The Phylogeny Fallacy and Evolutionary Causation

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Abstract: The use of evolutionary explanations to explain individual-level phenomena was reported by different authors as an explanatory mistake, named the Phylogeny Fallacy. The aims of this paper are twofold. First, I will argue that the Phylogeny Fallacy takes place within a particular stance about the causes of aptness in living systems, the so-called Two-Force Model, which states that there are two sorts of causes involved in the explanation of aptness: individual and populational causation. Secondly, I will introduce the One-Force Model of the Statisticalist School which claims that the unique level of causation is the individual level: all causes of aptness are proximate causes. I will argue that this framework may represent a suitable path to avoid the Phylogeny Fallacy. I will end by applying the analysis done to a specific research program, teleosemantics.

Keywords: Phylogeny Fallacy; Dichotomies; Evolutionary Causation; Explanation of Aptness; Statistical School; Teleosemantics.

1. Introduction

The limits of explanatory methods and research areas are always a rich and controversial topic in science. This is also the case in biology. What is the explanatory scope of populational explanations? What populational biology can and cannot explain? What can we know about evolution by understanding developmental processes? Does physiology inform us about evolutionary adaptations? There are a lot of issues behind these questions, and notably, there have been different answers throughout the history of biology.

This paper is concerned with a particular issue within the discussion of the epistemic boundaries of biological disciplines: the Phylogeny Fallacy. This term points to an explanatory error in biology: the use of populational explanation to address individual-level phenomena. The most common link to the Phylogeny Fallacy is based on various dichotomies related to the old nature versus

nurture debate. This issue has been extensively discussed by various scholars from different disciplines such as philosophy, cognitive science, and biology. In this paper, we will not address the discussion of the explanatory error behind the Phylogeny Fallacy. This problem has many faces. We will focus here on one specific issue: the relationship between the Phylogeny Fallacy and 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 about the causes of adaptive evolution: the so-called Two-Force Model.

The structure of the paper is as follows. In Section 2, I introduce the Phylogeny Fallacy and explain various reasons why a particular explanation may promote the fallacy. In Section 3, I will highlight the connection between the fallacy and the explanation of aptness. To do so, I will argue that the fallacy occurs in the context of the Two-Force Model. The Two-Force Model is about distinguishing different levels of causation (the individual and populational levels) involved in the explanation of aptness. I will point to two different types of Two-Force Models. First, the model adopted by the Modern Synthesis states that individual causation affects only affects a single life span and has no effect on populational causation. Second, a different Two-Force Model has been supported by recent trends in theoretical biology that assume that individual causation plays an important evolutionary role. In this framework, both levels of causation play a causal role in evolution. However, I will argue that both types of Two-Force Models may lead us to the Phylogeny Fallacy. In particular, I will note that emphasizing the causal role of developing organisms in evolution is not sufficient to avoid the Phylogeny Fallacy. 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, I analyze a specific case study, teleosemantics, in Section 5. The goal is to see how teleosemantic projects located in a Two-Force Model systematically support the Phylogeny Fallacy and, consequently, that a One-Force Model for teleosemantics may be a prominent and justifiable research project.

2. The Phylogeny Fallacy: conflating explanations

The Phylogeny Fallacy is a conflation between different biological explanations, especially when evolutionary explanations about populational processes are used to explain phenomena at the individual, ontogenetic level. A theory is subject to the Phylogeny Fallacy when evolutionary processes come into play to explain biological phenomena that do not fall within its explanatory framework.

We can present the fallacy following the classification of biological causes proposed by Ernst Mayr (1961, 1974). Evolutionary biology deals with evolutionary causes —so-called *ultimate causation*— and developmental biology and physiology (or "functional biology" in Mayr's terminology) deal with ontogenetic causes —*proximate causes*. He also attributed different explanatory roles for each kind of cause. Ultimate causation explains *why-questions*: why biological systems are organized in a specific functional and adaptive way. Proximate causes explain *how-questions*: how different parts of a living system interact to produce a functional or adaptive outcome. Without discussing the truthfulness of this dichotomy, it can help us to illustrate the fallacy. In causal terms, the fallacy concerns the conflation of ultimate causation with proximate causation. In explanatory terms, the fallacy consists of explaining how-questions by providing why-answers. It is also a conflation of disciplines, that is, to support that evolutionary biology can do the explanatory task of developmental biology and physiology. It is also a conflation of disciplines, that is, to support that evolutionary biology can do the explanatory task of developmental biology and physiology.

The Phylogeny Fallacy was launched by Lickliter and Honeycutt in 1989. We also find it earlier in developmental systems theory, most notably in the work of Susan Oyama (Oyama, 1985) and more recently in Ecological Developmental Biology (Lewontin, 2000). Nonetheless, the central arguments have come from embryologists and developmental psychobiologists devoted to the study of embryological processes in behavioral development and to addressing instinct and nativist theories in the XX century. The major players are Zing Yang Kuo (1921, 1922) at the beginning of the previous century, Lehrman (1953) in the mid-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 Phylogeny Fallacy by invoking Lorenz's distinction between instinct and acquired behavior. As he explains, this distinction concerns two types of learning processes involved in explaining adaptive behavior. On the one hand, we have evolutionary learning processes in which natural selection biases variation toward adaptive behavioral traits. The type of information involved here is phylogenetic information. On the other hand, ontogenetic learning regulates ontogeny based on environmental cues and behavioral feedback. Following Lorenz's terminology, the information involved is ontogenetic information. Instincts are developed through maturation, as a rigid process involving phylogenetic learning and established through phylogenetic information, whereas acquired traits are developed through ontogenetic information via context-dependent developmental pathways. The distinctions made by Lorenz's theory also lend themselves to an account of the Phylogeny Fallacy: using phylogenetic information to deal with ontogenetic information or using phylogenetic learning to explain ontogenetic information.

As shown in Table 1, there is a distinction between the individual and populational levels behind the Phylogeny Fallacy. Each of these levels provides different explanations for adaptive behavior.

The fallacy is about moving beyond the explanatory scope of the populational level to pursue individual-level phenomena. While it is not the goal of this paper to discuss the origin of the fallacy in evolutionary thought (see Keller (2010) for a wonderful paper on this topic), the explanatory power of evolutionary theory to help push its explanatory boundaries is an old scientific construct and was probably first introduced by Ernst Haeckel (1866: 7): "The theory of descent alone can explain the developmental history of organisms."

Individual Level	**Populational Level**
Proximate causes	Ultimate causes
Functional Biology	Evolutionary Biology
How-questions	Why-questions
Ontogenetic Learning	Phylogenetic Learning
Ontogenetic Information	Phylogenetic Information

Table 1: The demarcation between Individual and Populational levels in biological explanations.

I will not go into detail about the many problems associated with the Phylogeny Fallacy. I will only point out its main problem: its Explanatory Vacuity. In short, the problem is that appealing to evolutionary processes tells us nothing about the mechanisms of development. What do we know about development just by saying that a trait is the product of natural selection? As Oyama (1985: 159) stated, "[it] feels right, but it explains nothing" Dichotomous categories refer to traits of a population undergoing evolutionary processes, not individual developmental mechanisms of development. This is an idea advanced 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). If we want to explain developmental processes, it seems that looking into evolution is a vacuous strategy: "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 [as well as Lorenz's phylogenetic 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.

Notoriously, the central locus of the Phylogeny Fallacy lies in dichotomic thinking. Dichotomic thinking is the (explicit or implicit) use of dichotomic categories to explain the development of traits. Dichotomic thinking can be enclosed within the nature-nurture debate. Yet different concepts also fall on each side of the duplex, as is shown in Table 2. The logic behind these labels is to separate the causes of phenotypic outcomes. Some traits are innate, inherited, and biological, thus part of the nature of the species. Other traits are learned through environmental inputs, then part of our nurturing. Some traits are caused by evolution, while others are caused by ontogeny.

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

Table 2: Dichotomic categories. Different dichotomies are involved in the explanation of development. The left side refers to traits influenced, caused, or explained by individual-level processes, and the right side concerns traits influenced, caused, or explained by populational-level processes.

Many authors have argued that the use of dichotomic categories entails a fallacy —the Phylogeny Fallacy. It seems that we are not saying much about the causal processes that produce a trait just by saying that this is an innate or evolved trait; dichotomies are *explanatory vacuous* when we search for the mechanisms of development. Moreover, dichotomic thinking also has other problems. For instance, dichotomies may constitute a *semantic clutter* (cf. 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 dichotomic category. It has also been argued that dichotomous concepts 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.

Sometimes this fallacy is considered unproblematic if we can accept that all characteristics result from the interaction of various causes. This position might be called interactivism. However, it is

important to distinguish between two types of interactivism. *Type 1 Interactivism* -also called "consensus interactivism"- states that traits arise from the interaction of evolutionary causes (originating from the populational, historical level through hereditary channels) and ontogenetic causes (originating from the individual, ahistorical level through context-sensitive, environmentally-dependent ontogenetic processes). The Phylogeny Fallacy still holds in Type 1 Interactivism: even when causes interact, they are still delineated and levels of explanation are mixed: "This 'interactionist consensus,' however, perpetuates the nature–nurture debate by maintaining its inherent dichotomy" (Stotz, 2008: 360). On the contrary, *Type 2 Interactivism* argues that all traits arise from the interaction of causes at many levels of organization: Genes, genomes, cells, tissues, and the environment. It does not claim that some traits arose through evolution and others did not. In other words, ultimate causes do not play a causal role in proximate causes. Evolutionary explanations have no place in ontogenetic explanations. Type 2 Interactivism is safe from the Phylogeny Fallacy. The message to take away is quite simple: to explain development, one must understand interacting causes beyond dichotomies. Or, as Gottlieb says, "[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 deeply rooted in a division of the causes of adaptive behavior: individual-level causes and populational causes. The delineation of these two sources of causes is known as the Two-Force Model (Walsh, 2003). However, the Two-Force Model has been interpreted in different ways. The first, developed by the Modern Synthesis, denies any role for individual causation in evolutionary theory. The second, advocated by some critics of the Modern Synthesis, asserts that both levels of causation are involved in explaining adaptive evolution. Let's introduce the two.

3.1. Populational Causation and the Two-Force Model

In the context of the Modern Synthesis theory, the Two-Force Model receives a special interpretation. The main idea is that populational causes are involved in explaining adaptive evolution, while individual-level causes acting during the development of the organism play no role in evolution. As a result of central tenets of the Modern Synthesis, such as the exclusion of Lamarkian modes of inheritance, a reductive Genotype-Phenotype map, or the unbiased nature of variation, individual-level causation plays no role in evolutionary processes. Individual-level processes are involved in the acquisition of learned traits. However, learned or acquired traits are not passed on to the next generations. In this sense, development provides causes that explain some

adaptive traits of living systems, but only those traits that have been learned and do not affect natural selection processes. While Darwin enshrined the idea of natural selection as an adaptive process in evolution, the Modern Synthesis reading of Darwinism (i.e., neo-Darwinism) focused only on the genetic level, arguing that the association between selected phenotypes and inherited genes is transparent enough (i.e., there is a strong correlation between genetic variation and phenotypic variation) to exclude developmental analysis from evolutionary theory. In this sense, the Two-Force Model of the Modern Synthesis is related to the neglected role of developing organisms in evolutionary theory. As is widely recognized today in the context of the philosophy of biology, developmental processes have been black-boxed by the Modern Synthesis. In this scenario, developmental processes and individual causation can be ignored: It is enough to see what is going on at the genetic level to understand evolution, or as Maynard Smith said: "It is possible to understand genetics, and hence evolution, without understanding development" (Maynard Smith, 1982: 6). Consequently, as Brian Goodwin (1994: 1) says,

> "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."

However, it is important for our current discussion that various tenets of the Modern Synthesis are currently controversial. Extended inheritance systems have been found in all living systems (e.g., Jablonka and Lamb, 2014), developmental processes are not just about the expression of genetic information (e.g., Sultan 2015), and there are various sources of adaptively biased phenotypic variation involved in selection processes (e.g., West-Eberhard 2003). The idea that developing organisms are causally relevant in adaptive evolution is now supported by a growing wave of biological theories, motivated by the claim that "[t]he black box [of development] is now being opened to provide a more complete picture of what really happens" (Bateson & Gluckman, 2011, 17). New trends in biological theory call for a return of organismal causaton in evolutionary theory and the importance of an adequate understanding of development and organization properly (Baedke, 2018b; Bateson, 2005; Huneman, 2010; Nicholson, 2014). My goal is not to discuss the revived causal role of organisms in evolution or the various disputes over the status of the Modern Synthesis. Instead, I will argue here for two (conditional) theses. First, i*f these new waves in evolutionary thinking are still domiciled in a Two-Force Model, then we are in danger of falling into the Phylogeny Fallacy.* Second, *if we reject any Two-Force Model, the Phylogeny Fallacy would be avoided and reformulated by a new understanding of the causal structure of natural selection.*

3.2. Individual Causation and the Two-Force Model

How should we understand the causal role of organisms in evolutionary theory? One possible answer is to claim that evolutionary theory deserves to be evaluated, but without abandoning the Two-Force Model, i.e., that both individual and populational causes are responsible for the adaptive character of living systems. Indeed, this is a common position in the contemporary philosophy of biology (see discussion below). In addition to populational forces, development, and other individual-level processes are also evolutionary forces. This position entails a different Two-Force Model than that of the Modern Synthesis. As explained earlier, in the Modern Synthesis developmental causes have no influence on evolutionary dynamics. What occurs during the development of an organism has no effect beyond the generation of that organism. However, once individual causation is *also* established as a causal factor in adaptive evolution, once the organism reverts to biological theory, the Two-Force Model of the Modern Synthesis is altered. In this context, individual causation affects evolutionary causation.

As Denis Walsh (2003, 2015, 2019) details, those who defend a nonclassical Two-Force Model (those who oppose the Modern Synthesis) tend to ascribe a special role to individual-level processes. Here, several scholars (see Walsh (2002) for references) have located individual causation within a Two-Force Model. The explanatory role attributed to development in this context is to *constrain* the possible repertoire of phenotypes to be selected; in this reading, development *limits* what the environment can perpetuate or eliminate. In short, individual causation is a *bias* to the most important adaptive force: natural selection.

What is significant for our discussion, however, is that the problems remain even if we accept phenomena at the individual level as a factor in evolution. 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 fallacy. This position does not prevent invoking populational causation to deal with individual-level processes. In other words, the demarcation between two levels of causation is a prerequisite for the Phylogeny Fallacy. Furthermore, if we consider individual causation as complementary to populational causation, Type 1 Interactiovism still applies. In this scenario, developmental causation biases and constrains what can be selected during populational change: Adaptive change is due to the *interaction* between individual and populational causation.

There is another problem in these Two-Force Models that embrace the causal role of developing organisms in evolution, namely the Ontogeny Fallacy (Hochman, 2012). This fallacy is also the conflation of explanatory boundaries. In this case, it is about replacing evolutionary explanations with ontogenetic explanations; it is about emphasizing the individual level to understand populational phenomena. While the Phylogeny Fallacy attempts to explain developmental processes by looking at evolution, the Ontogeny Fallacy is about explaining evolution by looking into development. The main problem with this is that explaining what goes on during the lifespan

of an individual is something completely different from understanding how populations change throughout history. Remarkably, as noted by Hochman, the revival of organismal causation and the role of epigenesis in evolutionary theory, as noted by Hochman, may suggest that developmental answers may be appropriate for evolutionary questions. Thus, this is another possible fallacy enabled by the Two-Force Model: Proximate causation can be invoked to answer ultimate questions. In this sense, advocating organismic causation may lead one to engage in a new fallacy, the Ontogenetic Fallacy.

So we come to our first conclusion: *if the new trends in evolutionary thinking advocating organismic causation are still domiciled in a Two-Force Model, then we are in danger of falling prey to the Phylogeny Fallacy (and the Ontogeny Fallacy too).* This conclusion is relevant when we consider some of the reasons why the Modern Synthesis has been challenged. The nature-nurture distinction in the natural sciences was well established by the end of the XIX century and was integrated into evolutionary theory during the development of the Modern Synthesis, thanks to various tenets forged in that era, such as the exclusion of soft inheritance, the construction of a gene concept, and the reduction of variation to the genetic level. The rebirth of organismal causation challenged these doctrines. The take-home message, however, is that avoiding the fallacy is not just about challenging the Modern Synthesis and claims to introduce development as part of evolutionary thinking. The Phylogeny Fallacy, even if it arose in the Modern Synthesis niche, is not a problem of the Modern Synthesis itself, but of 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 that dichotomy, we must shift the role of organismic causation beyond the Two-Force Model. How, then, can we support organismal causation and the causal role of development in evolution without endorsing the Phylogeny Fallacy (or the Ontogeny Fallacy)? The answer to this question would lead us to our second conclusion: *If we reject the Two-Force Model, the Phylogeny Fallacy (and the Ontogeny Fallacy) would be avoided and reformulated under a new evolutionary framework.*

4. The Phylogeny Fallacy in the One-Force Model

There is an alternative to the Two-Force Model: the One-Force Model. As I will explain later, the One-Force Model, although it may seem a radical position, helps to clarify the different explanatory roles of each level of explanation in biology -the individual and the populational- by delineating different *kinds of explanations* in adaptive evolution -not different *kinds of causes* as in the Two-Force Model- and the different explanatory goals of each level of explanation. The distinctions that the One-Force Model makes are essential to avoid mixing explanations, i.e., to avoid the Phylogeny and Ontogeny fallacies.

4.1 The Statisticalist School.

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 its main references). On the one hand, the Causalist School (based primarily on the work of Elliot Sober (1984, 2013)) contends that natural selection should be understood as a force acting on populations and guiding them toward successful picks (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) for seminal papers) argues that the causes of natural selection are not at the populational level but at the individual level. There is no such thing as populational causation. The natural selection explanation provides *statistical explanations* for the effects of individual-level causation in population dynamics. Without developing a detailed defense of the Statisticalist School, let us present its basic principles.

The proposal of the Statistical School revolves around the concept of *fitness*, which is definitely central to any explanation by natural selection: populations change due to fitness differences, as Darwin taught us (Ariew and Lewontin, 2004). The statistical reading of natural selection, however, begins by highlighting the difference between two notions of fitness: trait fitness and individual fitness. The former properly pertains to population thinking and was introduced during the emergence of the Modern Synthesis through the famous mathematical insights into evolutionary theory of Fisher, Wright, and Haldane. In this sense, trait fitness concerns the fitness values of a population; it is concerned with its trait types, not its trait tokens. Individual fitness refers to the fitness value of each individual. It is about trait tokens, not trait types. Trait fitness indicates the survival and reproductive capacity of a population, while individual fitness refers to the survival and reproductive capacity of a single individual. This distinction involves distinguishing between the types of explanations associated with each notion 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 assessed *causally assessed*. Trait fitness is an average value in a given population, while individual fitness reveals the causes of an organism's survivability and reproductive capacity.

Following this distinction, the statisticalist thesis states that trait fitness is measured by averaging over the individual fitness of trait tokens. In short, trait fitness is an abstraction at the populational level of the individual fitness of the members of the population. Such an abstraction is a populational 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 populational 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 adopts a One-Force Model. Accordingly, the One-Force Model states that *all* causes of evolution lie at a unique 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 pertain to the individual level. The picture that emerges is that evolution is a populational consequence of what is going on 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 individual-level causes*.

It might seem that the Statisticalist School is a radical position. In fact, the One-Force Model is usually supported by those who believe that the Modern Synthesis should be seriously revised. However, the Statisticalist School proposes a valuable link between the explanatory framework of the Modern Synthesis explanatory framework and the new proposals based on individual causation. To this end, the Statisticalist School promotes a division of explanatory effort between the individual and populational levels. The division between the levels of explanation and the difference between the types of explanations motivates the division between different *explanatory tasks*. Individual-level analysis is devoted to understanding the causal processes in an organism during its lifespan. The populational level of analysis assesses the impact of individual-level processes on population changes using statistical methods. Causal explanations of development and statistical explanations of populational change have different explanatory roles associated with different levels of explanation. Each domain (with its own explanatory strategy) is irreducible to the other: we cannot understand evolutionary processes by studying individual-level causes, and we cannot understand individual-level phenomena by studying the populational domain. To understand the adaptive capacity of living systems, both types of explanations are inevitable:

> 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 division of explanatory tasks of the One-Force Model is crucial to avoid both the Phylogeny Fallacy and the Ontogeny Fallacy, as I will argue in the next section. 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 principally to populational biology) and emerging trends in theoretical biology that address the complexity and adaptability of developing organisms.

4.2 Reframing the fallacy: advantages of the One-Force Model

The statisticalist view of natural selection entails that the Phylogeny Fallacy must be redefined. As expected, the division of explanatory tasks concerns the nature of the explanation, not the nature of the cause, which is present in every explanation. As has been pointed out, the Statisticalist School assumes two types of explanations are required to understand adaptive evolution. Therefore, if we adopt the statisticalist view, the Phylogeny Fallacy should be redefined. It is not the mixing of causes, but the mixing of two different kinds of explanations, between statistical and causal explanations.

In this sense, the division of labor in explanation prevents us from mixing causes of aptness. The Phylogeny Fallacy as a mixture of populational and individual causation is not possible in the context of the Statisticalist School. We can still confuse statistical explanations with causal explanations. But even in this case, it is clear that a populational explanation cannot provide an individual-level explanation for the development of traits. The role of populational explanations is limited to statistical analysis. From a populational analysis, we can derive generalizations or idealizations (Neander, 2017b), average scores (Boorse, 2002), and statistical notions of what is normal or common (Millikan, 1989), but definitely not a causal explanation.

Moreover, the division of explanatory tasks also helps to avoid the Ontogeny Fallacy. That is, the Statsticalist School not only neglects the possibility of populational causes swamping individual-level explanations but also attributes an irreducible role to populational explanations. Such a role cannot in any way be reduced to individual-level explanations. The statistical analysis of populational biology, developed during the gestation of the Modern Synthesis, is critical to understanding how populations -not individuals- change over history.

Finally, 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 noted earlier, Type 1 Interactivism -and its link to the Phylogeny Fallacy- can be defended even by proponents of individual causation in evolutionary theory who support a Two-Force Model. However, once we abandon populational causes, Type 1 Interactivism no longer makes sense. On the contrary, Type 2 Interactivism seems to be motivated by the One-Force Model. According to it, different causes may interact in the formation of phenotypes, but all these causes belong to a proximate, individual level of analysis.

4.3 Interim summary

The purpose of this paper is to analyze the relationship between two central themes 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 emerged in the birth of modern evolutionary thought, new trends in theoretical biology emphasizing the role of individual causation in evolutionary theory still assume a Two-Force Model. The epistemological role of individual-level processes is not merely complementary to populational causation. If the Phylogeny Fallacy and Type 1 Interactivism are to be avoided, we must rethink the causal structure of natural selection to move beyond the Two-Force Model.

The second set of results emerges if we adopt the One-Force Model of the Statisticalist School. First, the Phylogeny Fallacy must be modified. We cannot invoke Mayr's distinction between two kinds of causes, since there is only one kind of causality: proximate causes. The fallacy consists of mixing two kinds of explanations: confusing statistical analysis with causal analysis. Second, splitting the explanatory domains (with their own explanatory tasks and methods) helps to avoid the Phylogeny Fallacy. Third, the One-Force Model also avoids the Ontogney Fallacy: statistical analysis of populational dynamics does not tell us the causal mechanism of development, and analysis of developmental mechanisms cannot serve as a substitute for an adequate populational explanation. Finally, the One-Force Model is fully consistent with and motivated by Type 2 Interactivism.

I conclude by analyzing a specific case study: teleosemantics, a relevant theory in the philosophy of mind and cognitive science. I will see how the analysis of the Phylogeny Fallacy and evolutionary causation can be applied to teleosemantics. In particular, I will see how mainstream teleosemantics projects that subscribe to a Two-Force Model endorse the Phylogeny Fallacy. Moreover, I will argue that a One-Force Model of teleosemantics -which is largely unexplored- could be a prominent 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, devoted mainly to understanding representational content from a naturalist standpoint (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 supports the Phylogeny Fallacy. Second, teleosemantics will help illustrate 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 addressing 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 mind. A representational system must function according to the biological functions it embodies, and the norms of representational systems (what representation *must* refer to) are based on this biological functionality.

Depending on which biological functional theory one chooses, different teleosemantic projects may emerge. I refer to as *mainstream teleosemantics* those teleosemantic theories that support the *Selected-Effect Theory of Functions* (SETF) for explaining representational content (cf. 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 as 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 good reasons to argue that mainstream teleosemantics supports the Phylogeny Fallacy.

There are at least two ways to argue that mainstream teleosemantics supports the Phylogeny Fallacy. The first and simplest reason is the ubiquitous use of dichotomies in the teleosemantic

literature. The result is a classic view, inherited from modern epistemologists, according to which some representations are innate and others are learned. In Table 3 we see various dichotomous expressions found in the core figures of teleosemantics.

Dichotomic Expressions	**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 (2017b: 166)
Innate capacities	Neander (2017b: 82, 101)
Innate information, representations	Neander (1995b: 111-112)
Innate, hard-wired belief-forming abilities	Papineau (1984: 557)

Table 3: Dichotomies in mainstream teleosemantics. A non-exhaustive list of dichotomic expressions found in the main teleosemantic projects. Reprinted with permission from Rama (2022).

In addition to these explicit uses of dichotomous categories, however, there is another important source of dichotomous thinking in teleosemantics: the distinction between p*hylogenetically selected functions* and *ontogenetically selected functions.* This distinction is also widely used in the teleosemantic literature. Let us explain the puzzle here.

As noted earlier, mainstream teleosemantics supports an evolutionary and populational perspective on biological functions: A trait must do what it was selected for during 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, not all representations can be adequately explained by an evolutionary perspective alone. I can point to at least three problems that teleosemantics attempts

to solve with ontogenetic functions. (I) *The problem of variation*: How is it possible that within one and the same species (e.g. between cultures) there are different representational capacities? II) *The problem of novelty*: How can new representations emerge in humans (e.g. ELECTRON, WIFI, BITCOIN) if such 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 capacities? Learning processes provide evidence that ontogeny 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 explanatory strategies and causal sources. Evolution explains some representations, while ontogeny explains others. This explanatory strategy highlights the relationship between dichotomies and the invocation of both types of functions. Biological, innate, or inherited representations are a source of evolutionary functions (SETF) or phylogenetic information (in Lorenz's sense). Cultural, learned, and environmentally induced representations are a product of ontogenetic functions (or ontogenetic information). For instance: "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. In the same vein, Neander claims: "[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 is that biological functionality is closely related to the explanation of aptness. 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 committing a pluralistic view of function to the Two-Force Model leads teleosemantics to endorse the Phyloegny Fallacy.

Interestingly, we can find different kinds of Two-Force Models in teleosemantics. Previously, I argued that the Two-Force Model of the Modern Synthesis rejects any causal role of individual-level processes in evolution, while some recent proposals in evolutionary biology accept that both individual and populational causation participates 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 as well. For example, the original work in teleosemantics developed by Karen Neadner and Ruth Millikan embraces the Modern

Synthesis Framework. In their proposals, ontogeny is limited to a single lifespan, without addressing the internal dynamics of development. Others, however, such as Fred Dretske (1988) and Nick Shea (2018), argue that both ontogeny and phylogeny influence evolutionary processes.

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) (cf. Neander (2017b: 82) for another example of Type 1: Interactivism).

5.2. One-Force Model for Teleosemantics

The result of this analysis is that teleosemantics can draw revenue from the embarrassment of a One-Force Model. This path would mean that both the Phylogeny Fallacy and the Ontogeny Fallacy are avoided. Moreover, such a teleosemantic project would be consistent 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) for some suggestions that are consistent with the ideas represented here). However, some ideas can be stated in advance. For example, SETF becomes a statistical theory of functions: 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, this would mean that it has contributed to increasing the average survivability of a population. The explanatory role of SETF cannot be to explain how a trait must work.

Alternatively, another theory of function must causally explain how a feature must work. Various proposals may fit well or poorly with teleosemantics. 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 is not suitable for understanding the normativity of representation (since there are no goals or purposes, traits do not have to function in a particular way to fulfill a goal -i.e., norms and proper functions are not natural kinds in themselves).

An important guiding idea in the search for a purely proximate explanation of biological (teleo)functions is that it may be inspired to some extent by new trends in theoretical biology. The various fields that challenge the Modern Synthesis and call for the irreducible causal role of organisms in adaptive evolution may be an appropriate place to look for the theory of functions that we need. For example, some scholars working in the evo-devo field 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) 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 considered the relationship between the causes of aptness in living systems and the Phylogeny Fallacy. A general overview would say that the fallacy results from a particular view of the causes of aptness, 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. I have focused on the explanatory model that underlies the fallacy: the delineation of levels of causation in explaining aptness, i.e., in explaining the functionality and adaptability of living systems that are not found in non-living systems and deserve a special field of research -a.k.a. biology. This idea has a clear historical side: the emergence of the nature-nurture dichotomy in biological science occurred when population biology emerged in biological science.

I have suggested that defending a One-Force Model is a simple way to avoid the Phylogeny Fallacy. I have presented the Statistical School as the framework that fulfills two important goals: (i) it promotes a One-Force Model for the causes of aptness and (ii) it extols the causal role of individual causation. I confine myself to introducing the statistical reading of natural selection and presenting its positive points with respect to the Phylogeny Fallacy without going into further reasons that support this position -i.e., without developing a solid defense of the Statisticalist School. However, the Statisticalist School and its purely proximal view of causality in biology offer a prominent way to resolve 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. In this sense, second, another positive point of the One-Force Model is that clear delineation of explanatory domains does not require delineation of causal domains. Each level of analysis -the populational and the individual- is not a distinct source of causation. They differ in their explanatory style. Individual phenomena involve causal processes, whereas population-based changes are described in statistical terms. The distinction between

explanatory strategies and explanatory tasks is crucial to avoid mixing explanatory domains. If we want to understand populational dynamics, we will provide a non-causal, statistical explanation of (trait) fitness differences in a population due to the presence or absence of a trait type. In contrast, to understand individual fitness, we need to show the various individual-level processes during an organism's lifespan that generate its survivability and reproductive capacities. Finally, this position also avoids 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). In this context, we say that the evolved population is not a ghost in the machinary of an organism that endows organisms with evolved causal powers. On the contrary, Type 2 Interactivism states that all causal factors in the development of a system are proximate causes.

Finally, I 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 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-forces model is advocated while individual-level processes are defended as a genuine element in the theory of functions. Be that as it may, the point is that teleosemantic projects systematically support the Phylogeny Fallacy in both cases. The use of dichotomies is ubiquitous in the teleosemantics literature, and the division of biological functions also leads to the fragmentation and mixing of individual and populational causes in teleosemantics. 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 has not yet been fully elaborated, I have pointed out several insights that can help build a teleosemantic project beyond explanatory fallacies.

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