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The Historical Transformation of Individual Concepts into Populational Ones: An Explanatory Shift in the Gestation of the Modern Synthesis

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Abstract: In this paper, I will conduct three interrelated analyses. First, I will develop an analysis of various concepts in the history of biology that used to refer to individual-level phenomena but were then reinterpreted by the Modern Synthesis in terms of populations. Second, I argue that a similar situation can be found in contemporary biological theory. While different approaches reflect on the causal role of developing organisms in evolution, proponents of the Modern Synthesis avoid any substantial change by reinterpreting and explaining individual-level phenomena from a population perspective. Finally, I will approach this debate by advocating the statistical reading of natural selection, which holds that explanations by natural selection are statistical. I will argue that the above historical conceptual reinterpretations belong to a new explanatory strategy developed by the Modern Synthesis based on population thinking. The reinterpretation of concepts at the individual level is part of the explanatory framework of the Modern Synthesis and the empty role of development within this framework. Moreover, the statistical perspective adopted here allows for the integration of two explanatory models: population-statistical and individual-causal. Finally, I will argue that this pluralistic framework can help to define the explanatory scope of the different biological approaches in order to achieve a coherent integration of development into evolution without rejecting population thinking.

Keywords: Conceptual Change in Biology; Evolutionary Explanations; Evolutionary Causation; History of Biology; Statisticalism.

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Introduction

This paper addresses different but interrelated themes in the history and philosophy of biology. The proposal is primarily metatheoretical. I will address how different biological theories and approaches have been linked throughout the history of biology. I will focus on two inextricably linked aspects of biological theories: their explanatory methods and the concepts that underlie their explanations. My central thesis is that the interpretation of biological concepts is related to the nature of the explanation.

This paper consists of three sections. In the first section, I will analyze a recurring pattern in the history of biology: the transformation of concepts originally used for individual processes into concepts related to population phenomena. This transformation took place during the gestation of the Modern Synthesis (hereafter: MS), especially in the first decades of the 20th century. I will focus on four biological concepts that have changed their meaning in the history of biology: the concept of heredity, the Baldwin Effect, Schamalhausen's stabilizing selection, and the concept of the reaction norm. In all cases, the conceptual reinterpretation has important theoretical implications. In particular, these conceptual changes enabled the integration into the MS of many developmental phenomena that were problematic for the foundations of the MS.

In the second section, I will delve into contemporary debates about the challenges that have confronted the MS in recent decades. The preceding analysis helps us to recognize a similar scenario that can be found in the history of biology. While critics of the MS argue that individual-level phenomena are central to understanding evolution, proponents of the MS claim that the explanatory role of individual development can be explained by population-level explanations. To illuminate this historical parallel, I will show how the same concepts analyzed in Section 1 are central to recent critics of the MS. I will also show how contemporary defenders of the MS manage to resolve this critique using the same explanatory strategy elaborated in Section 1.

Finally, in Section 3, I will link these historical and contemporary analyses to the debate about the causal structure of natural selection, i.e. how we must understand the nature of natural selection, its explanatory character, and its causal basis. There are two competing theories in the debate: the Causalist and Statisticalist Schools. After introducing each school, I will turn to evaluate its implication for the historical (Section 1) and contemporary (Section 2) analyses. Concerning the historical debate, if we follow the causalist position, we conclude that the conceptual reinterpretations during the gestation of the MS should be understood as transmission of causality: What is caused by individual development should be understood as caused by populational processes. However, if we take the statistical point of view, we can conclude that the semantic reinterpretation is part of an *explanatory shift*: phenomena caused by development are explained in *statistical terms* at the populational level. Supporting the statisticians' point of view, I conclude that the concept of reinterpretation of phenomena at the individual level should not be understood as a transfer of evolutionary causes between levels (individual and populational), but as the construction of a different explanatory strategy in population biology. In this sense, the current debates in evolutionary theory are also affected *if* we follow the statisticalist interpretation. First, contemporary defenders of the MS intend to provide statistical explanations for what should be causally explained. Second, the statistical interpretation of natural selection fits the populational, mathematical model of population genetics advocated by contemporary MS's proponents. Finally, the statistical reading does not neglect the central explanatory role of population explanations. Therefore, the statisticalist interpretation of conceptual change can lead to a parsimonious pluralistic framework for integrating new approaches in theoretical biology into the foundations of the MS.

1. The transformation of individual concepts into populational concepts in the history of biology

In this section, we will look at several examples of a similar phenomenon in the history of biology: how certain terms that referred to individual processes during ontogeny were reinterpreted in populational terms. As we will see in each example, this conceptual transition occurred in two steps: first, a particular biological phenomenon

originally explained in terms of developmental processes was reduced to the genetic level; second, the processes caused by individual ontogenetic processes were explained in populational terms by population genetics.

1.1 Heredity: From Epigenetics to Genetics

Ron Amundson (2005) traces the historical path from a developmental/epigenetic conception of heredity to the genetic (and popular) conception. The epigenetic concept of heredity was associated with the introduction of the term in biology – initially adopted as a metaphor from the social sciences. Heredity was born as the discipline dedicated to understanding the similarity between parents and offspring. The epigenetic concept of heredity holds that the similarity of traits across generations is explained by the transmission of developmental processes that build phenotypic traits from generation to generation. As Amundson (2005, 142) states, "[i]nheritance is the production of parent-offspring similarities, and this production takes place throughout epigenesis. Heredity is an epigenetic process." Traits are not skipped or copied at conception but reconstructed from generation to generation through the causal mechanisms of inheritance: "The causes of heredity are exactly the same as those of development" (Amundson, 2005, 143).

In the context of the debate between epigenetics and preformationism, studies of heredity within the epigenetic theory of inheritance have supported the epigenetic team: Similarity is a constructive process; it does not precede development. However, it would be wrong to claim that the epigenetic side of the debate has won the battle. The epigenetic theory of heredity was replaced by a preformationist theory in the twentieth century during the gestation of the MS. The abandonment of epigenetic theory resulted in a crude separation between inheritance and development: inheritance and development are different processes, they are explained in different ways and by different biological disciplines. Various findings were central to the construction of a genetic, preformationist theory of inheritance, such as the emergence of Mendelian genetics and the construction of the Wiessmann barrier, which is based on the separation between the germline and the somatic lines, or, in its modern version, on the separation between the genotypic space and the phenotypic space.

The resulting genetic theory of inheritance states that inherited material is transmitted at conception (Mamelli, 2005). Inheritance is not a transmission of developmental processes. Rather, it is a transmission of information units that are responsible for the structure of organisms: These units became known as genes. In other words, heredity and development are different biological phenomena. Moreover, they are explained in different ways. To explain heredity, we need to study the relationships between parents and offspring at conception. This is a task of *transmission genetics*. The effect of the transmitted genes during ontogenesis, however, is a task of *developmental genetics*. Since the birth of genetics and its integration into evolutionary theory, heredity has been decoupled from epigenetics. As Amundson notes, the separation of heredity and development was clearly stated by Morgan in his work *A Theory of the Gene*:

> Morgan eventually distinguished between two forms of genetics. One was transmission genetics, the Mendelian study. The other was developmental genetics, the study of the physiological action of genes in embryogenesis… Morgan derived two crucial points from this distinction. One was that *heredity is transmission genetics*. The second was that embryologists ought to turn their attention to developmental genetics (Amundson, 2005, 151, emphasis in the original).

The construction of a genetic theory of inheritance was a central point in the so-called black box of development. In evolution by natural selection, cumulative selection leads to adaptive complexity and speciation. This requires a solid link between what is inherited and what is selected. In the context of genetic inheritance theory, this link concerns the connection between genotypes and phenotypes. Consequently, we do not need to know how the Genotype-Phenotype map is traced. It is sufficient to use the genotypes as geolocators of the phenotypes: "[I]f a single allele can be regarded as the cause of pink eye color, then it is possible to causally explain adult

characteristics without any reference to the embryological processes that actually brought them about" (Amundson, 2005, 150). From this point of view, epigenesis becomes explanatorily vacuous for understanding evolution. The epigenetic theory of heredity was obsolete. The synthesis of this historical periplus is beautifully illustrated by Amundson:

> At a certain point in history, heredity-theorists stood at a semantic crossroads. Two parties to a theoretical dispute claimed the legitimate ownership of the term *heredity*. With the victory went the semantic spoils: *Heredity* now means what the winners of that theoretical debate took it to mean. The winners were geneticists, and heredity now means genetics. The losers in that debate were embryologists, who considered heredity to be a matter of embryological development (Amundson, 2005, 139-140).

Amundson illustrated how heredity was uncoupled from development and married to genetics. The next argument is the role of population genetics in understanding heredity. This historical phase is well analyzed in Evelyn Fox Keller's book *The Mirage of a Space between Nature and Nurture*. Subsequently, a new concept was introduced with the construction of population genetics: *heritability*. While heredity refers to the relationship between parents and offspring (a relationship between individuals), heritability "refers not to the quality of being inherited from parent to offspring, but to a *statistical quantity* associated with the ratio of genetic variation to phenotypic variation within a specified *population of organisms*" (Keller, 2010, 57, emphasis added).

In this quote, we find two core components of the conceptual shifts around heredity. First, inheritance has been delineated from the mechanisms of similarity (a.k.a. development): "Bluntly put, technical heritability neither depends on nor implies anything about, the mechanisms of transmission (inheritance) from parent to offspring [...] on the question of the mechanism of transmission, measures of heritability are simply silent" (Keller, 2010, 59, 68). The second point that Keller's analysis adds is that, unlike the traditional notion of heredity, heritability refers to a populational phenomenon; the technical term refers to the amount of inherited variation that is transmitted between members of a population. This is closely related – theoretically and historically - to the rise of population genetics as a statistical science. The study of inheritance in population biology simply calculates the persistence of variation between generations and explains it in terms of changes in gene pools. To understand heredity in evolution, we need to look at populations of genes. This means that firstly we need to understand heredity in genetic terms and secondly we need to construct a populational conception of heredity to deal with evolutionary processes - heritability.

1.2 The Baldwin Effect

The core of the Baldwin Effect is that learned and acquired traits are relevant for evolution. Organisms can improve their living conditions by producing new adaptive behaviors. These variations affect populational dynamics by changing the fitness values of the organisms. These variations can potentially be passed on to further generations and influence the evolutionary process. The Baldwin Effect has also been interpreted in different ways. Here too, the logic underlying the reinterpretation consists of a two-stage process: firstly, an organismic property – learning, adaptability, accommodation, plasticity - is reduced to the genetic level, and secondly, it is understood that the alleged organismic ability is ultimately an ability of an evolved population of genes. Organizational properties are subject to population biology.

The term "Baldwin Effect" was coined by Simpson (1955), who is in fact an MS's biologist, while at the same time de-emphasizing its relevance to evolutionary theory. In short, Simpson (1955) argued that if the Baldwin Effect is relevant in evolution, phenotypic variation already exists at the genetic level, or some kind of neo-Lamarckism must be defended. If one rejects the latter, as has been the case since the emergence of the MS and its genetic theory of inheritance, the Baldwin Effect appears to be genetically explainable without the need to add an ontogenetic cause to the theory of evolution: "It does not, however, seem to require any modification of

the opinion that the directive force in adaptation, by the Baldwin effect or in any other particular way, is natural selection" (Simpson, 1955, 116).

The first point of this strategy is to see behavioral plasticity as a consequence of genetic underpinnings. "If learned behaviors do become genetically underwritten, a population will be swapping 'short term and more plastic [learned behaviors] for long term, but more rigid adaptations,' (Simpson 1955, 116) thus subverting the very point of the Baldwin Effect" (Depew, 2003, 15). Instead of viewing ontogeny as a context-dependent process in which the organism's responses produce new phenotypic variations, as Baldwin originally intended, the explanatory burden is shifted to the genetic level. The second point is that natural selection acts on genes that produce plastic traits. Rather than assuming that developing organisms produce adaptive changes without a prior selection process specifying what outcome must be achieved, natural selection produces genotypes that can adapt to environmental conditions. In this sense, the evolutionary history of populations provides an adequate explanation for phenotypic plasticity without relying on developmental processes: Evolved genes are plastic, and then so are the phenotypic outcomes, not the other way around.

Simpson's original view of the Baldwin Effect was then incorporated into MS's thinking. Learned and plastic behavior is always within the scope of what natural selection has previously done to the genes. Here, too, the problem is that developmental processes are avoided in the study of evolution:
"In large part, that is because ontogenetic processes were off their screen; what Dobzhansky and Mayr [central architects of the MS] saw was only natural selection operating on adult phenotypes in populations" (Depew, 2003, 19).

The MS interpretation of the Baldwin Effect differs from Baldwin's insight. First, Baldwin was concerned with the individual development of behavior and its evolutionary effects. Furthermore, the Baldwin Effect is about the plasticity of behavior as a result of organisms confronting different environmental scenarios, whereas the MS considers it a phenotypic outcome that is genetically underpinned. These two points meant that the Baldwin Effect could be treated using the classical method of quantitative genetics so that Baldwin's controversial ideas about organismal regulation of development could be perfectly reinterpreted and anchored in the mainstream of evolutionary theory.

1.3 Stabilizing Selection

Let us now turn to a related but different concept from the Baldwin Effect: Schamalhausen's *stabilizing selection.* Schamallhausen's case is even more interesting because it involves a terminological ambiguity. Part of the acceptance of Schamalhausen's proposal by MS biologists is based on this ambiguity (Gottlieb, 1992, 126-136).

The core of stabilizing selection is not so far removed from the Baldwin Effect and certainly corresponds quite closely to Waddington's genetic assimilation. He attributes adaptability to organisms and claims that this is a factor in evolutionary theory. The phenotypic responses of organisms to environmental conditions can be stabilized in future generations by being controlled not by the environment but by the developmental system itself. Prior to genetic or environmental perturbations, organisms are able to stabilize the phenotypic outcomes produced. Phenotypic variants that are stabilized by ontogenetic processes can be inherited through epigenetic processes and further channeled through the germline. Stabilizing selection is therefore an ontogenetic process; it is a process of organismic ontogenetic stabilization that alters the selection pressure of the organism itself.

Interestingly, stabilizing selection, in contrast to genetic assimilation by Waddington, is described as part of the MS. What is the reason for this? In part, it is because of how the stabilizing selection was interpreted by the MS. There are two ways in which stabilizing selection was integrated into MS's thinking.

Dobzhansky, Schmalhausen's Russian colleague, accepted the idea of stabilizing selection. However, he goes beyond Schmalhaussen's interpretation by adopting the same strategy that we saw earlier in relation to heredity and the Baldwin Effect: He argues that stabilizing selection is caused by prior genetic selection; stabilizing selection was seen as a genetic, rather than an organismic, phenomenon. "Dobzhansky believed that this evidence of so-called 'genetic assimilation'" [and by extension, stabilizing selection] was really due to the selection of preexisting variants from the original population" (Gilbert, 1994, 145). To be accepted into the MS, it cannot be taken as an ontogenetic stabilization: according to the MS, "phenotypes produced by the environment are erroneously seen as non-genetic and thus have no place in the MS" (Gottlieb, 1992, 133). Rather than organisms adaptively stabilizing environmental pressures, it must be seen as genes already responding to different environments thanks to previous selection processes. Schmalhausen's idea "represents merely a degeneration of a part of an original adaptation" (Williams, 1966, 80).

The second reason to accept stabilizing selection within the MS concerns a terminological ambiguity: as Gottlieb (1992) explains, Schmalhausen used stabilizing selection to refer to two different biological phenomena. One is ontogenetic, which refers to the ability of organisms to influence selection processes by reacting to environmental conditions, the results of which can ultimately be internalized and passed on to future generations. However, Schmalhausen also used the term stabilizing selection to refer to the populational processes in which extreme variations are weeded out in order to stabilize the sample of possible trait types. According to the populational reading, "the stabilizing form of natural selection [understood in populational terms]… eliminates the most extreme forms of variation and builds up the mean or average form by selecting against the extremes at both ends of the distribution" (Gottlieb, 1992, 133). In this sense, the MS strategy adopted the populational interpretation of Schmalhausen, and the ontogenetic reading of stabilized selection was forgotten. Amundson elegantly summarizes this transition:

> The final irony came with the Synthesis reinterpretation of Schmalhausen's term for genetic assimilation, stabilizing selection. The meaning of this expression has completely changed within mainstream evolution discussions from Schmalhausen's intention. It now applies to selection for the mean in a population, as opposed to directional selection for extremes of a trait. This was not Schmalhausen's meaning. He had intended that *ontogenetic processes* were stabilized, and so buffered against either genetic or environmental perturbation. The Synthesized version of the expression removes all reference to ontogeny, and replaces it with a population–genetic definition of selection for the average phenotype (Amundson, 2005, 195-196, emphasis in the original).

1.4 The Norm of Reaction

The Norm of Reaction (NoR) refers to the ability of an organism to produce a repertoire of developmental outcomes with the same genetic toolkit in different environments. The NoR is defined by the interactions between genotype and environment (GxE). The genetic variable is fixed and phenotypic outcomes are analyzed in different environments. Importantly, although NoR plays a central role in the study of phenotypic plasticity, NoR is not associated with plastic responses (Schlichting and Pigliucci, 1999). While some phenotypic outcomes change under different environmental conditions, in other cases the outcome remains robust under changing environmental conditions. Both robustness and plasticity are a consequence of GxE interaction, in which the organism self-regulates its ontogeny and the outcomes it achieves (Bateson and Gluckman, 2011).

The term NoR was introduced by Woltereck in 1909. The English version is a translation of the original German term: *Reacktionsnorm*. Originally, the term referred to what most developmental biologists now understand by NoR: a property of a developing organism. Woltereck introduced the term to analyze the relationship between

genotype and environment. He proposed different *phenotypic curves* that are expressed under different environmental conditions. As you can imagine, the term refers to the characteristics of an organism's traits. We will see in Section 2 that this "…century-old norm of reaction concept… provides the essential data for eco-devo investigations" (Sultan, 2015: 21); i.e. that the original idea is what some areas of developmental biology today purport to introduce into contemporary evolutionary theory.

As Sarkar analyses, geneticists ignored the work that dealt with NoR, and those who referred to studies of GxE interactions did not use the term norm of response. During the first half of the 20th century, "[in] the West (that is, the US and Europe outside what became the Soviet Union)… [t]he norm of reaction (NoR) remained a relatively unknown concept during this period" (Sarkar 1999, 273). In the Soviet Union, however, the picture was different, and NoR was studied and viewed as an ontogenetic process from the beginning.

What is fascinating about this case is that the transfer of NoR from the individual to the populational level is linked to Dobzhansky's geographical (and socio-political) movement from the Soviet Union to the West (USA), i.e. the milestone of the MS. "The picture changes again when the Norm of Reaction was considered as hereditary units, as a property of genetic pools. The adaptive capacity of organisms was re-arranged as an adaptive capacity of hereditary units, thus GxE maps are understood in populational terms. *The norm of reaction became a property of genetic populations"* (Sarkar, 1999, emphasis added). Dobzhansky (1955, 3, emphasis added) makes his view explicit. First "…living *populations* occupy different adaptive peaks in the field of gene combinations. An array of related genotypes consonant with the demands of the environment is the adaptive norm of a population." Instead of considering possible adaptive peaks that an individual organism can achieve, the adaptive norm is considered as possible adaptive outcomes that a population can achieve. Consequently, adaptive norms are achieved and explained by population genetics; it is a product of natural selection not of natural development: "The adaptive norm of a *Mendelian population is a product of its evolutionary history*. Understanding the genetic architecture and the *origin of the adaptive norm* is a basic *problem of population genetics*" (Dobzhansky 1955: 3, emphasis added).

The path is quite similar and related to the previously presented understanding of plasticity. By considering NoR as heritable units and heritable units reduced to the germline, NoR is understood in genetic terms; not as the ability of organisms with the same genetic basis to develop different traits in different environments, but as a collection of possible outcomes that are genetically inherited. This move meant the possibility of translating GxE interactions to the populational level by understanding them as the product of evolutionary processes acting on gene pools. As Sarkar (1999) notes, the mechanism underlying an organism's ability to develop adaptively in a different context was removed and instead, NoR was understood as natural selection selecting for genes that could produce plastic traits.

To summarise this section, I have so far looked at four concepts that have undergone a similar transition. Originally they referred to ontogenetic processes, but as soon as they were reduced to the genetic level, they were explained in terms of populations. The developing organisms moved from the center of biological theory to the periphery. The explanatory role of developing organisms was overshadowed by the populational/genetic theory of evolution. Part of this theoretical construction involved the reinterpretation of biological phenomena to emphasize the explanatory role of organismal development. Amundson once again summarizes these ideas very aptly:

> The Synthesized version of the expression removes all reference to ontogeny, and replaces it with a population–genetic definition of selection for the average phenotype. Semantic modifications such as these are *specific examples* of the black boxing of embryology within the Synthesis. The black box is constructed out of population-level reinterpretations of concepts that were intended to refer to ontogenetic processes (Amundson, 2005, 195).

2. Contemporary Debates with a Historical Flavour

The black box of development meant that developmental processes are causally inert in evolutionary explanations. If a developmental process is responsible for producing new phenotypic variants, altering the fitness of an organism by plastic means, or establishing similarity between generations, then we must ultimately reduce these cases to population genetics. It seems that organismic phenomena are the relevant explanatory variable, but in reality, genes are the real explanatory unit. That was the MS strategy to dispense development from evolutionary theory. What is significant beyond this historical analysis is the fact that similar arguments are being waged in contemporary theoretical biology. As is well known, various disciplines primarily concerned with understanding developmental and physiological processes have questioned the foundations of the MS. Some arguments are empirical in nature -e.g. the gene-centered model of the MS cannot properly explain certain biological phenomena and therefore we need to look for epigenetic motifs. Other arguments are theoretical: certain tenets of mainstream evolutionary systems deserve to be revised, e.g. the central dogma of molecular biology. We will see how old debates analyzed in the previous section are revived in contemporary theoretical biology. In particular, the phenomena analyzed in Section 1 are of central importance to the critics of the MS. Old controversial phenomena have been revived thanks to new empirical advances. What is even more interesting, however, is the fact that today's defenders of the MS use the same strategy we saw in Section 1 to keep its foundations intact. The challenges of the MS have not found solid support in the life sciences at all, as critics have merely constructed “academic” (Gupta et al., 2017) or “career niches [... that] hinder progress in the field” (Welch, 2017, 265, 265). My aim, however, is not to enter the debate about whether the MS should be intact, revised, extended, or grossly modified. Rather, I want to point out the parallels between the historical analysis in Section 1 and contemporary debates about the foundations of evolutionary theory. To do so, I will show how the concepts presented in Section 1 are debated today and how the defenders of the MS manage to keep the black box closed.

2.1 Extended Inheritance and Replicator Biology

Eva Jablonka and Marion Lamb (2005, 2020) represent the milestone of extended inheritance systems. Among other things, they have worked to discover the many channels through which information flows between generations At the same time, the discovery of non-genetic forms of inheritance has been accompanied by various theoretical reflections on the nature of inheritance and its implications for evolutionary theory (see Gissis and Jablonka (2011) for a variety of topics around extended inheritance). Extended inheritance thus is retrieving the rejection of alternative, non-Mendelian theories of inheritance that were rejected from classical evolutionary theory (see Bonduriansky (2012) for different textbook examples that support the hard and mainstream view of inheritance).

Contrary to Weismannism, the central empirical fact is that inherited information is not transmitted exclusively via the germline. Other, non-genetic mechanisms are systematically involved in the similarity of traits across generations. Epigenetic, ecological, cultural, or symbolic systems also play a role in the maintenance of traits over time. While some forms of inheritance are only found in some species - e.g. symbolic inheritance - other forms are demonstrably ubiquitous: epigenetic systems of inheritance are present in all living systems. As Jablonka and Lamb (2020) clarify, the term "epigenetic" is used in the narrow sense to refer to the processes of protein expression in cell development as introduced by Nanney in 1958. In this narrow sense, epigenetics refers to the processes of cell formation, division, reproduction, and death. This narrow meaning differs from the broader meaning of the term “epigenetics", which encompasses any process related to the assignment of genes to phenotypes, as used for example by Waddington (see Stotz and Griffiths (2016) for a detailed conceptualization of epigenetics).

The theoretical implications of extended inheritance are manifold, but I will highlight only two here. First, the idea that the only explanatory role of heredity is to perpetuate the stability and reliability of traits across generations is a misconception. This is only one of its explanatory functions. The other role evidenced by extended systems is that heredity also is relevant in short-term evolutionary processes. As with genetic assimilation (see next section) or niche construction theory, developmental processes introduce new variants that require extended modes of inheritance to be maintained in future generations. In this case, extended systems might not be as reliable and stable as the genetic system, but still play a role in evolutionary processes. While genetic inheritance is suitable for explaining the maintenance of a trait over long periods, extended inheritance is suitable for maintaining developmental variants over short periods.

The second and philosophically deeper theoretical consideration concerns the nature of the theory of inheritance itself. In particular, following Jablonka and Lamb, we need to go beyond the replicator view of inheritance coined by the MS. The metaphor of the replicator is famously attributed to Richard Dawkins (1976), but the idea has been present since the early days of the MS (Ågreen, 2021) -for example, in Morgan's chromosome theory of inheritance (Walsh, 2015). The idea of genes as replicators is at the heart of the separation of inheritance and development. Replicators ensure that the same trait reappears in future generations *without us delving into the details of the mechanisms of similarity*. Replicator units of inheritance ensure that what is inherited constructs the phenotypes that have been selected, without explaining how this cross-generational similarity comes about. To overcome this view, extended systems advocate the old epigenetic view of inheritance ("epigenetic" now is used in its broad sense, as in Section 1.1). The goal of extended inheritance theory is not only to recognize multiple inheritance systems but also to (re)link developmental processes to inheritance systems. Extended inheritance theory, in contrast to the MS replicator view, is dedicated to understanding how inheritance systems are involved throughout development to produce cross-generational resemblance.

How does the MS deal with extended inheritance? There are several approaches. A first criticism is that extended inheritance is not a serious challenge for the MS, as extended systems only occur in some species. However, we have already seen that Jablonka and Lamb emphasize that epigenetic inheritance "has been found in all organisms in which it has been sought" (Jablonka and Lamb, 2020, 23). Another possible challenge is to deny the relevance of extended inheritance in evolution since the extended modes are not as stable and reliable as the genetic modes of inheritance. However, as we have seen, this criticism arises when we do not "recognize the evolutionary relevance of short-term environmental and epigenetic factors" (Sultan, 2017, 6). But even if we accept the evolutionary relevance and ubiquity of extended inheritance, there is a common way to integrate it into the field of the MS: namely, to assume some kind of extended replicator.

The strategy is to argue that extended inheritance can be adopted by the MS by embarrassing some kind of extended replicator: We need not link development to inheritance, but link inherited units (genetic or not) to developmental outcomes without bothering with developmental details. In principle, non-radical and nuanced versions of extended inheritance can accept a form of epigenetic (in the narrow sense) inheritance by incorporating non-coding regions of genomes and intracellular inheritance at conception. A clear case for this strategy is the Extended Replicator defended by Sterenly et al. (1996). As the name implies, this is an extension of replicator biology beyond DNA: replicator units are DNA plus something else. Nicholas Shea (2007, 2013) also assumes extended replication in his interesting proposal. His idea that inherited information represents instructions that can be read by the developmental system allows him to treat semantically the link between inherited units and phenotypic outcomes. Even some proponents of developmental systems theory (e.g. Griffiths and Gray, 1994) still invoke replication at the level of the whole developmental system, so they do not substantially alter the logic proposed by Dawkins, as Griesemer (2000) has correctly argued.

The relevant consequence of this position is that the assumption of a replicator position still maintains the demarcation between development and inheritance. As Shea noted, his (replicator) theory accepts non-genetic forms of inheritance but it "*does not depend at all on how the idiosyncratic complexities of development unfold.*

It requires *correlations between genotypes and phenotypes at the time of selection*, and can then treat *development as a black box*" (Shea, 2013, 476, emphasis added). The ability to preserve the replicator view is central to the defense of a populational approach to inheritance: we only need to know the frequency of variation of traits in a population; it does not matter whether they develop by genetic or non-genetic means; the way traits develop is not relevant to evolutionary theory. This is why today's defenders of the MS – who maintain a replicator view of inheritance - do not see extended inheritance as a real challenge: "The recent deployment of the concept of inclusive heritability in the context of an expanded and slightly modified *quantitative genetics* framework for analyzing evolutionary change" (Gupta et al., 2017, 495, emphasis added). In this picture, Dickins and Barton (2013) argue that epigenetic mechanisms are under genotypic control determined by prior selection processes; evolutionary questions cannot be answered by postulating non-genetic inheritance, they "only be answered in terms of the natural selection of genes, with everything ontogenetic treated as solely a proximately causal process" (Mesoudi et al. 2011, 4). In sum, "most neo-Darwinists would claim that the ability to adaptively switch epigenetic state is a property of the DNA sequence [...] and that any increase of adaptedness in the system has come about by a process of natural selection" (Haig, 2007, 423), there is "no challenge to the explanatory and conceptual resources of the MS, which are sufficient" (Dickins and Rahman, 2012, 2913).

However, these positions do not capture the revolutionary side of extended inheritance. From a historical perspective, we can say that the cellular theory of inheritance originally adopted by the MS is a kind of epigenetic theory. The Wiessmann barrier refers to cellular inheritance, while the molecularized version developed by Crick and Watson refers to molecular inheritance. Cellular inheritance was not considered extended inheritance at the time, even though chromosomes extend beyond DNA. This is a relevant difference (Noble 2021), but in both cases a replicator view was embarrassed. The fact that molecular or cellular inheritance can be understood in a replicator framework proves that the real challenge for the MS is not the plurality of inheritance systems, but the change of focus from a replicator to an epigenetic theory of inheritance. To summarise, not only experimental progress but also theoretical considerations are needed here. If we want to avoid these attitudes, it is important to emphasize that extended inheritance requires a new concept of inheritance that views inheritance as part of a theory of development (Jablonka, 2007): "[w]e need to return to an earlier, development- and organism-oriented view" (Jablonka and Lamb, 2020, 1) where "reproduction, inheritance, and development [were] all wrapped in one" (Gilbert, 2011, 121). This is the epigenetic conception of inheritance; this is in fact the revolutionary side of the novel concepts of inheritance.

2.2 Phenotypic Accommodation and Plastic Genes

Mary Jane West-Eberhard (2003) reintroduced the Baldwin Effect into modern evolutionary theory and linked it to Schamalhausen's stabilizing selection and, above all, to Waddington's genetic assimilation. The role of phenotypic plasticity in evolution, which West-Eberhard and others considered, gave rise to various discussions in contemporary biology. Unsurprisingly, the same picture emerges here. The old disputes about the role of plasticity in evolution can be found in the same place: one group focuses on ontogenetic explanations, and the other group postulates population-related explanations. The strategy of ousting the ideas of Baldwin and Schamalhaussen from evolutionary theory is now being used to treat phenotypic plasticity as an unproblematic phenomenon for the foundations of the MS.

The rise of phenotypic plasticity in recent evolutionary theory, now acknowledged as "a ubiquitous, and probably primal phenomenon of life" (Wagner, 2013, 216), is linked to a better understanding of developmental mechanisms. By overcoming the central dogma of molecular biology, development is seen as a context-dependent and contingent process rather than a rigid and determined process. Development is much more complex than the unfolding of DNA and we need to understand it seriously (Robert 2004) to avoid problematic simplifications. On the way to constructing phenotypes, developmental systems manage to adapt their trajectories and their outcomes to their (internal and external) life circumstances. This includes the ability to develop plastic phenotypes by plastic means, i.e. the ability to produce different (plastic) ontogenetic outcomes

and to find alternative (plastic) ways to produce phenotypic traits. The developing systems regulate their multiple developmental resources to produce a phenotypic outcome. In this context, plasticity refers to the way that the system produces context-dependent outputs by epigenetic means, insofar as "the central elements underlying many forms of plasticity are epigenetic processes" (Bateson and Gluckman, 2011, 43). That is why, West-Eberhard (2003, 33, emphasis added) defines plasticity as "the ability of an *organism* to react to an environmental input with a change in form, state, movement, or rate of activity".

West-Eberhard illustrates the importance of plasticity by proposing an evolutionary theory based on phenotypic and genotypic *accommodation*. In particular, she concludes that phenotypic evolution can dispense with genotypic change, that phenotypes are the leaders in evolution, while genes are followers, that is, the gene follows what the developing organisms do, rather than dictating to the developing organisms what they should do. In short, the core idea is that plasticity is a way to introduce phenotypic variants epigenetically. These variants can be epigenetically inherited and genetically assimilated (i.e. the developing system can find a way to use its genetic template to produce the new variant). Once assimilated, it becomes robust and potentially stably spread across the species. By showing that these cases are possible, she argues that genetic change is not the precursor to evolution, which contradicts the MS principle that evolutionarily relevant variation must be randomly genetically induced and genetically inherited (see for discussion Pigluicci et.al (2006)).

In contrast, MS's proponents argue that plasticity is not a major problem for the MS. Like Simpson, we currently find the same strategy to deal with plasticity within the MS rationale. In particular, plasticity is seen as coherent with mainstream evolutionary theory insofar as it is seen as the result of past selection processes. As Futuyma (2017, 6, emphasis added) stated, "genes are 'followers' only to the extent that genetic assimilation or accommodation 'fine-tunes' an adaptation that *had already evolved by selection and genetic variation*." The populational interpretation continues to be defended. Gupta et al. (2017: 495, emphasis added) also see no innovation in the current invocation of plasticity: "Almost from its inception, *quantitative genetics* has been concerned with what is now called […] phenotypic plasticity […] the *quantitative genetic* notion of genotype × environment interaction reflects an appreciation that there may be genetic variation for the degree and nature of phenotypic plasticity in a population." As this quote reflects, Baldwin's ideas are being incorporated into classical population genetics at the expense of viewing behavioral plasticity as a product of natural selection acting on genetic populations.

In conclusion, it is argued that the MS's analysis of variation in adaptive evolution need not to be changed; genetic selection is still the only adaptive force in evolution. Following Charlesworth et al. (2017, 1, 10), insofar as "allele frequency change caused by natural selection is the only credible process underlying the evolution of adaptive organismal traits [...] no radical revision of our [i.e. neo-Darwinian] understanding of the mechanism of adaptive evolution is needed." Once again, we have a split in interpretation as a result of a split in explanatory strategies. While contemporary defenders of the MS rely on the ability to treat phenotypic plasticity at the evolutionary level, viewing plasticity as a "quantitative trait" (de Jong, 2005, 101), those who emphasize the importance of organisms in evolution treat it as a product of individual causation and view plasticity as "an intrinsic property of organisms" (Sultan, 2021, 6).

2.3 Eco-Devo NoR and Populational NoR

NoR is of central importance in current approaches to phenotypic plasticity, especially in the new field of ecological developmental biology (Eco-Devo), where NoR plays a central explanatory and theoretical role (Gilbert and Epel, 2015; Sultan, 2015). The scene on NoR in contemporary biology has the same script. On the one hand, eco-devoists defend that the NoR is an individual-level phenomenon; it is not possible to reduce it to the genetic level and include it in population genetics. Eco-Devo NoR points to the causal abilities of organisms to develop differently in different environmental scenarios. As Sultan (2019, 113) points out,

> Recent studies of environmental effects and epigenetic modifications both across and within generations have provided stunning insights to the complexities of developmental causation. These insights make clear why the norm of reaction cannot be viewed as a genetically determined set of rules for development in specific environments. Instead [...] a genotype's realized norm of reaction takes shape actively, modulated by several interacting layers of environmental and epigenetic effects.

However, on the other hand, as Sultan acknowledges, we can also find several population interpretations of the NoR nowadays. Opponents of the eco-devo view of the NoR explain that "[a]bundant traditional theory, *based in population genetics*, describes how reaction norms evolve by selection on genetic variation, and there is abundant evidence of adaptation by natural selection on standing genetic variation" (Futuyma, 2017, 6, emphasis added).

Again, the rejection of the eco-devo view of NoR takes place in two steps. First, NoR is reduced to genetic space: "Evolutionary biologists have reconciled their awareness of genotype-environment interaction with a genetically determinist model for development by defining the norm of the reaction itself as a self-contained 'property of the genotype' —an 'environmental response program in the genes'" (Sultan, 2019, 112). Then, as a consequence of reducing NoR to the genetic level, it became subsumed to population genetics: "When the norm of reaction is viewed as a 'property of the genotype,' the organism's developmental plasticity becomes simply an 'extended phenotype' emanating from the genes, and the evolution of these response patterns can be understood in standard *population-genetic terms*" (Sultan 2019, 112, emphasis added). As the prominent evolutionary biology and contemporary defender of the MS, Douglas Futuyma, express, "as a graduate student, I learned that genotype-environment interaction was a staple in quantitative genetics" (Futuyma, 2017, 5). In this way, following Svensson's (2018, 8) extended use of quantitative genetics to defend the MS, "evolutionary quantitative genetics can be used to model reaction norm evolution, canalization, and phenotypic plasticity." As a result, the NoR could be "'successfully integrated into the MS.' Instead of natural selection on alleles that affect a trait's fixed value, selection could be understood to act on the alleles underpinning the norm of reaction" (Sultan, 2019, 112).

Let us summarize the picture. In Section 1, I presented four central phenomena at the individual level that were reinterpreted by the MS into populational concepts. Here I have seen how these concepts are now a contested area in the foundations of evolutionary theory. My analysis does not claim to be exhaustive. Surely other concepts have followed the same path in the history of biology. Also, there are other concepts that contemporary critics of the MS invoke, while defenders of the MS leave it at its foundations. My main point in this section is that the controversies lie in how these phenomena are interpreted and how they are to be explained. In this sense, the current situation is quite similar to the first half of the 20th century. In the next section, however, I would like to show that the interpretations of concepts are linked to explanatory methods. Scientific concepts are epistemic artifacts that are bound to their explanatory role. We will thus see how conceptual reinterpretations go hand in hand with an explanatory shift in the emergence of the MS.

3. Changing Forces or Changing Explanations?

There is an ongoing debate about the causal structure of natural selection (see Pence (2021) for a critical and up-to-date introduction to the topic). What are the causes of natural selection? What kind of explanation does natural selection provide? There are essentially two rival theories: the Causalist School and The Statisticalist School. I will present each school in the following Section 3.1. In Section 3.2 I will link this debate to the historical and contemporary analyses from Sections 1 and 2. Concerning the historical analysis, we will see how each school allows for a different interpretation of the conceptual changes made during the emergence of the MS. Following the Causalist School advocated by the MS, conceptual *changes correspond to changes in the causes of adaptive evolution*: the transfer of individual concepts to the populational level corresponds to the transfer of

causal powers from organisms to populations. However, following the statisticalist stance, I will argue that the conceptual changes during the emergence of the MS should not be understood as changes in the causes of evolution. Rather, the *conceptual changes in the history of biology resulted from a change in the method of explaining by the populational thinking of the MS.* The transformation of individual-level concepts into population-level concepts did not mean a shift in causes, but a shift in explanations. Finally, in terms of analyzing today's debate in evolutionary theory, each school seems to support different sides of the debate. While the Causalist School lends itself to the neglect of any explanatory role (causal or otherwise) for developing systems, the Statisticalist School is aligned with the call to include development as a central (causal) phenomenon in evolutionary theory (Rama, 2022, 2023). Moreover, the idea that population-level explanations are statistical fits with the statistical method of population biology and the kind of explanation advocated by the MS to reject development as part of evolutionary theory. This suggests a possible explanatory pluralism that will be outlined by the end of the paper, defining research questions for future work.

3.1 The Causalist School and the Statisticalist School

The Causalist School assumes populational forces produce evolutionary change (see for contemporary defenders of this position Abrams (2012), Millstein (2006), Ramsey (2016)). It understands natural selection as a force that acts at the level of populations and causes them to adapt, speciate, and die out. The primary source of evolutionary causality is Mayr's dichotomy between proximate and ultimate causes (Mayr, 1961, 1974). Evolutionary biology is dedicated to understanding the ultimate causes of evolution, relying primarily on the unique adaptive force: natural selection. As a populational and historical phenomenon, natural selection is responsible for the adaptation and diversity of living things. However, Elliot Sober is probably the main proponent of the causalist stance. Accordingly,

> …the population is an entity, subject to its own *forces*, and obeying its own laws. The details concerning the *individuals* who are parts of this whole are *pretty much irrelevant* [...] In this important sense, *population thinking involves ignoring individuals...* (Sober, 1980, 175, emphases added).

The crucial ingredient Darwin adds to the understanding of evolution is that evolution requires historical and populational explanations. We need to look at how populations change throughout history due to selection processes. Adaptations are defined at the populational level as processes arising from heritable variations in trait types with different fitness values. According to the causalist school, the population-based explanations for natural selection that Darwin introduced are causal. Population-related forces cause adaptations. A central claim of the causalist position is clearly expressed in Sober's quote: it is not necessary to deal with individuals in order to understand evolution. Organisms and their development can be completely ignored. The explanatory void of organismic development in evolution was presented as a black box: we only need to know how heritable genetic differences lead to different selection pressures on phenotypes. The link between genes and phenotypes - i.e. development - can be seen as a black box.

The Statistical School was a more recent alternative to the causalist picture (see Walsh et al. (2002), Matthen and Ariew (2002), and Walsh (2003) for seminal work). The statisticalist position is justified by two analyses. First, the explanatory methods of population biology are examined to see what kind of explanation is involved. The conclusion refers to the statistical foundations of population biology, forged during the first decades of the twenty century. According to this view, the population-based explanations for natural selection that Darwin introduced are statistical. Secondly, the statistical viewpoint is characterized by the idea that ontogenetic processes are the actual causal basis of evolution. There are no such things as populational causes in natural selection. All evolutionary causes lie at the individual level.

The analysis of the first point revolves around the concept of fitness. This concept is the key to explaining adaptive evolution. Accordingly, adaptive speciation, diversification, and extinction are due to fitness differences. Fitter traits that are maintained through inheritance systems lead to adaptations across generations. The Statisticalist School departs from the distinction between two notions of fitness: *trait fitness* and *individual fitness*. According to the Statisticalist School, natural selection is explained by the fitness of traits. Trait fitness is a populational concept. It refers to the fitness that a population has because its individuals possess a certain trait. It is a property of a trait type, not a property of tokens. In contrast, individual fitness refers to the fitness value of each individual. It is a property of tokens, not of types. The key difference between them is that, while "[t]rait fitness is the average survivability of a group of individuals possessing a type of trait" (Ariew, 2003, 562), individual fitness concerns those causal processes that produce the persistence and reproduction of an individual. This is at the core of statisticalism: trait fitness is statistically accessed, while individual fitness is causally accessed; trait fitness is a statistical property of populations, while individual fitness is a causal property of individuals (Ariew, 2003; Ariew and Lewontin, 2004).

This leads to the first statement: natural selection explanation describes the populational change by fitness differences, thus natural selection explanations are statistical. By describing how populations change due to fitness differences, natural selection explanations appeal to the notion of *trait* fitness. The way populations change due to their difference is not analyzed in terms of populational forces causing fitness differences but in terms of statistical differences between trait types. Once populational causes are removed, the statisticalist view concludes its second statement: "[t]here is one level of causation; all the causes of evolution are the causes of arrival and departure (the 'struggle for life') [...] It is 'proximate' causes all the way down" (Walsh, 2019, 238, 242). The only level of causation in adaptive evolution is the individual level, i.e. the processes that take place in a single life span.

The relationship between the two statements can be better understood by considering the relationship between the two levels of analysis: the individual and the popualtional. Following Walsh (2007, 2019), population-related changes are analytical (i.e. mathematical) consequences of individual causation. Triat fitness is an analytical consequence of individual fitness: individuals differ in their individual fitness and this leads to differences in the populations to which the organisms belong. The effects of individual fitness on the population level are analyzed statistically. Evolution is explained as a statistical effect of processes at the individual level, as an effect of individual causes at a populational level. Walsh et al. summarize their view as follows:

> In short, natural selection occurs only when the relative frequency of trait types changes in a population as a consequence of differences in the *average* fitness of individuals in different trait-classes. This is what we call the statistical interpretation of natural selection (Walsh et al., 2002, 464).

3.2 Conceptual Change as an Explanatory Shift

The aim now is to approach the historical and contemporary analyses done in Sections 1 and 2 from the causalist vs statisticalist debate. Let's start with the historical analysis in order to see how conceptual changes should be understood according to each school. Let us recapitulate from Section 1. The conceptual changes mentioned above concerned two levels: the individual and the population level; a concept initially used to refer to an individual-level phenomenon is reinterpreted to refer to a populational phenomenon. As we have seen, this means that what was explained at the individual level must now be explained at the populational level. Population-based explanations replace individual-level explanations. If we take the causalist point of view, we can conclude that *the conceptual changes presented earlier correspond to a change in the causes of evolutionary processe*s. In other words: What was caused by individual-level processes has been reinterpreted by the MS to mean that the actual source of causation is the population. Where someone saw individual causes, the MS

succeeded in finding the cause in the population. The shift from individual to population causation was the point at which the conceptual changes converged.

As we can see, the historical analysis of the previous section is linked to the black box of development. The neglected explanatory role of development is justified in part by shifting causation between the individual and populational levels. The MS strategy of reframing problematic cases such as the Baldwin Effect, stabilizing selection, epigenetic inheritance, or plasticity allowed for a fully population-based view of evolutionary explanations; in other words, the dispensability of individual-level causes.

Nonetheless, if we look at the statisticalist position, we reach a different conclusion, i.e. a different way of looking at the conceptual changes discussed in Section 1. Previously, we have seen that the conceptual interpretation analyzed in Section 1 can be understood within the causalist framework as a transfer of causes from the individual level to the population level. However, this transfer of causes does not make sense according to the statistical reading, for the simple reason that there is no such thing as populational causes. The MS strategy of reducing the explanation of phenomena at the ontogenetic level to the population level is not about causality. Rather, it is about a new type of explanation that was developed in the course of the gestation of the MS. This type of explanation concerns the statistical methods of population biology. I will not go into detail here about how population biology emerged from the mathematical insights of Fisher, Wright, and Haldane's biometric school and how this meant a particular way of understanding natural selection that differed from Darwin's original idea (Godfrey-Smith, 2009; Walsh, 2015). If we follow the statistical interpretation, it is sufficient to realize that the conceptual changes mentioned above entail a shift in explanations: What was explained causally has been explained statistically. While heredity originally referred to a causal process, the relevant concept for an evolutionary explanation was a statistical concept: heritability. While Baldwin and Schamalhausseen pointed to the ability of organisms to causally modify their own fitness by plastic means, the MS pointed to the statistical property of a population to produce plastic phenotypes. While the NoR referred to the causal processes linking the genotype of an organism to phenotypes produced in a different environment, the NoR has been reinterpreted to refer to the average phenotypic variation of a population with the same genetic toolkit under different environments.

We have seen that each school proposes a different way of understanding the conceptual changes during the emergence of the MS. Now let us turn to the question of the impact of the statisticalist position on the disputes analyzed in Section 2. A first observation is that the causalist position is a convenient terrain for keeping the black box of development intact. In other words, just as we can understand conceptual changes in the history of biology as causal transfer, we can also see that this interpretation can take place in today's debate; that is, contemporary defenders of the MS can reject the explanatory role of developing organisms in evolution by embarrassing the causalist position.

Here, however, we will follow the statisticalist interpretation of natural selection. I will highlight three important implications of this view for contemporary debates about the foundations of evolutionary biology. First, the statisticalist position fits the explanatory method used by the MS proponents to refute the challenges posed to them. As we argue in Section 2, the strategy is to argue that a quantitative analysis of population change is sufficient to explain extended inheritance, plasticity, or the NoR (among other phenomena, e.g. niche construction). Remarkably, the explanatory method used is a quantitative analysis: the statistical methods of population genetics. When the MS applies these methods, it seems that we are facing an explanatory shift, not a causal transmission. Therefore, understanding population genetic explanations in statistical terms fits the current strategy of the MS.

Second, statisticalism also fits with the new trends in theoretical biology that call for the integration of development into evolutionary theory. As mentioned in Section 2, the MS proponents emphasize that many biological phenomena deserve a proximate explanation for causal processes during ontogeny. Such a causal

interpretation is indeed a central thesis of statisticalism presented in Section 3.1: all causes of evolution lie at the individual level. Statisticalism, therefore enables and accepts the challenges to the MS based on extended inheritance, phenotypic plasticity, and NoR.

The first and second implications lead us to the third: the possibility of a pluralistic explanatory framework. As we have seen, statisticalism accepts the use of quantitative methods while recognizing causal support for evolution at the individual level. This may be interpreted in a contradictory way: we cannot accept both sides of the dispute. However, there is also a possible parsimonious interpretation that consists in adopting an explanatory pluralism. This suggests that both explanatory methods -the causal and statistical - which allow for different levels of explanation -the individual level and the population level - can coexist. This explanatory pluralism is explicitly embarrassed by the advocates of statisticalism: "The result is that evolutionary explanations are indispensable even if one knows the complete causal story about how each individual in a population lived and died. In other words, evolutionary explanations are not reducible to individual-level causal explanations" (Ariew 2003: 561). The idea, then, is that we need both populational-statistical and individual-causal explanations to understand evolution.

This pluralistic framework deserves further explanation (but see Walsh (2019)). Pluralism is not achieved by saying that every method of explanation is valid. It also raises many unresolved and difficult questions about how both explanatory perspectives (the populational-statistical and the individual-causal) can be coherently integrated. If we argue that both sides of the debate involve valid explanatory methods, we must make clear how they coexist without tension. The most important question is: *Does the distinction between the two levels of explanation fit the different bases of explaining heredity and plasticity at the population and individual levels?* In particular, many questions about the biological phenomena discussed here deserve special treatment concerning the goal of an integrated biological theory. Can the population explanation of heritability also include non-genetic forms of inheritance? Are environmental inheritance systems inevitably in tension with gene-based heritability analysis? If "quantitative genetics become liberated from the tyranny of genetic details in classical population genetics" (Svensson, 2018, 8), is it possible to take a statistical and population-based view of plasticity without reducing plasticity to a gene-based phenomenon? Can plasticity -as an organismic phenomenon- be integrated into the population-based foundations of the MS without neglecting its central causal role in evolution? These and many other questions need to be answered in order to specify the explanatory scope of any biological approach: what can and cannot be explained by the MS, and what can and cannot be explained only from the perspective of the individual level.

Another important consideration is whether explanatory pluralism allows for *conceptual pluralism.* It seems that explanations of plasticity at the population level and the individual level *refer* to different phenomena. One is the degree of variability of a population in different environments, the other is the ability of an organism to develop according to its environmental conditions. Heritability as a statistical and population-related concept does not have the same *meaning* as the individual and causal concept of inheritance. While this thesis deserves to be explored in detail, conceptual pluralism may also be a central element of an integrated biological framework.

To summarize, first, statisticalism allows us to understand the conceptual changes in the history of biology as part of an explanatory shift: the gestation of statistical, population-based methods during the emergence of the MS. Second, statisticianism also seems to be fertile ground for today's discussion of the foundations of evolutionary theory. First, it recognizes the kinds of methods used in population biology as statistical. Second, it accepts and motivates the need to understand causal developmental processes in evolution. Third, it allows for a pluralistic framework in which each explanatory method plays a different explanatory role. The integration of both perspectives and how each should be modified to fit into the other requires further research.

4. Conclusions

In this paper we have conducted three different but related analyses. First, I introduced four concepts -heredity, the Baldwin Effect, stabilizing selection, and the reaction norm- that have undergone a very similar path in the history of biology. Initially, they all referred to ontogenetic processes but were then reduced to the genetic level and finally transferred to the population level. This has contributed to organisms being regarded as second-class citizens in evolutionary biology.

Second, we saw in Section 2 that the historical disputes analyzed in Section 1 are quite similar to those in evolutionary theory today. I have presented three areas of current debates that are connected to and inspired by past approaches. We have seen how the extended theory of inheritance aims to return to the old epigenetic theory of inheritance, how phenotypic plasticity is postulated as an organismic phenomenon, as Baldwin and Schamlahauseen did, and how the eco-devo account of NoR aims to reclaim its original explanatory role. In this way, the contemporary critique of the MS aims to return to old ideas in biology by taking development out of the black box. Alongside this parallel, however, we have also seen that there is a clear parallel between how individual-level concepts have been pushed out of evolutionary theory and how contemporary defenders of the MS deal with all the efforts to hold back development in evolutionary theory. Just as the epigenetic theory has been dismissed since the emergence of genetic inheritance theory, today there are variants of replicator theories that have been proposed to dispute the revolutionary ideas of extended inheritance theory. Following the founders of the MS, phenotypic plasticity is nowadays dismissed with the argument that it is the result of past genetic selection. Finally, NoR is interpreted in the same way as the MS, by taking a population genetic approach and avoiding interpretation at the individual level. While the analysis in this section deserves more detail and further discussion with the current literature, the main goal was to show the parallelism between past and current arguments in evolutionary theory.

Finally, I linked the analysis to another central issue in the philosophy of biology, namely the ongoing debate about the causal structure of natural selection. I have argued that the causalist position promotes a different interpretation of the issues discussed in Sections 1 and 2. In terms of historical analysis, the causalist view means that the reinterpretation of concepts involves a transfer of causality. What is supposedly caused by processes at the individual level is actually caused by processes at the population level. On the contrary, the statisticalist view suggests that the conceptual changes conceal a shift in explanations. What is explained in causal terms is now explained in statistical terms. With this in mind, the analysis of today's debates is also influenced by the debate between causalists and statisticalists. The causalist position seems appropriate in an evolutionary framework where developing organisms remain in a black box. However, if we seek to justify the explanatory role of developing organisms in evolution, statisticalism should be advocated. In this sense, statisticalism ascribes a central role to developmental processes: they are the causal basis of evolutionary theory.

Although I have not put forward an explicit and detailed defense of statisticalism, the Statisticalist School, as argued at the end of Section 3, has at least three advantages. First, understanding conceptual change as part of explanatory change fits the explanatory role of statistical explanation in population biology. The explanatory method used by the MS to understand plasticity, heredity, and the NoR is at the center of population biology and its statisticalist (and original) foundations. Second, statisticalism ascribes an important explanatory role to developmental processes. In this sense, it embraces the recent experimental and theoretical advances that have motivated a revision of the current evolutionary theory. A third advantage is that statisticalism also ascribes an indispensable and irreducible explanatory role to population biology and its statistical method. While the consequence of this point will be explored in future work, statisticalism emphasizes that a populational, historical, and statistical perspective is essential in evolutionary theory. This is certainly a plausible pluralistic framework to connect the MS with the new trends in evolutionary theory. In other words, the realization that different types of explanations are required in evolutionary theory can be linked to different evolutionary approaches. While this general goal is ambitious, we can see here how the recognition of different types of

explanations is linked to various open questions regarding the concepts discussed here that will be explored in future research.

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