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Evolutionary causes as mechanisms: a critical analysis

Saúl Pérez-González[1] · **Victor J. Luque**[1]

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Abstract In this paper, we address the question whether a mechanistic approach can account for evolutionary causes. The last decade has seen a major attempt to account for natural selection as a mechanism. Nevertheless, we stress the relevance of broadening the debate by including the other evolutionary causes inside the mechanistic approach, in order to be a legitimate conceptual framework on the same footing as other approaches to evolutionary theory. We analyse the current debate on natural selection as a mechanism, and extend it to the rest of the evolutionary causes. We focus on three approaches that we call the stochastic view, the functional view, and the minimalist view. We argue that all of them are unable to account for evolutionary causes as mechanisms. It is concluded that the current mechanistic proposals cannot be accepted as a common framework for evolutionary causes. Finally, we outline some guidelines and requirements that any mechanistic proposal should meet in order to be applied to evolutionary theory.

Keywords Mechanism · Natural selection · Evolutionary cause · Function · Stochastic

1 Introduction

Evolutionary biologists sometimes talk about evolutionary causes as mechanisms acting upon populations. For example, it is said that natural selection "is a powerful mechanism of evolution" (Herron and Freeman 2014, 227), that it "is an effective mechanism for producing adaptation" (Bell 2008, 499), but at the same time there

✉ Saúl Pérez-González
saul.perez@uv.es

[1] Departament de Filosofia, Universitat de València, Av. Blasco Ibañez, 30, 46010 Valencia, Spain

are other "mechanisms [that] might overcome the limitations of natural selection" (Hamilton 2009, 368). Following this use, some philosophers have tried to develop a mechanistic approach for those causes, mainly focusing on natural selection. This could be a promising strategy for addressing some traditional topics in philosophy of biology. For instance, biologists and philosophers have argued for years on the (lack of) existence of biological laws and how this affects the scope of evolutionary explanations (Beatty 1995; Elgin 2006), since the traditional notion of scientific explanation (Hempel 1965) required laws as part of the *explanans*. The mechanistic account instead does not demand to appeal to laws in order to explain a phenomenon. It would also underline the link between evolutionary biology and other fields of science where the mechanistic approach is generally accepted (e.g. molecular biology).

Following the mechanistic approach to causal evolution, there has been a discussion about whether mechanistic proposals can account for evolutionary causes as mechanisms. This debate was introduced by Skipper and Millstein (2005), who claimed that the main notions of mechanism [those of Glennan (2002) and Machamer, Darden, and Craver (henceforth MDC) (2000)][1] did not suit natural selection. They identified three kinds of problems of those notions of mechanism in order to account for natural selection: problems related with parts or organization (i.e. their characterizations of mechanisms' parts or organization do not suit natural selection's parts or organization), problems related with productive relationships (i.e. they cannot account for some productive relationships that occur in natural selection), and problems related with regularity (i.e. natural selection does not meet MDC requirement of regularity).[2] Although Skipper and Millstein focused exclusively on natural selection, the same kind of problems would be found by Glennan's and MDC's proposals in order to account for other evolutionary causes (DesAutels 2018). For instance, evolutionary causes such as migration or genetic drift often depend on passive processes (e.g. lack of rainfall) that do not suit their accounts of productive relationships in mechanisms.

Our aim in this paper is to analyse current mechanistic approaches to causal evolution (those that have been developed after Skipper and Millstein's initial critique), which mainly focus on natural selection, and explore their validity for accounting for evolutionary causes. We argue that those approaches have to face important difficulties, and therefore in their current formulation are unsuitable for evolutionary theory. The structure of the paper is as follows. Section 2 presents the four main causes of evolution. Sections 3–5 analyse the three main current mechanistic approaches to

[1] These notions of mechanism, which were analysed by Skipper and Millstein, are ontic. They are based on the idea that mechanisms are part of reality and exist independently of us. However, Daniel J. Nicholson (2012) has proposed an epistemic notion of mechanism for biology and claimed that it can account for natural selection. Nicholson defines mechanisms as "epistemic models that enable the explanation of how phenomena are causally brought about" (Nicholson 2012, 161). Although Nicholson considers that his notion of mechanism allows us to understand natural selection as a mechanism, understanding natural selection (or another evolutionary cause) as a mechanism in this sense is problematic and does not suit biologists' ideas.

[2] Skipper and Millstein (2005) consider that only MDC's proposal faces problems related with regularity. Regarding Glennan's proposal, they argue that it "holds promise for capturing the way in which natural selection is regular" (Skipper and Millstein 2005, 342).

evolutionary causes. They respectively address what we call the "stochastic view", the "functional view", and the "minimalist view". We offer reasons why none of them can account neither for natural selection, nor for evolutionary causes in general. Finally, Sect. 6 concludes by underlying the potential benefits of the mechanistic proposal and noting some requirements that should be met by a mechanistic approach for evolutionary causes.

2 Evolutionary causes

Evolutionary theory usually establishes four main causes of evolution: mutation, migration, natural selection, and genetic drift.[3] Traditionally, the term mutation refers to an error in the replication of a nucleotide sequence. Nevertheless, currently "mutation" includes any alteration of the DNA molecule or any alteration of the genome such as inversions, deletions, translocations, duplications, etc. (Futuyma 2013). Migration refers to the movement of individuals between populations. These movements incorporate new genes to the gene pool of one population from one or other populations –technically, this incorporation is called gene flow, which requires not only migration but also mating in order to establish those new genes in the population (Conner and Hartl 2004). On the other hand, modern formulations of evolution by natural selection usually require three conditions: variation, heredity, and fitness differences. These are the so-called Lewontin's conditions for evolution by natural selection, heritable variation in fitness (Lewontin 1970). Although this formulation is not perfect (see Godfrey-Smith 2007), it is sufficiently general and used by researchers. Thus, we can use Lewtonin's conditions as a starting point. If in a population we have organisms that differ in their traits (variation), some of these organisms have more offspring than others due to these trait differences (fitness differences), and these traits are transmitted to the next generation (heredity), then evolution by natural selection occurs. Finally, allele and trait frequencies are also influenced by several factors that affect their trajectories in a stochastic way as a result of sampling process (Conner and Hartl 2004). This sort of randomness is called "genetic drift", an indiscriminate sampling process that produces specific effects on a population (Millstein 2002).

Unfortunately, there is no consensus among philosophers about how we should understand these different causes of evolution. Thus, a great number of different interpretations of causal evolution have been postulated: (1) the force interpretation, championed by Sober (1984); (2) the manipulationist approach based on the work of Woodward (2003) (see Reisman and Forber 2005; Shapiro and Sober 2007; Clatterbuck 2015); (3) the causal process approach, specially defended by Millstein (2006, 2013); (4) the counterfactual approach, developed by Glennan (2009) and Huneman (2012); (5) the probabilistic approach, elaborated in different ways by Abrams (2015), Razeto-Barry and Frick (2011), and Otsuka (2016); (6) and finally the mechanistic approach.

[3] There is disagreement among some philosophers on this causal view, arguing instead for a "statistical view" of evolution (Matthen and Ariew 2002, Walsh et al. 2002). In as much we accept that these phenomena are all causal, then we ask whether there can be a mechanistic account for them.

Until now, the mechanistic approach has mainly focused on natural selection (Barros 2008; DesAutels 2016; Illari and Williamson 2010). New mechanists have been arguing about how that approach suits natural selection, almost without devoting attention to the other evolutionary causes. However, the alternative approaches (e.g. the force interpretation, the counterfactual approach, etc.) aspire to locate the different evolutionary causes in a single framework. Thus, for example, the force interpretation conceptualizes the causes of evolution as forces, interacting analogously as Newtonian forces do in physical systems. This single framework aspiration results from the fact that biologists consider that all evolutionary causes are processes of the same kind (i.e. processes capable of producing changes in allele or trait frequencies, and whose (combined) effects are mathematically tractable), and that a common conceptual framework allows researchers to deal with those causes, their dynamics, and their relationships in a more effective way. Given this situation, we consider that the mechanistic approach must take on the challenge of conceptualizing all evolutionary causes as mechanisms, in order to be a legitimate conceptual framework on the same footing than the other approaches. In fact, the will of generality is already present in the new mechanistic philosophy itself (see Machamer et al. 2000; Illari and Williamson 2012; Glennan 2017). Moreover, Lane DesAutels (2018) has recently advocate the viability of the mechanistic approach as a common framework for some evolutionary causes –although this interesting first tentative does not address the issue in an extensive way. In what follows, we will analyse the current debate regarding the mechanistic interpretation of natural selection and extend it to all evolutionary causes. We will consider the main mechanistic proposals that have been developed after Skipper and Millstein's initial critique to the mechanistic approach.

3 Mechanistic proposals based on a stochastic view of mechanisms

After the initial critique by Skipper and Millstein on the mechanistic approach to natural selection, different kinds of mechanistic proposals have been raised. One sort of proposal is based on the idea that, due to the fact that natural selection is not regular, it could be possible to account for it as a mechanism by means of a notion of stochastic mechanism. This idea is proposed by Skipper and Millstein themselves after presenting their critique. In this sense, they said:

> In the context of natural selection, the most general statement we can make concerning directions for further work on the new mechanistic philosophy is this: to capture natural selection, a main evolutionary mechanism, a conception of stochastic mechanism as a non-unique causal chain is required in which change is produced by virtue of the ways in which property differences among members of a population in the context of some environment affect properties of that population. (Skipper and Millstein 2005, 345)

Barros (2008) has followed this advice and has developed a notion of stochastic mechanism in order to account for natural selection. Barros considers that by

lowering the demands for regularity it is possible to understand natural selection as a mechanism in a sense close to that posed by Glennan and MDC. He defends that the regularity that must be demanded of a mechanism depends on the type of explanation in which it is integrated. He argues that "[t]he degree of regularity needed to make a satisfactory scientific explanation depends in part on whether the explanation is being made ex post or ex ante" (Barros 2008, 309). He divides mechanistic explanations between ex post, which just explain the occurrence of a phenomenon after its occurrence, and ex ante, which "seek to explain both how a phenomenon has occurred in the past and to predict how it will occur in the future" (Barros 2008, 309). Barros claims that ex post explanations are always deterministic and make reference only to deterministic mechanisms. However, within ex ante explanations, he distinguishes between deterministic and stochastic explanations, depending on what kinds of predictions they support. Ex ante deterministic explanations are ex ante explanations which appeal to a deterministic mechanism and which support predictions with certainty. Ex ante stochastic explanations are ex ante explanations which appeal to a stochastic mechanism and where the "outcome can be predicted in advance in terms that are probabilistic" (Barros 2008, 311). Stochastic mechanisms can be biased, if they give the result a probability greater than 50%, or unbiased, if they give the result a probability equal to or less than 50%. It would be possible to distinguish between ex ante biased stochastic explanations and ex ante unbiased stochastic explanations. Natural selection would be a biased stochastic mechanism and would be involved in ex ante biased stochastic explanations. As a biased stochastic mechanism, which is present in ex ante biased stochastic explanations, natural selection just has to work more than half of the time in the same way under the same conditions.

The main problematic aspect of Barros' proposal is the idea that populations are component entities of natural selection. Barros considers that natural selection is a two-level mechanism. In order to understand natural selection in mechanistic terms it is necessary to appeal to both individual level and population level mechanisms. At the population level, natural selection is a mechanism that has populations as components. Barros understands a population as "an abstract entity that describes a group of individual organisms" (Barros 2008, 316). Understanding natural selection as a mechanism whose component entities are populations gives rise to different problems. Firstly, it would produce an identification between the subject of the overall phenomenon of the mechanism and the components of the mechanism. The mechanism would connect initial population-level conditions with final population-level conditions, through different states at the population level. This goes against the idea, which underlies the mechanistic approach, that a mechanism builds a link between a phenomenon of certain level and lower-level entities and activities (Machamer et al. 2000, 19). Presenting a mechanism means to open a black-box (a connection between an input and an output whose structure is considered inexistent or irrelevant) and the aim of a mechanism is to provide "a fine-grained as well as tight coupling" (Hedström and Swedberg 1998, 25) between the input and the output. Actually, a population-level mechanism of natural selection would not be a mechanism, but a succession of causal relations at a certain fixed level.

Secondly, it would mean accepting that a real concrete system can be entirely composed of abstract entities and their relations. We can divide the notions of mechanism

between ontic notions, which consider that mechanisms are concrete things in the real world and exist independently of us, and epistemic notions, which consider that mechanisms are pieces of scientific reasoning. Barros is aligned, like Glennan and MDC, with the ontic conception of mechanisms and considers that mechanisms are concrete things. His idea that a mechanism could be composed of populations, which he defines as abstract entities, and their relations implies assuming that a real concrete system could be entirely composed of abstract entities and their relations. It would create several problems regarding mechanisms' ontological status.

It could be though that a different notion of stochastic mechanism, which eludes the diverse problems of Barros' proposal,[4] could correctly account for natural selection and the other evolutionary causes. However, it is not at all clear if it is the case. Every attempt at understanding evolutionary causes as mechanisms by means of a notion of stochastic mechanism would be built on the assumption that they are stochastic, but we consider that this idea could be problematic too. Certainly, genetic drift is traditionally portrayed as a stochastic factor of evolution, producing unpredictable fluctuations in allele frequency (Gillespie 2004; Rice 2004). However, the stochastic character of other evolutionary causes such as natural selection, migration or mutation is at least dubious.

Several authors in this debate (Barros, Skipper, Millstein…) have accepted the stochastic nature of natural selection. However, we think that defenders of the mechanistic view should consider that the stochastic character of natural selection and other evolutionary causes is not sufficiently supported, especially from the mathematical apparatus of Population Genetics. Population Genetics textbooks usually start formulating the Hardy–Weinberg law. This law assumes: random mating, discrete generations, no mutation, no migration, no random genetic drift (infinite population size), and no natural selection. Therefore, by relaxing these assumptions we can elaborate dynamic models in order to predict the allele frequencies provided that one or more evolutionary causes are acting on populations. For differences in fitness –natural selection– one of the simplest examples is one locus with two alleles, A and a, with frequency p and q (respectively), non-overlapping generations, and with constant genotypic fitnesses w_{AA}, w_{Aa}, w_{aa}. The model deals with viability selection, where w is the average probability of survival from zygote to reproductive age. Assuming Hardy–Weinberg equilibrium before selection, the frequency of A in the next generation is

$$p' = \frac{w_{AA}p^2 + w_{Aa}pq}{\bar{w}}$$

where $\bar{w}$ is the mean population fitness ($w_{AA}p^2 + 2w_{Aa}pq + w_{aa}q^2$). The expected change in the frequency of A is

[4] Joyce C. Havstad (2011) has also raised some other problems of Barros' proposal. She noted that the model of the mechanism of generalized natural selection proposed by Barros is too general and due to this fact is unable to distinctively characterize natural selection. The model proposed by Barros is not a model of generalized natural selection but a model of selection in general, which fits any selective process.

$$\Delta_p = p' - p = p\left(p\frac{w_{AA}}{\bar{w}} + q\frac{w_{Aa}}{\bar{w}} - 1\right)$$

We can reduce the portion of the brackets as

$$\Delta_p = p\left(\frac{w^* - \bar{w}}{\bar{w}}\right)$$

where w^* is the marginal fitness of allele A, i.e., a measure of its average fitness, taking into account the frequencies of the other alleles present in the genotypes in which A is present (Charlesworth and Charlesworth 2010). This model of natural selection is a deterministic one. That is, the changes in allele frequencies must be viewed as part of a deterministic process. Although this is one of the simplest models, it shows that natural selection is modelled deterministically in Population Genetics. This is also the case for migration and mutation. Mutation is introduced by specifying the rate at which each allele (A and a) mutates to the other. In the same way, migration is introduced by specifying the rate at which a proportion of individuals immigrates or emigrates. Thus, we can combine all these different evolutionary causes, elaborating more deterministic models. For example, the *mutation-selection equilibrium* model

$$\hat{p} = \sqrt{\frac{\mu}{s}}$$

where μ is the mutation rate and s is the strength of selection, shows how the action of natural selection against a deleterious allele is compensated by a high mutation rate of change from the normal allele to the deleterious, the population being in equilibrium (Rice 2004). Similarly, the effects of selection and migration can be represented as

$$\Delta p_1 = \frac{p_1}{\bar{w}}a_A - m(p_1 - p_2)$$

where p_1 and p_2 are the frequency of the allele in each population, a_A is the average excess of fitness of the allele A, and m is the migration rate (Templeton 2006). However, when drift is introduced, the effects of these evolutionary causes are represented as a stochastic model calculating the probability distribution of populations. Thus, for the equilibrium probability distribution of allele frequency under selection, mutation, migration, and drift we obtain

$$\hat{\Psi} = C\bar{w}^{2N_e}(1-p)^{4N_e(u_1+m-mp_I)-1}p^{4N_e(u_2+mp_I)-1}$$

where C is a constant, and N_e the effective population size, u is the mutation rate, and mp_I is the migration rate times the frequency of the allele A among immigrants (Rice 2004). This is standard in any Population Genetics textbook (Charlesworth and Charlesworth 2010; Ewens 2004; Rice 2004). We start with a deterministic theory, which includes models of selection, mutation, and migration. Thereafter, we construct a stochastic theory including random genetic drift by postulating a finite

population size, and now evolution is considered as a stochastic process, where deterministic models are replaced by stochastic models, like Markov chain theory and Diffusion theory, combining deterministic and stochastic processes.

Certainly, it could be argued that the fact that some evolutionary causes (natural selection, migration, and mutation) are modelled deterministically in Population Genetics does not refute by itself their stochastic interpretation. That deterministic character could be an idealizing assumption of the models that belies biological reality. Furthermore, not every theoretical work models those evolutionary causes in a deterministic way.[5] As Millstein et al. (2009) claim, although mathematical models are relevant for understanding biological concepts, "models themselves are not the ultimate resource" (Millstein et al. 2009, 6). In order to accurately understand evolutionary causes is necessary to also take into account the historical and contemporary biological practice. Nevertheless, we consider that biological practice also casts doubt on the stochastic interpretation of some evolutionary causes. When biologists deal with real populations in their day-to-day practice, they often understand evolutionary causes such as natural selection, migration, and mutation in a deterministic way. They consider that those causes are deterministic processes that influence the change in trait frequencies, and that the stochasticity of populations' evolutionary path is mainly a product of genetic drift. In this sense, evolutionary biologist Graham Bell says: "Drift and selection are not alternatives. All populations are finite, and few are completely devoid of variation in fitness, so both drift and selection will almost always occur together. The most important effect of drift is that it makes the outcome of selection less predictable. The expected response to selection per generation in a finite population is $sp(1-p)$, its deterministic value, but through sampling error it will deviate from this precise value, the variance of this deviation being $p(1-p)/N$" (Bell 2008, 69).[6] Actually, this deterministic conceptualization[7] was already present in some classical works regarding evolutionary theory. For example,

[5] There are some theoretical works where multiple evolutionary causes, and not only genetic drift, are modelled stochastically. For example, Rice and collaborators (Rice 2008; Rice and Papadopoulos 2009; Rice et al. 2011) have developed a stochastic version of the Price equation that can deal with random variables as stochastic fitness and stochastic migration, and therefore allowing to model selection and migration as stochastic processes. However, we consider that those theoretical works do not accurately represent biologists' ideas about the nature of evolutionary causes. Biologists often understand evolutionary causes other than drift as deterministic processes. From our point of view, modelling stochastically those evolutionary causes is probably just a result of epistemological limitations. This seems the case of Rice and collaborators, inasmuch they talk repeatedly about our epistemic problems to study evolutionary systems and the necessity to construct stochastic models to supply those shortcomings. For example, they say that "we can not know with certainty how many descendants an individual will leave or what they will look like until after reproduction has taken place" (Rice and Papadopoulos 2009, 2); and that "[b]ecause we can not know with certainty how many descendants each individual in a population will have, we need to treat fitness as a random variable -having a distribution of possible values" (Rice et al. 2011).

[6] It is necessary to point out that we are not claiming that any deviation from fitness expectations should be attributed to drift. That deviation could be the result of other elements (selective pressures and/or other evolutionary causes that were not taken into account, a spurious statistical correlation, etc.).

[7] Another remarkable example of this deterministic conceptualization is provided by Brian Charlesworth: "In the era of multi-species comparisons of genome sequences and genome-wide surveys of DNA sequence variability, there is more need than ever before to understand the evolutionary role of genetic

in the well-known experiment by Dobzhansky and Pavlovsky (1957), it is argued that the apparent stochasticity of natural selection is a consequence of the finite size of populations (i.e. genetic drift). Dobzhansky and Pavlovsky tracked the frequencies of two genetic types, PP and AR, which are different inversions on the third chromosome in *Drosophila pseudoobscura*. They replicated twenty populations, ten with a large initial population (of about 4000 flies) and ten with a small initial population (of about 20 flies). They found that the outcome of natural selection in the studied populations was conditioned by genetic drift. Although, "the environments being reasonably uniform in all experimental populations, the outcome of the selection processes in the replicate experiments should also be uniform" (Dobzhansky and Pavlovsky 1957, 316), the finite size of the populations (i.e. genetic drift) prevent the outcomes from being uniform. They also showed that, as the population size decreases and the influence of genetic drift increases, the deviations become greater, increasing the fluctuations across generations.

Defenders of the stochastic view of evolutionary causes could argue, as Skipper and Millstein (2005) do regarding natural selection, that several identical real populations under the influence of the same evolutionary cause would, in all likelihood, differ in their evolutionary outcomes. The structure of the argument would be as follows. Imagine that we have one hundred populations of an organism (like finches), with the same population size, the same distribution of beak lengths, and all located in the same environment, and that one evolutionary cause such as natural selection, mutation, or migration acts upon them. It would be expected that, despite the fact that the evolutionary cause is equally acting in all of them, the evolutionary outcomes of the various populations would not be equal (i.e. the distribution of break lengths in the next generation will differ). It could seem that this kind of argument supports the idea that those evolutionary causes are stochastic processes whose outcomes are not fully predictable. However, we think that it is inaccurate. From biologists' point of view, that difference in populations' outcomes would not be a consequence of the stochastic nature of those evolutionary causes, but an effect of genetic drift (Hansen 2017). The populations' evolutionary outcomes would differ just because, since those populations are finite, genetic drift affects them.

On balance, to claim that natural selection and other evolutionary causes such as migration and mutation are stochastic processes needs more support and deal with the theoretical mathematical models developed in the last century. The stochastic view, in order to become a common framework and account for evolutionary causes as stochastic mechanisms, requires conceptualizing evolutionary causes as stochastic processes. A process can only be understood as a stochastic mechanism if it is stochastic. Although some evolutionary causes –like genetic drift– fit into that framework, others –like migration or natural selection– are less clear. Therefore, we argue that both parts, defenders and critics of the mechanistic approach, must do not take for granted the stochastic nature of some evolutionary causes and

Footnote 7 (continued)
drift, and its interactions with the deterministic forces of mutation, migration, recombination and selection." (Charlesworth 2009, 195).

discuss more deeply on it. This also undermines Skipper and Millstein's argument that natural selection is not regular enough and does not meet MDC's requirement of regularity. Their critique, which motivates Barros' proposal, is not justified enough. Although this gives new chances for the mechanistic approach, it still has to handle with other evolutionary causes (essentially, genetic drift) that are unquestionably considered stochastic. A defender of the mechanistic view could answer that it is possible, as Barros shows, to hold a pluralistic mechanistic framework and consider that mechanisms could be deterministic or stochastic. Following this line of argumentation, it could be argued that all evolutionary causes are mechanisms, although some are deterministic mechanisms and other are stochastic mechanisms. However, if this position was taken, the question would be whether in that scenario evolutionary causes would actually be allocated in a single framework. There would be some relevant differences among them (e.g. grade of regularity, type of connection among its components, predictability, etc.), and it is not clear which similarities would be underlined by this framework.

4 Mechanistic proposals based on a functional view of mechanisms

There is a strong connection between the notion of mechanism and the notion of function. Several definitions of mechanism have been elaborated on the basis that a mechanism is something that performs a function. The most relevant of them is the notion of mechanism proposed by Bechtel and Abrahamsen (2005). They define a mechanism as a "structure performing a function in virtue of its component parts, component operations, and their organization" (Bechtel and Abrahamsen 2005, 423). More recently, Garson (2013) has also defended a functional notion of mechanism.

Recently some authors have tried, inspired by functional notions of mechanism and based on the consideration that natural selection performs a function, to respond to the Skipper and Millstein's initial critique and defend the possibility of understanding natural selection as an MDC mechanism. Illari and Williamson (2010) have argued that natural selection is decomposable into parts (entities and activities) and has organization. In a similar line of argumentation, Lane DesAutels (2016) has argued that natural selection meets the requirement of regularity imposed by MDC.

Illari and Williamson argue that for a complex system to be a mechanism it has to perform a function. In this sense they affirm: "mechanisms are mechanisms for a phenomenon. In that sense, mechanisms have functions" (Illari and Williamson 2010, 283). If the mechanism is integrated into a system, "function" must be understood as "causal-role" function (Cummins 1975). If the mechanism does not refer to a system that integrates it, then "function" has to be understood on the basis of the notion of Craver (2001) of "isolated description". In the case of natural selection, they consider that it does not refer to a system that integrates it and "function" must be understood on the basis of the notion of "isolated description".

Craver (2001) considers that there are three ways to describe the activity of a mechanism: contextual description, isolated description, and constitutive

description. Isolated description consists of describing the activity of the mechanism regardless of context (Craver 2001, 64). From this perspective the activity of the mechanism: (1) does not refer to a context, (2) does not refer to objects beyond the limits of the mechanism, (3) it is something that the mechanism produces by itself, and (4) allows setting the active, spatial and temporal limits of the mechanism. Illari and Williamson appeal to a notion of function derived from this type of description. According to this notion, which is not made explicit by Craver, the function of a mechanism is the activity assigned to it by means of an isolated description. They refer to this activity as the "characteristic activity" of the mechanism. From this approach the function of the heart would be to contract. In the case of natural selection, they consider that its function –characteristic activity—is the production of adaptations.

Illari and Williamson argue that by taking the function of a mechanism as a reference it is possible to decompose it into entities and activities. They consider that the components of a mechanism have to be identified and individualized on the basis of their functions. In this sense they say: "successful structural decomposition is into functionally relevant parts" (Illari and Williamson 2010, 284). In relation to the components of the mechanisms "function" must be understood as a "causal-role" function. Regarding the function of the entities they affirm: "The function of an entity is the role it plays in the overall behaviour of the mechanism" (Illari and Williamson 2010, 285). Besides, regarding the activities, they maintain a similar position and affirm: "[a]ctivities are individuated in a similar way to entities in the hierarchy of mechanisms. Activities are identified in terms of their contribution to the behaviour of the phenomenon to be explained" (Illari and Williamson 2010, 285). The function of a component is always dependent on the function of the mechanism, since the function of the component is characterized on the basis of its contribution to the function of the mechanism. Therefore, since the individualization of the components depends on their function, the individualization of the components depends on the function of the mechanism.

They consider that insofar as natural selection has a function, it is decomposable into parts. In the case of natural selection, the components would be sub-mechanisms that are integrated into it and contribute to its function. Examples of such component entities of natural selection would be populations, organisms, cells, etc. Examples of activities would be reproduction, recombination, and so on.

Illari and Williamson also defend that by taking as a reference the function of a mechanism it is possible to attribute organization to it. They define the organization of a mechanism as "whatever features exist by which the activities and entities each do something and do something together to produce the phenomenon" (Illari and Williamson 2010, 289). The organization can be of different types: spatiotemporal organization, feedback, control systems, part-whole, etc. They consider that natural selection, insofar as it has a function, is an organized mechanism. With regard to the type of organization that occurs in natural selection, they point out that "no one form of organization is present in all cases of natural selection but only organization understood at a certain level of abstraction" (Illari and Williamson 2010, 290). However, with regard to the concrete cases of natural selection they affirm: "natural

selection in the concrete case does show spatiotemporal organization" (Illari and Williamson 2010, 290).

DesAutels takes as his starting point the thesis defended by Illari and Williamson. His argument assumes the following considerations: (1) mechanisms have a function, (2) natural selection has a function (to produce adaptation), and (3) natural selection is decomposable into entities and activities. On the basis of these considerations he argues "natural selection only fails to be regular in ways that are unthreatening to its status as MDC mechanism" (DesAutels 2016, 16). Firstly, DesAutels considers that the regularity demanded can be understood as process regularity or as product regularity. The process regularity consists in that "the constituent entities and activities of a mechanism behave in roughly the same way each time the mechanism operates" (DesAutels 2016, 16) and product regularity consists in that "the output of a mechanism is roughly the same" (DesAutels 2016, 16). He considers that the relevant regularity to be an MDC mechanism must be the process regularity, due to the fact that the paradigmatic example of an MDC mechanism (protein synthesis) is process regular but not product regular. Natural selection is not product regular, as noted by Skipper and Millstein (2005, 343). However, natural selection is process regular.

Secondly, DesAutels distinguishes between internal and external sources of irregularity. The irregularity of a mechanism may be due to elements internal to it or elements external to it. The delimitation of a mechanism (to establish what is internal and what is external to it) depends on its function. In this respect he says: "entities and activities are constitutive of a given mechanism just in case the mechanism could not serve its function without them" (DesAutels 2016, 18). In order to be an MDC mechanism the relevant aspect is not to be irregular due to internal sources. The criterion for being regular cannot be regularity regarding external sources because even the most regular mechanisms (e.g. synaptic transmission, protein synthesis, DNA replication, etc.) may be affected by external sources of irregularity.[8] Skipper and Millstein (2005, 343) reveal the irregularity of natural selection over external sources of regularity, but not over internal sources.[9]

The authors whose proposal is based on a functional view of mechanisms consider that natural selection has a primary function: producing adaptations. As we have seen, this idea is the cornerstone of their approach and all their arguments ultimately rely on it. Nevertheless, we consider that producing adaptations is not the function of natural selection. Illari and Williamson say: "Natural selection having an isolated description is no problem. Natural selection explains adaptation, because natural selection characteristically produces adaptation. So natural selection is a mechanism *for* adaptation" (2010, 283). DesAutels agrees: "Quite clearly,

[8] We think that, in the case of natural selection, those external sources of irregularity identified by DesAutels –i.e. "non-critical environmental features which are not constitutive of the token mechanism [selection]" (2016, 19)– are very likely to be just instantiations of drift processes.

[9] DesAutels also proposed a third distinction. He distinguishes between abstract and concrete regularity. Abstract regularity is the ability of a type mechanism to subsume different token mechanisms. On the other hand, concrete regularity is the capacity of a type mechanism to give a detailed account of the token mechanisms that it subsumes.

natural selection is a system *for* something: it is that which brings about adaptation" (2016, 14). This move is quite natural. Organisms appear to be designed for their environments. This apparent design, attributed to a supernatural designer for centuries, needed a naturalistic explanation. Darwin gave us that naturalistic explanation where the process of natural selection progressively improves organism's suitability to their environment, and therefore giving them the appearance of design. Since Darwin published his theory, natural selection has been strongly connected to the concept of adaptation. Nevertheless, our aim in this part of the section is to challenge this deep connection, and thus partially undermining the functional argument of natural selection as a mechanism for adaptation.

The original formulation of natural selection by Darwin was strongly influenced by William Paley's "argument from design" (Darwin 1958; Ayala 2004) and the demographic works of Robert Malthus (Darwin 1958; Eldredge 2005). Thus, Darwin focused on explaining how highly functional organs like the human eye (Paley) could have emerged from the struggle for existence. This struggle would be an ineludible result of lack of resources in nature due to the geometrical growth of populations (Malthus). Following this line of argumentation, some authors (for instance Dennett 1995) proposed to define fitness as an individual's capacity to solve "design-problems" set by the environment, i.e. its capacity to fit in that environment. This engineering approach has been very successful in some areas (like behavioral ecology), and especially in popular explanations of the theory of natural selection. This engineering view is based on the idea that organisms evolve traits that maximize the ability of a population of those organisms to increase in size, i.e. traits that increase overall population growth rate (Rice 2004).

However, theoretical and experimental advances over the last century show a subtle but critical distinction between the efficiency of an individual and its capacity to fit or adjust in a particular environment, i.e. to solve the "design-problems". Imagine a female lion –a type of lion– with the best possible characteristics to deal with all the challenges of its environment: a short, thin coat to carry high temperatures; a strong and robust body structure capable of dealing with all kinds of blows, falls, and collisions; sharp fangs to tear flesh; strong claws to catch prey; sexually attractive to seduce male lions; etc. This (type of) lion is perfectly adapted to its environment. Nevertheless, imagine that this (type of) lion is sterile. If that is so, then its fitness (i.e. its reproductive efficacy) would be null because it does not leave offspring and therefore its genes do not pass to the next generation. Natural selection would act against this type of lions –against the infertile trait–, although their suitability to the environment is impressive.[10] It is necessary to understand that, ultimately, natural selection acts on one trait –fitness, i.e. reproductive success– and it may act indirectly on other traits.[11] The idea is that natural selection does not necessarily

[10] Certainly, there are examples of sterile organisms with positive fitness, like in eusocial insect colonies, where some individuals forego their reproductive capacity in order to support the reproduction of their relatives. These types of organisms successfully pass their genes to the next generation. In our example, there is no such behavior.

[11] As Gillespie says: "To a geneticist, fitness is just another trait with a genetic component. To an evolutionist, it is the ultimate trait because it is the one upon which natural selection acts" (Gillespie 2004, 59).

lead to adaptation, as Darwin thought, but always leads to non-random reproductive success.[12] That is, adaptation is a potential (but not necessary) outcome of the process of natural selection (Gould and Lloyd 1999). Here is an example by Richard Lewontin:

> A mutation that doubled the egg-laying rate in an insect, limited by the amount of food available to the immature stages, would be very rapidly spread through the population. Yet, the end result would be a population with the same adult density as before but twice the density of early immatures and much greater competition among larvae stages. Periodic server shortages of food would make the probability of extinction of the population greater than it was when larvae competition was less. Moreover, predators may switch their search images to the larvae of this species now that they are more abundant, and epidemic diseases may more easily spread. It would be difficult to say precisely what environmental problem the increase of fecundity was a solution to. (Levins and Lewontin 1985, 81)

Those mutant insects mentioned by Lewontin are not better adapted because there is no improvement in their performance regarding environmental challenges. That is, there is no better fit to their environment because they have double offspring but also double mortality. Now imagine that some of those mutant insects suffer another mutation that doubled the efficiency for metabolizing food. That would be an adaptive improvement and it would be reflected in the population census, increasing the population size. This is what Darwin had in mind and why the term *fitness* was used, since *to fit* makes reference to the grade of adaptedness of an individual to its environment. In other words, natural selection does not necessarily optimize any individual trait—i.e. an optimal trait value that maximizes the fitness associated with that trait–, and when it does it is under rare circumstances such as when the trait has no phenotypic variance at equilibrium, and there is nonlinearity between the trait and fitness at equilibrium (see Crow and Nagylaki 1976; Templeton 2006, for mathematical details).

In summary: "The point here is that the dynamics of selection depend on the dynamics of population growth, and this critically affects the outcome of selection. Survival of the fittest is just one of many possible outcomes. There is more to selection and evolutionary success that increase of the better-designed phenotypes" (Michod 1999, 27). Of course, there are in the literature definitions of adaptation where the most adapted phenotype is always chosen by natural selection (see Reeve and Sherman 1993). Nevertheless, we think that they are misleading, since there are conditions where natural selection operates and the best-adapted organisms do not succeed.[13] Therefore, one might say that that undermines the functional argument

[12] De Jong endorses a similar claim: "Differences in adaptation will lead to fitness differences, but fitness differences are not necessarily associated with differences in adaptation" (De Jong 1994, 20).

[13] Another example of this is a particular type of selection, the so-called called "survival of the first", where a best adapted type cannot invade a population because the population growth is superexponential (see Michod 1999, chap. 3; Nowak 2006, chap. 2, for mathematical details). It could be argued that the fact that natural selection does not always produce adaptation, does not imply that adaptation is not the

of natural selection as a mechanism for adaptation. In our opinion it is true that a mechanism always needs the same function, but it certainly isn't true that if there is a different function, there is no mechanism. We have tried to show that the function of natural selection is not what Illari, Williamson and DesAutels thought it was, but this is quite different to there not being any function at all. It could be argued that the proper function of natural selection is to increase the reproductive success. However, this change has a cost for the functional view, because changing the function implies changes in the entities and activities that compose the mechanism, as well as its organization and boundaries, inasmuch all those elements rely upon the assumed function of the mechanism.

Illari and Williamson—and DesAutels to the extent that he probably follows them in this aspect–, are not only wrong when they claim that the function of natural selection is the production of adaptation, they are also wrong when they consider that, if we defined the function of a mechanism as the activity assigned to it by means of an isolated description (characteristic activity), then the function of natural selection would be the production of adaptation. Often, when an isolated description of a process of natural selection is presented, the activity assigned to it is not the production of adaptation, but the increase of reproductive success.

The functional view, in order to be applied to evolutionary causes, needs to assign functions to them. The problem is that each evolutionary cause brings about several outcomes and it is difficult to identify one of them as its function. Mutation is usually considered the ultimate source of genetic variation. So, a defender of the functional view may claim that the function of mutation is to provide genetic variation. However, a crucial problem arises very quickly when we try to assign functionality to particular outcomes of migration and drift: there are too many. Migration and drift have several impacts on populations. Migration introduces genetic variation in a population. It also reduces the level of differentiation among populations, homogenizing the populations of a species. In addition, migration is crucial for hybridization and for the process of speciation. Meanwhile, drift plays a major role in the survival of new mutations and their possible fixation in a population. It removes genetic variation in a population, but at the same time increases the level of differentiation among populations. It also, as a random process, affects the predictability of a population. In addition, it should be recalled that all evolutionary causes affect gene frequencies (another outcome). Therefore, migration and drift produce several outcomes upon populations, and this is also true for mutation and selection. Mutation alters the change in gene frequency, but also increase variation within subpopulations and decreases the variation among subpopulations. Likewise, natural

Footnote 13 (continued)
function of natural selection. In order for a mechanism to have a function it does not have to always succeed at implementing that function. For example, heart's function is to pump blood at a specific rate, but sometimes it does not pump blood at that rate. Nevertheless, it does not seem the case for natural selection and adaptation. When a heart does not pump blood at a proper rate, doctors consider that it is not producing its function in a correct matter. However, when natural selection is acting but it does not produce adaptation, evolutionary biologists do not consider that it is not producing its function in a correct matter, as long as there is non-random differential reproductive success.

selection alters the variation within and among subpopulations, as well as changing the gene frequencies. Thus, the functional view must contend with the problem of attributing a particular function for each evolutionary cause in spite of the great variability of outcomes. This problem could not be resolved by means of considering that the function of an evolutionary cause is its characteristic activity (i.e. the activity assigned to it by means of an isolated description), because the production of all the outcomes noted above is assigned to the causes precisely when they are described in isolation.

The fact that several phenomena or outcomes are attributed to each evolutionary cause is also problematic for the functional view in another related sense. A mechanism is always a mechanism for some phenomenon (Glennan 1996, 52; Bechtel and Abrahamsen 2005, 422; Craver 2007, 123). The defenders of the functional view consider that the identification and delimitation of a mechanism depends on the phenomenon for which it is responsible. A mechanism is composed of those entities, activities, and organizational features that contribute to the phenomenon for which it is responsible. It implies that the mechanism responsible for each phenomenon or output is different. Hence, from their proposal, the mechanism responsible for each outcome attributed to a particular evolutionary cause would be a different one. But it does not suit evolutionary biologist ideas. They consider that the *same* particular evolutionary process is responsible for all those outputs. Figure 1 shows a particular model of migration, a circular stepping-stone model, where migration occurs only between adjacent demes. In this case, the entities and activities that contribute to each output attributed to a particular case of migration are not the same. Therefore,

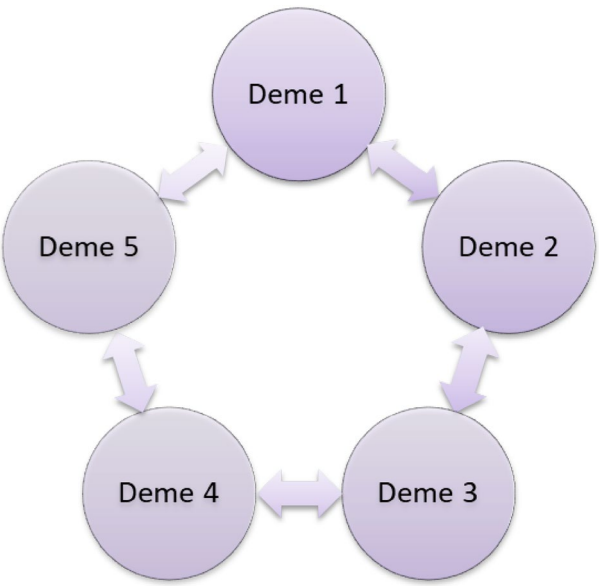

Fig. 1 This image represents a circular stepping-stone model, where migration occurs only between adjacent demes. A particular phenomenon of migration generates two different outcomes (i.e. changes in two demes). The entities and activities responsible for those outcomes are not exactly the same

from the functional approach's point of view, the mechanism responsible for each outcome would be different. On the contrary, evolutionary biologists consider just one evolutionary cause for all outputs attributed to a particular case of migration.

In order to avoid those problems, the defenders of the functional view could answer that the phenomenon for which a particular evolutionary mechanism is responsible is the conjunction of the outputs attributed to it by biologists. In that case, the mechanism would be composed of every entity, activity, and organizational feature that contribute to any of that outcomes. However, this approach does not suit biologists' ideas and in our view it seems an artificial and ad hoc construction. Biologists consider those outcomes as different phenomena produced by the same cause, not as parts of the same phenomenon.

5 Mechanistic proposals based on a minimalist view of mechanisms

Glennan (2017) has recently adopted a new approach in order to account for the diverse kinds of mechanisms (including evolutionary mechanisms). He has assumed a minimalist view. Glennan has abandoned his previous notion of mechanism (see Glennan 1996, 2002) and developed a minimal characterization which aims to be "broad enough to capture most of the wide range of things scientists have called mechanisms" (Glennan 2017, 18). Thus, he defines a mechanism for a phenomenon as follows:

> A mechanism for a phenomenon consists of entities (or parts) whose activities and interactions are organized so as to be responsible for the phenomenon. (Glennan 2017, 17)

Glennan considers that a mechanism is composed of organised entities and their activities. Entities are "objects—things that have reasonably stable properties and boundaries" (Glennan 2017, 20) while activities are "a kind of process—essentially involving change through time" (Glennan 2017, 20). Activities that involve more than one entity (pushing, eating, hitting…) are called interactions. Regarding mechanisms' organization, Glennan considers that it has a horizontal dimension (i.e. spatio-temporal and causal organization among the mechanism's components) and a vertical dimension (i.e. relation between the mechanism as a whole and its component activities).

Although Glennan's new minimal notion of mechanism aims to account for mechanisms across the sciences, it does not suit evolutionary mechanisms. Glennan considers mechanisms in general and his notion include general characteristics that are often present in mechanisms. Nevertheless, his proposal does not take into account the specific traits of evolutionary causes. In fact, this new version of Glennan's proposal faces the same kinds of problems (i.e. problems related with parts or organization, and problems related with productive relationships) that the previous version of it (see Sect. 1). In the first place, Glennan's notion of mechanism's component entity (or part) does not suit evolutionary causes' parts. Glennan characterizes mechanisms' component entities as "stable bearers of causal powers" (Glennan 2017, 35). He considers that entities must have reasonable stable properties and

boundaries. The stability required to entities depends on the mechanism in which they are involved. A component entity of a mechanism is stable enough if its properties and boundaries remain stable while the phenomenon for which that mechanism is responsible is taking place. For example, component entities of a circulatory system (heart, lungs, veins…) are stable enough. Their properties and boundaries remain stable while blood circulates and transports blood cells, carbon dioxide, hormones, nutrients, and oxygen. However, the application of this requirement of stability to evolutionary causes' parts would be problematic. Firstly, it would be very difficult to determine which degree of stability must be required to a part of an evolutionary cause. Evolutionary causes are often responsible for several phenomena with different timescales. As it has been argued (see Sect. 4), each evolutionary cause is responsible for several phenomena or outcomes. For instance, genetic drift is responsible for the fixation of certain new mutations, the removal of genetic variation in populations, the increment of differentiation among populations, etc. Likewise, mutation is responsible for the change in gene frequency, the increment of variation within subpopulations, the decrease of variation among subpopulations, etc. Phenomena for which an evolutionary cause is responsible often do not have the same duration. Frequently, one phenomenon for which the cause is responsible has finished while others are still taking place. For example, a new mutation may have already been fixated in a population by drift while the increment of differentiation among populations is still taking place. This plurality of phenomena and timescales means that there is not a unique phenomenon's timescale associated to each particular evolutionary process. Therefore, it would not be clear which timescale should be the reference for the requirement of stability and which degree of stability should be demanded. Secondly, properties of parts of evolutionary causes do not always remain stable while those causes and phenomena for which they are responsible are taking place. Evolutionary causes' parts often undergo changes during the evolutionary processes. For example, consider genetic drift and migration. Individual organisms are component parts of those processes. In genetic drift, individual organisms are subject to environmental influences which do not depend on their physical characteristics. Likewise, migration involves individual organisms that move from one population to another. However, individual organisms' properties do not always remain stable while drift or migration processes are taking place. In this manner, phenotypic plasticity (Pigliucci 2001) may change some individual organisms' properties such as colour skin, height, or even reproduction mode during a process of drift or migration. This is also the case for natural selection. At the present time, there is a debate about which are the parts of natural selection (see Glennan 2009; Millstein 2006; Otsuka 2016). Some authors consider that parts of natural selection are individual organisms while other authors argue that they are populations. Nevertheless, in both cases parts of natural selection would not be stable enough. Individual organisms' properties may change while a process of natural selection is taking place. Those changes may be produced by phenotypic plasticity, but they may also be a consequence of certain circumstances during organisms' lifespan. Imagine an individual male organism that, during a process of sexual selection, loses an eye fighting against a competing male. Although that wound may not affect its reproductive fitness, some of its properties would have significantly changed during

the evolutionary process. Likewise, populations' properties and boundaries change while a process of natural selection is taking place. There is always a change in the mean (or higher moments) distribution of certain traits in populations during processes of natural selection. Besides, populations' boundaries change because of births and deaths.

Regarding problems related with productive aspects, Glennan's notion of activity does not account for many productive relationships that occur in evolutionary processes. Glennan characterizes productive relationships within mechanisms as activities. Activities are processes that involve change through time, in which at least one entity (or part) is engaged. One trait of activities is that they "require entities (parts, components) to act and be acted upon" (Glennan 2017, 31). However, this characterization of activities does not fit many productive relationships within evolutionary processes. Productive relationships among parts of evolutionary causes are possible even if none of them has been acted upon. For example, consider a process of frequency-dependent selection. Imagine a population of birds composed by two types: green and blue. Predators see much better green organisms than blue ones, making them decrease their frequency, and increasing blue organisms' frequency. In this example, there is a productive relationship between both bird types. Blue type has increased its frequency because green types are easier to see by predators. Nevertheless, blue types have not been acted upon by green types. This productive relationship within a process of natural selection does not suit Glennan's notion of activity.

In addition to the previously outlined difficulties, Glennan's proposal would also face the functional view's problem regarding the identification and delimitation of evolutionary causes (see Sect. 4). As functional view's advocates, Glennan argues that a mechanism is always a mechanism for a phenomenon and that "[a] phenomenon is what is used to identify and delimit its mechanism" (Glennan 2017, 23). He considers that a mechanism is composed of those entities, activities, and organizational features that contribute to the phenomenon for which it is responsible. It would mean that the mechanism responsible for each phenomenon or outcome is different. However, as it has been noted, this approach does not suit evolutionary biologists' ideas about the identification and delimitation of evolutionary causes. They do not consider that for each outcome of causal evolution there is a different evolutionary cause which is responsible for it. For them, a particular evolutionary cause may be responsible for several outcomes.

6 Conclusion

As we have noted, there is no consensus about how the causes of evolution should be understood (see Sect. 2). Thus, we consider that it is relevant to study whether a mechanistic framework can account for evolutionary causes. If it were successful, it would bring us some benefits. Some of those would be (1) it would make possible to explain without laws (which are not present in some cases of evolution); (2) it would offer helpful possibilities of representation (e.g. spatial representation, colours, etc.) which are not available in other frameworks (e.g. probabilistic, counterfactual, etc.); (3) the appellation to mechanisms may be used to support causal

claims; (4) mechanisms would connect different ontological levels; and (5) it would reveal an underlying link between evolutionary biology and other fields of biology where the mechanistic framework is generally accepted (e.g. molecular biology, biochemistry, etc.).

However, as we have showed through the present article, the mechanistic framework is faced with outstanding difficulties. Current mechanistic approaches to causal evolution have problems for accounting for natural selection and the other evolutionary causes as mechanisms. The stochastic view, in addition to unjustifiably assume the stochastic character of natural selection, must admit either that not all evolutionary causes are mechanisms or that evolutionary causes are mechanisms of very different types—which would make difficult to account for their relations and dynamics. Likewise, the functional view erroneously postulates that natural selection's function is to produce adaptation and faces the problem that evolutionary causes produce several outcomes and it is not clear which would be their function. Finally, the minimalist view, which considers mechanisms in general, does not take into account the specific traits of evolutionary causes and cannot account for their parts and productive relationships.

Given the issues above noted, it is possible to raise some requirements that a fruitful and encompassing mechanistic approach for evolutionary theory should meet. Firstly, it should account for all evolutionary causes as similar processes in order to satisfy biologists' considerations, and therefore it should not accept excessive heterogeneity on what is considered an evolutionary mechanism. Secondly, it should find out common characteristics not based in regularity. A particular kind of regularity (to be stochastic, to be deterministic, etc.) cannot be among the requirements for defining evolutionary mechanisms. Thirdly, it should take into account the interactions between different evolutionary causes. This is something that no one has faced, since almost all discussions have been around natural selection. A mechanistic common framework demands specifying how interactions among evolutionary mechanisms are, using a common measure for all mechanisms, etc. Related to this, evolutionary mechanisms should be conceptualized in an operative way, allowing us to build tractable (mathematical) models. Thus, these models would show the dynamics among evolutionary mechanisms, underline relationships, etc., offering the kind of things that a common framework does. Therefore, an excessive heterogeneity would violate that feature. Finally, a mechanistic approach could take into account several hierarchical or ontological levels. This is important because there is no consensus about whether the causes of evolutionary change belong to the level of individuals (Bouchard and Rosenberg 2004), or whether the causes of evolution should be better understood as population-level causes that act on the entire population (Millstein 2006). In contrast, mechanisms are able to deal with different hierarchical levels, showing at the same time individual and population-level interactions. Nevertheless, this is conditional upon the satisfaction of previous points. That is, taking into account different levels could lead to a proliferation of variables –because you are explicitly exposing individual and population interactions of several mechanisms–, potentially producing an inoperative framework (for instance,

mathematically intractable, requirements of too much data, etc.). This unpleasant result would reduce its attractiveness as a common framework for biologists.[14]

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[14] There is a related worry. It might also be the case that a mechanistic approach is able to conceptualize evolutionary factors as mechanisms, in an isolated way, but it is not able to express the interactions between different mechanisms.

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