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

Preprint · February 2024

Tiago Rama
Universidad de la República de Uruguay

The Historical Transformation of Individual Concepts into Populational Ones:

An Explanatory Shift in the Gestation of the Modern Synthesis

Tiago Rama[1]

Accepted Manuscript for *History and Philosophy of Life Science* (2024)

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, a similar situation can be found in contemporary evolutionary theory. While different approaches reflect on the causal role of developing organisms in evolution, proponents of the Modern Synthesis refrain from any substantial change by reinterpreting and explaining individual-level phenomena from a population perspective. Finally, I will approach these historical and contemporary debates by arguing for the statistical reading of natural selection, which holds that explanations by natural selection are statistical. My main conclusion is that the historical conceptual reinterpretations belong to a new explanatory strategy developed by the Modern Synthesis based on population thinking. Adopting the statistical point of view has three advantages for the issues discussed in this paper. First, understanding historical conceptual change as part of an explanatory shift fits with the emergence of population biology as a discipline that employs statistical methods. Second, concerning current debates in evolutionary biology, the statisticalist reading can validate the goal of both sides of the dispute. It ascribes an invaluable role to the population statistical explanation of the MS and also commends the study of developmental and organismal causes of adaptive evolution. Finally, the division of explanatory roles in evolutionary biology, embarrassed by statisticalism, can be related to the different interpretations that important biological concepts have undergone throughout history and contemporary biology, i.e., that the division of explanatory roles allows for a division of conceptual interpretations.

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

---

[1] Department of Philosophy, University of the Republic, Montevideo, Uruguay. Corresponding email: trama.folco@gmail.com

Table of Contents

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. The central idea 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 nowadays. While critics of the MS argue that individual-level phenomena are central to understanding evolution (usually referred to as the Extended Evolutionary Synthesis (EES)), 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, causalism and statisticalism. After introducing each theory, I will turn to evaluate its implication for the historical (Section 1) and contemporary (Section 2) analyses. As far as the historical debate is concerned, if we follow the causalist position, we conclude that the conceptual reinterpretations during the gestation of the MS should be understood as a transfer 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 reinterpretations are part of an *explanatory shift*. Phenomena caused by development are explained in *statistical terms* at the population level. The main conclusion of this article is that the reinterpretation of concepts referring to individual-level phenomena should not be understood as a transfer of evolutionary causes between levels (individual and population) but as the construction of a different explanatory strategy in population biology. Consequently, the current debates in evolutionary theory are also affected *if* we follow the statistical interpretation. We can appreciate that current supporters of the MS provide statistical explanations for the phenomena that should be causally explained. Furthermore, a central claim made by the statisticalist view is that we must divide the explanatory labor in evolutionary theory between population-statistical explanations and individual-causal explanations, and that, importantly, each type of explanation roughly corresponds to a different biological framework –the MS and its opponents. At the end of the paper, I will extend this idea by suggesting that the different semantic attributions of central concepts in evolutionary biology are related to the division of explanatory labor, i.e., that biological concepts are understood differently depending on which type of explanation we choose. To summarize, adopting the statisticalist position has three advantages:[2] First, it captures the historical transition from individual to population concepts as part of an explanatory shift in population biology. Second, the statistical interpretation of natural selection fits the populational, mathematical model of population genetics espoused by contemporary proponents of the MS. Finally, the statistical reading does not neglect the central explanatory role of population explanations. The division of explanatory labor –and its connection to the "division of semantic attributions" mentioned above– enables both explanatory approaches in evolutionary biology.

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. These conceptual transitions 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 a genetic 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, "inheritance 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

---

[2] I thank an anonymous reviewer for making this point explicitly and more clearly than in the first manuscript.

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 theory of inheritance, such as the emergence of Mendelian genetics and the construction of the Weismann barrier.

The resulting genetic theory of inheritance states that inherited material is transmitted at conception (Mameli, 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. As a result, heredity and development are different biological phenomena. 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*. 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 step regards the role of population genetics in understanding heredity. This historical phase is well analyzed in Evelyn Fox Kelle'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 (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. 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.

The term "Baldwin Effect" was coined by Simpson (1955), one of the architects of the MS, while at the same time de-emphasizing its relevance to evolutionary theory. In short, Simpson 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 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. In front of genetic or environmental perturbations, organisms can 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 went beyond Schmalhaussen's interpretation by adopting the same strategy that we saw earlier concerning heredity and the Baldwin Effect. He argued 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 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 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 necessarily 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 -as 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. 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 analyzes, geneticists ignored the work that dealt with NoR, and those who referred to studies of GxE interactions did not use the term *norm of reaction*. 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.[3] "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.

---

[3] Geographical factors are also an interesting aspect of historical analysis in evolutionary biology. As a detailed review by Depew (2011) shows, even among the founders of MS, there is a plurality of positions that correspond to the geographical settings and specific academic niches in which the MS originated. While the plurality of views on certain topics within the MS is relevant (as Depew has analyzed, Huxley's frameworks have several differences with Dobzhansky's view), the analysis conducted here does not rely on these differences.

To summarize 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. 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 (Rama, 2024b). We will see how old debates analyzed in the previous section are revived in contemporary biological theory. In particular, the phenomena analyzed in Section 1 are of central importance to the critics of the MS.[4] Old controversial phenomena have been revived thanks to new empirical advances.

---

[4] Other concepts also deserve a similar analysis, such as the concept of *niche construction*. Moreover, a specific concept that fits the analysis carried out here is that of epigenetics, i.e., the process that maps genes into phenotypes. We can perform a similar historical analysis –again following Amundson (2005, 223, 224)– by looking at Waddington's epigenotype and how Mayr "accepted" it by collapsing the epigenotype with the genotype, using the same strategy we analyzed in Section 1. Similarly, the discovery of genetic regulatory networks (GRNs) was also the subject of controversy and one of the first motivations to question central ideas of MS when we turn to today's debates. In this context, a first, still ongoing lecture on GRNs preserves the core of the Central Dogma, which views development as the expression of an evolved program (e.g., Waters, 2007; Shea, 2007; Rosenberg, 1998). However, GRN has been shown to be part of a more complex system in which non-genetic sources of information –nuclear, cytoplasmic, intercellular, or exogenous– are also crucial for DNA expression and cell fate (e.g., Griffiths and Stotz, 2013; Wagner, 2014). A detailed analysis of this case is beyond the scope of this article, but some of these ideas are included in the analysis of epigenetic inheritance (Jablonka and Lamb, 2008). An analysis of the various

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. My aim, however, is not to enter into the debate about whether the MS should be intact, revised, extended, or grossly modified. There are various ways of interpreting this scenario, from those who call for a reconfiguration of evolutionary theory to those who leave the building blocks of the MS enterprise intact. Even within the EES, there are different positions, and not everybody would accept all the thesis related to the EES. Rather, I want to show 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 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. As well, their discoveries have 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 numerous, 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 is also relevant in short-term evolutionary

---

interpretations of epigenetics in the history of biology would enrich the ideas presented here. I thank an anonymous reviewer for pointing this out.

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 they 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. Shea (2007, 2013) also assumes extended replication. His idea that inherited information represents instructions that can be read by the developmental system allows him to semantically treat 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 theory accepts non-genetic forms of inheritance but "*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; 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). Dickins and Barton (2013) also argue that epigenetic mechanisms are under genotypic control determined by prior selection processes. Evolutionary questions cannot be answered by postulating non-genetic inheritance; they are going to "be answered in terms of the natural selection of genes, with everything ontogenetic treated as solely a proximately causal process" (Mesoudi et al., 2013, 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). As a result, 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 in the early years of neo-Darwinism, is a kind of epigenetic theory. The Weismann 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 cells extend beyond DNA. This is an important difference (Noble, 2021). The central point is that 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; 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 the revolutionary side of the concept of extended 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 one. 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 an apt phenotypic outcome (Rama, 2024a). 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. 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 Pigliucci 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 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 [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; 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). 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. My main point in this section is that the controversy lies 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. What are the causes of natural selection? What kind of explanation does natural selection provide? There are essentially two rival theories, causalism and statisticalism. In Section 3.1 below, I will introduce each theory. In Section 3.2, I will link this debate to the historical and contemporary analyses from sections 1 and 2. I will point out three advantages of adopting the statisticalist position for our purposes. First, in Section 3.2.1, which deals with the historical analysis, we will see how each theory allows for a different interpretation of the conceptual changes that were made during the emergence of the MS. According to causalism, *conceptual changes* correspond to *changes in the causes of adaptive evolution*: the transfer of individual concepts to the population level corresponds to the transfer of causal forces from organisms to populations. Following the statisticians' point of view, however, 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 result from the change in explanatory method done by the population-based thinking of the MS*. The transformation from individual-level concepts to population-level concepts did not mean a shift in causes, but a shift in explanations. This interpretation fits with the emergence of population biology at the beginning of the twentieth century as a discipline that uses statistical methods to explain population dynamics. Second, in Section 3.2.2, when analyzing the debate in evolutionary theory today, each theory seems to support different sides of the debate. While causalism favors the neglect of any explanatory function (causal or otherwise) for developing systems, statisticalism is aligned with the call to include development as a central (causal) phenomenon in evolutionary theory (Rama, 2022, 2023). Adopting a statisticalist viewpoint enables us to assign a central explanatory role to each side of the debate. Statisticalism requires both the quantitative analysis of population biology developed by the MS and the need to view developmental and organismal processes as causal bases of adaptive evolution. Finally, in Section 3.2.3, I will argue that the division of explanatory roles between population-statistical and individual-causal explanations allows us to assume a division of semantic interpretation as well, i.e., the idea that terms can be interpreted differently depending on the type of explanation. The coexistence of different explanatory frameworks in evolutionary biology allows the coexistence of different semantic attributions to biological concepts.

3.1 Causalism and Statisticalism: An Overview

Causalism assumes populational forces produce evolutionary change (see for contemporary defenders of this position Abrams (2012), Millstein (2006), Ramsey (2016), Pence (2021)). It understands natural selection as a force that acts at the level of populations and causes them to adapt, speciate, and die out. 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, emphasis 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 causalism, 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 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.

Statisticalism is a more recent alternative to the causalist picture (see Walsh et al. (2002), Matthen and Ariew (2002), and Walsh (2003) for seminal works). 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 twentieth century. According to this view, the population-based explanations for natural selection that Darwin introduced come to be 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. Statisticalism departs from the distinction between two notions of fitness: *trait fitness* and *individual fitness*. 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; see Otsuka (2016) for a critical analysis of this distinction).

This leads to the first statement: natural selection explanations describe the population change by looking at trait fitness differences; thus, natural selection explanations are statistical. The way populations change due to their differences 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.

The relationship between the two statements can be better understood by looking at the relationship between the two levels of analysis. Following Walsh (2007, 2019), population-related changes are analytical (mathematical) consequences of individual causation. Trait fitness is an analytical consequence of individual fitness. Individuals differ in their fitness, and this leads to differences in the populations to which the organisms belong, which are assessed through the use of mathematical calculations. This defines the nature of explanation in population biology (Walsh et al. (2017)). Population biology is based on a set of theorems, equations, and calculi that can go beyond what happens to particular individuals and provide an explanation for the properties of the population (see Millistein (2006) and Matthen and Ariew (2009) for a debate and Otsuka (2016) for a critical review of this point). It is not the fact itself of using statistical calculus that makes population biology a statistical discipline, but the fact that population patterns are explained by abstracting from the individual level. A population is viewed as an average, an epistemic abstraction that is explained not by looking for individual causes, but by looking for abstract properties of the population, such as trait fitness. Walsh et al. (2002, 464) 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.

3.2 Conceptual Change as an Explanatory Shift

The aim now is to consider the historical and current analyses carried out in sections 1 and 2 from the perspective of the debate between causalists and statisticians. Each position has been defended and criticized many times. Causalism has its own advantages (e.g., the account of genetic drift developed by Millstein (2002), Milstein et al. (2009), or the appropriateness of causal analysis involving population forces in real evolutionary explanations (Otsuka, 2016)) and points out the weaknesses of statisticalism (e.g., how can we explain the fact that populations are real entities composed of individuals (Pence, 2021). Although the aim is not to provide further arguments on the many interrelated issues, my core claim is that statisticalism has at least three advantages for the purposes of this paper. It explains the conceptual changes as part of the statistical method employed by the MS (Section 3.2.1); it recognizes and justifies the explanatory frameworks supported by both the MS and the EES in contemporary debates (Section 3.2.2); it allows different semantic ascriptions to coexist as part of different explanatory frameworks (Section 3.2.3).[5]

3.2.1 The Explanatory Shift in the History of Biology

Let us start with the historical analysis to see how to understand the conceptual shifts in each theory. The conceptual changes mentioned in Section 1 concerned two levels: a concept originally used to refer to a phenomenon at the individual level is reinterpreted to refer to a population phenomenon. Population-based explanations replace individual-level explanations. If we adopt the causalist viewpoint, we can conclude that the *conceptual changes outlined earlier correspond to a change in the causes of evolutionary processes*. In other words, what was caused by individual-level processes was reinterpreted by the MS to mean that the actual source of causation was the population. Where someone saw individual causes, the MS succeeded in finding the cause in the population. The shift from individual to population-based causation was the point at which the conceptual changes converged.

However, if we consider the statisticalist position, we come to a different conclusion. We have already seen that the conceptual interpretation analyzed in Section 1 can be understood in 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 population-level 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 and found its origins in the emergence of population biology itself through the mathematical insights of the biometric school of Fisher, Wright, and Haldane 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 suffices to recognize that the conceptual changes mentioned above entail a shift in explanations. What was explained causally became a statistical explanation. While inheritance 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. Whereas the NoR referred to the causal processes linking an organism's genotype 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 in different environments. In this sense, statisticalism fits

---

[5] I thank an anonymous reviewer and the editor-in-chief for pointing out the need to make this point more clearly and to make explicit the different advantages that each theory has in the various debates.

the explanatory method used by population biology to reduce individual-level phenomena to population-level phenomena.

3.2.2 Current Disputes Through the Statisticalist Lens

We have seen that each theory proposes a different way of understanding the conceptual changes during the emergence of the MS. Let us now turn to the question of what influence the statisticalist position has 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 change in the history of biology as causal transmission, 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, or equivalently, by arguing that development and organismal causation have nothing to do with evolutionary biology.

However, adopting a statisticalist position allows for a different interpretation of the current scenario in the philosophy of evolutionary biology. In particular, statisticalism is able to recognize the importance of both explanatory models and thus does justice to the motivation of both the MS and EES. On one side of the debate, the explanatory method used by MS's proponents to invalidate the challenges they face relies precisely on statistical explanations of population trends. As we explained 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). When MS biologists currently apply these methods, they are proposing explanatory shift, not causal transmission: the challenging phenomena presented by the EES are resolved by treating population change in statistical terms.

Statisticalism justifies the explanatory role of population thinking in the MS, but without removing the importance of individual causation in developmental and physiological processes. On the other side of the debate, therefore, 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 EES emphasizes that many biological phenomena deserve a proximate explanation in terms of causal processes during ontogeny. Such a causal interpretation is indeed a central thesis of statisticalism: 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.

As a result, the causal processes outlined here must be understood at the individual level and considered for a full evolutionary explanation. Keeping causation at the individual level prohibits using the reductive strategy of the MS that has led evolutionary biology to dispense with the study of development. Research into the developmental mechanism of the transmission of traits or the question of how an organism adapts its developmental course to environmental conditions is at the heart of the EES and is also vindicated by statisticalism.

3.2.3 The Division of Explanatory Labor and the Division of Semantic Interpretations

This brings us to the third consequence of endorsing the statisticalist position: the possibility of linking various semantic ascriptions of biological terms to the various explanatory methods in evolutionary biology that statisticalism supports. This is related to the second point, i.e., the fact that statisticalism accepts the use of quantitative methods of the MS and also recognizes the causal support for evolution at the individual level that the EES demands. A key idea in this context is the division of explanatory labor between population-level statistical explanations and individual-level causal explanations. This idea is well elaborated and defended in several articles (Ariew, 2003; Walsh, 2019; Walsh et al., 2002; Ariew et al., 2015). It states that both explanatory methods –causal and statistical– that allow for different levels of explanation –the individual level and the population level– can coexist: “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). We need both populational-statistical and individual-causal explanations to understand evolution.

The implication relevant to our discussion is that if we adopt this “division of explanatory labor” we can understand and explain the reason why biological concepts are interpreted differently. This means that we might also adopt a “division of semantic interpretations”. In other words, the acceptance of different explanatory methods is linked to the acceptance of different semantic interpretations of biological concepts. This proves the link between scientific explanation and conceptual interpretations in general, and in particular the different interpretations of biological concepts in distinct explanatory frameworks in biology. In this context, the biological concepts discussed here have been interpreted differently in that they have been part of different explanatory approaches. 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 does not have the same *meaning* as the concept of inheritance. The former refers to the populational property of a trait type being maintained across generations due to the genetic components of that population, whereas the latter refers to the causal processes between parents and offspring that are responsible for cross-generational similarity. Thus, when MS biologists seek to explain a biological phenomenon using population biology, they adopt a statistical interpretation of the concepts involved in their explanations, whereas the EES would adopt an individual-level interpretation for their causal explanations.

Just as statisticalism accepts and requires both methods of explanation, we can accept different interpretations of biological concepts under statisticalism. Note that this does not mean that one interpretation can replace the other. Rather, each interpretation is relevant and valid for understanding adaptive evolution. We cannot reduce the population interpretation of plasticity to the interpretation at the individual level or vice versa. The division of explanatory and interpretive frameworks in biology certainly requires further elaboration and detailed analysis of each biological phenomenon. If we argue that both sides of the debate contain valid explanatory methods, we need to make clear how they can coexist without tension. In particular, many questions about the biological phenomena discussed here deserve special treatment concerning the goal of an integrated biological theory. Can the population-based explanation of heritability also include non-genetic forms of inheritance? Are environmental inheritance systems necessarily 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 it 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 to specify the explanatory scope of any biological approach, i.e., what can and cannot be explained by the MS, and what can and cannot be explained only from the perspective of the individual level.

4. Conclusions

In this paper, we have conducted three different but related analyses. First, I introduced four concepts 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 several areas of current debates that are connected to and inspired by past approaches. 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. 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 have linked the analysis to another central issue in the philosophy of biology, namely the ongoing debate about the causal structure of natural selection. Although I have not put forward an explicit and detailed defense of statisticalism, I argued that it has at least three advantages for a better understanding of the phenomena discussed here. First, it fits with the explanatory role of statistical explanation in population biology to understand conceptual change as part of explanatory change. The explanatory method employed by the MS to understand plasticity, heredity, and the NoR is at the heart of population biology and its statistical (and original) foundations. The main conclusion overall is that the conceptual changes are part of an explanatory shift from the individual to the population level, rather than a causal transfer. 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 current evolutionary theory. A third advantage is that statisticalism ascribes an indispensable and irreducible explanatory role to both population biology (and its statistical method) and the individual analysis of organismic and developmental causes. This division of explanatory causes is linked to the division of semantic interpretations between individual and population interpretations. This move makes it possible to accept both types of interpretations of biological concepts and opens the door to a possible path of parsimony for the integration of the MS and the EES based on the recognition of a diversity of semantic interpretations as part of a diversity of explanatory frameworks.

Acknowledgements: I gratefully acknowledge the useful comments and suggestions of two anonymous reviewers, which were taken into account in improving an early version of this article. I thank Sergio Balari for discussing the ideas presented here. I am also grateful for the feedback of participants at HOPOS 2022 and CLEVOL 2021, where the ideas of this paper were debated. This work was supported by the ANII (Uruguay) through grant PD_NAC_2023_1_176930 and by the Sectoral CSIC (Uruguay) through grant 22520220100257UD.

References

Abrams, M. (2012). Measured, modeled, and causal conceptions of fitness. *Frontiers in Genetics*, 3, 196.

Ågren, J. A. (2021). *The Gene's-Eye View of Evolution*. Oxford University Press.

Amundson, R. (2005). *The Changing Role of the Embryo in Evolutionary Thought*. Cambridge University Press.

Ariew, A. (2003). Ernst Mayr's ultimate/proximate distinction reconsidered and reconstructed. *Biology and Philosophy*, 18(4), 553–565.

Areiw, A. & Lewontin, R. (2004). The Confusions of Fitness. *The British Journal for the Philosophy of Science*, 55(2), 347–363.

Ariew, A., Rice, C., & Rohwer, Y. (2015). Autonomous-statistical explanations and natural selection. *The British Journal for the Philosophy of Science,* 66(3),

Bateson, P., & Gluckman, P. (2011). *Plasticity, Robustness, Development and Evolution.* Cambridge: Cambridge University Press.

Bonduriansky, R. (2012). Rethinking heredity, again. *Trends in ecology & evolution,* 27(6), 330-336.

Charlesworth, D., Barton, N. H., and Charlesworth, B. (2017). The sources of adaptive variation. *Proceedings of the Royal Society B: Biological Sciences*, 284(1855), 20162864.

Dawkins, R. (1976). *The selfish gene*. Oxford New York: Oxford University Press.

Depew, D. (2003). Baldwin and his many effects. Evolution and learning. In M. B. Weber & D. Depew (Eds.), *Evolution and Learning: The Baldwin Effect Reconsidered* (pp. 3–31). MIT Press.

Depew, D. J. (2011). Adaptation as process: the future of Darwinism and the legacy of Theodosius Dobzhansky. *Studies in History and Philosophy of Science Part C: Studies in History and Philosophy of Biological and Biomedical Sciences*, 42(1), 89-98.

de Jong, G. (2005). Evolution of phenotypic plasticity: patterns of plasticity and the emergence of ecotypes. *New Phytol*, 166, 101-118.

Dickins, T. E. and Barton, R. A. (2013). Reciprocal causation and the proximate–ultimate distinction. *Biology & Philosophy*, 28, 747-756.

Dickins, T. E., & Rahman, Q. (2012). The extended evolutionary synthesis and the role of soft inheritance in evolution. *Proceedings of the Royal Society B: Biological Sciences*, 279(1740), 2913-2921.

Dobzhansky, T. (1955). A review of some fundamental concepts and problems of population genetics. *Cold Spring Harbor Symposia on Quantitative Biology*, 20, 1–15.

Futuyma, D. (2017). Evolutionary biology today and the call for an extended synthesis. *Interface Focus*, 7(5), 20160145.

Gilbert, S. F. (1994). Dobzhansky, Waddington and Schmalhausen: Embryology and the modern synthesis. In M. B. Adams (Ed.) *The evolution of Theodosius Dobzhansky: Essays on his life and thought in Russia and America* (143-154). Princeton University Press.

Gilbert, S. (2011). The decline of soft inheritance. In S. Gissis and E. Jablonka (Eds.) *Transformations of Lamarckism: from subtle fluids to molecular biology* (pp. 121-125). MIT Press.

Gilbert, S. F., & Epel, D. (2015). *Ecological Developmental Biology: The Environmental Regulation of Development, Health, and Evolution*. Sunderland, MA: Sinauer.

Godfrey-Smith, P. (2009). *Darwinian populations and natural selection*. Oxford University Press.

Gottlieb, G. (1992). *Individual development and evolution: the genesis of novel behavior*. New York: Oxford University Press.

Gissis, S and Jablonka, E. (Eds.) (2011) *Transformations of Lamarckism: from subtle fluids to molecular biology*. Vienna Series in Theoretical Biology. The MIT Press.

Griesemer, J. (2000). Development, culture, and the units of inheritance. *Philosophy of science*, 67, 348–368.

Griffiths, P. & Gray, R. (1994). Developmental Systems and Evolutionary Explanation". *Journal of Philosophy*, 91(6), 277–304.

Griffiths, P., & Stotz, K. (2013). *Genetics and philosophy: An introduction*. Cambridge University Press.

Gupta, M., Prasad, N. G., Dey, S. Joshi, A. and Vidya, T. N. C. (2017). Niche construction in evolutionary theory: the construction of an academic niche?" *Journal of Genetics*, 96(3), 491–504.

Haig, D. (2007). Weismann rules! OK? Epigenetics and the Lamarckian temptation. *Biology & Philosophy*, 22, 415-428.

Jablonka, E. (2007). The developmental construction of heredity. D*evelopmental Psychobiology*, 49(8), 808–817.

Jablonka, E. & Lamb, M. (2005). *Evolution in four dimensions: genetic, epigenetic, behavioral, and symbolic variation in the history of life*. Cambridge, Mass: MIT Press.

Jablonka, E., & Lamb, M. J. (2008). The epigenome in evolution: beyond the modern synthesis. *Информационный вестник ВОГиС*, 12(1-2), 242-254.

Jablonka, E. & Lamb, M. (2020). *Inheritance systems and the extended synthesis*. Cambridge University Press.

Keller, E. F. (2010). *The Mirage of a Space between Nature and Nurture*. Duke University Press.

Mameli, M. (2005). The inheritance of features. *Biology and Philosophy*, 20, 365-399.

Matthen, M. & Ariew, A. (2002). Two ways of thinking about fitness and natural selection. *The Journal of Philosophy,* 99(2), 55–83.

Matthen, M. and Ariew, A. (2009). Selection and causation. *Philos Sci* 76:201–224

Mesoudi, A., Blanchet, S., Charmantier A., Danchin, E., Fogarty, L., Jablonka, E., Laland, K. N., Morgan, T., Müller, G., Odling-Smee, F. J., and Pujol, B. (2013). Is non-genetic inheritance just a proximate mechanism? A corroboration of the extended evolutionary synthesis. *Biological Theory*, 7, 189-195.

Millstein, R. (2002). Are random drift and natural selection conceptually distinct? *Biol Philos* 17(1):33–53

Millstein, R. (2006). Natural selection as a population-level causal process. *The British Journal for the Philosophy of Science,* 57(4), 627–653.

Millstein, R., Skipper R. A. J., Dietrich, M. R. (2009). (Mis)interpreting mathematical models: drift as a physical process. *Philos Theory Biol* 1:e002

Nanney, D. L. (1958). Epigenetic control systems. Proceedings of the National Academy of Sciences, 44(7).

Noble, D. (2021). The illusions of the modern synthesis. *Biosemiotics*, 14(1), 5-24.

Otsuka, J. (2016) A critical review of the statisticalist debate. *Biol Philos* 31, 459–482.

Pence, C. (2021). *The causal structure of natural selection*. Cambridge Elements in the Philosophy of Biology.

Pigliucci, M., Murren, C. J., & Schlichting, C. D. (2006). Phenotypic plasticity and evolution by genetic assimilation. *Journal of Experimental Biology*, 209(12), 2362-2367.

Rama, T. (2022) *Agential Teleosemantics*. Doctoral Dissertation. Autonomous University of Barcelona.

Rama, T. (2023). Evolutionary causation and teleosemantics. In J. M. Viejo and M. Sanjuan (Eds.), *Life and Mind: New Directions in the Philosophy of Biology and Cognitive Sciences* (pp. 301-329). Cham: Springer International Publishing. DOI: 10.1007/978-3-031-30304-3_14

Rama, T. (2024a). The Explanatory Role of Umwelt in Evolutionary Theory: Introducing von Baer's Reflections on Teleological Development. *Biosemiotics,* 1-26. DOI: 10.1007/s12304-024-09569-8

Rama, T. (2024b). Is a Cognitive Revolution in Theoretical Biology Underway? *Foundations of Science,* 1-22. DOI: 10.1007/s10699-024-09950-3

Ramsey, G. (2016). The causal structure of evolutionary theory. *Australasian Journal of Philosophy,* 94(3), 421–434.

Robert, J. S. (2004). E*mbryology, Epigenesis and Evolution: Taking Development Seriously.* Cambridge: Cambridge University Press.

Rosenberg, A. (1997). Reductionism redux: computing the embryo. *Biology and Philosophy,* 12, 445-470.

Sarkar, S. (1999). From the Reaktionsnorm to the adaptive norm: the norm of reaction, 1909–1960. *Biology and Philosophy,* 14(2), 235–252.

Schlichting, C. D. & Pigliucci, M (1998). *Phenotypic evolution: a reaction norm perspective*. Sinauer associates incorporated.

Shea, N. (2007). Representation in the genome and in other inheritance systems. *Biology & Philosophy,* 22(3), 313–331.

Shea, N. (2013). Inherited representations are read in development. *The British journal for the philosophy of science,* 64.1, 1–31.

Simpson, G. G. (1953). The Baldwin Effect. *Evolution,* 7(2), 110.

Sober, E. (1980). Evolution, population thinking, and essentialism. *Philosophy of Science,* 47(3), 350–383.

Sober, E. (1984). *The nature of selection: evolutionary theory in philosophical focus*. Chicago, University of Chicago Press.

Sterelny, K., Smith, K. & Dickison M. (1996). The extended replicator. *Biology and Philosophy,* 11(3), 377–403.

Stotz, K., & Griffiths, P. (2016). Epigenetics: Ambiguities and implications. *History and Philosophy of the Life Sciences*, 38(4), 1–20.

Sultan, S. (2015). *Organism and environment: ecological development, niche construction, and adaption.* New York, NY, United States of America: Oxford University Press.

Sultan, S. (2017). Developmental plasticity: re-conceiving the genotype. *Interface Focus*, 7(5), 20170009.

Sultan, S. (2019). Genotype-environment interaction and the unscripted reaction norm. In T. Uller & K. N. Laland (Eds.), *Evolutionary Causation: Biological and Philosophical Reflections* (pp. 109–127). Cambridge, MA: The MIT Press.

Svensson, E. I. (2018). On reciprocal causation in the evolutionary process. *Evolutionary Biology,* 45(1), 1-14.

Wagner, A. (2013). *Robustness and Evolvability in Living Systems.* Princeton, NJ: Princeton University Press.

Wagner, G. P. (2014). *Homology, genes, and evolutionary innovation.* Princeton University Press.

Walsh, D. (2003). Fit and diversity: Explaining adaptive evolution. *Philosophy of Science,* 70(2), 280–301.

Walsh, D. (2007). The pomp of superfluous causes: The interpretation of evolutionary theory. *Philosophy of Science*, 74(3), 281–303.

Walsh, D. (2015). *Organisms, Agency, and Evolution*. Cambridge University Press.

Walsh, D. (2019). The paradox of population thinking: First order causes and higher order effects. In T. Uller & K. N. Laland (Eds.), *Evolutionary Causation: Biological and Philosophical Reflections* (pp. 227–246). Cambridge, MA: The MIT Press.

Walsh, D., Lewens, T. & Ariew, A. (2002). The trials of life: Natural selection and random drift. *Philosophy of Science,* 69(3), 429–446.

Walsh, D. M., Ariew, A., & Matthen, M. (2017). Four pillars of statisticalism. *Philosophy, Theory, and Practice in Biology*, 9(20171201).

Waters, C. K. (2007). Causes that make a difference. *The Journal of Philosophy*, 104(11), 551-579.

West-Eberhard, M. (2003). *Developmental plasticity and evolution*. Oxford New York: Oxford University Press.

Williams, G. C. (1966). *Adaptation and natural selection: A critique of some current evolutionary thought*. Princeton, Princeton University Press.