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Abstract: The use of evolutionary explanations to explain phenomena at the individual level has been described by various authors as an explanatory error, the so-called Phylogeny Fallacy. In this paper, this fallacy will be analyzed in the context of teleosemantics, a central project of the philosophy of mind whose main aim is to naturalize intentional systems by appealing to their biological teleofunctions. I will argue that those teleosemantics projects that invoke evolutionary functions generally commit the fallacy. First, I will point to various arguments in the literature that point to this fallacy. However, a more general argument will also be made. To illustrate this puzzling scenario, I will present two *desiderata* that any teleosemantic project must fulfill. I will argue that naturalizing intentionality based on natural selection creates an explanatory gap between types and tokens. This gap prohibits an adequate explanation of the *desiderata*. To close the gap, teleosemantics invokes replicator biology, a view of inheritance that has already been identified to commit the fallacy. This leads teleosemantics into a complex situation: to close the gap, it must commit the fallacy.

Keywords: Phylogeny Fallacy; Replicator Biology; Actuality *Desideratum*; Historical *Desideratum*; Proximate/Ultimate Causes; Teleosemantics.

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The theory of descent alone can explain the developmental history of organisms.
Haeckel, 1866, 7

1. Introduction

The limits of scientific explanations have always been the subject of intense debate. This is also the case in biology. What is the scope of a particular biological explanation? What can be known about development when looking at evolution? What can heredity tell us about developmental processes? Does development inform us about evolutionary pathways? What a particular explanation does and does not explain is a complex question. Here we will analyze a controversial aspect of biological explanations: the Phylogeny Fallacy; i.e. the use of evolutionary explanations in developmental biology. We will restrict our analysis to one particular area of research, teleosemantics. Thus, while various analyses concern biological traits in general, our main target concerns intentional systems: those systems that teleosemantics attempts to explain by invoking a naturalized theory of biological teleofunctions. This paper aims to provide a critical review of teleosemantics and trace some conclusions that might help to take crucial steps toward the naturalization of intentionality.

The paper is organized as follows. In Section 2, I introduce the Phylogeny Fallacy and explain various reasons why a given explanation is subject to this fallacy. In Section 3, I analyze the literature on teleosemantics and show various papers that engage in the fallacious reasoning of the Phylogeny Fallacy. However, to provide a general argument that goes beyond a specific literature review, in Section 4 I will present two central *desiderata* that any teleosemantic project must fulfill: the *historical desideratum* and the *actuality desideratum*. In Section 5, I show that the evolutionary roots of teleosemantics are confronted with a puzzling scenario: If it wants to do justice to the *desiderata*, then it must commit the fallacy. Or, if teleosemantics does not commit the fallacy, then a central explanatory goal of teleosemantics remains unexplained as the product of an explanatory gap between population and individual characteristics. These constraints on mainstream teleosemantics might hint at the naturalization of intentionality, i.e., new biological roots for teleosemantics.

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 (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*. 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 account for 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 throughout 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, this (fallacious) argument has been constructed (Keller, 2010; Rama, forthcoming). 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). Accordingly, 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 phenotypic outcomes. 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 that 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 to explain the development of traits, such as innate – learned, acquired – instinctive, inherited – environmental, biologically determined – culturally determined, or fixed – plastic. Crucially, each side of each duplex is linked to the different levels of explanation mentioned above. The first element of each duplex refers to phenotypes that are influenced, caused, or explained by population-level processes, and the second element of each duplex refers to phenotypes that are influenced, caused, or explained by individual-level processes.

In this sense, 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, 1970) 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.

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).

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. Thus, the explanatory logic behind developmental dichotomies is to separate the causes of phenotypic outcomes. 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 individual 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. In this vein, 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; developmental 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 Teleosemantics

3.1 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). 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 and explained by natural selection processes: 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, at least, two reasons to argue that mainstream teleosemantics engage in the fallacious argumentation of the Phylogeny Fallacy. In both cases, I will suggest an “argument by ostension” (Neander, 2017): I will indicate those texts in mainstream teleosemantics that commit the 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. Nativism is usually defended in teleosemantics, an innateness is systematically attributed so several traits, such as innate perceptual-cognitive mechanisms (Millikan, 2006, 109), skills (Millikan, 2000b, 54), behavior (Dretske, 1988, 123), capacities (Neander 2017, 82) sensory-perceptual systems, (Neander 2017, 82), information (Neander, 1995a, 111), representations (Neander, 1995a, 112), beliefs (Papineau, 1984, 557), mental states (Marinez, 2013, 448), functions (Artiga, 2010, 206), or mechanisms (Artiga, 2010, 206). As expected, innateness is associated with the evolutionary level of explanation and explained in terms of evolved genetic programs; these innate traits

are “genetically programmed systems” (Millikan, 2000a, 86), “explained in terms of such genetic selection” (Papienau, 2016, 118), “causally explained by the genes inherited” (Dretske, 1988, 92), “[explained by] genes coding for behavior” (Dretske, 1988, 125), “rigidly programmed behavior” (Dretske, 1988, 125), or “strongly phylogenetically determined” (Artiga, 2021, 2). In sum, 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.

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 the acquisition of traits. 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, 2017, 153).

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 (2017, 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.

4. The Historical *Desideratum* and the Actuality *Desideratum*

In the previous section, we saw that mainstream views of teleosemantics commit the Phylogeny Fallacy. We have established this through a literature review, that is, by examining key teleosemantic texts and looking for developmental explanations based on evolutionary explanations. The argument presented in the previous section certainly demonstrates the explicit link between teleosemantics and the use of developmental dichotomies associated with the proximate/ultimate distinction. However, this "argument by ostension" has only shown that *some* teleosemantic projects commit this fallacy. The point now is to see whether all teleosemantic projects based on evolutionary functions are destined to commit the fallacy. This does not mean that all projects must explicitly endorse developmental dichotomies or the distinction between ontogenetic and phylogenetic functions. It only means that teleosemantics' explanatory strategy for naturalizing intentionality is systematically engaged in the fallacious argumentation of the Phylogeny Fallacy. To accomplish this goal, a literature review cannot serve as an argument: We need to identify what central theoretical commitments cause teleosemantics to engage in the fallacious reasoning of the Phylogeny Fallacy. If this is the case, if there is a basic assumption in teleosemantics that can lead to explanatory error, then teleosemantics faces a serious problem that affects more than just a few particular authors.

To this end, in this section, I will first present two central *desiderata* for any teleosemantic (naturalistic) project. At issue is something that a teleosemantic project must account for in order to provide a proper naturalization of intentionality. I will introduce them and show in the next section how a central theoretical principle supported by teleosemantics prevents an adequate explanation of these *desiderata*, *unless* this teleosemantics commits the fallacy.

The naturalization of teleology has always been a headache for philosophers and theorists of biology. It seems perfectly natural to give living systems goals and purpose, but there are serious obstacles to doing so in scientific explanations. The main puzzle of teleological explanations concerns causality. Precisely, the *temporality of teleological causation.* Accordingly, teleological explanations cite the effect of an activity to explain this activity. The heart pumps blood *because* pooping blood has certain functional effects. However, the "because-relation" of the previous section seems to involve backward causal relations. Supposedly, scientific explanations cannot violate the *Causal Asymmetry Principle* (Potochnik, 2017): causal relationships are always asymmetrical, past events cause future events, not the other way around. While the foundations of science require that present events cause future ones, in teleological explanations it seems that present events (e.g. the pumping of blood) are caused by future ones (the effects of pooping blood). Certainly, this inversion of causal relations in scientific explanations is untenable, because it presupposes that the causal relations are symmetrical.

Vitalists argued that the causal structure of teleological explanations calls into question the naturalization of teleology. Others argue that teleological explanations are not causal explanations (e.g. Walsh (2012)). However, the most common scientific strategy to deal with this conundrum is to argue that teleological explanations can indeed be formulated in terms of past events (Neander, 2018). It *seems* that we explain a current event by citing future events but if we uncover the proper causal structure of teleological explanations, we will find that past events give rise to current events. It *seems* that teleological explanations presuppose symmetrical causation but the causal structure of teleology actually preserves the principle of asymmetry. What is needed is to uncover such causal structure. So what is the causal structure of teleological explanations?

There are many answers to this question. However, our aim of analysis in the context of teleosemantics concerns the naturalization of teleology (and intentionality) on the basis of evolutionary processes. Here we find the central key for dealing with this question: recursive *A-B-C-A* chains. These chains are looped sequences of events. Crucially, if the effect of event *A* is the cause of another event *B*, which in turn causes *C*, and *C* causes *A*, then *A-B-C-A* chains are recursive processes in which causes and effects interact according to the asymmetric principle. From an evolutionary perspective, the effect of past selection leads to new organisms that are also under selection control. In this context, the key is heredity. Inheritance enables recursivity: trait *A* in one generation produces a series of effects by which it is selected so that *A* will reappear by inheritance in the next generation. So asymmetric causal relations are preserved insofar as the *effects of past events cause current events*. The key message is well expressed by Nicholas Shea (2018, 59): "Without the historical angle we would be back to the mystery of teleological causation, the mystery of how it is possible to explain a cause in terms of the type of effect it is likely to produce." Following Rama (2022, 65), we will call this the *Historical Desideratum*:

> (HD) *Historical Desideratum*: Without a historical dimension concerning an adaptive bias operating in the past, there is no teleological analysis in the present.

Teleosemantic projects based on evolutionary processes can definitively explain HD. This is their main advantage and has helped to overcome other alternatives to biological theories of function (such as Cummins' theory (1975)). From this evolutionary perspective, the effects of past selection cause current events. As Macdonald and Papineau (2006, 10–11; emphasis in original) wrote:

> On this account of function, functions are the upshot of prior processes of selection. A trait has a function if it has been designed by some process of selection to produce some effect. In the central cases, where the traits in question are biological *adaptations*, the selection process will be non-intentional natural selection. An effect of a trait counts as its function if the trait has a certain history: in the past possession of that trait produced the relevant effect, which in turn had the consequence of facilitating the reproduction of items with that trait. In such cases, it is natural to adopt teleological terminology, and say that, in the normal case, the trait exists *because* of an effect the trait can produce, or *in order to* fulfill its function.

So if we want to know why an organism has the functions and goals that it has, we have to take a look into the past. However, it is also important that teleological explanations based on past events are able to explain the current functioning of a trait. This is the *Actuality Desideratum*:

> (AD) *Actuality Desideratum*: a teleological (and thus teleosemantics) account must be capable of identifying the actual, intrinsic properties of a (token) intentional/teleological system *X*.

Since the primary goal of teleosemantics is to achieve naturalization, let us examine intentionality, although the argument is also applicable to teleological explanations. The reasons why the AD needs to be explained are fairly clear. Suppose we have two distinct systems (tokens), *X* and *Y*. *X* is an intentional system, say a particular human, while *Y*, say a rock, is not an intentional system. Teleosemantics, using a naturalistic language, must precisely determine the distinguishing features between *X* and *Y* that make the former an intentional system and the latter a non-intentional system. What inherent features of *X* are missing in *Y*? What are the differences between *X* and *Y* that make the former an intentional system and the latter not? It is not only important to determine the intrinsic attributes of *X* that give it intentionality, but also to clarify why the lack of such attributes in *Y* makes it non-intentional. Why is the movement of planets non-intentional while human behavior is intentional? What properties do intentional systems exhibit that are absent in planetary motion? If we can

elucidate the intentionality of *X* using naturalistic explanations, we show that intentionality is an inherent aspect of nature and that intentional explanations are scientifically valid.

There is another way of pointing out the necessity of accounting for the AD. As explained above, a central aim of all teleosemantic projects is to account for the normativity of an intentional system. In other words, the representation of the world involved in intentional cognitive processes must be subjected to normative evaluation. A clear case is that of misrepresentation: when a system misrepresents the world, i.e. when the system does not follow the norm of proper functioning. The decisive factor is that the errors are made by individual systems, by tokens. Something happens to a particular individual that has caused him to make a mistake. This proves the requirements of an intrinsic analysis of the properties of individual intentional systems, as demanded by AD. Suppose now that we have two intentional systems, *x1* and *x2*, belonging to the same species *X*. How can we judge, for instance, that *x2* works well, while *x1* does not work well, without looking at the intrinsic differences of these systems? What are the differences between x1 and x2 that made the latter being mistaken? How do we assess normative valuations of tokens without looking at the individual level of analysis? In short, we need to explain the intrinsic properties of a system that make them intentional. We need to account for the AD.

Is it possible to provide an evolutionary explanation of teleology based on past natural selection that explains the intrinsic intentional properties of certain tokens? In other words: Is it possible to provide an evolutionary explanation of teleology that is suitable for the HD and can also explain the AD?

5. The Explanatory Gap and The Phylogeny Fallacy

We pointed out that mainstream teleosemantics is suitable for dealing with the HD. Past selection of populations explains proper functions. Riddles come when we look at the AD. I will show in Section 5.1 that there is an explanatory gap which is based on the population perspective of the SETF, and the need to explain the AD; a gap between what the SETF explains and what it is needed to explain. In Section 5.2, I will argue that this gap is closed by committing the Phylogeny Fallacy.

5.1 Populations and Individuals, Types and Tokens

The first step in my argument begins by pointing out that the teleological functions, and hence the biological norms of the SETF are defined at the level of populations. This is explicitly assumed by mainstream teleosemantics. First, because natural selection is a population process: it explains how heritable fitness differences in populations lead to adaptations. Second, the population approach of the SETF is central to its solution to the normativity of intentionality. As explained above, the idea of teleosemantics is that a biological theory of teleological functions could play the role of explaining the normativity of intentional systems in a naturalistic way. The SETF does this by appealing to natural selection. The SETF thus defines norms at the population level: a trait must function according to the role it has played during selection processes. We are referring here to trait types, not tokens. Tokens do not evolve, populations do. Biological norms in the SETF are therefore properties of trait types, i.e. properties of a population. This allows SETF to define errors in the mis-instantiation of a type: when a token does not function according to the type to which it belongs. An error occurs when a token does not match its type.

The distinction between type and token is related to the delimitation of levels presented in Section 2. Types refer to populations, tokens to individuals. So we can expect what is at stake here: is it possible to explain the AD by starting from a population-based theory of biological functions? Can we explain the intrinsic properties of tokens by defining proper functions as a property of types? How can we explain the intrinsic properties of a token that makes a mistake if the norms are defined at the population level?

Natural selection itself cannot answer these questions. They all require an explanation of individual traits and processes, not population processes and trait types. Natural selection may be able to explain why a trait type works the way it does, but it is not adequate to explain how a trait token works a certain way. To summarize, the fact that natural selection explains populations and not individuals introduces an explanatory gap between solving HD on the basis of evolutionary selection processes and AD's call for individual-level analysis. From the perspective of the SETF, what is the difference between an intentional system *X* and a non-intentional system *Y*? In contrast to *Y*, *X* has its origin in a phylogenetic journey shaped by natural selection, argues the SETF. However, this explanation falls short because it does not reveal the inherent features of *X* that give it intentionality. What insight does natural selection offer to understand the inherent properties of an intentional system? Unfortunately, not much. We can know nothing about the intrinsic properties of a living system if we say that it evolved by natural selection. We get no new information about the differences between the two systems that evolved by natural selection just because we say they evolved by natural selection. The inadequacy of natural selection in dealing with individual traits is accepted and is crucial to population thinking in explaining natural selection: "The population is an entity, subject to its own forces, and obeying its own laws. The details concerning the individuals who are parts of this whole are pretty much irrelevant...In this important sense, population thinking involves *ignoring individuals*" (Sober, 1980, 370, emphasis in the original). Therefore, we conclude that the historical and population-based account of the SETF is unable to resolve AD (Bickhard (2003) comes to a similar conclusion by stating that the SETF of mainstream teleosemantics is causally epiphenomenal at the individual level).

We can illustrate this argument with the help of the distinction between proximate and ultimate causal explanations. This will also help us understand, in the next section, why mainstream teleosemantics, in attempting to naturalize intentionality, is implicated in the false argument of the Phylogeny Fallacy. We can understand that the AD requires proximate explanations: we need to understand the proximate causes and processes that make a given system intentional. However, since natural selection provides ultimate explanations, the SETF is inadequate to explain AD. Explaining evolution by natural selection is not the same as explaining the actual properties of a given system that make it intentional. Merely stating that a trait has evolved by natural selection says nothing about the proximate mechanisms that make a system intentional. Intentionality, like any trait, must be something lying to an individual, being the effect of proximate causes and the proximate cause of other effects.

My argument is somehow intertwined with the imaginary experiment of Swampman's case. The swampman emerges as a replica of a human being produced by a sudden, random event, such as an arbitrary collision of atoms. Both the human and swampman exhibit identical behavioral and physiological characteristics. Swampman, however, lacks an evolutionary history and therefore lacks intentionality. The classic problem is that this is a counter-intuitive conclusion, as there is no intrinsic difference between the swampman and the human. However, we can note something else: namely the inability of SETF to explain the intrinsic properties of humans. As explained above, the explanation that a trait has evolved by natural selection is not an explanation of the proximate causes and processes that operate in individuals. This deficiency arises because the SETF fails to get "under the skin" of the system and examine its intrinsic properties. Thus, if we want to explain the intrinsic qualities of an intentional system, we need to understand the proximate causes and proximate mechanisms that make a particular system intentional. Not only is the SETF unable to point out the proximate causes and processes that leave the swampman without intentionality, but it also cannot tell us the proximate causes and processes that make a human an intentional system.

5.2. Bridging the Gap with Replication Biology

The connection between the explanatory gap of the previous section and the Phylogeny Fallacy is quite simple: to close the gap, we must engage in the misleading reasoning of the Phylogeny Fallacy within the SETF. If we

try to explain the proximate mechanisms and causes of intentional systems by appealing to explanations of natural selection, then we are trapped in the fallacious reasoning of the Phylogeny Fallacy. In other words, the Phylogeny Fallacy is committed when one attempts to explain the properties of trait tokens on the basis of an explanation of the properties of trait types. We can recognize the problems with this situation by invoking Mayr's distinction between proximate and ultimate causal explanations. When the SETF invokes natural selection to explain the AD, ultimate causation is conflated with proximate causation and evolutionary explanations replace true proximate explanations.

The goal of naturalizing intentionality leads it to engage in the misleading argumentation of the Phylogeny Fallacy. That is, proponents of the SETF implicitly assume that it is possible to explain misrepresentation (and thus the AD) from the population perspective of natural selection. If mainstream teleosemantics naturalizes intentionality, as its proponents intend, then they must conflate explanatory domains by invoking ultimate explanations in proximate explanations, even if this is a hidden assumption within mainstream teleosemantics. Thus, in contrast to the ostension argument presented in Section 3, here I do not refer to explicit uses of developmental dichotomies or interactionist ideas. Rather, I want to show that the population perspective of the SETF must commit this fallacy in order to achieve its explanatory aim.

There is indeed a more explicit way to show how mainstream teleosemantics commits the fallacy. This concerns the endorsement of a replicator view of biology, in particular a replicator theory of heredity. The core idea is quite simple: if the problem is to reconcile the population perspective of the SETF with the individual-level analysis required by the AD, then we need an appropriate theory of inheritance to bridge this gap –to link types with tokens. Replicator theory is the view of inheritance supported by the MS. The idea of replicators in biology is famously attributed to Richard Dawkins, but in fact, it was already developed during the emergence of the MS in the first steps of neo-Darwinism by Haeckel and Weismann (see Ågren (2021) for a recent conceptual and historical analysis). The idea is that evolution can be subsumed under certain "units". These units must be responsible for producing the raw material for natural selection: They must produce phenotypic variation, they must lead to fitness differences, and they must be inherited. These units came to be called genes –whatever piece of matter genes are. If we argue that the units are replicated generation after generation, we can explain how cumulative selection processes are possible: Evolution could be defined as a change in the units of replication.

The problem with the replication point of view is that the link between inherited units and phenotypic outcomes remains unexplained. It is merely postulated that we can understand evolution by linking inherited inputs to phenotypic selection without knowing how the intrinsic properties of the input-output relationship come about (Rama, 2024a, 2024b). Here we find several ideas that in one way or another express the notion that development can be explained by evolution, as expressed in Heackel's epigraph. For instance, Mayr's view, or any other explanation of development based on inherited information, has been labeled as misleading reasoning in which developmental explanations are replaced by evolutionary explanations: "Mayr's insistence on keeping the causes of development and evolution strictly separate looks rather ominous in light of the fact that Mayr himself was guilty of conflating them. He used to explain development with reference to the existence of an evolved genetic program" (Stotz, 2019, footnote 4). As Stotz correctly points out, the idea that development is explained by an evolutionary genetic program, or that development is the unfolding of instructions that have arisen through natural selection processes, leads to a conflation of different levels of explanation.

A direct link between replicator biology and mainstream teleosemantics is that most teleosemantic theories endorse the MS framework. This thus directly commits teleosemantics to adopting a replicator stance and accepting the positive and negative consequences. However, it has also been made clear that the concept of replication is at the heart of teleosemantics: "[c]entral to the etiological [those teleosemantic projects based on the SETF] account is the idea that individuals gain functional traits as a result of being replicated" (Macdonald and Papineau, 2006, 12; emphasis in the original). The most explicit development of the replicator concept in teleosemantics is attributed to Millikan (1984, Chapters 1 and 2), who uses the concept of copying to define the

natural functions in selection processes. The result is that mainstream teleosemantics must resort to the notion of replication to explain the intrinsic properties of intentional systems in order to bridge the gap between types and tokens. To explain individual properties (required by the AD) on the basis of population properties (natural selection), we must invoke replicator explanations of development based on inherited information, we must engage in the misleading reasoning of the Phylogeny Fallacy.

It is important to emphasize that this problem arises not only in teleosemantic theories in cognitive science but also in teleosemantic approaches in biology that adopt the SETF. Teleosemantic approaches in biology attempt to explain organic processes by invoking a teleosemantic view of inherited information. Here the term semantics is used in a broad sense (probably too broad) to capture information and signaling processes in organic activities. In this sense, information teleosemantics in biology can pursue various goals: understanding biological communication, genetic regulation, plastic behavior, or how inherited information is related to development, among many other questions. As already mentioned, Mayr's view of genetic programs is that of a replicator and uses the concept of information for teleonomic explanations. From this point of view, we can therefore understand that genetic information specifies the content of the phenotypes that must be constructed. The idea of replicator units can be understood in a teleosemantic context if the inheritance units are informationally defined. As a result, "the concept of information is used in biology only for causes that have the property of intentionality [...] A DNA molecule has a particular sequence because it specifies a particular protein, but a cloud is not black because it predicts rain. This element of intentionality comes from natural selection" (Maynard-Smith, 2000, 189-190). All in all, development is the execution of inherited information designed by natural selection: Development reads evolution.

We can therefore point to two important consequences. First, the problem of the Phylogeny Fallacy in teleosemantics is a consequence of its biological foundations: using replicator notions to link the population account with individual phenomena is not an appropriate strategy. Second, the problem of the teleosemantic conception of information –in biology or cognitive science– is not empirical but theoretical. This means that the problem is not caused by reductive genetic claims such as Mayr's. Rather, the problem is that evolved information is invoked to explain developmental processes, i.e. to argue that inherited information provides instructions for development. Recognizing this is central to evaluating the approaches that take an extended replicator view as problematic, i.e. those that argue that inherited information is not only genetic but also epigenetic. Even if we were to recognize non-genetic forms of inheritance, the extended view retains the same explanatory logic: Inherited information in development, whether genetic or not, can be understood in a teleosemantic (or teleonomic) way because it is the product of natural selection. Probably the most robust extended replicator view of teleosemantic information is proposed by Nicolas Shea (2007, 2011, 2013), who claims that inherited information (genetic and non-genetic) can be understood teleosemantically insofar as it represents the phenotypic outcomes that natural selection has previously picked up. The source of intentionality is a selection process in the population, and supposedly we can explain the link between inherited information and phenotypic outcomes (development) as a product of natural selection. Griffiths (2013) has already argued that Shea's view is subject to the Phylogeny Fallacy for precisely the reasons I discuss here: Explaining the relationship between inherited inputs and phenotypic outcomes as a product of natural selection is not the same as explaining the causal relationship between inputs and outputs (i.e. development). Advocating an extended replicator does not provide us with a causal picture of the intrinsic properties that emerge during an individual's lifespan.

6. Conclusions

I started this paper by introducing the Phylogeny Fallacy, the distinction between different levels of explanations, and its connection with developmental dichotomies. Then I show that mainstream teleosemantics systematically commits the fallacy: levels of explanations are mixed and developmental dichotomies are usually central in

teleosemantics. However, I intend to show that this is not a coincidence or something specific to the principal teleosemantic theories produced till now: I intend to show that the Phylogeny Fallacy is a problem for any teleosemantic theory based on the SETF. My argument points out that the fallacy is committed when we intend to solve an explanatory gap: we need to explain the intrinsic properties of particular intentional systems (the AD) but the SETF is a theory about properties and processes of trait types. To solve this gap we should attribute natural selection to the capacity to explain individual-level processes. The conclusion is that the biological foundations of mainstream teleosemantics face a serious problem: the advantages of anchoring teleology in past evolutionary events have the negative consequence of abandoning the possibility of accounting for the AD.

Is there any possible solution? Is it possible to account for both *desiderata*? There seems to be a tension between both *desiderata.* One looks for explanations based on past events, while the other demands explanations based on current events. We saw that the commitment to natural selection seems to be suitable for one *desideratum* but not for the other. The explanatory gap between types and tokens is not possible to close without committing the Phylogeny Fallacy. So we need to provide a historical explanation not based on trait types; historical explanations should not be based on population biology. Moreover, we need to provide an explanation based on proximate causes, so that we can point out the actual mechanism operating on individuals and account for the AD. So we need a *historical explanation based on proximate causes*. While the scope of this paper transcends the aims of answering this question properly, lineage explanations (Calcott, 2009), central in evo-devo, might be a suitable way of preserving the historical side of teleosemantics without abandoning the proximate level of analysis (Brown, 2021).

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