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Universidad de la República de Uruguay
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Explanatory Domains and Reciprocal Causation: How (not) Integrate Development and Evolution

Abstract: A common explanatory error in science concerns the conflation of the epistemological roles between two domains. Here we will address a specific case: when explanations of development replace evolutionary explanations or vice versa. Ernst Mayr famously distinguished between proximate and ultimate causal explanations in biology. His view was central to the Modern Synthesis' exclusion of development from evolutionary theory. Nonetheless, the explanatory role of developmental processes in evolution is a central theme in current theoretical biology which has prompted several revisions of Mayr's distinction. Here we will review these reviewers to determine whether the integration of development and evolution is based on an appropriate reinterpretation of Mayr's distinction. In many cases, revisionists suggest an interactionist alternative, in which proximate and ultimate causes interact to produce evolved traits. The most frequent interactionist account relies on the idea of reciprocal causation. We will argue that this perspective is still problematic and that the boundaries between explanatory domains are crossed. Instead, we should rethink Mayr's distinction by adopting an alternative view of evolutionary causation, the so-called Statisticalist view, which maintains that the only level of causation is the individual level. By ruling out two different levels of causation, this framework is appropriate to avoid fallacious explanations and reconsider reciprocal causation entirely as a proximate phenomenon. We introduce the concept of "statistical reciprocity" to explain the statistical effects of reciprocal causality in populations and outline some ideas of "population ontogenetics" as a prominent framework for unifying development and evolution beyond interactionist positions.

Keywords: Evolutionary Causation; Phylogeny and Ontogeny Fallacies; Proximate-Ultimate Distinction; Reciprocal Causation; Population Ontogenetics.

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> It is true that Darwin, when considering natural selection, leaves out of account the causes which have produced the alterations in separate individuals [...] To Darwin it was of less immediate importance to discover these causes than to find a *rational form* in which *their effects* become fixed, acquire permanent significance.
> Friedrich Engels, 1878, 82-83, emphasis added.

1. Introduction

Ernst Mayr (1961, 1974) systematized the prevailing opinion on biological causes and biological explanations. Mayr established a taxonomy of biological causes, different explanatory goals associated with the study of each type of biological cause, and different disciplines in biology dealing with each explanatory goal. Accordingly, we must distinguish between ultimate causation and proximate causation. Ultimate causation involves evolutionary processes acting on populations on a phylogenetic time scale. Proximate causation is present in individual-level processes that act on organisms on an ontogenetic time scale. Ultimate causal explanations answer *why-questions*: Why are biological systems organized in a certain functional and adaptive way? Proximate causal explanations deal with *how-questions*: How different parts of a living system interact to achieve a functional or adaptive outcome. Finally, he assigned each type of question to different biological domains. Evolutionary and population biology provide ultimate explanations for why-question, while developmental biology and physiology provide proximate explanations for how-questions.

Mayr's view of biological explanations represents "the epistemological statement of the modern synthesis" (Dickins and Barton, 2013, 2), it has been central to explaining why development should not be integrated into evolution (Amundson, 2005), and "has acted to stabilize the dominant evolutionary paradigm against change" (Laland et al., 2011, 1512). The core idea is that only population-based causes involved in natural selection processes are necessary to explain adaptive evolution. Population-based causes provide the ultimate explanation for the adaptive nature of living organisms. However, Mayr did not ascribe the same explanatory role to the proximal, individual causes: causes at the individual level, which act during the development of the organism, play no role in evolution. This view of evolutionary causation is central to the emergence of the so-called black box of development: the assertion that developmental processes involving proximate causes need not be understood to explain adaptive evolution.

The black box of development was built on central tenets of the Modern Synthesis (hereafter: MS), such as the exclusion of Lamarckian modes of inheritance, a robust Genotype-Phenotype Map, or the unbiased nature of variation. It is important to note that Mayr systematized a view of biological explanations that was already present in biology. While Darwin anchored the idea of natural selection as an adaptive process in evolution, the MS's interpretation of Darwinism focused only on the genetic level. An important step towards a pure population perspective of evolution was the reduction of the core components of natural selection to the genetic level. Thus natural selection occurs in populations when there is heritable variation in fitness in gene pools. According to this paradigm, causal explanations of adaptive evolution fall within the domain of population genetics. To understand evolution, it is sufficient to see what is going on at the genetic level, or as Maynard-Smith (1982, 6) stated: "It is possible to understand genetics, and hence evolution, without understanding development". "Mayr championed the view that evolutionary biologists could legitimately jump from genotypes to fitnesses, whilst ignoring all the biology in-between" (Laland et al., 2013, 7), and therefore "Mayr's dichotomy reflects a mid-twentieth-century consensus within evolutionary biology [...] regarding the basic mechanics of evolution and the explanatory adequacy of population genetics" (Brown, 2021, 4).

This paper is motivated by current philosophical debates about the foundations of evolutionary theory. In particular, the MS has been severely challenged in the last decade by an increase in conceptual and empirical

knowledge supporting the idea that developmental processes must be at the heart of evolutionary theory. This has led to a revision of evolutionary theory and the way in which the explanatory domains in biology have been defined.

The first aim of this paper is to review the reviewers: to promote a critical analysis to assess whether the alternative proposals to Mayr's distinction are on the right track. We will argue that many proposals –which we refer to as adopting the "integration-as-interaction" strategy– are still problematic. In particular, we will examine the alternative views of biological causation that are based on reciprocal causation to show some of their weaknesses. The second aim is to find a solution to these problems by proposing an alternative way of rethinking Mayr's distinction based on the so-called Statisticalist view.

The paper is structured as follows. In Section 2, after clarifying the notion of explanatory domains, we will point out various explanatory errors that can arise when we separate ultimate and proximate causation. In Section 3, I will introduce the strategy of "integration-as-interaction" in the context of the Extended Evolutionary Synthesis and the ongoing goals of integrating development and evolution by reviewing Mayr's distinction. We will argue that this position is problematic, as it still suffers from the explanatory shortcomings mentioned above. We will show a clear case where this problematic strategy is applied: reciprocal causation. In Section 4, we will present an alternative view of evolutionary causation –the so-called Statisticalist School– and the revision of Mayr's distinction proposed in this framework. We will argue that this position seems to be a better way to integrate development and evolution and to avoid explanatory errors.

2. Explanatory Domains

Mayr's distinction between proximate and ultimate levels and the different types of questions they answer demarcated two different explanatory domains. Since then, Mayr himself has played the role of a guardian, criticizing various scientists who, in his opinion, have mixed up the ultimate and proximate levels (Beatty, 1994). Mayr also assigns specific fields or disciplines to each explanatory domain (evolutionary and functional biology). However, biological fields are usually concerned with different epistemic tasks, and the boundaries of each field are relative to the different variables under consideration. Interfield theorists such as Darden and Maull (1977) note that many fields in biology have emerged from work on bridging different fields, so a single field may belong to different explanatory domains. Thus, rather than using fields as delimiters of domains, we confine ourselves here to identifying explanatory domains based on the *nature of the explanation and the facts explained* –-and concomitantly, what kind of explanation is *not* given and what remains *un*explained.[1]

Here we would like to discuss two specific *explanatory errors*: the phylogeny fallacy and the ontogeny fallacy. Both cases involve the conflation of explanatory domains –the invocation of one kind of explanation to take on the epistemological task of the other; or in other words, when we overstep the epistemological boundaries. The most commonly discussed fallacy is that of the phylogeny fallacy, baptized by Lickliter and Berry in 1990. It was first pointed out by embryologists (Kuo, 1921, 1922; Gottlieb 1997) and developmental psychobiologists (Lehrman, 1953; Micheal and Moore, 1995). A characteristic of this fallacy is that it refers to evolution in order to explain a developmental process. Thus, in order to illustrate the phylogenetic fallacy, we need to trace some epistemic boundaries and cross their borders. In this case, we must distinguish between two levels of causal explanation: the population level and the individual level. Someone engages in the false argument of the phylogeny fallacy when uses population-level causal explanations to address phenomena that should be explained by individual-level causal explanations.

[1] The names of many fields in biology explicitly show how the merging of two existing fields creates a new interfield area (e.g. "evo-devo", "population genetics", "molecular epigenetics", "biochemistry"); see Edna-Suárez (2009) for a recent historical discussion. I thank Sergio Balari for his comments on this point.

As we can see, this characterization of the phylogeny fallacy corresponds to Mayr's distinction. We commit the fallacy when we use ultimate explanations to deal with proximate causes, when we give why-answers to how-questions. As has been reported by many, the phylogeny fallacy is linked to the use of developmental dichotomies (Rama, forthcoming). Developmental dichotomies are those dichotomous categories associated with the nature-nurture dichotomy that are used to categorize phenotypes (or their causes) into developmental or evolutionary terms, such as innate-learned, inherited-acquired, biological-cultural, and others. Innate traits are supposedly explained by evolution, while learned traits are explained by ontogeny. The point here is that the use of developmental dichotomies is one of the ways to commit the phylogeny fallacy. This is because, by using developmental dichotomies, we are providing an evolutionary explanation for a developmental mechanism that transcends the boundaries of evolutionary biology; that is, we want to explain how development unfolds by saying that it is evolutionarily predetermined. Thus nativism says nothing about the correct developmental mechanisms; or as Oyama (1985, 159) claimed, "[it] feels right, but it explains nothing." We do not explain how developmental causes produce a trait just because we say that it is innate. As Griffiths and Stotz (2013, 23) recently noted: "The idea of genetic information, like the idea of innateness, is a Trojan horse that helps to disguise an evolutionary explanation as a developmental explanation, and obscures the fact that no actual explanation of development has been produced"

Keller (2010) rightly notes that the use of developmental dichotomies entails two distinctions. First, between individuals and populations, and second, between an explanation for a trait and an explanation for the difference between traits. When we ask for explanations of development, we want to know how developmental processes work: we are asking about phenomena on an individual level and we intend to explain a particular characteristic. However, when we say that a trait is innate, caused by evolution, or inherited, we are not dealing with a specific trait, but we qualify a trait as part of a population – we point to the causes in the population that produce this trait type (not token). In doing so, we shift the nature of the explanation: we explain *how-much-questions* instead of *how-questions,* i.e. we ask how much a cause influences the development of a trait instead of how this cause is involved in developmental mechanisms.

How-much-questions are not proper explanations of development. They are also associated with a soft interactivist position, sometimes referred to as consensus interactivism, which we refer to here as *Type 1 Interactivism* (Rama, 2022). Type 1 Interactivism holds that the emergence of all traits is explained by the interaction of evolutionary and developmental causes, inherited and environmental causes. Innate causes and learned causes produce traits; the issue is to determine how much a certain cause influences a trait. "This 'interactionist consensus,' however, perpetuates the nature-nurture debate by maintaining its inherent dichotomy" (Stotz, 2008, 360). Under this view, *development and evolution interact in the explanation of development.*

So far we have identified the phylogeny fallacy. The ontogeny fallacy, introduced by Hochman (2012), is the inverse of the phylogeny fallacy: the use of explanations of development as a substitute for evolutionary explanations. Remarkably, this fallacy is not particularly explored in biology (Hochman has analyzed specifically how Lickliter and Honeycutt (2003) commit this fallacy). In this paper, we will address the ontogeny fallacy. However, it is important to keep in mind several ideas related to the phylogeny fallacy. In particular, we will see in the next section how various recent proposals that revise Mayr's distinction still embrace Type 1 Interactivism by supporting the idea that *development and evolution interact in the explanation of evolution*.

3. Reviewing Reviewers: How not to Reframe Mayr's Distinction

We have seen that Mayr's distinction was central to the exclusion of development from evolutionary theory. In this section, we will first briefly introduce the ongoing debates that call for an Extended Evolutionary Synthesis (EES), and the demand to rethink Mayr's distinction in order to integrate development and evolution. After

introducing this new perspective, we will identify some problems it still faces. In particular, we will discuss those proposals that revise Mayr's distinction by advocating an "integration-as-interaction" strategy, principally by embracing the notion of reciprocal causation. The critical analysis here does not necessarily apply to the EES as a whole. It applies only to those who still distinguish between individual and population causation and claim that the integration of these causes is achieved by advocating an interactivist position: Development and evolution interact to cause adaptation and diversity in nature.

3.1. Mayr's Distinction from an Extended Position

Various tenets of the MS are currently controversial. There are new approaches that are more or less opposed to the foundations of the MS. The most consolidated proposal comes from the EES: It aims to extend the MS principles to explain phenomena for which there is no adequate explanation. The reason for this revolt in biological theory is that explaining natural selection processes solely in terms of gene populations entails simplistic and incorrect assumptions about central biological processes in evolution, and therefore "[t]he black box [of development] is now being opened to provide a more complete picture of what really happens" (Bateson and Gluckman, 2011, 17). For example, extended inheritance systems have been found in all living systems (Jablonka and Lamb, 2014). The genetic view of inheritance mischaracterizes inheritance processes and does not adequately account for epigenetically mediated intergenerational relationships. Moreover, developmental processes are not only about the expression of genetic information, but various non-genetic developmental resources are causal specifiers of developmental outcomes (e.g. Griffiths and Stotz, 2013). In this sense, the Genotype-Phenotype Map is less robust than the MS argues: Genotypes are not geolocators of phenotypic outcomes because outcomes are produced by multiple complex causal networks involving different levels of organization (from genes to exogenous causes). In contrast to an unbiased view of variation, there are also multiple sources of adaptively biased phenotypic variation, such as plasticity, niche construction, or self-organization (e.g. Sultan, 2015). We will not go into the discussion of the various biological processes appealed to by EES. Rather, we will discuss below the implications of this extended framework for the distinction between direct and ultimate causation. The main result of this revolt is that the idea that developing organisms are causally relevant in adaptive evolution is now supported by a growing wave of biological theories (Rama, 2024a).

This extended view has spawned several critical revisions of Mayr's ultimate-proximate distinction (Laland et al, 2011, 2013; Laland and Sterelny, 2006; Brown, 2021; Vromen, 2017; Calcott, 2013; Haig, 2013; Ramsey and Aaby, 2022; Buskell, 2019; Corning, 2019; Scholl and Pigliucci, 2014; Otsuka, 2014; Svensson, 2018; see Uller and Laland (2019) for an edited volume on recent approaches to evolutionary causation). There are several reasons to argue that proximate causes must be included in evolutionary explanations: As West-Eberhard has expressed (2003, 11): "The proximate‐ultimate distinction has given rise to a new confusion, namely, a belief that proximate causes of phenotypic variation have nothing to do with an ultimate, evolutionary explanation." The general consensus of most critics of Mayr's analysis is that proximate causes are also relevant evolutionary explanations. Therefore, Mayr's distinction deserves reformulation: "Progress within biology demands dismantling of Mayr's identification of proximate with ontogenetic processes and ultimate with evolutionary processes" (Laland et al, 2011, 1516).

3.2 Integration as Interaction

How should we restructure biological explanations and biological causation in light of recent challenges to the MS? This is a question that asks about the explanatory role of developing organisms in evolutionary theory: In what ways must developmental processes be included in ultimate explanations?

One possible answer is to argue that Mayr's proximate-ultimate distinction deserves evaluation, but without abandoning the distinction between individual and population causation. In this model, both individual and

population causes are explanatorily relevant for explaining adaptive evolution. In addition to population forces, development and other processes at the individual level are also evolutionary causes of fit and diversity. This position differs from that of the MS insofar as, in the MS, developmental causes have no influence on evolutionary dynamics. However, once individual causation is also established as a causal factor in adaptive evolution, the MS view is affected. This new view advocates a particular strategy for integrating development and evolution, the "integration-as-interaction" (hereafter: I-I) strategy. The I-I strategy states that development and evolution interact; proximate causes interact with evolutionary causes to produce fit and diversity in nature.

The I-I strategy may be a common way to think about evolution from an extended perspective, as also noted by Walsh (2019). It can be implicitly assumed when we intend to integrate development and evolution. However, it is a misguided reconstruction of biological explanations: The I-I strategy conflates explanatory domains. Fundamentally, the logic of dichotomizing levels of causation remains under an interactionist framework (Dickins and Barton, 2013; Svensson, 2018; Corning, 2019): "For two things to interact they must, logically, be distinct things. In talking about such interactions, they therefore have to make the proximate–ultimate distinction" (Dickins and Barton, 2013, 4). Consequently, in this context, many ideas present in the phylogeny fallacy also apply to the ontogeny fallacy that the I-I strategy commits. Crucially, Type 1 Interactivism is assumed, but at the evolutionary level. While we have seen that this interactionism is advocated to explain development, it is also supported by the I-I strategy to explain evolution: Developmental causes and evolutionary causes interact to produce adaptive evolution. This explanatory error is as flawed as explanations of development based on nativist ideas. This is the inverse of using evolution in explanations of development, i.e. the ontogeny fallacy. What does it mean that development and evolution interact? Certainly, development influences evolution, but in what way do population causes interact with individual causes? How can we determine whether an evolved trait was caused by development or by evolution? It seems like we are asking for how-much-questions, as Keller said. Does it make sense to ask how much development causes the evolution of a certain trait? Walsh (2019, 237) is clear on this position:

> The revised two-force picture [under the I-I strategy] encourages us to ask: "how much of evolutionary change is due to individual-level processes and how much to selection?" But one cannot partition causal responsibility between the first order causes of population change and their higher order effects in this way [...] [The problem] lies in construing individual-level causes and ensemble-level processes as somehow on an ontological par, as interacting causes of ensemble change.

The interactivist view of biological causation is sometimes hidden in the various disciplines associated with the EES, but in other cases it is explicit. Let us take a classic example that favors extended views: phenotypic plasticity. According to this view, plasticity shows that development is not encoded in traits, but is generated epigenetically through the regulation of various exogenous factors. Compare trait X, which arose plastically, with trait Z, whose origin is due to a random genetic mutation. Should we say that X was more influenced by development than Z? Do populational causes affect Z more than X? Once again, this question sounds nonsensical. Did not Z come about through a development process just like X? Isn't X selected in a population just like Z? If we assume that plastic traits are more proximate than robust traits, we still retain the dichotomous thinking typical of Type 1 Interactivistm (Bateson and Gluckman, 2011; Schwab et al., 2019).

For the same reasons, the revival of the inheritance of acquired character seems to preserve dichotomic notions from the past. Certainly, we can ask whether variants induced by epigenetic factors can be inherited. The problem lies in understanding that these traits are acquired, in contrast, we suppose, of genetically-induced traits. Moreover, constructivist and holistic views of development in the EES (such as developmental systems, post-genomics, or systems biology) remind us that traits are caused by multiple factors, so it makes no sense to classify traits on the basis of developmental resources, much less to say that some traits are acquired and others are not. The division into innate and acquired makes no sense in development. So why should it be maintained

in evolution if all traits depend on genetic and environmental factors? Does it make sense to ask how much an environmental cause must influence a trait for it to be an acquired character?

The aim is not to give an overview of the various places in which the I-I strategy is supported. We will rely on the alternative to the Mayr view that is most clearly endorsed: reciprocal causation.[2] Various areas of the EES, such as niche construction, eco-devo, evo-devo, and extended inheritance, take "a reciprocal view of the interaction of proximate and ultimate factors" (Laland et al., 2011, 1514). It is the key concept used by those reviewing Mayr's distinction from an extended position. As expressed in the closing remarks of the influential paper by Laland et al. (2011, 1516), "biological sciences might now be better served by a new "reciprocal" conception of causation."

3.3. What's Wrong With Reciprocal Causation?

Organisms interact with their environment. This is almost trivial. What is relevant is that reciprocal interactions between organisms and the environment influence central components of evolution that the MS has completely reduced to the genotypic space: reciprocal causation generates variation, alters the environment, increases individual fitness, allows developmental processes to be adaptively modulated, and is involved in the reconstruction of traits in future generations. What is wrong with reciprocal causation? There is nothing wrong with reciprocal causation if we understand it as a proximate process in which proximal mechanisms are coupled to the environment in different ways throughout an individual's lifespan. In this reading, reciprocal causation is a kind of *Type 2 Interactivism*, which states that several *proximate causes* interact in ontogenetic processes. The activity of the organism is produced by several endogenous and exogenous causal factors. Reciprocal causation is a rich concept. It also proves that Mayr's ideas about explanatory domains are misguided: "When there is reciprocal causation, proximate details are required to answer ultimate questions" (Calcott, 2013, 775), thus the development of organisms does have evolutionary consequences.

The problem is that reciprocal causation is taken as the alternative model. In this context, the I-I strategy is a common way to understand the challenges to the MS. Revisionists of Mayr's view usually intend "to deny the strong causal autonomy entailed by the proximate-ultimate distinction" (Brown, 2021, 8) and "replace Mayr's uni-directional view on the relation between ultimate and proximate causes by the bi-directional one of reciprocal causation" (Vromen, 2017, 1). Reciprocal causation is understood as the interaction between proximate and ultimate causes: "[O]n a reciprocal view of the interaction of proximate and ultimate factors" (Laland et al., 2011, 1514), proximate and ultimate causation *interact* in the explanation of adaptive evolution. To understand adaptive evolution properly, we need to include "a story of *reciprocal interaction* between evolutionary dynamics and the mechanisms of development" (Calcott, 2013, 776). In this context, the role of development in evolution is seen as an *interaction:* developmental causes interact with evolutionary causes, and the explanatory role of development is to be found in these reciprocal interactions: "When we take reciprocal causation seriously, we can show that the interaction between development and evolution can make a difference to the evolutionary trajectory of a lineage" (Calcott, 2013, 776). This image is frequent in niche construction theory, where "evolution and development are perceived as interactive processes" (Laland et al., 2008, 553). Also, some perspectives on phenotypic plasticity seem to support this view: "Adaptive evolution proceeds through repeated bouts of reciprocal causation between developmental plasticity, processes of inheritance, and natural selection" (Uller and Helantera, 2019, 369); "there is a reciprocal relationship between the organism-initiated phenotypic novelty and natural selection" (Ramsey and Aaby, 2022, 8).

[2] In a recent and interesting essay, Hazelwood (forthcoming) comes to a conclusion similar to mine by analyzing the place of reciprocal causation in various views of evolutionary causation. Although he provides a different argumentation and pursues different goals, he shows further reasons why reciprocal causation can be problematic in the philosophy of biology.

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Most understand reciprocal causation as a proximate phenomenon, so this is not a new interpretation (Baedke et al., 2021; Rama, 2024b). The problem, however, is that some argue that the interactions between organisms and the environment entail reciprocal causation of development and evolution. It is even common for both interpretations to be defended together in the same work as if their differences had gone completely unnoticed. What is at stake here is that reciprocal interaction at the proximate level is lumped together with reciprocal interaction between individual development and population evolution. This conflation is based on a misconception of ultimate causation: the proximal causal relationships between organisms and the environment are transformed into causal relationships at multiple levels between the proximal causes of individuals and the ultimate causes of the environment. The environment seems to be understood simultaneously as an ultimate and a proximate cause, as a populational cause and as an individual cause. How is it possible? Consider the problem: a blackbird has built a nest on the lemon tree in my garden. The garden's lemon tree cannot produce many lemons because the buildings cast shadows, so it is trying to develop an appropriate morphology to receive light. I think my cat has a romance with the neighbor's cat and uses the lemon tree to reach the neighbor's house. There will probably be newborn kittens sooner or later. Does my cat interact with evolution to reproduce? Does the blackbird interact reciprocally with ultimate causes? Does the lemon tree interact with natural selection to get light? It seems that organisms simply interact with their surroundings: my cat with the cat, the blackbird with the lemon tree, and the lemon tree with the buildings. Why should we say that in all these cases organisms interact with evolution or development interacts with natural selection? Why should we believe that "their actions [are] simultaneously proximate and ultimate causes" (Ramsey and Aaby, 2022, 10)? Definitely, explanatory externalism –MS's assertion that external environmental pressures are the only direct force of evolution– should be questioned. But if we understand external pressures as a population force, it is difficult to figure out what it means for individuals to interact with a population force. And if explanatory externalism is understood to mean that environmental pressure is an individual-level cause, then selection is caused by proximate causes, and thus there is no reciprocal causality between proximate and ultimate causes at all.

Phenotypic plasticity, sexual selection, or niche construction are proximate phenomena, and we need to assess how these phenomena have evolutionary consequences. However, if we accomplish this task by treating an individual's environment as the ultimate cause, we would be left with how-much questions in evolution: how much a proximate or ultimate factor causes the evolution of a trait, how much development or evolution causes the evolution of a trait, how much proximate causes or ultimate causes cause the evolution of a trait. We have already seen that these questions are nonsensical. Reciprocal causation as a model for defining explanatory domains is anchored in Type 1 Interactivism: development and evolution interact to cause evolution.

In my view, reciprocal causation has nothing to do with the distinction between proximate and ultimate explanations (i.e. with the distinction itself). However, proponents of the I-I strategy maintain both levels of causation. To "undermine false dichotomies […] something new is needed'' (Amundson, 2005), and Laland et al. (2008, 553) "suggest that this something new is reciprocal causation". Reciprocal causation understood under the I-I lens essentially preserves the dichotomies. And furthermore, it conveys a wrong idea of how the elements of the dichotomy are related. The understanding of reciprocal causation under the I-I strategy is based on the notion that the organism is the proximate cause and the environment is the ultimate cause. However, an organism's environment is a proximate cause, as is any cause related to an individual's life history. External selection pressure on an individual is one of the proximate mechanisms and is involved in the explanations for an organism's survival and reproduction.

We have thus identified two kinds of Type 1 interactivism: Interactivism to explain development and Inteactivism to explain evolution. A curious thing about both kinds of interactivism is that they set the causes opposite to each other! That is, the developmental dichotomies in the developmental explanation state that the internal cause (genes, heredity, nature) is the evolutionary, ultimate cause and the external cause (environmental influences in learning processes) is the ontogenetic, proximal cause. On the other hand, reciprocal causality in the evolutionary explanation states that the internal cause (developing organisms) is the ontogenetic, proximal

cause and the external cause (selection pressure) is the evolutionary, ultimate cause: the inner cause in development is the evolutionary cause, while the inner cause in evolution is the developmental cause; the external cause in development is the proximal cause, while the external cause in evolution is the ultimate cause.

The problem here lies not in the concept of reciprocal causation itself, but in how it is interpreted. In particular, how we understand things that exhibit reciprocal causation. The task now is to figure out how reciprocal causation as a proximate phenomenon affects the ultimate explanation without mixing up the explanatory domains –without claiming that my cat has a romance with evolution. To accomplish this task, we need an alternative view of the explanatory domains in biology.

4. Beyond Levels of Causation

The Statisticalist School provides an alternative to Mayr's distinction. As we shall see, a distinction between proximate and ultimate *explanations* is retained (but modified), but the distinction between proximate and ultimate *causes* is abandoned: All causes of evolution are proximate causes. The Statsitcalist School provides a different way of understanding development in evolution that goes beyond Mayr's two levels of causation. As we will explain later, the statisticalist position, though it may seem radical, helps to clarify the different explanatory roles of each level of explanation in biology – individual and population– by delineating different *kinds of explanations* in adaptive evolution –not different *kinds of causes*. We argue that these distinctions are essential to allow for a proper integration of development and evolution, and that the phylogeny and ontogeny fallacies are avoided in this framework. Finally, we will analyze how reciprocal causality should be understood from this perspective by introducing the idea of "statistical reciprocity" and how development and evolution could be integrated by introducing the idea of "population ontogenetics".

4.1 Individual Causes, Population Effects

There is an ongoing dispute about the causal structure of natural selection (Pence, 2021). On the one hand, the Causalist School (drawing primarily on the work of Elliot Sober (1984)) argues that natural selection should be understood as a force acting on populations, steering them towards successful peaks (population maintenance) or desert valleys (extinction). In contrast, the Statistical School (see the seminal work of Walsh et al. (2002) and Matthen and Ariew (2002)) argues that the causes of natural selection are not at the level of populations but at the level of individuals. *There is no population causation*. Rather, the explanation of natural selection provides *statistical explanations* for the effects of individual-level causes on population dynamics. Without developing a detailed defense of the Statisticalist School, we will present its basic principles.

The Statistical School's proposal revolves around the concept of *fitness*, which is central to any explanation of natural selection: adaptive changes in the population require fitness differences, as Darwin taught us (Ariew and Lewontin, 2004). We must differentiate between two notions of fitness: *trait fitness* and *individual fitness*. The former refers to a property of a population and was introduced during the emergence of the MS by the famous mathematical insights of Fisher, Wright, and Haldane's theory of evolution. In this sense, trait fitness refers to the fitness values of a population, not an individual; it refers to a property of trait types, not trait tokens. Individual fitness, on the other hand, refers to the fitness value of each individual. It refers to trait tokens, not trait types.

This distinction between different concepts of fitness is associated with different types of explanations. While "[t]rait fitness is the *average* survivability of a group of individuals possessing a type of trait' (Ariew 2003, 562, emphasis added), individual fitness concerns the causal processes in a single lifespan that produce a certain reproductive and survival capacity in an organism. The difference is that trait fitness refers to a statistical measure and individual fitness is evaluated causally. Trait fitness is an average value in a given population,

whereas individual fitness indicates the causes of an organism's ability to survive and reproduce: “Evolutionary explanations differ in kind from proximate explanations. Evolutionary explanations are statistical, they range over the ensemble of individuals, taken as a class. Proximate explanations are individual level causal explanations ranging over individual life histories” (Ariew, 2003, 561).

Based on this distinction, the statisticalist thesis states that trait fitness is measured by averaging the individual fitness of trait tokens; “[a]s an average, trait fitness does not reflect a property that any individual necessarily possesses” (Ariew, 2003. 562), it does not provide information about traits tokens. In short, trait fitness is an abstraction of individual fitness at the population level. Individuals vary in their fitness values, and to properly explain these patterns and commonalities of variation, we need to provide statistical explanations that relate to population-level properties. This abstraction is defined in terms of a population average, a statistical measure. As adherents of the Statisticalist School claim, trait fitness is a *mathematical consequence* (Walsh, 2015) of individual fitness, a *statistical effect* (Walsh, 2007) at the population level of what happens to organisms. As Walsh (2003, 464; emphasis in original) summarizes it, “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 view of natural selection is based on a One-Force Model (Walsh, 2019). Accordingly, the One-Force Model states that all causes of evolution lie at a single level of analysis: the level of the individual. As Walsh asserts, “[t]here is one level of causation; *all the causes of evolution are the causes of arrival and departure…* It is `proximate' causes all the way down” (Walsh, 2019, 238, 242, emphasis in the original); all causes of adaptive evolution affect the individual level. Evolution is a population-related consequence of what happens at the individual level. Such a consequence is analyzed statistically. The change in the structure of a population is *a higher-order statistical effect on the causes at the individual level* (Walsh, 2019).

4.2 The One-Force Model: Reformulating Mayr’s Dichotomy

It may seem that the Statisticalist School is a radical position. Indeed, it is usually supported by those who believe that the MS should be seriously revised (and not just amended). However, the Statisticalist School proposes a valuable link between the MS explanatory framework and the new proposals based on individual causation (the EES). To this end, the Statisticalist School advocates splitting explanatory efforts between the individual and population levels. Ariew (2003) dealt with the task of reformulating Mayr’s distinction: Since there is only one level of causation, the distinction between two causal levels of analysis is untenable. Instead, Ariew suggested, the appropriate division of levels of explanation is a division of *types of explanation*, not of *types of cause*, thus “the individual level causal vs. statistical level evolutionary distinction should replace Mayr’s proximate-ultimate distinction” (Ariew, 2003, 557). Individual-level analysis is devoted to understanding the causal processes in an organism over its lifetime. At the population level, the effects of individual-level processes on changes in the population are analyzed using statistical methods. Mayr’s dichotomy should not be changed by adopting the I-I strategy. We need to redefine the dichotomy not in terms of levels of causality, but in terms of different levels of explanation associated with different types of explanation.

As Ariew notes, each level of explanation has its own explanatory domain: causal explanations of development and statistical explanations of population change have different explanatory roles and different epistemic boundaries. For sure, “this is not to say that one type of explanation is more important than the other, but that they are two entirely distinct and irreducible forms of explanation” (Ariew, 2003, 563). Each domain (with its own type of explanation) is irreducible to the other: we cannot understand evolutionary processes only by studying the individual-level, and we cannot understand individual-level phenomena only by studying the population domain. To understand the adaptability of living systems, both types of explanations are essential: “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; emphasis added).[3]

4.3 Avoiding Fallacies

The Statisticalist position seems apt to avoid the explanatory fallacies discussed in this article. We cannot conflate proximate and ultimate causation simply because there is no such difference. We cannot explain development in terms of ultimate causation because there is no such thing as ultimate causation. None of the causes of development interact with evolution during phylogenetic processes. The ontogeny and phylogeny fallacies –as merging levels of causation– are not possible in the Statisticalist School. The role of population-based explanations is limited to the statistical analysis of population changes. Population-based explanations do not provide causal explanations. From a population analysis, we can derive generalizations or idealizations (Neander, 2017), averages (Boorse, 2002), and statistical ideas about what is normal or usual in a population (Millikan, 1989), but definitely not causal explanations (Rama, 2023). For the same reasons, Type 1 Inetractivism is not possible in this framework. Instead, we should adopt Type 2 Interactivism: Traits are produced by multiple interacting developmental resources at the proximate level.

However, it is important to note that in this framework we can still make explanatory errors that arise when we mix different domains of explanation. This situation is inevitable once we draw epistemic boundaries between explanatory domains. This situation is salutary in science: the possibility of mixing domains is inherent once we have domains of explanation. The problem with Mayr's distinction is not the distinction itself, but the fact that development remains harmless in evolutionary theory and that Type 1 Interactivism is usually (but not necessarily) used to explain development from the MS perspective. Likewise, it is not a fallacy to argue that there are proximate and ultimate causes; the problem is to support the interactionist picture of the I-I strategy. Finally, it is not a fallacy to separate proximate causal explanations and ultimate statistical explanations. If we take the statisticalist position, the fallacy is reformulated not in terms of levels of causal explanation but in terms of levels and types of explanations: we commit the fallacy when we provide statistical, population explanations to deal with proximate, individual causal phenomena. The relevant point, however, is that if we reformulate the fallacy from this perspective, we gain a valuable explanatory purchase. First, as mentioned above, the classical way of thinking about this fallacy (in terms of levels of causation) is not possible. Second, the possibility of falling into such a fallacy is certainly lower: if we accept that natural selection provides statistical answers to population questions, why would anyone use it to explain development? In other words, differentiating explanatory domains in terms of types of explanation prevents us from crossing their epistemic boundaries, whereas differentiating explanatory domains based on causal levels (as the I-I strategy does) tends to mix explanatory domains.

Importantly, developmental dichotomies can also be reformulated from a statistical perspective. One of the problems with developmental dichotomies is that they are intended to deal with how-questions by providing how-much answers. Answers based on developmental dichotomies are decoupled from an adequate explanation of causal mechanisms. We think that how-much-questions can be understood in statistical terms and that it describes population properties: How much variability is there in a trait in a population? The relevant point is that how-much-questions are different from how-questions as statisticalists intend: the former is a statistical problem of population biology, the latter is a causal problem of developmental biology, so it is clear that how-much-questions should not be confused with how-questions.

As mentioned in Section 2, the problems with developmental dichotomies are diverse, so we are not claiming that developmental dichotomies could be valid within the statisticalist framework. We just want to make clear

[3] An open question that, to my knowledge, has not yet been addressed by proponents of the Statisticlasit School is to find a proper place for lineage explanations in its taxonomy of explanatory domains (Calcott, 2009).

that the statisticalist framework is also suitable for explaining the difference between how-much and how-questions in terms of types of explanation. This might suggest that developmental dichotomies describe properties of populations: they attribute a statistical property to a trait within a population. This is certainly not enough to make developmental dichotomies valuable: When philosophers and scientists say that a trait is innate, they are usually saying more than just a statistical statement: they are talking about causes. Be that as it may, if one retains developmental dichotomies (they should be called something like "population dichotomies" indeed) as references to statistical properties of populations, one may be able to avoid erroneous uses of these categories.

4.4 Reciprocal Causation and Statistical Reciprocity: Towards Population Ontogenetics

Reciprocal causation should be viewed solely as a proximate phenomenon: individual organisms interacting with their environment. The question therefore arises as to how reciprocal causation affects the ultimate explanations from a statisticalist viewpoint. Or more generally, to include other processes at the individual level that cannot be reduced to genes: How does the integration of development and evolution influence ultimate explanations? An important point to make before answering this question is that the reason for defending the Statisticalist School is not that development is a causal factor in evolution. The Statsiticalist School is based on epistemological analysis of explanations for natural selection. Certainly, the Statsiticalist School is motivated by the EES (and related approaches) and intends to assign a central explanatory role to development. But in one very important respect, the Statisticalist School is quite compatible with the MS: we can accept statisticalism even if we adopt a gene-centered stance. In this picture, the evolutionary causes are still ontogenetic, and the explanations of populations are quantifications over ontogenetic causes. The subtle and essential difference is that, under the MS framework, ontogenetic processes can be reduced to the genetic level. Therefore, the core component of natural selection (inheritance, variation, and fitness) can be understood in genetic terms. Population biology becomes population genetics. The statistical calculation in population biology only captures the changes in the genetic composition of a population. Although this seems to contradict his view of 1961 and 1974 –in that the ultimate explanation is not causal– Mayr himself seems to accept a statistical position in other works: "Individuals, or any kind of organic entities, form populations of which we can determine the arithmetic mean and the statistics of variation. Averages are mere abstractions; only the individuals of which populations are composed have reality" (Mayr, 1959, 2).

If we understand this point, we can better answer our question of how the rejection of gene-centrism and the introduction of development in evolution modifies *ultimate explanations*. To answer this question from a statistical perspective, we must first answer the question of how the rejection of gene-centrism and the introduction of development in evolution change *proximate explanations*. We need to understand the proximate causes in order to calculate their effects on populations. In other words, gene-centrism allows certain explanations for population change (certain formulas, such as Fisher's Theorem or Price Equation (Queller, 2017)), while the rejection of gene-centrism may require other explanations for population change (new formulas, new theorems, new equations). The introduction of development into evolutionary theory means that the elements of natural selection cannot be understood in genetic terms: Variation, inheritance, and fitness are ontogenetic phenomena. This is where reciprocal causation finds the expected explanatory purchase: as an evolutionary cause, i.e. as part of the processes that influence natural selection by producing variants or novelties, influencing inheritance, or altering individual fitness.

If we include development as a causal factor of evolution (including genes, of course), we get something that could be called "population ontogenetics": the mathematical study of the statistical effects of ontogenetic processes on the population (Walsh et al., 2017), "a *rational form* in which *their effects* become fixed" (Engels, 1878, 82). Note that the difference between the genetic and ontogenetic views in population biology is based on the reducibility of the components of natural selection to the genetic level. But we can understand both views to mean that population biology is the mathematical study of the statistical effects of individual processes on the population. In this sense, population ontogenetics justifies the population-based thinking of the MS, while

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properly capturing the importance of ontogeny that EES thinks about. We still can say, paraphrasing Dobzhansky (1937), that “evolution is a change in the [onto]genetic composition of populations. The study of mechanisms of evolution falls within the province of population [onto]genetics.” The central question with regard to the integration of MS and EES is therefore: *What calculi are needed? What theorems do we need to understand changes in the ontogenetic structure of populations?*

This view is completely free of the explanatory fallacies discussed here. It avoids Type 1 Interactivism and does not conflate the how-much questions (statistical) with the how-questions (causal). Reciprocal causation is a proximate phenomenon. It concerns proximal causes beyond Mayr's genetic metaphors. Consequently, reciprocal causation also concerns ultimate explanations. However, it should not be seen as an alternative to Mayr’s dichotomy. Rather, reciprocal causality leads to a modification of population biology: We can refer to the population effect of reciprocal causation as "statistical reciprocity.” In this sense, there is a link between the two levels of explanation and different interpretations of reciprocal causation (Rama, forthcoming): one interpretation is that it is a proximate phenomenon, and the other interpretation, "statistical reciprocity", is that reciprocal interactions have statistical effects in population biology. Statistical reciprocity must therefore be included in the theorems needed to understand changes in the ontogenetic structure of populations. For example, in the context of niche construction, we can note that Odling-Smee's initial work was primarily aimed at showing that niche construction has consequences for the mathematical calculation of population change and that his efforts were directed towards the introduction of reciprocal interactions into population biology (Odling-Smee et al. (1996) or Day et al. (2003), see Wade and Sutan (2023) for a more recent approach in this direction). Statistical reciprocity shows that externalism is not sufficient for ultimate explanations: population ontogenetics must include the effects of reciprocal causality in its calculus.

5. Conclusion

This article was intended to provide a critical review of Mayr’s dichotomy. This goal was achieved in two steps. First, we reviewed some reviewers of Mayr’s dichotomy. We argued that a common strategy for integrating development and evolution is to claim that development interacts with evolution (the I-I strategy). As we have already pointed out with regard to interactionist explanations of development, interactionism in evolutionary theory “fills right [it seems as if we are integrating development into evolutionary theory], but it explains nothing [about how proximate causes affect ultimate explanations]” (Oyama, 1985, 159). The I-I strategy is explicitly adopted by those who claim that proximate and ultimate causation reciprocally interact. However, it is also important to analyze whether it is present in other fields aligned with the EES, such as evo-devo or extended inheritance theory.

The second step was to show an alternative view that is free of explanatory errors, the so-called Statsticalist School. Epistemic boundaries are not only relative to the level of analysis, but are also based on different types of explanation: population biology and developmental biology do not differ in their causal support, but in the type of explanation that each discipline provides. We have argued that this division of explanatory domains is apt to avoid explanatory fallacies and to appreciate the epistemological difference between how-much and how-questions. Finally, we have argued that reciprocal causation is entirely a proximal phenomenon. It motivates a reconsideration of Mayr’s distinction, but it cannot be regarded as an alternative. Rather, reciprocal causation affects proximal mechanisms and therefore has population-level implications: causal reciprocity between organism and environment must be mapped into “statistical reciprocity” in population biology. This view is free of the explanatory errors discussed here and is fully compatible with Type 2 Interactivism.

For sure, the integration of development and evolution is a more difficult task. Certainly, this project deserves further work and many questions need specific and well-developed answers. One of the difficulties is that the importance of individual and population explanations seems to be assigned to different biological theories: the

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EES and the MS, respectively. The issue is therefore not only how development and evolution are related, but also how these theories can be mixed coherently. Statisticalism seems suited to the task (Walsh, 2015, 2019), insofar as its reconstruction of Mayr's dichotomy preserves the types of explanations that each framework regards as crucial: the MS is mainly devoted to understanding population change through the use of mathematical methods, while the EES is mainly concerned with the study of developmental mechanisms. This appears to be a possible route to integration (Walsh and Rupik, 2023). However, it is important to point out that parsimony pluralism requires more than the delineation of explanatory domains. In particular, explanatory domains are not autonomous. The fact that the MS and the EES may be intended for different questions does not mean that their answers do not influence the other domain; after all, pluralism requires a common theoretical framework into which each perspective can fit. This is the aim of *population ontogenetics*: the EES changes the understanding of proximate causation beyond the gene-centered view, and it consequently affects population models of the MS; evolutionary causes cannot be fully captured by population genetics. A central open question is how the reconfiguration of proximate causation affects population biology: what formulas, equations, and calculations are needed to integrate development into ultimate explanations?

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