, 2017).

Of course, early theorists did not call it *evolvability*. They called it a host of names, such as "evolutionary plasticity" (Dobzhansky 1937: 126–127), "adaptability" (Baldwin, 1896; Osborn, 1896), or "flexibility" (Mather, 1943: 44). Or they would allude to evolvability when they would reason about the adaptive consequences of sexual reproduction (Fisher, [1958] 1999: 50; Williams, 1971; Maynard Smith, 1978). But the salient commonality was that they all recognized evolvability early on as something of a paradox for evolutionary theory; that there was something special about evolvability that did not fit within the conventional Darwinian interpretation of evolution. As Graham Bell described the situation:

> It was the very success of this attack [on group selection] which led population biologists to realize how embarrassing sex is. Most supposedly altruistic behaviours were quickly found either to have concealed advantages for the individual, or else to be directed towards the welfare of closely related individuals. But sex appeared to fit into neither of these categories: if it permits the rapid mobilization of genetic variation, then this may be a matter of vital concern for the population, but does not in itself concern the individual. Evolutionary biologists thus found themselves on the horns of a dilemma: either the apparently unsatisfactory hypothesis of group selection was indeed an adequate explanation for the maintenance of sexuality, or else a quite different hypothesis framed in terms of natural selection must be sought (Bell 1982, 47).

Sex is a matter of vital concern precisely because of the emergent evolvability benefit ascribed to higher-level entities such as populations.

I think the historical uncertainty surrounding the evolvability concept is important to note, something that is not currently acknowledged by progressives or conservatives alike, outside as well as within the evolvability research front. Conservatives tend to not see natural selection as "a forward-looking force" precisely because of the reductive atmosphere that affected attitudes on higher-level selection for generations to come, whereas progressives call for new theory that typically exists outside the traditional gene-centered bounds.

This is why genetic evolvability is such an important concept because it demonstrates the need for progress that progressives are calling for, but within the traditional viewpoint and vernacular that conservatives can appreciate. As noticed here by Ågren, this is how you actually cause radical change in the sciences, not in the form or function of the EES.

Within the evolvability research front, progressives are actively squandering a massive opportunity to solve one of the longest-running and severe explanatory problems in evolutionary biology, *the paradox of sex.* Genetic evolvability is crucial to explaining the long-term evolutionary trajectories of species; explaining the existence and persistence of genetic evolvability mechanisms that conserve,

produce, or domesticate new or "better" genetic variations. Yet this brings up another vital point that Agren touches on, *how to provide a unified taxonomy for evolvability*.

To be fair, the evolvability research front has been building a unified taxonomy for all evolvability-like phenomena in the last few years (Hansen et al., forthcoming). And in many regards, they have been successful. However, I think the one area that they have overlooked, *genetic evolvability*, is exactly the area that could lead to their success and status in greater biology and would allow for a lot of unanswered questions surrounding evolvability to be resolved, such as the question of *the evolution of evolvability* that asks *what are the causal mechanisms that govern evolvability* (e.g., Pigliucci, 2008; Payne & Wagner, 2018; Hansen et al., *in press*)? Genetic evolvability clearly demonstrates the vital role that higher-level selection plays in the maintenance of evolvability mechanisms over time, thus bypassing the question of *the evolution of evolvability* altogether.

The analogies that Ågren draws to the issues of social evolution are therefore curious and also welcomed because they exhibit the power of historical and philosophical perspectives to solve age-old conceptual issues that have incessantly plagued scientists. Recent philosophical work on biological individuality (Pradeu, 2016), causality (Anjum & Mumford, 2018), and the levels of biological organization (Brooks et al., 2021) provide a useful framework to solve many of these issues. And novel perspectives on pluralism should also take center stage in these conversations. Whatever way we can "retain a meaningful pluralism of approaches" in the evolvability research front should always take precedence over division. Yet one thing is certain, whatever sort of pluralism we agree upon to resolve the generality issue should keep genetic evolvability at its conceptual and theoretical core.

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