The Phylogeny Fallacy and Evolutionary Causation

Abstract: The use of evolutionary explanations to explain phenomena at the individual level has been described by various authors as an explanatory error, named the Phylogeny Fallacy. This paper will not address the debates about the fallacious nature of the Phylogeny Fallacy. Rather, I will analyze various positions regarding causal explanation in biology in order to evaluate which of them are prone to commit this fallacy. I will argue that this fallacious reasoning takes place within the framework of a particular stance on the causes of adaptive evolution in living systems, the so-called Two-Force Model, which holds that there are two sorts of causes involved in the explanation of adaptive evolution: individual and populational causation. The best-known Two-Force Model is adopted by the Modern Synthesis theory and is well represented by Mayr's distinction between proximate and ultimate causality. However, Mayr's distinction has been criticized and reformulated by new trends in biology that claim that proximate causes are also part of evolutionary explanations. Here I argue that many of Mayr's critics still support a Two-Force Model, and therefore tend to commit the fallacy. I introduce a different perspective on causal explanation in biology: the One-Force Model of the Statisticalist School, which claims that the only level of causation is the individual level; all causes of adaptive evolution are proximate causes. By ruling out two different levels of causation, I will argue that this framework can be an appropriate way to avoid fallacious explanations. Finally, I will apply the analysis carried out to a specific research program, teleosemantics.

Keywords: Phylogeny Fallacy; Developmental Dichotomies; Evolutionary Causation; Proximate/ultimate distinction; Statistical School; Teleosemantics.

1. Introduction

The boundaries of explanatory methods and fields of research are always a rich and controversial topic in science. This is also the case in biology. What is the scope of populational explanations? What can developmental biology tell us about evolution? What can we know about evolution if we understand developmental processes? Does physiology give us information about evolutionary adaptations? There are many problems behind these questions and, above all, there have been different answers throughout the history of biology.

This article focuses on one particular issue in the discussion about the epistemic boundaries of biological disciplines: the Phylogeny Fallacy. This term refers to an explanatory error in biology: the use of causal explanations at the population level to explain phenomena at the individual level. The Phylogeny Fallacy can occur when we assume a distinction between causal levels of explanation: individual causal explanations and population causal explanations. This demarcation between causal levels is associated with various developmental dichotomies related to the nature-nurture dichotomy, such as innate-acquired, inherited-environmental, and many others. While I will detail the explanatory mistake of the Phylogeny Fallacy and its relationship to the developmental dichotomies, I will not get into a detailed discussion of whether or not the Phylogeny Fallacy is actually a fallacy. This topic has been extensively discussed by various scientists from different disciplines such as philosophy, cognitive science, psychobiology, and biology. I will focus here on one particular topic: the relationship between the Phylogeny Fallacy and various positions on the causal structure of natural selection, a relationship that has not yet been explored in detail. I will relate the fallacy to a particular model of the causes of adaptive evolution: the so-called Two-Force Model (Walsh, 2019).

The structure of the paper is as follows. In Section 2, I introduce the Phylogeny Fallacy and explain various reasons why a particular explanation engages in this fallacious reasoning. In Section 3, I will show the connection between the fallacy and a particular position on the causal structure of evolutionary theory. I will argue that this fallacy is committed by those causal explanations that assume a Two-Force Model for the causes of adaptive evolution. The Two-Force Model is about distinguishing different levels of causation, the individual and the population level. I will refer to two different types of Two-Force Models. First, the model adopted by the Modern Synthesis and well represented by Mayr's proximate/ultimate distinction. It states that individual causation affects only a single life span and has no influence on population causation. Second, a different Two-Force Model is supported by recent trends in theoretical biology that assume that individual causation plays an important evolutionary role and that seek to criticize and reshape Mayr's distinction. In this framework, both levels of causation play a causal role in evolution. However, I will argue that both types of Two-Force Models can lead one to commit the Phylogeny Fallacy. In particular, I will point out that emphasizing the causal role of developing organisms in evolution *is not* sufficient to avoid fallacious explanations. Rather, it is necessary to abandon the Two-Force Model and move to a One-Force Model. The One-Force Model will be presented in Section 4 in the context of the Statisticalist School, a specific position in recent debates about the causal structure of natural selection. I will show how the Phylogeny Fallacy and other problems can be avoided by adopting this position. Moreover, the causal role of individual-level processes becomes even more relevant than in Two-Force Models. To conclude this paper, in Section 5 I analyze a specific case study, teleosemantics. The aim is to show how teleosemantic projects situated in a Two-Force Model systematically engage in the fallacious explanations of the Phylogeny Fallacy and

that, consequently, a One-Force Model for teleosemantics might be an important and warranted research project.

2. The Phylogeny Fallacy: Conflating Causal Explanations

The Phylogeny Fallacy was a term introduced by Lickliter and Berry in 1990. However, we have also found several areas of research that pointed to the presence of this fallacy in biology, such as Developmental Systems Theory –most notably in the work of Susan Oyama (Oyama, 1985), Developmental Psychobiology (Michel and Moore, 1995) and, more recently, Ecological Developmental Biology (Lewontin, 2000). The Phylogeny Fallacy is a conflation between different *biological explanations*; it is an explanatory error. Moreover, it is a mixing of different *levels of biological explanation*. To understand the fallacy, we must therefore first distinguish between two levels of explanation: Population-level explanations and individual-level explanations. Population explanations in biology describe how populations change in the course of phylogenetic history. Explanations at the individual level, on the other hand, explain how an organism changes in the course of its ontogenetic history. Another element in understanding the fallacy is that it is a conflation between different *levels of causal explanation*: between causal explanation at the population level and causal explanation at the individual level.

The explanatory error of the Phylogeny Fallacy lies in the fact that we cannot answer individual-level questions by giving population-level answers about evolutionary processes. Individual-level questions, which relate primarily to developmental biology, ask about the causal mechanisms that give rise to a trait. They require an explanation of how different processes and causes interact at different ontogenetic stages to produce a particular phenotypic outcome. Population-level explanations are concerned with the changes in the population over the course of evolutionary history that produce particular adaptive phenotypic outcomes. The nature of the causal explanations provided by the two levels of analysis is different: one level of analysis is concerned with the mechanism of development and the other with the evolution of populations. As Griffiths (2013, 29) notes, "an evolutionary explanation of a development mechanism is not the same thing as a mechanistic explanation of development" (Griffiths, 2013, 29): explaining that a particular developmental mechanism has evolved is not the same as explaining how that mechanism works in developmental processes; claiming that a trait has evolved does not inform us about the processes that build that trait during ontogeny; or "discovering that there is a "gene for" a trait or that the trait is "genetically encoded" will never be more than a starting point for the elucidation of an actual developmental mechanism" (Griffiths, 2013, 24). What do we know about developmental processes just because we say that a trait is the product of natural selection? As Oyama (1985, 159) stated, "[it] feels right, but it explains nothing." Thus, when we replace explanations of development with evolutionary explanations, we commit the Phylogeny Fallacy, a

misleading line of reasoning. A theory commits the Phylogeny Fallacy when evolutionary explanations come into play to explain individual-level phenomena, i.e. biological phenomena that do not fall within its explanatory scope.

The blending of evolutionary and ontogenetic explanations is not new in the history of biology. Since the early days of evolutionary theory, the (fallacious) argument –arguing evolutionary explanations are sufficient to explain developmental phenomena– has been constructed (Keller, 2010). This position was clearly defended by Haeckel (1866, 7): "The theory of descent alone can explain the developmental history of organisms." Nowadays, however, the characterization of the Phylogeny Fallacy in terms of causal explanations can be understood and is usually discussed in the context of the classification of biological causes proposed by Ernst Mayr (1961, 1974). Evolutionary biology is concerned with evolutionary causes –so-called *ultimate causation*– and developmental biology and physiology (or "functional biology" in Mayr's terminology) with *proximate causes*. He also argued that each kind of cause belongs to different explanations with different explanatory tasks. Ultimate causality explains *why-questions*: why are biological systems organized in a particular functional and adaptive way? Proximate causes explain *how-questions*: how different parts of a living system interact to produce a functional or adaptive outcome. Later, in Sections 3 and 4, I will engage in a discussion of the validity of this dichotomy. Until then, Mayr's distinction can help us illustrate the fallacy: The fallacy consists in conflating ultimate causation with proximate causation; it consists in explaining how-questions by giving why-answers. As Lickliter and Berry (1990, 349) assert:

> This conceptual dichotomy [between proximate and ultimate causation] is a deeply engrained habit of thinking and is characterized by the belief that aspects of development are determined by either (a) events which occurred earlier in the development of the individual, or (b) preontogenetic factors which operated on the ancestors of the individual. We term this conceptual framework, with its implicit predeterminism, the "phylogeny fallacy."

Different theories can commit the Phylogeny Fallacy. However, the most common theories subject to this fallacy are those that invoke *developmental dichotomies* inherent in the nature-nurture debate: the (explicit or implicit) use of the nature-nurture dichotomy (and related dichotomies) to explain the development of traits. There are several interrelated dichotomies used in explanations of development, as shown in Table 1. Indeed, the central arguments that have pointed to the explanatory error of the Phylogeny Fallacy have come from embryologists and developmental psychobiologists who have challenged the instinct and nativist theories of ethology (Rama, 2018). The most important players are Zing Yang Kuo (1921, 1922) at the beginning of the last century,

Lehrman (1953) at the middle of the twentieth century, and Gilbert Gottlieb (1997) at the end of the century. The classical nativism and instinct theory in ethology was proposed by Konrad Lorenz. Indeed, we can also introduce the distinction between levels of explanation by invoking Lorenz's distinction between instinct and acquired behavior: instinct behavior is explained in one way, acquired behavior in another. According to Lorenz, each type of behavior is explained by different causal learning processes. On the one hand, we have evolutionary learning processes in which natural selection drives variation in the direction of adaptive behavior. On the other hand, ontogenetic learning regulates ontogeny on the basis of environmental cues and behavioral feedback. Instincts are explained by phylogenetic learning processes, while acquired traits are explained by ontogenetic learning. The explanatory distinction that Lorenz's theory makes is also a distinction between causal levels of explanation, and therefore the possibility of engaging in the fallacious reasoning of the Phylogeny Fallacy is given: The fallacy is to use explanations based on phylogenetic learning to explain individual-level processes. As shown in Table 2, there are several ways to distinguish between the individual and population levels.

Individual Level	**Populational Level**
Learned	Innate
Acquired behavior	Instinct behavior
Environmental	Inherited
Cultural	Biological
Nurture	Nature
Plastic	Fixed

Table 1: Developmental dichotomies. Several dichotomies are at play in explaining development. The left-hand side refers to phenotypes that are influenced, caused, or explained by processes at the individual level, and the right-hand side concerns phenotypes that are influenced, caused, or explained by processes at the population level.

Individual Level	**Populational Level**
Proximate causes	Ultimate causes
Functional Biology	Evolutionary Biology
How-questions	Why-questions

Ontogenetic Learning	Phylogenetic Learning

Table 2: The demarcation between Individual and Populational levels in biological explanations.

Following Lorenz or other theories that advocate developmental dichotomies, the development of some traits can be explained by evolutionary theory, while other traits are explained by development, by proximate explanations. Instincts or innate traits are said to depend not on ontogeny but on phylogeny, while acquired traits are a product of developmental processes. Note that the reason why the use of developmental dichotomies entails committing the Phylogeny Fallacy is that developmental dichotomies are based on a demarcation between causal levels of explanations. In other words, as can be seen in Table 1 and Table 2, developmental dichotomies are based on the dichotomous view of causal factors in biological explanations. Thus, the explanatory logic behind developmental dichotomies is to separate the causes of phenotypic outcomes. These dichotomies are also introduced in a subdivision between the levels of causal explanations. Some traits are innate, inherited, and biological, i.e. part of the nature of the species; they are caused by the history of the species. Other traits are learned through environmental inputs, so they are part of our nurturing; they are caused by the history of individuals. Some traits are caused by the evolution of populations, while others are caused by the ontogenesis of individuals.

Developmental dichotomies have been strongly criticized in recent biological theories. There are several problems. For instance, developmental dichotomies may constitute a *semantic clutter* (see Mameli, 2007, 2008; Mameli and Bateson, 2006, 2011; Bateson and Mameli, 2007; Griffiths, 2002; Wimsatt, 1986; Lorenzo and Longa, 2018) insofar as it seems that there are plenty definitions for each developmental dichotomy (e.g. Wimsatt (1986) had reported 28 different meanings of the word “innate”). It has also been argued that developmental dichotomies are usually based on some *empirical inadequacies* (see Oyama et.al., 2001; Michel and Moore 1995; Gottlieb, 1997; Lewontin, 2000), such as the notion of genes as the only source of information in development, the adoption of a purely genetic view of inheritance, or the support for a theory of phenotypic variation exclusively based on chance. However, these controversies are not our point of discussion. Instead, many authors have argued that the use of developmental dichotomies entails a fallacious argumentation —the Phylogeny Fallacy (Griffiths, 2013; Lickliter and Berry, 1990; Michel and Moore, 1995; Lorenzo and Longa, 2020, Rama, 2022; Oyama, 1985).

As mentioned earlier, the problem with using developmental dichotomies is that we do not say much about the causal processes that produce a trait just because we say it is an innate or evolved trait; dichotomies are explanatorily vacuous when we are looking for the mechanisms of development. This is the explanatory error of the Phylogeny Fallacy: using a populational causal explanation (e.g. that a trait *x* is innate) to explain a developmental question: how *x* develops. This was the central idea advocated by Kuo a century ago: "To call an acquired trend of action an

instinct is simply to confess our ignorance of the history of its development" (Kuo, 1921, 650). We say nothing about developmental mechanisms when we say that a trait is an instinct, innate, or inherited. If we want to explain developmental processes, it seems that looking into evolution is a pointless strategy to explain developmental processes: "The use of the distinction generates in researchers the false illusion that certain important empirical questions have already been answered" (Bateson and Gluckman, 2011, 129). In this sense, the evolutionary explanation of development is not, in fact, a proper explanation of development. As Griffiths and Stotz (2013, 23) recently argued: "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".

Sometimes the Phylogeny Fallacy is not seen as a fallacious reasoning if we accept that all traits result from the interaction of different causes. This position could be called interactivism. However, it is important to distinguish between two types of interactivism. *Type 1 Interactivism* –also called "consensus interactivism"– holds that traits arise from the interplay of evolutionary causes (emanating from the populational, pre-ontogenetic level) and ontogenetic causes (emanating from the individual, ontogenetic level). However, this interactivism is still problematic: even if we argue that the causes interact, we are still dealing with two different levels of causal explanation: "This 'interactionist consensus,' however, perpetuates the nature–nurture debate by maintaining its inherent dichotomy" (Stotz, 2008, 360). In sum, those who assume Type 1 Interactivism are still committing the Phylogeny Fallacy. Instead, *Type 2 Interactivism* proposes a comprehensive framework for understanding the development of traits, emphasizing the interplay of multiple levels of organization, including genes, genomes, cells, tissues, and the environment. Type 2 Interactivism states that all traits emerge from complex interactions among various proximate causal factors; it emphasizes the importance of proximate causes and their interactions throughout ontogeny in shaping developmental outcomes. By focusing on these proximate causes and their interactions, Type 2 Interactivism aims to provide a more adequate understanding of developmental mechanisms; it advocates for a holistic approach to understanding development, emphasizing the interconnectedness of various causal factors beyond simplistic dichotomies and evolutionary explanations. As developmental psychobiologist Gilbert Gottlieb said, "[t]he developmental analysis begins where the nature-nurture debate ends" (Gottlieb, 1992, 157-8).

3. The Phylogeny Fallacy in the Two-Force Model

We have seen that the Phylogeny Fallacy is prone to occur when we divide between levels of causation: individual-level causes and population-level causes. The delineation of these two sources of causes is known as the Two-Force Model (Walsh, 2019). However, the Two-Force Model has been interpreted in different ways. The first, developed by the Modern Synthesis (henceforth: MS),

denies any role for individual causation in evolutionary theory. The second, advocated by some critics of the MS, claims that both levels of causation are involved in explaining adaptive evolution. The debate about the causes of adaptive evolution has been intense in recent years. The influential Mayr's dichotomy has been revisited to examine the relationship between proximate and ultimate causation. I will present the two positions on the Two-Force Model in terms of understanding proximate and ultimate causation. I will argue that recent efforts to assume reciprocal causation between populational and individual causes are insufficient to avoid the Phylogeny Fallacy. In Section 4, I will argue that Mayr's proximate/ultimate causation should be abandoned if we aim to avoid the Phylogeny Fallacy.

3.1. Ultimate Causation and the Two-Force Model

In the context of the theory of the MS, the Two-Force Model receives a special interpretation. This is, in fact, the interpretation of causal explanations proposed by Mayr (a central architect of the MS). The core idea is that population-related causes are involved in explaining adaptive evolution. Population-based causes provide the ultimate explanation for the adaptive nature of living organisms. However, as an advocate of the Two-Force Model, Mayr does not ascribe the same explanatory role to proximal, individual causes: causes at the individual level, which act during the development of the organism, play no role in evolution. According to this picture, proximate causation plays no role in evolutionary explanations of population change. This has contributed to the emergence of the so-called black box of development: the assertion that developmental processes involving proximate causes do not need to be understood to explain adaptive evolution.

The black box of development has been built over central tenets of the MS, such as the exclusion of Lamarckian modes of inheritance, a robust Genotype-Phenotype map, or the unbiased nature of variation. While Darwin anchored the idea of natural selection as an adaptive process in evolution, the MS's interpretation of Darwinism (i.e. neo-Darwinism) focused only on the genetic level. A key step towards a pure population perspective of evolution was the reduction of the core components of natural selection to the genetic level. Natural selection occurs in populations when there are *heritable variations in fitness* in the population. Firstly, inheritance is completely restricted to *genetic* inheritance systems, neglecting the possibility of extended forms of inheritance. Variation is understood to be the emergence of unbiased traits as a result of random *genetic* variation. Finally, it is assumed that there is a strong correlation between genetic variation and phenotypic variation (i.e. developmental outcomes are genetically determined) so that the fitness of phenotypic outcomes can be traced back to their *genetic* basis. According to this paradigm, causal explanations of adaptive evolution fall within the domain of population genetics: causal explanations of adaptive evolution refer to the evolutionary history of a population of genes. To understand evolution, it is enough to

see what is going on at the genetic level, or as Maynard Smith said: "It is possible to understand genetics, and hence evolution, without understanding development" (Maynard Smith, 1982, 6).

In this scenario, however, it is not assumed that the proximal cause influences adaptive evolution. Rather, the ontogenetic causes that produce adaptive changes do not influence evolutionary processes. The causal effects of adaptive development do not extend beyond a single lifespan. As mentioned above, this is a consequence of the central tenets of the MS. First, if any kind of Lamarkism is denied, the epigenetically induced variants would not be inherited and therefore have no effect on evolutionary change. Furthermore, the assumption that the Genotype-Phentype Map is robust (i.e., that phenotype outcomes are specified by genetic inputs) must mean that phenotypic variation affecting selection pressure is found at the genetic level. Proximate causes play no role in evolutionary explanations in this framework: acquired traits do not affect evolution. As mentioned above, this Two-Force Model of the MS is related to the neglected role of developing organisms in evolutionary theory. Consequently, as Brian Goodwin (1994, 1) claims,

> Something very curious and interesting has happened to biology in recent years. Organisms have disappeared as the fundamental units of life. In their place we now have genes, which have taken over all the basic properties that used to characterize living organisms [...] Better organisms made by better genes are the survivors in the lottery of life.

As mentioned above, Mayr's delineation of ultimate and proximate causation (and his interpretation of these causes) fits the Two-Force Model of the MS. As Brown (2020, 4) puts it, "Mayr's dichotomy reflects a mid-twentieth-century consensus within evolutionary biology, sometimes referred to as the MS, regarding the basic mechanics of evolution and the explanatory adequacy of population genetics." In this sense, proximate cause analysis refers to the "decoding of the programmed information contained in the DNA code" whereas evolutionary explanation looks for the emergence of genetic codes during population change –i.e. "the laws that control the changes in these codes from generation to generation" (Mayr, 1961, 1502).

3.2. Proximate and Ultimate Causation in the Two-Force Model

For our current discussion, however, it is important to note that various tenets of the Modern Synthesis are currently controversial. In the contemporary philosophy of biology, there is an ongoing debate about the foundations of evolutionary biology. In this context, there are new approaches that are more or less opposed to the foundations of the MS. The most consolidated proposal comes from the *Extended Evolutionary Synthesis* (Pigliucci and Müller, 2010; Laland et al., 2015; Huneman and Walsh, 2017): 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 the

explanation of natural selection processes solely in terms of the population of genes entails simplified and incorrect assumptions about biological processes and leads to many biological phenomena that are not properly explained; as a result, "[t]he black box [of development] is now being opened to provide a more complete picture of what really happens" (Bateson & Gluckman, 2011, 17). For example, extended inheritance systems have been found in all living systems (e.g. Jablonka and Lamb, 2014). The genetic view of inheritance mischaracterizes inheritance processes and does not properly account for epigenetically mediated cross-generational 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 Gentype-Phentype Map becomes 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 several sources of adaptively biased phenotypic variation involved in selection processes, such as plasticity, niche construction, or self-organization (e.g. West-Eberhard 2003). I will not get into the discussion of the various biological processes involved in the revision here. Rather, I will discuss below the implications of this extended framework for the distinction between proximate and ultimate causation. The most important 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 (Baedke, 2018; Bateson, 2005; Huneman, 2010; Nicholson, 2014).

As expected, this extended view has fostered several critical revisions of Mayr's ultimate-proximate distinction (Laland et al, 2011, 2013; Brown, 2020; 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). As a result of this revision, many have adopted a different Two-Force Model. I will call it the *Extended Two-Force Model*, which is motivated by (some of) those theories that seek to extend the MS framework. However, I will argue that the Extended Two-Force Model also tends to commit the Phylogeny Fallacy. So let us see in what ways the revision of proximate and ultimate causation can still be problematic.

There are several reasons for arguing 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 main motivation is that those proximate phenomena that cannot be understood through a gene-based explanation can influence evolutionary dynamics (Laland et al., 2011). Organisms and their selection pressures interact in a reciprocal and non-random way: selection pressure is not the only causal force, but organisms also

deal with their living conditions and therefore change their selection pressure. Debates on *reciprocal causation* point to the importance of causal relationships between organisms and their selection pressures in the context of evolutionary theory (Lewnotin, 2000; Baedke et al., 2021; Svensson, 2018; Buskell, 2019). For example, niche construction processes alter environmental conditions and modify the fitness outcomes of individuals; phenotypic plastic changes respond adaptively to the external and internal living conditions of organisms; symbiotic and cooperative relationships also demonstrate the reciprocal interaction between living systems and their environment, as do many other phenomena associated with the Extended Evolutionary Synthesis. Critics of Mayr's distinction propose various alternatives, from abandoning the concept of "ultimate" explanation (Haig, 2013), to diversifying the various biological explanations beyond Mayr's dichotomy (Calcott, 2013), to reinterpreting the nature of the answers given by each level of causal explanation (Scholl and Pigliucci, 2014), or diversifying evolutionary causes without adopting Mayr's position (Laland et al, 2013), among other possible positions (in Section 4 we will see a further reinterpretation of Mayr's distinction). However, the general conclusion reached by 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).

How should we understand the causal role of organisms in evolutionary theory in light of recent challenges to the MS? One possible answer is to adopt an Extended Two-Force Model: claiming that Mayr's proximate-ultimate distinction deserves evaluation, but without abandoning the Two-Force Model. 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 forces. This position entails a different Two-Force Model than that of the MS. As already explained, in the MS, developmental causes do not influence evolutionary dynamics. What happens during the development of an organism has no effect beyond the creation of that organism. However, as soon as individual causation is *also* established as a causal factor in adaptive evolution the Two-Force Model of the MS changes.

Certainly, not all critics of Mayr's distinction explicitly (but implicitly they usually do) endorse this Extended Two-Force Model. However, this extended view is a common way to understand the challenges to the MS: "to deny the strong causal autonomy entailed by the proximate-ultimate distinction" (Brown, 2020, 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, 2016, 1). "[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; each level of causation is not autonomous and independent from the other. 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).

The Extended Two-Force Model is an answer to the question of the explanatory role of development in evolutionary theory. Denis Walsh (2003, 2007a, 2015, 2019) has been critical of this answer. We will see in the next section that Walsh and colleagues develop an alternative position. However, what is important for our discussion is that the problems remain even if we agree with this position: The assumption of an Extended Two-Force Model still supports a distinction between levels of causation, and therefore, there is the possibility of engaging in the fallacious reasoning of the Phylogeny Fallacy. In other words: If we still hold to a Two-Force Model, it is not enough to reconsider the role of development in evolution to avoid the Phylogeny Fallacy. The Extended Two-Force Model assumes that both ultimate and proximate causation are relevant for evolutionary explanations. However, as mentioned above, this view can lead us to adopt the problematic position of mixing causal levels of explanation. Given the explanatory error of the Pylogeny Fallacy, this position is still problematic: the Phylogeny Fallacy is not a problem specific to the MS view of evolutionary causes (expressed in Mayr's view of proximate-ultimate causes), but a problem of any position that advocates a Two-Force Model for the causes of adaptive evolution.

Moreover, as explained earlier, the Extended Two-Force Model is a particular position about the palace of development in evolutionary theory. As mentioned earlier, this position assumes that developmental causes *interact* with evolutionary causes. This position was labeled Type 1 interactivism in Section 2, and we say that this interactivism is problematic in that it also leads us to a fallacious argumentation. If we assume that adaptive evolution is due to the *interaction* between individual and population causation, we are committed to the idea that adaptive traits result from a mixture of causes.

Furthermore, there is another problem with the Extended Two-Force Model, namely the *Ontogeny Fallacy* (Hochman, 2012). This fallacy also involves the conflation of causal levels of explanation. In order to commit this fallacy, we must therefore first assume a distinction between causal levels of explanation. The Ontogeny Fallacy is the inverse of the Phylogeny Fallacy: the invocation of individual, proximate causal explanations to explain population-level phenomena. In this case, the fallacy occurs when we replace evolutionary causal explanations with ontogenetic causal explanations. The reason why the Ontogeny Fallacy involves a fallacious argumentation is the same

as the Phylogeny Fallacy: the explanatory framework of a particular explanatory level is exceeded. The main problem here is that explaining what goes on during an individual's lifespan is completely different from understanding how populations change over the course of history. Ontogenetic explanations of proximate causes cannot replace population-based explanations.

Remarkably, the revival of organismic causation and the role of epigenesis in evolutionary theory, as noted by Hochman, may suggest that developmental answers may be appropriate for evolutionary questions. The Ontogeny Fallacy may occur within the framework of an Extended Two-Force Model: If we assume that evolutionary explanations are explained by both individual and population causes, we mix different levels of explanation; in this case, we attribute an explanatory role to individual causal explanations that goes beyond their explanatory scope. However, within the Two-Force Model of the MS, we cannot engage in this misleading reasoning because proximate causes are not involved in evolutionary explanations.

The first conclusion of my analysis, then, is that any theory that assumes a Two-Force Model is vulnerable to the fallacious reasoning of the Phylogney Fallacy. This fact has already been well recognized in the context of the Two-Force Model of the MS. However, we have seen here that reframing the distinction between proximate and ultimate may still face the problem of Phylogeny Fallacy. This is the case with the Extended Two-Force Model: if the extended view of evolution that advocates organismic causation is still domiciled in a Two-Force Model, then we are in danger of engaging in the fallacious reasoning of the Phylogeny Fallacy (and the Ontogeny Fallacy too). This conclusion is relevant when we consider some of the reasons why the MS has been challenged. The distinction between nature and nurture was well established in the natural sciences by the end of the 19th century and was integrated into evolutionary theory during the development of the MS, thanks to various principles forged in that era. The rebirth of organismal causation challenged this doctrine. The most important message, however, is that avoiding the fallacy is not just about questioning the MS and claims about the introduction of development as part of evolutionary thinking. The Phylogeny Fallacy is not a problem with the MS itself, but with any theory that supports a Two-Force Model. Thus, if we are to overcome the dichotomous view of nature and nurture and the Phylogeny Fallacy inherent in this dichotomy, we must shift the role of organismal causation beyond the Two-Force Model. So how can we support organismal causation and the causal role of development in evolution without engaging in these fallacious arguments? The answer to this question requires the adoption of a One-Force Model on the causes of adaptive evolution.

4. The Phylogeny Fallacy in the One-Force Model

There is an alternative to the Two-Force Model: the One-Force Model. The One-Force Model is a different answer to the question of the explanatory role of developing organisms in evolutionary theory; it is a different way of understanding development in evolution that goes beyond the Two-Force Model. As I will explain later, the One-Force Model, though it may seem a radical position, 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* as in the Two-Force Model. Each kind of explanation is associated with different explanatory goals and different levels of explanation. I argue that the distinctions made by the One-Force Model are essential to avoid the Phylogeny and Ontogeny fallacies.

4.1 The Statisticalist School: Proximate Causation and the One-Force Model

In general, the One-Force Model is supported in the context of an ongoing dispute about the causal structure of natural selection (see Pence (2021) for a recent introduction to the topic and key references). On the one hand, the Causalist School (drawing primarily on the work of Elliot Sober (1984, 2013)) holds 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 population level, but at the individual level. *There is no such thing as populational causation*. The explanation of natural selection provides *statistical explanations* for the effects of individual-level causes in population dynamics. Without developing a detailed defense of the Statisticalist School, let us present its basic principles.

The Statistical School's proposal revolves around the concept of *fitness*, which is central to any explanation of natural selection: populations change due to differences in fitness, as Darwin taught us (Ariew and Lewontin, 2004). However, the statistical reading of natural selection begins by emphasizing the difference between two notions of fitness: *trait fitness* and *individual fitness*. The former refers to thinking in terms of populations and was introduced during the emergence of the Modern Synthesis through the famous mathematical insights into evolutionary theory by Fisher, Wright, and Haldane. In this sense, trait fitness concerns the fitness values of a population; 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 connected with different types of explanations associated with each concept of fitness. 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 particular

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, while individual fitness reveals the causes of an organism's survivability and reproductive capacity: "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 the individual fitness of the members of the population 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, 2007b) at the population level of what happens to organisms. As Walsh summarizes it,

> In short, natural selection occurs only when the relative frequency of trait types changes in a population as a consequence of differences in the *average* fitness of individuals in different trait-classes. This is what we call the statistical interpretation of natural selection (Walsh, 2003, 464; emphasis in the original).

This view of natural selection is based on a One-Force Model. Accordingly, the One-Force Model states that all causes of evolution lie at a single level of analysis: the individual level. As Walsh claims, "[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. The picture that emerges from this is that 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 Delimitation of Explanatory Tasks: Reformulating Mayr's Dichotomy

It may seem that the Statisticalist School is a radical position. Indeed, the One-Force Model is usually supported by those who believe that the MS should be seriously revised. However, the

Statisticalist School proposes a valuable link between the explanatory framework of the MS and the new proposals based on individual causation. To this end, the Statisticalist School advocates a division of 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 subdivision of levels of explanation is a subdivision of *kinds* of explanation, not *causal* explanation. Under this line, he argued, "the individual level causal vs. statistical level evolutionary distinction should replace Mayr's proximate-ultimate distinction" (Ariew, 2003, 557). Analysis at the individual level is devoted to understanding the causal processes in an organism during its life span. At the population level, the effects of processes at the individual level on changes in the population are analyzed using statistical methods. Mayr's dichotomy should not only be modified by extending the Two-Force Model of the MS. We need to redefine the dichotomy not in terms of levels of causation, but in terms of different levels of explanation associated with different kinds of explanation.

Crucially, Ariew associates the difference between kinds of explanation and levels of explanation with different explanatory tasks: Causal explanations of development and statistical explanations of population change have different explanatory roles, which are associated with different levels of explanation. For sure, "[t]his 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 explanatory strategy) is irreducible to the other: we cannot understand evolutionary processes by examining causes at the individual level, and we cannot understand phenomena at the individual level by examining the population domain. In order to understand the adaptability of living systems, both types of explanations are essential:

> On my view evolutionary explanations are statistical explanations of population-level phenomena to be distinguished from `proximate' or individual level causal explanations. 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; emphasis added).

The partitioning of the explanatory tasks of the One-Force Model is crucial to avoid both the Phylogeny Fallacy and the Ontogeny Fallacy. Moreover, by assigning a specific explanatory role to each level of analysis, it suggests a possible mediating and systematic way to understand the relationship between the Modern Synthesis (which is primarily devoted to population biology) and

emerging trends in theoretical biology that address the complexity and adaptability of developing organisms (Rama, forthcoming).

4.3 Avoing Fallacies

As far as the Phylogeny Fallacy is concerned, adopting the One-Force Model avoids the possibility of engaging in the misleading reasoning of the Phylogeny Fallacy. This becomes clearest when one realizes that we cannot mix different levels of causal explanations because there are no different levels of causal explanations. There is only one level of causal explanation: the individual level. In this context, the division of explanatory tasks refers to the kind of explanation, not the kind of cause. As mentioned above, the Statisticalist School assumes two types of explanation required to understand adaptive evolution. Thus, if we adopt the statisticalist view and support a One-Force Model, the Phylogeny Fallacy cannot occur. The Phylogeny Fallacy as a mixture of population and individual causation is not possible in the context of 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, 2017b), averages (Boorse, 2002), and statistical ideas about what is normal or common in a population (Millikan, 1989), but definitely not causal explanations.

Moreover, and for the same reasons, the division of explanatory tasks also helps to avoid the Ontogeny Fallacy. That is, the Statsticalist School not only neglects the possibility that populational causes override individual-level explanations but also ascribes an irreducible role to populational explanations, as mentioned in the previous subsection. The clear distinction between levels of explanation and their corresponding explanatory role proves that proximate causal explanations cannot replace the explanatory role of population-level explanations. The statistical analysis of population biology developed during the development of the MS is crucial to understanding how populations –not individuals– change throughout history. Advocating an organismic view of evolutionary causation does not mean neglecting the need for population thinking.

Another positive point of the One-Force Model is that it is perfectly compatible with Type 2 Interactivism, but not with Type 1 Interactivism. As mentioned above, Type 1 Interactivism –and its connection to the Phylogeny Fallacy– can even be defended in the context of an Extended Two-Force Model. However, once we abandon populational causes, Type 1 Interactivism no longer makes sense: populational causes do not interact with individual causes. On the contrary, Type 2 Interactivism seems to be motivated by the One-Force Model. Accordingly, various causes can

interact in the development of phenotypes, but all these causes belong to a proximate, individual level of analysis. The causal explanation of individual processes involves only proximate causation.

4.4 Interim conclusion

This essay aims to analyze the relationship between two central issues in the contemporary philosophy of biology. The debate about the causal structure of natural selection and the Phylogeny Fallacy. While the nature-nurture debate and the Phylogeny Fallacy arose at the birth of modern evolutionary thought (Keller, 2010), recent trends in theoretical biology that emphasize the role of individual causation in evolutionary theory still assume an Extended Two-Force Model. However, we have found that the commitment to a Two-Force Model tends to provide fallacious explanations for the explanatory scope of each causal level of explanation. If the Phylogeny Fallacy and Type 1 Interactivism are to be avoided, we need to rethink the causal structure of natural selection to move beyond the Two-Force Model.

The second set of results arises if we adopt the One-Force Model of the Statisticalist School. The Phylogeny Fallacy cannot occur under this perspective. We cannot rely on Mayr's distinction between two kinds of causes, since there is only one kind of causality: proximate causes. The division of explanatory domains (with their own explanatory tasks and methods) helps to avoid the Phylogeny Fallacy: Statistical analysis of population dynamics cannot provide a causal analysis of the mechanism of development. Moreover, the One-Force Model also avoids the Ontogney Fallacy: the analysis of developmental mechanisms cannot serve as a substitute for an adequate explanation of changes in populations. As mentioned above, the key step is that the division into explanatory kinds is associated with corresponding explanatory domains: each level of explanation has its own irreducible explanatory task. Fallacies would be avoided if we advocate that "evolutionary and proximate (more precisely, individual-level causal) explanations are distinct, irreducible, and both indispensable. Both answer different questions in ways that cannot be answered by referring to the other" (Ariew, 2003, 560).

To conclude this paper, I will analyze a specific case study: teleosemantics, an important theory in the philosophy of mind and cognitive science. I will see how the analysis of the Phylogeny Fallacy and evolutionary causality can be applied to teleosemantics. In particular, I will show how mainstream teleosemantics projects that advocate a Two-Force Model systematically engage in the fallacious argumentation of the Phylogeny Fallacy. Moreover, I will argue that a One-Force Model of teleosemantics –which is largely unexplored– could be an important new framework for a teleosemantic program.

5. The Phylogeny Fallacy in Teleosemantics

5.1 Mainstream Teleosemantics and the Phylogeny Fallacy

Teleosemantics is a central area within the philosophy of cognitive science, mainly concerned with understanding representational content from a naturalistic point of view (see Papineau (2017) and Schulte and Neander (2022) for introductions to teleosemantics). I will analyze teleosemantics for three reasons. First, I will argue that mainstream teleosemantics engage in the fallacious reasoning of the Phylogeny Fallacy. Second, teleosemantics will help to clarify the connection between the Phylogeny Fallacy and the various positions regarding the causal structure of natural selection. Finally, I will suggest that adopting a One-Force Model might help us avoid the Phylogeny Fallacy in a teleosemantic project.

The naturalistic root of teleosemantics comes from biology itself. A classic problem in the study of mind and behavior concerns the possibility of understanding the normativity of mental representation from a naturalistic standpoint. Several old questions revolve around the normativity of content and its centrality in dealing with mental processes as a phenomenon governed by internal, intentional, and goal-directed states. Without going into these complex issues, the strategy of a teleosemantic project is to understand normativity in the science of mind in terms of biological normativity. In other words, to apply a biological concept of normativity and natural functions to the realm of the mind. A representational system must function according to the biological functions it embodies, and the norms of representational systems (what the representation *must* refer to) are based on this biological functionality.

Depending on which biological functional theory one chooses, different teleosemantic projects can emerge. I refer to those teleosemantic theories that support the *Selected-Effect Theory of Functions* (SETF) to explain representational content as *mainstream teleosemantics* (see Neander, 1991; Millikan, 1984; Papineau, 1984). The core idea of SETF is that the function of a trait is defined by its evolutionary history: A phenotype must do what it was selected for by natural selection. The history of populations (the history of trait types) explains the function of current trait tokens. In this sense, my visual representation system works well when it represents the world the way the visual systems of my ancestors did. Mainstream teleosemantics is a rich framework for understanding mental representations from a naturalistic perspective. It has several advantages and applications that still make it the most prominent naturalization project of intentionality. However, we have two reasons to argue that mainstream teleosemantics engages in the fallacious argumentation of the Phylogeny Fallacy.

The first and simplest reason is the ubiquitous use of developmental dichotomies in the teleosemantic literature. The result is a classic view according to which some representations are innate and others are learned. In Table 3 we see various developmental dichotomies found in the core figures of teleosemantics. When mainstream teleosemantic theories attempt to explain the normativity of traits by appealing to these dichotomies, they commit the explanatory error of resorting to population-level phenomena to explain the normativity of individuals. The critique of developmental dichotomies analyzed in Section 2 applies to any project that includes developmental dichotomies in its explanations, and thus also applies to mainstream teleosemantics.

Developmental Dichotomies	**Reference**
Innate perceptual–cognitive mechanisms	Millikan (2006, 109)
Genetically programmed systems	Millikan (2000a, 86)
Innate skills, abilities	Millikan (2000b, 54. 63, 65)
Genes coding for behavior	Dretske (1988, 123, 125)
Behavior causally explained by the genes inherited	Dretske (1988, 92)
Innate behavior	Dretske (1988, 123)
Rigidly programmed behavior	Dretske (1988, 125)
Innate sensory-perceptual systems	Neander (2017a, 166)
Innate capacities	Neander (2017a, 82, 101)
Innate information, representations	Neander (1995a, 111-112)
Innate, hard-wired belief-forming abilities	Papineau (1984, 557)

Table 3: Developmental dichotomies in mainstream teleosemantics. A non-exhaustive list of developmental dichotomies found in mainstream teleosemantic projects. Reprinted with permission from Rama (2022).

In addition to these explicit uses of developmental dichotomies, however, there is another important source of explanations in teleosemantics that deal with a mixture of population-level and individual-level explanations: the distinction between *phylogenetically selected functions* and *ontogenetically selected functions*. This distinction is also frequently used in the teleosemantic literature. Let us explain the puzzle here.

As mentioned above, mainstream teleosemantics advocates an evolutionary and population-based perspective on biological functions: A trait must do what it was selected for in evolution. However, ontogenetic functions are also part of mainstream teleosemantics. The commitment to ontogenetic functions is usually related to the inadequacy of solving certain problems that arise from a purely evolutionary perspective. In other words: as teleosemanticists recognize, not all representations can be adequately explained by an evolutionary perspective alone. There are at least three problems that teleosemantics attempts to solve with ontogenetic functions (Papineau, 2017). (I) *The problem of variation:* How is it possible that there are different representational capacities within one and the same species (e.g. between cultures)? II) *The problem of novelty:* How can new representations arise in humans (e.g. ELECTRON, WIFI, BITCOIN) if these representations were not subject to slow and gradual natural selection processes? III) *The problem of environmental dependence:* How can we explain the role that experience plays in the acquisition of representational skills? Learning processes provide evidence that ontogenesis plays a role in determining content. Given these scenarios and the need for an ontogenetic dimension, ontogenetic functions came into play.

The result is a mixture of causal explanatory strategies. Evolution explains some representations, while ontogenesis explains others. This explanatory strategy highlights the relationship between developmental dichotomies and the invocation of both types of functions. Biological, innate, or inherited representations are a source of evolutionary functions (SETF); they are the result of phylogenetic learning processes (in Lorenz's sense). Cultural, learned, and environmentally induced representations are a product of ontogenetic learning processes. For example: "In the case of *innate abilities,* no matter what dispositions a mechanism happens to have, what determines its abilities is what it *was selected for doing*. In the case of *learned abilities,* what natural selection selected for was the ability to learn in a certain way. It selected for mechanisms that became tuned through *interaction with the environment* to do things of useful kinds" (Millikan, 2000b: 63; emphasis added). Evolutionary functions explain innateness, ontogenetic functions explain acquisition. Neander also claims in this sense: "[W]hile the functions can be determined by phylogenetic natural selection, operating on a population over generations, they can also be refined or altered by ontogenetic processes involved in development or learning (Neander, 2017a: 153).

This pluralistic view of functions assumes a Two-Force Model. The reason for this is that biological functionality is closely linked to the explanation of adaptive evolution (Ariew, 2003). The functional role of traits makes organisms suitable for their living conditions. Different theories of function provide different explanations for why the traits of organisms are functionally adapted to sustain and reproduce life. We can therefore conclude that a pluralistic view of function in the Two-Force Model leads teleosemantics to mix population-level and individual-level explanations, thus committing the Phylogeny Fallacy.

Interestingly, we also find in teleosemantic the endorsement of the different kinds of Two-Force Models analyzed in Section 3. Previously, I argued that the Two-Force Model of the MS rejects any causal role of individual-level processes in evolution, whereas some recent proposals in evolutionary biology accept that both individual and populational causality is involved in explaining adaptive evolution. In both cases, a Two-Force Model is advocated, but there are different positions regarding the explanatory role of individual causation. We also find this situation in teleosemantics. The original work in teleosemantics by Karen Neadner and Ruth Millikan, for example, is anchored in the Modern Synthesis framework. In their proposals, ontogenesis is limited to a single lifespan without taking into account the internal dynamics of development. However, others, motivated by ideas of the Extended Evolutionary Synthesis adopt an Extended Two-Force Mode. This is the case of Shea (2013), who developed a teleosemantic theory in the context of extended systems of inheritance. However, as Griffiths (2013) correctly argues, Shea's position is still problematic: evolved information transmitted by inheritance channels (genetic or not) does not account for a proper explanation of development. Therefore, Shea's view, even if motivated by extended views in biology, is not enough to avoid the fallacy. This evidences that the analysis of Phylogeny Fallacy and evolutionary causation properly applies to the teleosemantic context: in the same way that revising Mayr's distinction under an Extended Two-Force Model does not avoid the fallacy, the revision of mainstream teleosemantics under an extended view is not enough to avoid conflating explanations.

Moreover, as expected, the relationship between ontogenetic and phylogenetic functions takes the form of Type 1 Interactivism: Representations are the product of both evolutionary and ontogenetic functions, as Millikan puts it: "Inner states, such as the perceptual and cognitive states of organisms, can have proper functions that vary as a *function of environmental input* to the *genetically programmed* systems responsible for producing them" (Millikan, 2000a: 86; emphasis added). Dretske also adopts a Type 1 Interactivism: "The old nature-nurture dichotomy is too simple. Behavior is the product of a dynamic interaction between genetic and environmental influences. The *innate and instinctive is inextricably intertwined with the learned and the acquired*..." (Dretske, 1988, 31; emphasis added; see Neander (2017a: 82) for another example of Type 1 Interactivism). As explained in Section 2, Type 1 Interactivism is not free of problems: The assumption of Type 1 Interactionism also means that populational causes are mixed with individual causes in the explanation of (cognitive) phenomena at the individual level. To avoid this interactivist stance, we need to move to a purely proximate explanation of biological functions, as analyzed in the following section.

5.2. One-Force Model for Teleosemantics

The upshot of this analysis is that teleosemantics can capitalize on the embarrassment of a One-Force Model. This route would mean that both the Phylogeny Fallacy and the Ontogeny Fallacy are avoided: If we start from a One-Froce Model we cannot engage in these fallacious arguments. Moreover, such a teleosemantic project would be compatible with Type 2 Interactivism. A detailed elaboration of this perspective is beyond the scope of this paper (but see Bickhard (2003), Mossio et.al. (2009), and Rama (2021, 2022, 2023) for some suggestions that are in line with the ideas presented here). However, some ideas can be mentioned in advance.

For example, one consequence of adopting the statisticalist position is that SETF becomes a statistical functional theory: It describes natural selection processes that are accounted for by statistical analyses according to the Statistical School (Rama, 2023). If a trait type has been selected in the course of evolution, it means that it has contributed to increasing the average survivability of a population. As a result, we obtain a statistical analysis of normativity rather than the purported causal analysis that mainstream teleosemantics intends. Therefore, the explanatory role of the SETF cannot be to explain how a trait must function, it is not suitable for the naturalization of intentionality.

Alternatively, another theory of function must causally explain how a feature must function. Various proposals may fit teleosemantics well or poorly. Cummins' theory of function, for example, is based entirely on a proximate level of analysis and Type 2 Interactivism (Cummins, 1975). However, Cummins' proposal is not about teleological functions, so it does not lend itself to understanding the normativity of representation (since there are no goals or purposes, properties do not have to function in a certain way to fulfill a goal –i.e. norms and appropriate functions are not natural kinds per se). Even though Cummins' analysis is entirely based on proximate causation, we should understand how biological functions relate to an organism's goal in order to have a normative analysis of how a trait functions.

An important guiding principle in the search for a purely proximate explanation of biological telefunctions is that it can be inspired to some extent by new trends in theoretical biology. The various fields that challenge the MS and call for the irreducible causal role of organisms in adaptive evolution may be an appropriate place to look for the theory of functions we need. For example, some scientists working in the field of evo-devo have proposed several theories of function (Amundson and Lauder, 1994; Balari and Lorenzo, 2013; Love, 2007). Similarly, the organizational theory of functions (Mossio et.al., 2009; McLaughlin, 2000, Rama and Barandiaran, forthcoming) is motivated by various approaches in systems biology, autonomous systems theory, self-organizing systems, and other fields concerned with the complexity of developing organisms.

6. Conclusions

In this paper, we have examined the relationship between the causes of aptness in living systems and the Phylogeny Fallacy. In a general overview, I would say that this misleading reasoning tends to occur when we adopt a particular view of the causes of adaptive evolution, namely the Two-Force Model. I have not gone into detail about the problems of the Phylogeny Fallacy –but have pointed to the main literature that deals with it. My aim was not to give further reasons why mixing populational and individual explanations leads to fallacious reasoning. Rather, I have focused on the explanatory model that underlies this argument: the delineation of causal levels in the explanation of adaptive evolution. The assumption of a Two-Force Model implies the separation of causal explanations between different levels and therefore allows for the blending of the explanatory domains of each level of explanation. This idea also has a clear historical side: the emergence of the nature-nurture dichotomy in biological science occurred with the emergence of population biology in biological science (Keller, 2010). To analyze the connection between the Phylogeny Fallacy and evolutionary causality, I looked at recent debates about the validity of Mayr`s distinction in the context of the Extended Evolutionary Synthesis. I concluded that many critics of Mayr's view still hold some sort of Two-Force Model. I called it the Extended Two-Force Model –the position that claims that proximate and ultimate causes interact in explaining adaptive evolution. I have argued that this position, even if it recognizes the neglected explanatory role of developing organisms in evolution, is still problematic: the demarcation between levels of causation is still defended and the possibilities of engaging in the fallacious reasoning of the Phylogeny Fallacy are enabled by this position.

I have suggested instead that the defense of a One-Force Model is a way to avoid the Phylogeny Fallacy. I have presented the Statistical School as the framework that accomplishes two important goals: (i) it promotes a One-Force Model of the causes of aptness and (ii) it prizes the causal role of individual causation. I confine myself to introducing the statistical reading of natural selection and presenting its positive points concerning the Phylogeny Fallacy without going into further reasons supporting this position –i.e. without developing a solid defense of the Statisticalist School. However, the Statisticalist School and its purely proximate view of causality in biology provide an excellent way to avoid the Phylogeny Fallacy. First, since there is no subdivision of causal domains, it makes no sense to say that causal domains could be merged into a particular biological explanation. This also allowed us to avoid the Ontogeny Fallacy: ontogenetic causal explanations cannot replace populational explanations. In this sense, secondly, another positive point of the One-Force Model is that a clear delineation of explanatory domains does not require a delineation of causal domains. Each level of analysis –the population level and the individual level– is not an independent source of causality. They differ in their method of explanation. Individual phenomena are accounted for in terms of causal explanations, while population-related changes are described in

statistical terms. The distinction between explanatory strategies and explanatory tasks is crucial in order to avoid mixing up the areas of explanation. If we want to understand population dynamics, we provide a non-causal, statistical explanation for the differences in (trait) fitness in a population due to the presence or absence of a trait type. In contrast, if we want to understand individual fitness, we need to identify the various processes at the individual level during an organism's lifespan that generate its survival and reproductive capacities. Finally, this position also avoids supporting Type 1 Interactivism, a type of interactivism that has "not been enough to drive away the ghost of dichotomous views of development" (Gray, 1992, 172). The evolved population is not a ghost in the machinery of an organism that endows organisms with evolved causal powers. On the contrary, Type 2 Interactivism holds that all causal factors in the development of a system are proximate causes.

Finally, I have applied my analysis of the Phylogeny Fallacy and the causal structure of natural selection to a particular area of research: teleosemantics. I conclude that the ideas I have arrived at in my analysis can be applied to teleosemantics. First, most attempts at teleosemantics assume a Two-Force Model. In many cases, this is a classical view adopted from the Modern Synthesis. In other cases, however, a Two-Force Model is advocated, while individual-level processes are defended as a genuine element in a theory of functions. Be that as it may, the point is that teleosemantic projects systematically commit the Phylogeny Fallacy in both cases. The use of developmental dichotomies is pervasive in the teleosemantic literature, and the division of phylogenetic and ontogenetic functions leads to fragmentation and conflation of individual and population causes in teleosemantics as well. On the other hand, I have suggested that a One-Force Model is an appropriate way to avoid the de Phyloegny Fallacy in teleosemantics. Although this path is not yet fully elaborated, I have pointed out some insights that can help build a teleosemantic project beyond explanatory fallacies.

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