Inclusive Fitness Teleology and Darwinian Explanatory Pluralism: A Theoretical Sketch and Application to Current Controversies

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Abstract Formal Darwinism (FD) [Grafen (2002) J Theor Biol 217:75–91; (2007) J Evol Biol 20:1243–1254.] is a theoretical framework for articulating optimization models in behavioral ecology and allele dynamics modeling in population genetics. It yields a teleology centered on inclusive fitness maximization ("IF teleology"), which captures the many aspects of teleology in Darwinian thinking [Huneman (2019b) Stud Hist Philos Sci Part C 76:101188. 10.1016/j.shpsc.2019.101188] and supports an explanatory pluralism in evolutionary biology. Based on this framework, the present chapter intends to show how the major distinctions regarding kinds of explanation identified in evolutionary biology can be connected and systematized through such explanatory pluralism. Then I will show that it can be redescribed in terms of Aristotle's four causes, and finally, it makes sense of the use of two distinct notions of causation. The rest of the paper analyses two examples where this FD-based pluralism and the correlated use of IF teleology allow one to cast a light on current controversies regarding evolutionary theory: the disputed need to overcome the Modern Synthesis of evolution because of non-genetic inheritance, biased variation, or niche construction; and the opposition of kin selection and multilevel selection regarding the evolution of altruism.

Keywords Explanation · Natural selection · Inclusive fitness · Teleology · Causation · Pluralism · Aristotle

1 Introduction

In this chapter, I will argue that in the context of the "Formal Darwinism" elaborated by Alan Grafen (2002, 2007, 2015), a framework for thinking of explanatory and causal pluralism in evolutionary biology can be designed. Formal Darwinism (FD) is

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a view that establishes isomorphisms between this teleology, mathematically understood in terms of "optimization programs," and the population genetics, which models allele dynamics in a population at the genetic level (those isomorphisms hold between programs and the Price equation, see Grafen 2002, 2007, 2014; Huneman 2014a, 2015). The FD-based pluralism articulates teleology and mechanisms of efficient causes. In Huneman (2019b), I already argued that the teleological kind of explanation could be accounted for in terms of "inclusive fitness," where inclusive fitness (of an organism) measures the benefit of a trait or a strategy as the contribution to the offspring directly produced by the focal organism, plus the contribution to offspring produced by other organisms, weighted by the relatedness coefficient.[1] This teleological explanation based on inclusive fitness was there shown to capture the two dimensions of *design* and *contrivance* proper to living organisms—namely, organisms seem to be designed, and their parts are contrived toward an apparent purpose. This, as Okasha (2018) demonstrated, makes for a "'unity of purpose," which in most cases allows the biologist to describe organisms as agents that maximize some magnitude related to survival and reproduction and named fitness.

Besides, various distinctions of explanatory strategies, and causal concepts, have been proposed in evolutionary biology: Mayr's division of ultimate and proximate causes, functions vs. mechanisms, Tinbergen's "four questions" (Tinbergen 1963), and others. In this chapter, I will use Formal Darwinism as a tool for pluralism to systematically assess and articulate those divisions. I will start by sketching the main distinctions between explanatory types in evolutionary biology. Then in a second section, I will trace them back to an explanatory or causal pluralism first stated by Aristotle, who (according to most of the usual translations of his *Physics*) spoke of the "four causes," even though one may consider that he meant here "the four explanantia," *aitiai*. Given that Aristotle's distinction is highly concerned with what we call the difference between final causes or teleology and efficient causes or mechanisms, the IF teleology understood as the genuine Darwinian teleology, according to Huneman (2019b), will in the third section proves instrumental in making sense of this quadripartition in a Darwinian context. Explanatory pluralism is not an epistemic virtue by itself, and that it is not philosophically or scientifically productive unless a formal and conceptual articulation is provided for the different modalities of causation, which is done here.

In the last two sections, I wish to show that this pluralism may be a fruitful framework by considering two major controversies in evolutionary biology. These controversies concern the purported need to expand or extend the classical Modern Synthesis framework (Müller 2017) and the controversies over the proper account of the evolution of prosocial traits such as altruism of hymenopteran insects (who do

[1] On inclusive fitness, see Birch (2017b), which explores all dimensions of Hamilton's paper's legacy, who coined the main guidelines of the philosophy of social evolution, including this notion of inclusive fitness and the parent notion of kin selection (see below Sect. 5.2.). The coefficient of relatedness is notably difficult to evaluate and even define, but it is mostly thought to measure the statistical association between individuals at a specific locus of their genome.

not reproduce but work for the queen) or monkeys emitting alarm calls for the tribe at the cost of their lives.

Those are huge controversies, and this chapter will not solve them. However, it will focus on some implicit assumptions therein regarding causal concepts and explanatory perspectives and argue that the IF teleology, brought in the debates, can contribute to making sense of some theoretical divides and overcome disagreements due to the fact that authors talk past each other.

2 Darwinian Teleology and the Pluralism of Causes

By providing an isomorphism between the Price equation in population genetics (an equation analytically describing the change of phenotypic value or allele frequencies between two generations in functions of their frequency and fitness; see Gardner 2008) and "optimization programs" in behavioral ecology (namely, a mathematical description of the hypothetical choice of the best fitness-enhancing strategy by the organism), Formal Darwinism (Grafen 2002, 2007) allows an explanatory pluralism in evolutionary biology. Phenomena can equivalently be understood through optimization schemes or in terms of allele dynamics, and explanations can—depending upon available information and the nature of the explananda—be run at the gene or the organism level (Huneman 2014a, 2014b). This pluralism allows for making sense of a specific kind of teleological explanation, in terms of maximization of inclusive fitness, called IF Teleology (Huneman 2019b). The genuine Darwinian teleology is indeed based on this notion of maximizing inclusive fitness. Such account of teleology allows one to make sense of what I called "intrinsic teleology" (Huneman 2019b) approaches, such as self-organization approaches, or organizational views of functions (e.g., Mossio et al. 2009). It also allows the understanding of plasticity as leading evolution by biologists such as West-Eberhard (2003) or philosophers such as Walsh (2015) and of the etiological theory of functions (e.g., Wright 1973; Millikan 1984; Neander 1991; Griffiths 1993). It makes sense of traits that are apparently not benefitting their bearers, such as the peacock's tail or the worker bee's sterility, and indifferently understands teleological statements regarding organisms or traits (or alleles) (Huneman 2019b). A conceptual analysis of teleology in terms of IF maximization, embedded within the explanatory pluralism based on Formal Darwinism, provides us with a framework to revisit the various notions of causation used by biologists. I will do this in this section.

Since Aristotle, philosophical reflection on causation and explanation has been accompanied by the awareness that causation occurs in many senses. Aristotle famously distinguished four kinds of causes (*aitiai*[2]). Modern science is said to have restricted the "four causes" to the latter, namely the antecedent efficient cause,

[2]Even though it can also be translated by "explanation." See above.

mostly understood as a mechanical impetus. Nevertheless, since then, biologists struggled with causal claims that seem to exceed this restriction, especially by allowing for final terms—goals of *embryological processes, adaptations* that seem to require explanation through design, as Paley famously argued (Huneman 2015, on this argument), *functions*, which explain X by invoking some effect of X, therefore exemplifying the logics of final cause. Analytic philosophers put a great weight of attention on functional statements—especially since the paraphrases by Nagel (1961) or Hempel (1959) in mechanistic terms seemed inconclusive (McLaughlin 2001). However, Kant's major attempt to account for biological judgment represented a first significant treatment of the same problem (Kant 1790; Ginsborg 2014; Huneman 2006).

In this section, I will show that FD pluralism, essentially tying IF teleology with dynamics, can be integrated within a general scheme of explanatory pluralism in evolutionary biology and pinpoint the specific role of teleology. The main idea in the two next sections will be that acknowledging Formal Darwinism and the foundations it provides for Darwinian teleology allows one to build a framework encompassing the main explanatory differences in evolutionary theory.

In the current terms of the philosophy of science, explanatory pluralism means that several types of explanation for aspects of a single phenomenon are different but together legitimate. In this chapter, following Aristotle and the usual translation of "*aitia,"* I consider that explanations search for causes (Salmon 1984; Woodward 2003) so that explanatory pluralism here goes with causal pluralism. In other contexts, I have challenged the exclusivity of causal explanations, understood in the sense of causation entirely restricted to production or to a sense that is based on production,[3] for instance, in Huneman (2010a, 2018). Here I equate causation in a very general sense with explanation. This chapter contributes to an updating of Aristotle's four causes, which justifies my take here. Moreover, given that his quadripartition of causes is very general, I will start by considering biology in general, even though most of my argument will concern evolutionary biology. If one agrees that, following the usual credo, "Nothing makes sense in biology except under the light of evolution," then the proper Darwinian teleology provides us with the genuine meaning of biological teleology, and I can legitimately proceed from evolutionary biology to biology.

To this extent, I start by reviewing five explanatory/causal pluralisms seen in the literature about biology (Sect. 1), and then I will consider the role of Formal Darwinism and IF teleology in their context (Sect. 2).

A. A major variety of pluralism goes with the difference stated by Ernst Mayr (1961) between *"proximate" and "ultimate" causes*. Proximate causes occur in an individual organism's lifetime, whereas ultimate causes pertain to past generations of populations of the organism's species (see Beatty 1994; Ariew 2003). Evolutionary investigations search for ultimate causes, while physiological or

[3] See Glennan (2017) for an account of how production will always be the fundamental meaning of causation.

molecular genetic investigations search for proximate causes. It has been argued that advances in Evo-Devo and molecular genetics challenge this distinction because, for example, if development (embryogenesis, or, more ecologically, niche construction) is relevant to evolution, then the two kinds of causes overlap (Laland et al. 2014; see also Pigliucci and Scholl (2015) for a more nuanced reading of Mayr). However, I think that this difference is still to be considered as a reference point; it is indeed still used to organize textbooks, lectures, or other presentations.

B. A second difference between kinds of causes holds between a *function* and the *mechanism* that realizes the function. The mechanisms of the cells, glands, and vessels in the kidney explain why the elimination of toxins occurs. The fact that the function of kidneys is eliminating toxins explains why kidneys are here. Therefore, functions and mechanisms are two kinds of causes, so indissociably related that they look like two faces of the same medal.

   A caveat here: philosophers argued over the concept of function for decades; some favor the etiological concept of function, according to which "F is the function of X" means "F has been selected for doing X" (Wright 1973; Neander 1991). This has several shortcomings (e.g., Walsh 2002; Enç 2002), and many refined versions of the etiological view have been proposed, including Griffiths (1993), Kitcher (1993), Garson (2017), Huneman (2013a). The other prominent family of accounts of functions says that "F is the function of X" means that (a) there is a system (X) in which an R-ing activity is carried on, (b) F is part of S, and X is the contribution of F to R-ing, and (c) X contributes to explain R-ing in a causal way (Cummins 1977, 2002). Therefore, this second account of functions ascribes to functional concepts another explanatory role than the one I considered here (namely, explaining the presence of X), which is the explanatory role considered in etiological views of function. Thus, the current division (B) of causes concerns only the function in the sense of etiological theories.

C. A third difference is precisely the one on which Formal Darwinism focuses: *optimization vs. dynamics*; both would explain why kidneys eliminate toxins, but the selective stories at stake are told in different perspectives, dynamics, and optimization, which ultimately resort to, respectively, a physical and an economical language. It is unclear whether optimization approaches are causal explanations—it has been argued, by Rice (2012) and Huneman (2018), that they are indeed not mechanisms and possibly not causes. In any case, this divide concerns a central explanatory dualism in evolutionary biology.

   Lastly, dynamics and optimization seem to be more related to the "ultimate causes," *sensu* Mayr, because they are about evolution. Proximate causes—for example, mammal maternal care behavior—can be understood in terms of a decision-making process oriented towards maximization of a proxy for fitness. However, at the same time, they can be understood in terms of dynamics (such as a process of behavioral conditioning).

D. This difference between types of causes also cuts across another important distinction, which is sometimes believed to make sense of the difference between disciplines like population genetics and disciplines like behavioral

ecology. The former considers *how a trait evolves*, namely, by selection or by drift, or by mutation or migration; therefore, they do not consider *why the trait is here for*. The latter asks about the reasons *why those traits or these genes have the fitness they have*; they investigate the "causes of selection" (Wade and Kalicz 1990). In fitness terms, this means that some approaches enquire about the causes of fitness (behavioral ecology), and therefore about adaptation; others, like population genetics, investigate the dynamics of evolution and thereby take fitness values as given, notwithstanding their causes. Thus, their models can be applied to very different ecological situations, provided that the distribution of fitness values is always similar, as well as the population structure, even though they do not consider the nature of adaptation (namely, what adaptations are for, ecologically speaking), which will vary according to ecological settings.

E. Finally, biological explanations can be either more *focused on traits* or more *focused on organisms*. This difference pervades all the three other elements of the above distinctions. Such difference when it comes to teleology is refracted into the difference between the "intrinsic teleology" account (which includes the organizational account of function, *sensu* [Mossio et al. 2009]), and the selected effects (or etiological) account of functions, because the former is focused on organisms as teleologically oriented, while the second is focused on traits (as targets of selection), as explained in Huneman (2019b).

To sum up, any causal explanations can be either ultimate or proximate (A); and ultimate explanations can either focus on traits or organisms (E), and they can be couched either in terms of optimality considerations or in terms of gene dynamics (C). Optimality generally focuses on the reasons for fitness values, while gene dynamics is concerned with the processes through which evolution occurs (D). Finally, something can be, in general, explained by a function, or the explanation can appeal to a mechanism that explains the function (B). Any genuine explanation can be classified according to each of these distinctions (Fig. 1). The next section will detail the relations between these dimensions of distinctions.

3 Formal Darwinism and Explanatory Pluralism

In this section, I argue that the Formal Darwinism (hereafter FD), a theoretical framework first designed by Grafen (2002, 2007, 2014), shows how all these distinctions can be interrelated, and finally, how an explanatory pluralism proper to evolutionary biology could be articulated.

Regarding distinction **D** (*how a trait evolves/why is it for*), the first thing is that the dynamics of gene frequencies in evolutionary theory mostly corresponds to the *working of selection.* In contrast, the optimization approach corresponds to the *reasons for selection*: it wonders why the fitness (inclusive) is higher with this trait.

However, FD shows how these two questions are related: the workings of selection as a dynamics of allele frequency change exactly underpin the realization

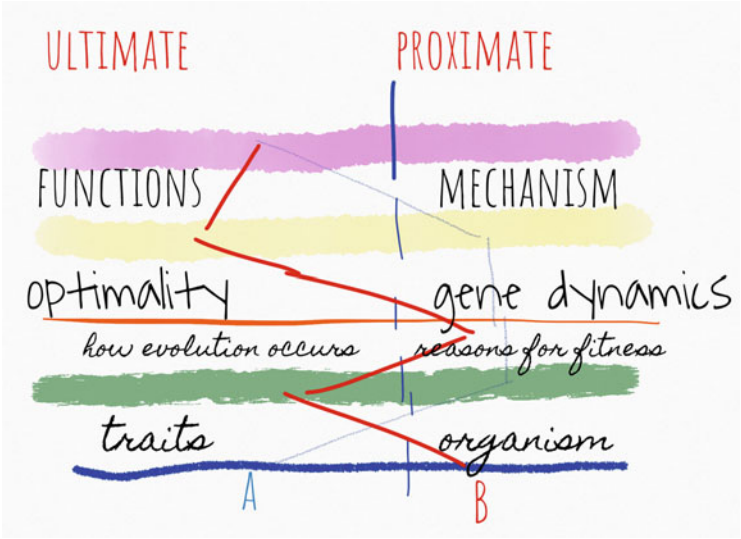

Fig. 1 The five dimensions of explanatory pluralism (and how two given explanations A and B behave regarding those distinctions)

of an optimum (under constraints) by an organism seen as a strategy-choser (see Okasha (2018) for a systematic investigation of this model of the strategy-choser). Hence it can account for the perspective difference **D** within a unified setting—"account for," here, meaning that it shows how the two members of the distinction define legitimate explanations likely to be articulated together in a non-reductive way.

Regarding **E** (*trait focused—organism focused*), FD establishes a link between organism-focused and trait-focused approaches because it explains why the emergence of traits occurs in a designed way, i.e., by contributing to the overall design of the organism (as argued in Huneman 2019b). It yields a genuine teleology—IF teleology—that embraces both trait-oriented perspectives and organism-oriented perspectives. Hence it accounts for **E** within a unified framework, which is the same framework articulating the two sides of **D**.

As to **B** (mechanism-function), teleological explanation pertains to the function of traits; the function/mechanisms distinction often seems to correspond to the distinction **A** between ultimate and proximate causes.[4] In general, however, FD shows that, within the same functional attribution, a more fine-grained distinction stands between a particular mechanism or dynamics of allele frequencies and the teleological explanations that uncover the function's target. In other words, the etiological

[4] As is apparent in the example of the kidneys: their function is eliminating toxins; the mechanism is a complex dynamic of filtering that implies the osmotic properties of cell membranes, and that can be studied at various levels of integration—tissues, cells, metabolic pathways—within the lifetime of the organism.

function F—as when one says "X has the function F"—is carried on by a molecular, cellular, and physiological mechanism. Nevertheless, this function itself results from another kind of mechanism, namely the population-level dynamics of gene frequencies across generations. In turn, such a mechanism can also be explained in terms of teleology, namely, optimization of inclusive fitness or IF teleology (Huneman 2019b). Therefore, the pair "mechanisms/functions" indicates an explanatory difference more complex than a mere correspondence with the pair "ultimate/proximate" explanation (e.g., of the kidney), since the "ultimate" part of the explanation—the "function" in an etiological sense—can be understood simultaneously in terms of a population-level mechanism (namely, as an allele frequency change) and as an optimization.

Such optimization is the maximization of inclusive fitness, which has been shown to be the most systematic sense of teleology in a Darwinian context (Huneman 2019b). Thus, finally, when it comes to the difference **A** between ultimate and proximate in general, dynamics and optimization as understood by FD *both* pertain to ultimate causation. Thereby the distinction **C** further divides each member of distinction **A** into two sub-categories: *dynamical* and *optimal*. The category "Ultimate cause" therefore has to be divided into a dynamic of alleles, which is, so to say, a "*proximate ultimate*" cause, and IF teleology, which is an "*ultimate ultimate*" cause, and both are connected through the isomorphisms stated by Formal Darwinism.

Thus, there are two lessons from this examination of the relationship between differences in explanatory types. First, the five explanatory pluralisms **A-E** that pervade evolutionary biology, and even biology in general (whence one considers distinction **A**), can be *articulated through FD.* Secondly, the same reasoning shows that there are *grades of teleologically ultimate explanations* and that IF provides the "ultimate ultimate" cause in biology.

4 The Four Causes, Revisited

Aristotle famously divided causes into four kinds: *efficient* (material events which produce the consequent); *formal* (the essence of the event, i.e., the usual answer to the question "what is it?"); *final* (the goal of the event); *material* (the substance of the event).[5] Those categories have been mainly given up with the scientific revolution

[5] Here is the text where Aristotle first set this distinction. "In one way, then, that out of which a thing comes to be and which persists is called a cause, e.g., the bronze of the statue, the silver of the bowl, and the genera of which the bronze and the silver are species. In another way, the form or the archetype, i.e., the definition of the essence, and its genera, are called causes (e.g., of the octave the relation of 2:1, and generally number), and the parts in the definition. Again, the primary source of the change or rest; e.g., the man who deliberated is a cause, the father is cause of the child, and generally what makes of what is made and what changes of what is changed. Again, in the sense of end or that for the sake of which a thing is done, e.g., health is the cause of walking about. ("Why is

and the change in the ideas of explanations, causation, and law brought about by modern physics; however, the Aristotelian inspiration may sometimes help draw the conceptual space of a scientific field. Especially, Tinbergen explicitly thought about it by distinguishing four causes, even though they do not correspond one-to-one to Aristotle's causes (Tinbergen 1963[6]). According to him, a biological behavior calls for explanations in terms of its development, its mechanisms, its evolutionary history, and finally, its adaptive meaning or function. Here, "function" would correspond to the final cause, "development" to the efficient cause, "mechanism" to the material cause ("formal cause" is harder to get into the picture).

In the case of Formal Darwinism, gene frequencies' dynamics would correspond to the "efficient cause" since it is the temporal process that brings about the trait along evolutionary time. The "formal cause" would be the function of the trait, namely *what it does* that explains *why it is here.* Furthermore, the "final cause" would be the maximization of inclusive fitness, which is even more general than the function itself. In this case, according to our typology, *population genetics* would consider both the material and the efficient causes (respectively, the alleles and their dynamic); the *behavioral ecology* considers the formal cause, namely the function, and the final cause—namely, to leave as many offspring as possible through direct and indirect ways, i.e., maximizing inclusive fitness.

Thus, within evolutionary biology, FD allows us to recover a quadruple partition of causes that updates an Aristotelian-style metaphysical division. Moreover, as in Aristotle's physics, all those explanations are compatible: taken together, they constitute a complete explanation of the phenomenon under focus.

However, the pluralism we just considered until now was about *types of causes*, like Aristotle's partition, which could map onto *types of explanations*. Nevertheless, there is another philosophical pluralism, which concerns the *concept of causation* (or the nature of causation itself, if one wants) (e.g., Godfrey-Smith 1994). Here, the main distinction is about two extensive families of theories of causation, which corresponds to two intuitions: "A causes B" can mean (1) that "if A had not occurred, B would not have occurred," or it can mean (2) "there is a physical process through which B stems from A." The first families can be called "difference-making causation" (Menzies 2004). They comprehend the so-called counterfactual theories of causation, first elaborated by Lewis (1973) (e.g., Collins et al. 2004), the "manipulationnist" theories of causation—namely, an intervention on variable A changes, *mutatis mutandis*, variable B—, and the probabilistic theories of causation—i.e., the difference A makes on B is probability raising. The second family of accounts is the "process causation," first elaborated by Salmon's theory of transmitted mark (Salmon 1984), then refined (Dowe, 2000). Hall (2004) argued that the two families, corresponding to the two very general concepts of causation, are

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he walking about?" We say: "To be healthy," and, having said that, we think we have assigned the cause.)" (Physics, 194b24-195a3, tr. Barnes.)

[6] See also Hladký and Havlíček (2013) on the relation between this quadripartition and Aristotle's four causes.

heterogeneous—especially, features of the latter such as locality are not possessed by the former and reciprocally. Glennan (2017) argued that Salmon's focus on fundamental physics is unnecessary and that the process account may hold even though not all causal processes stand at the level of particle physics. In any case, if Hall is right regarding concept dualism, this entails a general pluralism in scientific explanations, when causal statements can sometimes be uttered according to one concept but not to the other.[7]

This difference directly impinges upon evolutionary biology. Actually, Formal Darwinism states an equivalence between these two kinds of causation: *dynamics* of gene frequencies concerns causation in the sense of a *process*, while *optimization* clearly concerns causation in the sense of *difference-making*, since the traits that make the highest difference upon inclusive fitness will evolve. Therefore, the *explanatory pluralism* stated above in Sect. 1, which systematizes the distinctions **A–E**, is supplemented along another dimension by a *causal pluralism* in the form of equivalence between difference-making and process causation in evolutionary biology, to the extent that equivalences do hold between behavioral ecology and population genetics (instantiating those two distinct concepts of causation) established by FD.

Until then, I proposed that the pluralism proper to FD, especially the kind of teleology defined on this basis (IF Maximization), makes room for a reassessment of Aristotle's causal pluralism. The major distinctions regarding explanations in biology (labeled **A–E**) were translated into a unity of teleology and mechanisms based on the ultimate teleology provided by FD, namely IF teleology. IF also allows one to make sense of the uses of two concepts of causation, the difference-making concept and the process concept, and provides possible connections between those two causal schemes.

To sum up this examination of the relation between Aristotle's classical theory of causes and the current practical distinctions one can make between various explanatory types in evolutionary biology, I will say that various kinds of explanations, distinguished by considering the pluralism involved by FD, could be rethought along the lines of Aristotle's division of causes. The last section of this paper will now apply this view in order to show that it could contribute to solving current controversies in the field because many debates are either triggered by confusion between kinds of causes or by a disagreement about which one should be in priority handled, for methodological or pragmatic reasons.

[7] In Huneman (2012, 2013b), I argued that natural selection could be understood as a causal explanation when causation is taken in the sense of difference-making, and I defended an account of this causation in terms of counterfactuals—but not in the sense of production processes.

5 Pluralism and the Current Controversies

5.1 Pluralism and the Current Controversies: (a)—The So-Called Extended Synthesis

There is an ongoing controversy about whether one should extend or expand evolutionary theory (Pigliucci and Müller 2011, Pigliucci 2007, Jablonka and Lamb 2005, Laland et al. 2014; Müller 2017) in the light of findings by Evo-Devo or behavioral ecology or ecology; or not (e.g., Lynch 2005; Wray et al. 2014).[8] The alternative theories are very varied. However, they all insist, in different ways and with distinct emphases, that the Modern Synthesis cannot integrate new findings of developmental and molecular biology and genomics. These findings include the non-genetic forms of inheritance such as parental effects (Bonduriansky and Day 2009) or epigenetics (Jablonka and Raz 2009; Danchin et al. 2011; Danchin and Pocheville 2014); the role of organisms in shaping their environment (niche construction, Odling-Smee et al. 2003); the complexities of genomic systems (Griffiths and Stotz 2013); or the prevalence of phenotypic plasticity (West-Eberhard 2003) and developmental biases (Raff 1996; Brakefield 2006; Uller et al. 2018). Many of these claims challenge the gene-centered view of the Modern Synthesis, which conferred to population genetics the crucial role of modeling the process of evolution by natural selection.[9] On the contrary, they call for a "return of the organism" (Bateson 2005; Huneman 2010b). I already argued that FD pluralism might conciliate the organism-centered and the gene-centered views because it holds together gene dynamics and organismal teleology (see Huneman 2014b). Here, I consider another avenue of conciliating claims regarding the pluralism of explanation and causation, which is the focus of this chapter.

As summed up by Huxley to Mayr in 1951, the position that the alternative to MS intends to challenge is the following: "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."[10] Among many biologically disputed issues, one philosophical argument coined by Modern Synthesis's challengers is about causes of adaptation: the claim is that cumulative selection on small variation is not the actual cause of adaptation. Based on the interpretation of natural selection called "the statisticalist interpretation" (Walsh et al. 2017), which is out of the scope of this chapter, Denis Walsh developed a very sophisticated

[8] Various approaches to the alternative theories are collected in Huneman and Walsh (2017), in which. I tried to show which empirical data would be required to trigger a real revolution of the explanatory scheme proper to the Modern Synthesis, rather than a piecemeal rearrangement. The perspective chosen in this chapter does not contradict this more extended argument.

[9] For example: "The core of the synthetic theory is pretty much just the theory of population genetics" (Beatty 1986, p. 125).

[10] Julian Huxley to Ernst Mayr, 3 September 1951. Papers of Ernst Mayr. HUGFP 14.15 Box 1. Harvard University Archives, Cambridge, MA.

argument according to which natural selection causes the spreading of adaptive traits but does not cause their emergence (see also Walsh 1998). Instead, the latter is due to some individuals' developmental process, especially adaptive plasticity (Walsh 2010) or possibly self-organization (Walsh 2003). In the introduction of their extended synthesis book, Pigliucci and Müller (2011) try to give a general account of the rationale for extending the evolutionary synthesis. The general idea is that Darwin and the Modern Synthesis thinkers had a *statistical* view of evolution by natural selection, mainly counting representations of genes generation after generation because they did not know the mechanisms of variation and production of new traits. However, now that we can access these mechanisms, the overall picture of evolution and adaptation changes: hence we switch from a "statistical" conception of causation in evolution to a "mechanical" conception of causation.[11] As they say, "the shift of emphasis from statistical correlation to mechanistic causation arguably represents the most critical change in evolutionary theory today." Interestingly, this distinction between views is grounded on the distinction stated above between the concepts of causation: according to Pigliucci (2007), the "extended synthesis" would move us towards a process view of causation and explanation, instead of the difference-making view, implicit in any probabilistic account. Thus, for the same reason, the FD pluralism sketched here allows one to conciliate them and solve the dispute, as I will indicate now.

Even if they are quite different, both reformist claims—Walsh and the 'statisticalists', Pigliucci, Müller and the Extended synthesis—mentioned here say that evolution by natural selection, according to MS, is a *statistical* explanation. However, the underlying mechanisms are the genuine *causes* of adaptation (for Walsh) or evolution (for Pigliucci and Müller). This view overlooks the complex picture of evolution by natural selection provided by FD. There is, in fact, a dynamic of gene frequencies, which corresponds to a statistical explanation and especially brackets the variation mechanisms. Even so, the inference that new science of mechanisms would give a better or more accurate account of adaptation or evolution is not justified, since we just saw that this statistical explanation is equivalent to another one couched in non-statistical terms: the teleological one, or *optimization*. Therefore, the need to extend the MS cannot be attributed to the fact that MS was only a dynamic of genes, statistically modeled, because this is only one side of the FD equivalence. On the other side, IF teleology explains adaptations as such, as it unravels the final cause that accounts for why a given trait is there—namely, its maximizing inclusive fitness.

To be more precise, the emphasis put by Walsh (2010, 2015), as by many biologists and philosophers who support a radical reform of the Modern Synthesis, on mechanisms of variation versus natural selection, envelops the assumption that natural selection only explains the *spreading* of the traits, but not their *origin*. There is a longstanding debate on this issue (Neander 1995; Walsh 1998), opposing what

[11] I gave a direct extended critique of this argument in Huneman (2019a), based on analysis of some explanatory practices in postgenomic evolutionary biology, but here I focus on the metaphysics of causation.

Neander has coined the "Negative view" (i.e., the above-mentioned assumption about what selection explains) and a "Creative view" of selection. I will not survey it here; let us just notice that the Darwinians of the Modern Synthesis were concerned by this issue and that, as Beatty (2016, 2019) has argued, one of the hallmarks of their view is the commitment to the idea of a "creativity of natural selection." This commitment means that for them—and against, first, the Mendelians and then, some the opponents of Mayr or Dobzhansky, such as Goldschmidt—natural selection was not only a sieve that prevents the less fit from spreading and therefore lets the fittest spread. It also shapes the adapted traits because it constructs the gene pool from which new variants are built across generations.

The FD pluralism advocated here includes a teleological-explanation-based account for this "creativity" view. Why? The whole population genetics modeling of the action of selection can be seen as a *statistical* explanation of what happens. Therefore, it is mostly concerned by the *spreading* of the adapted traits since those traits are originally brought about by mutation and recombination in some genotypes. However, if we switch to a teleological view—and this perspective is always available, because of the nature of IF teleology, as rooted in the equivalences between allele dynamics and optimization (Grafen 2002, 2007)—, then, the adapted traits themselves (and not their spreading) are here because they maximize inclusive fitness. The teleological argument indeed is formulated as: "since trait X maximizes inclusive fitness, it will be the strategy adopted by organisms of the kind considered, in the environment and population under focus"; and under this view, no population-level process statistically described is among the explanantia. Hence, against the "negative" view, which is presupposed by the arguments of those who think Modern Synthesis should be overcome by a mechanistic and non-statistical understanding of adaptation, natural selection really explains the fact that the adaptive traits are there.[12]

To sum up, here, the IF teleology allows one to downplay one of the epistemological arguments put forth by defenders of an alternative to the Modern Synthesis—namely, the opposition between a supposedly statistical knowledge in MS and a mechanistic knowledge brought for by its alternatives—, because not all aspects of the overall explanatory picture of MS have been considered by such critical account.

5.2 *Pluralism and the Current Controversies: (b)—Explaining Altruism: The Multilevel/Kin Selection Controversy, Sketching an Answer*

For three decades in evolutionary biology, a huge debate has been going on about what is the cause—or, at least, what best explains—some prosocial traits, i.e., the

[12] In fact, to be successful, the argument should consider cumulative selection underlying complex adaptation as the trait maximizing inclusive fitness. However, this issue is not central here. The importance of cumulative selection to justify teleology is highlighted in Huneman (2019b)

traits that are costly for their bearer and beneficial for others (often called *altruistic*, see West et al. 2007). The apparent evolutionary problem here is that these altruist traits should be counter-selected, but in fact, they are everywhere: sterile workers in hymenopterans insects, alarm calls in antelope or monkeys, helping behaviors in many species, etc. The emergence and maintenance of these traits raise, therefore, a profound problem for classical Darwinism.

Two main theories have been advanced to understand this: the first one (suggested by Hamilton 1963) is the kin selection/inclusive fitness theory, according to which a trait evolves if its benefit is higher than the cost mitigated by the "relatedness." A proxy for relatedness is kinship. However, relatedness is, in general, more complicated than this (Taylor and Frank 1996; Frank 2006). The famous "Hamilton's rule" summarizes this account of altruism: $b > rc$—where b is the benefit for the receiver, c the cost paid by the focal actor, and r their relatedness (even though one can model kin selection by considering the payoff undergone by the focal actor due to the actions of the others modulo relatedness (Taylor et al. 2007)). The rule can compare the two components of inclusive fitness, namely the direct ($-c$) and indirect (br) fitness payoffs.

The other view is called Multilevel Selection (MLS) theory (Sober and Wilson 1998; Wilson 2001; Okasha 2006; Damuth and Heisler 1988). It is grounded in the following idea: if one supposes that evolution occurs in a population divided into groups, altruists compared to selfish individuals or behavioral strategies score worse. However, groups including many altruists fare better than groups with a lower proportion of altruists since altruists, per definition, invest resources, energy, and time in the group's welfare. Therefore, evolution can be considered as resulting from the addition of intragroup competition (which favors selfish elements, see e.g. Burt and Trivers 2006) and intergroup competition (which favors altruists): when the second term is higher, altruism can evolve.

Some authors have claimed that one of the following views is just a particular case of the other. Sober and Wilson (1998) held that multilevel selection is the most general theory and includes kin selection as a case where groups are kin groups; Nowak (2006) claimed that kin selection is only one among other explanations of cooperation and that it is overstated (Nowak et al. 2010). West et al. (2007), Lehmann and Keller (2006), Lehmann et al. (2007), and others argued that the evolutionary mechanism is always a kin selection. In turn, Abbot et al. (2011), West et al. (2010), Ferrière and Michod (2011) argued that the kin selection/inclusive fitness theory is the most powerful and accurate one.[13]

The main argument of MLS tenants is that it captures the real causal structure of evolution, while kin selection may correctly represent what happens to genes, but does not capture anything except a shadow of the causal processes (Sober and Wilson 1998). Interestingly they invoke the same distinction between causal and pseudo-causal distinction (mentioned above Sect. 5.1) as Walsh (2003) did

[13] Nevertheless, see Birch (2017a) for a comprehensive account of what Nowak et al. (2010) really meant about the kin selection and how the controversy mostly focuses on something else.

regarding adaptations. In effect, they say that what causes the evolution of altruism is, in real life, competition between groups, while, by overlooking real interactions between organisms, the kin selection view cannot see this. This view justifies their "averaging fallacy" argument, which in essence means that computing the fitness of organisms by averaging their reproductive success in different contexts (i.e., groups) may give a correct estimation of final gene frequencies. However, it neglects the causes of this final frequency, i.e., their belonging to specific groups, since, by definition, the averaging neglects this fact. This argument connects to Sober and Lewontin (1982) critique of genic selectionism, where allelic selectionist is said to be blind to the level of real causes, which stand at the level of the genotypes; the classic example here is the case of the superiority of heterozygotes, as happens with recessive alleles for malaria resistance in sickle cell anemia.[14]

On the contrary, supporters of inclusive fitness would say that the causes of evolution are occurring at the level of alleles increasing or decreasing in frequency in accordance to their contribution to the fitness of the relatives so that in all cases presented by supporters of MLS (e.g., Traulsen and Nowak 2006), what really happens is a process of kin selection (Lehmann et al. 2007). They add that multilevel selection is most of the time mathematically intractable (West et al. 2007), which provides a substantial methodological advantage to kin selection models. Notwithstanding this argument, the two camps clearly do not focus on the same causal aspects: the MLS supporters argue that the causal story is the competition between groups, so their causally relevant facts are intergroup competition and intragroup competition; the kin selection theorists argue that the causally relevant facts are direct fitness benefits and indirect fitness benefits. Therefore, in the first approach, what is crucial to determine is the population's partition into groups. In the second one, what is crucial is relatedness because it allows one to define and measure indirect payoffs. Furthermore, it seems that relatedness is tractable in an easier way than group partitioning, which could explain that most accounts of social evolution are couched in terms of kin selection (West et al. 2010).

However, there are equivalences between these stories (for an in-depth analysis of those equivalences, see Birch 2017a). As Kerr and Godfrey-Smith (2002) have shown, a multilevel approach of evolution, in general, is mathematically equivalent to an approach where the fitness of individuals is contextualized over groups and then computed across generations. More simply, the more the intergroup competition increases relative to the intragroup competition, the more Multilevel Selection one has for the prosocial (altruists) traits: but this means that the between-group variance relatively overcomes the intragroup variance. This fact, in turn, entails that relatedness increases in each set of individuals interacting with a focal individual and then in general, which in the end means that kin selection increases; and reciprocally (Frank 2006; Foster et al. 2006; West et al. 2007). Let us call (E) this equivalence.

[14] Because being heterozygote is a property of the genotype, not the allele; hence the relevant causal property stands at the genotypic, not the allelic, level.

Therefore, both camps claim that they provide a causal story of social traits' evolution but disagree on the main causal fact—group partition vs. relatedness. Central to such dispute is the status of causal explanations in biology, even if it is often not made explicit. MLS theory is said to capture the causal story because here causation is thought in terms of mechanisms: the process by which some groups are superseding other groups, then changing the frequencies of social individuals. According to Darwinian cultural evolutionists like Boyd and Richerson, this is well exemplified by two ethnic groups previously living in Africa, the Nuer and the Dinka, in Sudan. Nuers did not have the same policy for redistributing cattle since they were more involved in providing cattle for the whole tribe, beyond what one would need for oneself; as a result, between 1820 and 1890, they took over most of the Dinka's territory and increased by four times their territory (Kelly 1985).

The kin selection view is, in turn, articulated in terms of variables making differences to outcomes: if one increases or decreases the relatedness, then, whatever the ecological processes occurring in the population—competition, predation, etc.,—the expected outcome (i.e., relative frequencies of social individuals) increases or decreases.

Thus, besides all methodological and biological differences between kin selection approaches and MLS approaches of altruism, a difference between two conceptions of causation also stands between them: a process view, on the one hand, a difference-making view on the other (according to the distinction made in Sect. 3). Thus, it seems that the dispute about which is the real causal story and, therefore, the best account is doomed to go on forever since the two sides talk past each other. However, given that I previously articulated a pluralistic view of causal explanations in evolutionary biology (Sect. 3), there might be a way to make sense of that controversy.

So how to conciliate kin selection models and MLS models from the viewpoint of the pluralism of explanation concepts? First, recall that kin selection theory can be easily formulated in terms of inclusive fitness theory (Hamilton 1963; Birch 2017b): the former is a story about *dynamics of gene frequencies* since the fitness of alleles is computed in the model; the latter is the same story understood in terms of *optimization of the organism's strategy*: altruists evolve if and only if they maximize inclusive fitness, which is, remember, the addition of what the focal strategy brings to other organisms (mitigated by relatedness), and the cost the organism incurs (both expressed in fitness units). The former formulation, namely kin selection, stands at the level of *alleles*, while the latter, inclusive fitness, considers *organisms* since it computes inclusive fitness at their level.

However, as I said, the inclusive fitness account also pertains to a difference-making view of causation. As we have seen (Sect. 3), the FD equivalences entail that such causal approach can be articulated to causal explanations of another nature, namely process causation, which is at stake in models of alleles dynamics. Now, this equivalence will allow making sense of the controversy about the kin selection and MLS. How?

In effect, given the equivalence mentioned above (E) between varying relatedness in the kin selection perspective and modifying the intergroup/intragroup competition

Table 1 Typology of explanations of social evolution in terms of causal types

	Concept of causation	Level	Type of explanation
Inclusive fitness	Difference making	Organisms	Teleological
Kin selection	Process	Genes	Mechanical
MLS	Process	Organisms	Mechanical

ratio in the MLS view, the latter appears to be, at the level of organisms, the causal explanation in terms of causation-as-processes that precisely corresponds to the difference-making view of inclusive fitness. Ultimately, our framework, therefore, provides a pluralist explanation of the evolution of social traits at three levels (Table 1). Let us unpack it now. MLS is a causal (*sensu* processes) explanation at the level of organisms; through (E), it is equivalent to a causal (*sensu* processes) explanation at the level of alleles, namely kin selection models; and the latter, through the FD equivalence, corresponds to a causal explanation (*sensu* difference-making), stated at the level of organisms, namely the inclusive fitness approach. The two equivalencies (FD and E) about models finally support a pluralism of causal concepts and explanation levels. Furthermore, the teleological explanation, namely the IF teleology, is the more encompassing one since it can account for different kinds of processes at both levels (organisms, alleles). In other words, it is the most general account of the evolution of social traits—that is, the most generic one, since it can be predicated based on a variety of different genetic make-ups that satisfy distinct models of kin selection.

Therefore, the pluralism about the evolution of social behavior based, in the present approach, upon the FD pluralism, will allow for *various grades of genericity.* IF teleology approach is the most generic, and then, details about the genetic make-ups of altruism and selfishness will allow for more realistic models that, in turn, model more possible structures of the allele dynamics—namely, the various kin selection models of different gene pools.

Of course, this is not supposed to close the controversies about social evolution. I just suggested that considering the general framework for explanatory pluralism provided by FD to evolutionary biology may help to identify the issues where discussants talk past each other and the issues where they agree (or do not disagree) with each other even while not acknowledging it. This is especially true if one acknowledges the legitimacy of Darwinian teleology, understood as IF teleology, which constitutes the "ultimate ultimate causation." In those debates, equivalence (E) has often been appealed to for supporting a pluralism between explanations of social evolution. However, putting FD into the picture allows one to provide a more complete table of explanatory types, understand bridges between distinct explanations that use various causation concepts and stand at distinct levels, and finally, allows for genericity grades that differently realize the same IF teleology explanation.

6 Conclusions

In this paper, I considered the uses of a view of evolutionary theory based on Formal Darwinism, which confers an overarching role to a specific teleological explanation understood in terms of the maximization of inclusive fitness. On this basis, I presented a general scheme for making sense of explanatory pluralism in evolutionary biology, integrating five classical views of explanatory differences, and then, both the four types of causes and the two notions of causation, and sketched the position occupied by the Darwinian IF teleology into this scheme.

This is not only a general investigation of the scientific image of biology aimed at philosophers; more importantly, it is intended to provide a framework in which some controversies can be solved, and some current challenges to classical evolutionary thought can be addressed and assessed. This is why I indicated how an awareness of such explanatory and causal pluralism—updating (for the former) the Aristotelian quadripartition—may help biologists in dealing with two massive controversies in evolutionary theory. These controversies concern the call for an alternative explanatory scheme integrating non-genetic inheritance, facilitated variation or niche construction, and the debates about the proper model and theory for accounting for the evolution of prosocial traits. Because Aristotle's distinctions have been fruitful in understanding science across the ages, I think that an attempt to update these distinctions by considering various explanatory practices in evolutionary biology could be useful.

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Editorial Note: The Evolution of Meaning—From Neo-Darwinism to Biosemiotics

Richard Theisen Simanke

Huneman's chapter presents an original, thoughtful, and compelling argument for explanatory pluralism in the biological sciences. Assuming Formal Darwinism as a theoretical framework and focusing on the crucial concept of inclusive fitness maximization, the author argues that this perspective provides a consistent strategy for articulating the different causal pluralisms proposed within the field of evolutionary theory. Aristotle's theory of causation—arguably the first systematic model of explanatory pluralism in the history of philosophy of science—is called upon as a historical point of comparison, and the author presents his argument as reconstruction and updating of the Aristotelian views, especially on the complementary role played by teleology and mechanism in causation. Underlying the author's views is

the conviction that explanatory pluralism is not an epistemic virtue by itself and that it is not philosophically or scientifically productive unless one provides a formal and conceptual articulation for the different modalities of causation. The chapter still contains a consistent proposal not only to articulate the different kinds of causes within each pluralistic model but also to relate these models to one another.

Since this collection's general subject matter addresses both evolutionary biology and biosemiotics, it seemed adequate to add here some remarks concerning how an argument for explanatory pluralism and the integration of different theoretical models relates to the biosemiotic view of living beings as meaning-producing entities and natural interpretive systems.

First of all, one must note that Neo-Darwinian evolutionism, however prevalent it may be in the context of contemporary life sciences, is but one of the great paradigms or theoretical models that one can distinguish in this field. Biosemiotics is another model of comparable scope and complexity, with all the doctrinal and methodological implications entailed by such condition. These models foster the emergence of research programs on specific questions in the field of biological knowledge and the formation of research communities endorsing these views of life and the corresponding conceptions of science. Other such models can be mentioned, like those defined by the core concepts of autopoiesis or artificial life.

All these theories organize biological research, with repercussions extending from the more concrete and applied issues to the more general and abstract ones. These more abstract questions touch the borders between biological sciences and the philosophy of biology, including the problem of defining *life* as such. Many authors (El-Hani 2008; Emmeche 1997) have argued that these paradigms provide the possibility for a situated and circumscribed definition of the very concept of life, relatively to the theoretical model informing the investigation of its phenomena. This strategy could overcome the supposed intractability of defining life, often rejected as a metaphysical and unscientific problem. Thus, from the viewpoint of artificial life, being alive is defined as a property of systems capable of reacting automatically and adaptively, in an open-ended way, to unpredictable changes in their environments (Bedau 1996). For autopoiesis, living beings are organizationally closed but structurally open (both materially and energetically) networks, whose components produce the network itself and its boundaries and are, in turn, recursively produced by them (Maturana and Varela 1980). From a Neo-Darwinian perspective, if one wishes to define life in accordance to the major theoretical principles (which is not something mandatory to do in this framework), life would consist in the property of being self-replicating entities likely to evolve through random variation of inheritable traits and a posteriori natural selection of those traits favoring survival and differential reproduction (Dawkins 1976, 1983). Finally, biosemiotics sees life as meaning-production through interpretation of natural sign-systems or, in Claus Emmeche's (1998, p. 11) synthetic formulation, as the "*functional interpretation of signs in self-organised material code-systems making their own Umwelts"* (author's emphases).

One of these models' common denominators is that they all recognize the evolution of species, even if departing from the orthodox Neo-Darwinian view of evolution, at least in some of its aspects. Biosemiotics also acknowledges the species

transformation over time but changes the focus from the evolution of structures, functions, and behavior to the evolution of systems of signs and the organisms' interpretation capacities. At least since Terrence Deacon's works, the integration of evolutionary and semiotic perspectives has come to the foreground of the debate concerning human evolution and its distinctive features (Deacon 1997; Schilhab et al. 2012). In this context, Huneman's equally integrative approach, even if formulated from within the Darwinian evolutionary framework, can contribute to bringing closer two of the main theoretical models in contemporary biological sciences with their respective conceptions about the fundamental nature of living beings, as seen above.

Particularly significant is Huneman's recovery and updating of Aristotelian biological philosophy. The rediscovery of Aristotle's philosophy of nature is a striking feature in contemporary science, especially biology, although not exclusively (Feser 2019; Simpson et al. 2018). Besides providing a representative historical illustration for the kind of explanatory pluralism claimed by Huneman, Aristotelian naturalism makes it possible to consider the teleological dimension of biological explanation in terms compatible with a scientific attitude and thus reconcile two types of explanatory strategies—mechanism and finalism—often regarded as incompatible.

As intentional acts, the symbolic communication and sign interpretation privileged by biosemiotics contain a dimension of intentionality in the phenomenological sense of the term, even if considered natural phenomena (Hoffmeyer 2012). Thus, they require some teleology modality, even if it is not the transcendent finality presupposed by vitalist and metaphysical views of life, evolutionary or otherwise. Again, reference to classical conceptions of life and nature can provide a model to conceive of the immanence of meaning in the lifeworld without simply anthropomorphising it. As Merleau-Ponty remarks in the opening of his courses on nature at the Collège de France, referring to classical Greek thought:

> There is nature wherever there is a life that has a meaning but where, however, there is not thought (. . .). Nature is what has a meaning, without this meaning being posited by thought; it is the autoproduction of a meaning. Nature is thus different from a simple thing. It has an interior, is determined from within (. . .). Yet nature is different from man; it is not instituted by him (. . .). (Merleau-Ponty 2003, p. 3)

There are elements in this passage that allow for adding a biosemiotic and autopoietic perspective to the scientific knowledge of nature—and of life, in particular. The evolutionary element—generally absent from classical thought—complements and makes this multidimensional theoretical model more comprehensive. However, if Neo-Darwinian evolutionism must participate in this process, one must trim some of its edges and challenge the necessity of some of its doctrinal commitments. It is to this task that Huneman's work presented here represents an invaluable contribution.

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