Evolutionary Causation and Teleosemantics

Abstract Disputes about the causal structure of natural selection have implications for teleosemantics. Etiological, mainstream teleosemantics is based on a causalist view of natural selection. The core of its solution to Brentano's Problem lies in the solution to Kant's Puzzle provided by the Modern Synthesis concerning populational causation. In this paper, I suggest that if we adopt an alternative, statisticalist view on natural selection, the door is open for two reflections. First, it allows for setting different challenges to etiological teleosemantics that arise if a statisticalist reading of natural selection is right. Second, by providing a different solution to Kant's Puzzle based on individual causes of evolution, statisticalism promotes a different answer to Brentano's Problem, what I label as Agential Teleosemantics.

Keywords: Evolutionary causation; Causalist vs Statisticalist; Etiological Teleosemantics; Agential Teleosemantics; Biological Agency.

0. Introduction

In the first paragraph of most papers about teleosemantics, this approach is usually introduced as the main attempt to naturalize intentionality. The reason is that its proposal is rooted in a biological account of natural teleology. This work is about such roots. I approach this issue from current debates between the statisticalist and causalist views on the causal structure of natural selection. I aim to argue that the statisticalism vs. causalism debate has important consequences for teleosemantics. Here I adopt a statisticalist viewpoint. The implications are twofold: a critique of teleosemantics and a reconstruction of it. First, it signifies different challenges to etiological (mainstream) teleosemantics –insofar as it is based on a causalist view of natural selection. Second, the statisticalist view allows for the development of an alternative solution to the naturalization of intentionality based on a different view on the causes of adaptive evolution.

A central issue around the statisticalism vs. causalism debate concerns the explanatory role of individual organisms in evolution. As remarked by many, the Modern Synthesis has managed to eliminate organisms from their explanation of evolution. This gave rise to black-boxing development (Hamburger 1980). Accordingly, evolution can be explained by looking into sub-organismal units of replication –genes- being arranged in supra-organismal units –populations- adaptively biased by natural selection. Organisms are not part of the picture. Even though the statisticalism vs. causalism debate concerns the nature of populational explanations –whether they are causal or statistical, different positions regarding the explanatory role of organisms in evolution provide support for either side.

I outline two implications for teleosemantics from a statisticalist viewpoint: challenging teleosemantics and resetting it. I propose two readings of these implications. The *weak implication* is formulated as a conditional: *if* the statisticalist view is correct, *then* teleosemantics must be challenged and reframed from a statisticalist perspective. The *strong*

implication is to argue for the antecedent of the conditions, i.e. that the statisticalist view is correct, and therefore, to defend the consequent. I will support the strong implication, even though I find the weak implication a valuable analysis to situate teleosemantics in debates about the nature of natural selection. As I will argue towards the end of the paper, support for statisticalism comes from recent proposals in organismal agency and its roots in individual-level causation.

In section 1, I start by introducing Brentano's Problem on intentionality and Kant's puzzle on teleology, in order to present the core of the teleosemantic project and the form it has been taking since its origin. In section 2, I present the causalist view and its relation with etiological teleosemantics. In section 3, I move to a different thesis concerning the causes of natural selection, what is known as the Statisticalist School. After presenting it in detail, I put forward two challenges to etiological teleosemantics that arise if we accept statisticalism. Finally, in section 5, I present an alternative teleosemantic project motivated by the Statisticalist School, where the causes of evolution are seen as ontogenetic in nature and related to the *adaptive agency* of individual organisms. I conclude with an outline of the core tenets of *Agential Teleosemantics.*

1. Brentano's Problem through Kant's Puzzle: setting teleosemantics' core

1.1 Brentano's Problem and Kant's Puzzle

Brentano's Problem is a good starting point to understanding the contemporary issues about the naturalization of intentionality. Intentionality concerns the capacity of certain natural states (paradigmatically, cognitive states) to be about or refer to something else. Brentano's view placed intentionality in a paradoxical situation. Intentional explanations are explanatory useful and indispensable to understand goal-directed behavior, yet intentional explanations seem not to be aligned with the foundations of modern science. This implied, as Brentano emphasized in his *Psychology from an Empirical Standpoint*, that intentionality cannot be naturalized. There cannot be a science of goal-directed behavior that involves intentionality.

The issue turns around causation. To see this clearly, let's present the contemporary version of Brentano's Problem under the view of cognitive science and the philosophy of mind. In a nutshell, the mainstream view since the Cognitive Revolution in the mid-twentieth century posits that goal-directed behavior could be explained by appealing to the processing of representations. The idea is that animals are able to represent the world in a certain way. Such representations are achieved by complex mechanisms of perception and categorization. The information reached about the environment is processed in such a way that the animal behaves according to both its representation of its environmental circumstances and its inner goals (desires, needs, emotions, etc.). Since then, different disciplines within cognitive science have emerged incorporating some form of representationalist talk, and their inquiries are devoted to understanding how animals are able to represent the world, process such contentful information, and behave intelligently and adaptively.

Note first, as Brentano did, that intentional states could be about, for instance, non-existent objects or future scenarios. However, if we explain behavior based on how the organism responds to the world, it seems difficult to figure out how we can provide causal explanations involving non-existent objects. Shortly, the trouble arises when the causal chains underpinning certain goal-directed behavior involve a non-existent object. Non-existent objects are not part of causal chains; non-existent things have no causal powers, but it seems that representations of such non-existent objects do have causal powers. This is usually presented as a mismatch between the existence of the intentional object and the existence of the world object; that is, there could be an intentional object (a representation) without reference. This mismatch needs to be accounted for insofar as it seems that representations of non-existent entities are necessary to explain certain behaviors. The principal trouble connected with this mismatch is latent in the issue of misrepresentation –a highly discussed issue in the teleosemantic literature. Intentional states could misrepresent the world. We have false beliefs, hallucinations, perceptual errors, conjectures, imaginations, false scientific theories, and so on. Even in the simplest cases, such as the famous case of frogs trying to catch flies, misrepresentation is present. In this sense, we can say that intentionality transcends what is actual. Let´s call Causal Mismatch the mismatch between the intentional-object and the world-object, insofar as such relationships seem not to be suitable to be understood in terms of causal chains.[1]

Brentano's Problem (under a contemporary reading) arises when we appreciate the following two points: First, intentionality explains. Intentional states are systematically used to predict, understand and systematize goal-directed behavior in a truthful way. These kinds of explanations are usually labeled as *Folk Psychology*. However, to refer to them, I will opt for the name *Folk Intentionality,* to emphasize the parallelism with *Folk Teleology* (cf. below) and to provide a broader view of Brentano´s Problem without assuming that whatever is intentional is also psychological. Moreover, since the begging of the cognitive revolution in the mid-twentieth century, practically the whole cognitive enterprise is grounded in *Folk Intentionality* in a way that intentional talk plays a central explanatory role in any of the subfields of cognitive science –this includes also non-classical accounts of intentionality within the so-called post-cognitivism, but clearly not some eliminativist positions concerning intentionality. However, secondly, the Causal Mismatch is not present in other sciences like physics or chemistry. These sciences were taken as the paradigm of scientific progress and the foundational roots of modern sciences since Descartes. Supposedly, in physics and chemistry, there are no gaps in the causal connections that lead to the *explanandum*. Step-by-step causal

[1] It is relevant to remark that the connection between causation and misrepresentations takes place within teleosemantics' aim of naturalizing intentionality. In this sense, teleosemantics was born against two classical approaches to representational content. One is the idea that the content of a representation is determined by its relation with other representations –known as intensionalist theories. The difficulty of this attempt is naturalism, insofar as we cannot be naturalists and simultaneously anchor intentionality in other intentional stuff. In this view, misrepresentation cannot be a problem of causation insofar as content is not determined by reference but by how representations relate with each other. Teleosemantics opposes this view; it provides a referentialist theory of content. However, the second theory that teleosemantics rejects is the causalist one, which is also referentialist. Even if causalist theories of content pursue a naturalistic view of intentionality, they seem incapable of dealing with misrepresentations. In this sense, teleosemantics emerged as the attempt to solve misrepresentation and simultaneously accept the causalist view of intentional explanations defended by causalist theories. Therefore, misrepresentation becomes primarily an issue of causation once we stress the commitment of teleosemantics to naturalism. I thank an anonymous review for noting the importance of this clarification.

interactions, even if complex, upward or downward, produce the phenomenon to be explained. Here lies what is sometimes labeled as the Explanatory or Causal Asymmetry principle in science (Bromberger 1966, Potochnik 2017): step-by-step causal chains go from the past to the present, from the *explananda* to the *explanandum*. We can, therefore, explain the behavior purely based on neurophysiological causation; i.e. on those causal processes taking place in the nervous systems that step-by-step produce a particular behavioral outcome. However, such kinds of neurophysiological explanations would not involve any sort of intentionality, and consequently the behavior explained could not be treated as goal-directed.

Brentano's problem lives in this contradictory scenario. On the one hand, intentionality is explanatory central in the cognitive enterprise, but intentional states cannot be understood under the same foundations of modern science. In other words, if causal interactions between nerve cells produce a certain behavioral outcome, and (ontological dualism aside) intentional states are neural states, how is it possible that intentional explanations give rise to the Causal Mismatch?

Kant's Puzzle concerns teleology. Its structure parallels that of Brentano's Problem. I am not going to expand very much here because I will retake this issue in the next section. Teleology concerns the capacity of certain systems to have a certain goal or purpose to fulfill. Teleological explanations in biology, therefore, appeal to what a certain system pursues in order to account for the functioning, activities, and behavior of such a system. Kant's position concerning teleology parallels Brentano's position concerning intentionality. For Kant, organisms cannot be understood without appealing to teleology. We cannot understand how organisms work, their complex organization, and their aptness to the environment eschewing teleological talk. However, he did not believe that teleological explanations could be a genuine part of science. According to Kant, in his *Critique of Judgment*, there cannot be science for purposive natural systems. Why is this so?

The reason, again, turns around causality. The kinds of explanations involved in teleology seem not to be aligned with the kinds of explanations accepted in science, even though teleology appears to be inexorable in our understanding of nature. To appreciate the paradoxical situation, let's remark first that teleology, just like intentionality, offers us, and scientific practice, a useful and veridical way to predict, explain, systematize, and interact with living beings. To unify terminology, I will call *Folk Teleology* these kinds of explanations. The second point to recognize is the status of the causal bases posited in teleological explanations. As noted, teleological explanations appeal to purposes and goals. Nonetheless, such purposes concern future stages, possible outcomes to be obtained. How is it possible that future stages explain current activities? Here causation enters the scene. An outcome cannot cause those processes that produce it. While, as explained before, science is built from step-by-step forward causal interactions –in biology, the psychochemical causes of physiological processes-, teleological explanations seem to involve backward causation. This character of teleological explanation violates what the Explanatory or Causal Asymmetry principle. The causal interaction underpinning any scientific explanation must run in one direction, from previous states of affairs to future states of affairs, not the other way around; the asymmetry regards

the fact that causal interactions —even if complex, upward or downward— are unidirectional in time.

We can therefore see Kant's Puzzle also as a problem of naturalization. As with the case of intentionality, there is a tension between what is explanatorily desirable —Folk Teleology— and an uncomfortable situation with the causal basis of scientific explanations —Causal Asymmetry. The project of naturalizing teleology aims at figuring out how teleology can be legitimated by science without involving old-fashioned causation.

We can appreciate many connections between intentionality and teleology. Particularly, here I shall focus on problematic aspects of both phenomena. These connections are presented in figure 1. First, both riddles —the problem and the puzzle— take place when we appreciate that intentionality and teleology are explanatory necessary but still explanatory illegitimate. This creates a gap between the kinds of explanations and causations reputable in science —Causal Mismatch and Causal Asymmetry— and the advantages of assuming teleological and intentional positions —Folk Intentionality and Folk Teleology. Is it possible to bridge this gap in naturalistic terms without throwing the baby out with the bathwater?

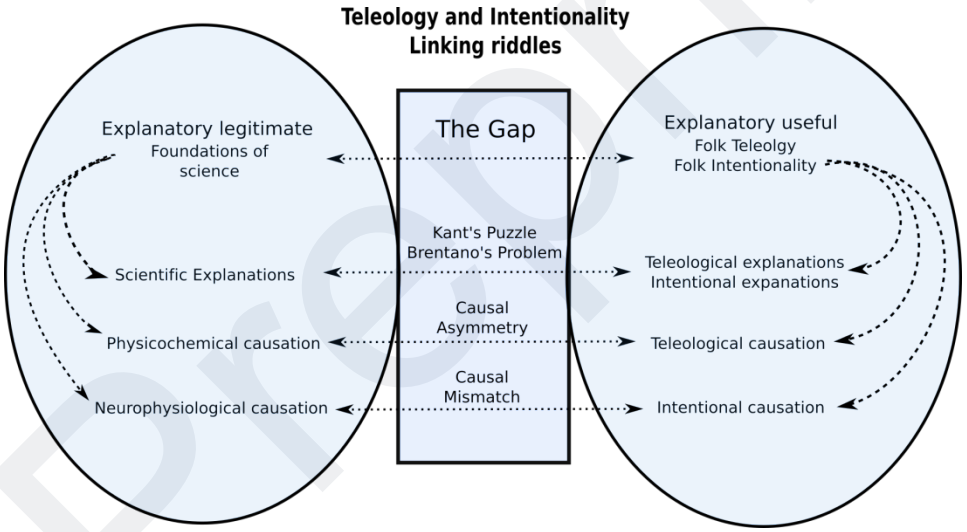

Figure 1: The connections between Kant's Puzzle and Brentano's Problem. Both concern a tension between useful and legitimate explanations according to the foundations of science. This gives rise to a gap that naturalist projects aim to bridge. It consists in explaining how teleological causation could be compatible with physicochemical causation such that the principle of Causal Asymmetry stands, and accounting for intentional causation in terms of neurophysiological causation to solve the Causal Mismatch

1.2. Teleosemantics' core and etiological teleosemantics

Teleosemantics' core is to provide a naturalist solution to Brentano's Problem on the basis of *any* naturalist solution to Kant's Puzzle.[2] This involves two important theses. First, intentional states have teleological functions: any cognitive phenomenon performs a certain task directed towards the fulfillment of a certain goal. Second, intentional states, as natural phenomena, can be analyzed with the same tools as other natural traits, in a way that the analysis of teleological functions done in biology also encompasses that of cognitive functions. These theses are not going to be discussed here insofar as they are generally accepted within teleosemantics. Before moving on, note however that I remarked that *any* solution to Kant's Problem can be the key to unknot Brentano's Problem from a naturalist standpoint. Somehow this is true if we accept both theses. This just stresses the connection between teleology and intentionality without presupposing any particular solution to Kant's Puzzle. Thus, this can be a classical etiological account or any other attempt to deal with Kant's Puzzle. By the end of the paper, I will argue that there are other sources of teleology beyond natural selection that do not face the problems that I will present later on. With these remarks in mind, I just wish to highlight that the Teleosemantic Project is not committed to any particular teleological position (although historically it appears to be so).

The Teleosemantic Project is first of all an attempt to bridge the Intentional Gap. The building blocks of the bridge are provided by biology. Mainstream teleosemantics is etiological teleosemantics. It is based on a teleological theory of trait functions —cognitive functions included. It, therefore, relies on a particular biological framework that provides a specific solution to Kant's Puzzle.

What is the solution to Kant's Puzzle endorsed by etiological teleosemantics? I am going to get into specific details in section 4. For the time being, it is enough to start first by referring to its teleological basis: natural selection. The etiological theory of functions ascribes functions to traits according to the effects that they produce. Etiology can take many forms. Standard views in etiological teleosemantics are based on the Selected-Effect Theory of Functions (SETF), which is (in general) rooted in natural selection (Millikan 1989, Neander 1991). The idea is that the function of a trait is defined by those effects that such trait has had during the evolutionary process of natural selection. The function of the heart is to pump blood insofar as the effects of pumping blood make hearts being selected. Note that this theory of functions is teleological: it explains why a trait is present in nature by positing a certain purpose that it should fulfill. From this basic framework, teleosemantics extends the analysis to the cognitive domain. The core idea is that the Causal Mismatch is possible insofar as cognitive functions are selected-effect functions. Therefore, malfunctioning is possible. This means that there is room for misrepresentation. If a trait token (a cognitive function of a certain individual) does not do what it was determined by the trait type it belongs to (the evolved type), then it is possible to misrepresent the world, make errors, or produce maladaptive behaviors. Crucially, no intentionality seems to be involved in the explanation of the Causal Mismatch. This is good

[2] Certainly, there could be a non-naturalist teleosemantic project. For instance, if we adopt a theological view of organism design, we can define the function of representational systems but in this case not from a scientific perspective. Moreover, as pointed out by an anonymous reviewer, there might be an attempt to solve Brentano´s Problem from the notion of biological function with any specific solution to Kant's Puzzle, such as for instance, claiming that the notion of function is fundamental. As the reviewer recognizes, the teleosemantic project that departs from such a position may not be considered naturalist.

news because the main task is to solve Brentano's Problem in naturalistic terms. An obvious requirement for such an aim is that the explanation of intentionality does not presuppose intentionality. If we are capable of understanding the nature of intentional states in terms of evolutionary biology, the requirement is fulfilled (but see Fodor and Platelli-Palmarini 2010).

2. Evolutionary causation: the Causalist School

Etiological teleosemantics has been both strongly criticized and passionately defended. As a result, novel and interesting proposals have been emerging since its inception (cf. Shea 2018, Neander 2017a, Millikan 2017, for some recent proposals). Yet, most of (but not all, see e.g. Bickhard 2003, Mossio et al. 2009) the challenges it confronts are not related to its biological foundations (its source of Natural Teleology), rather they come from the teleosemantic analysis of representational content. My contention is that, if we accept a non-causalist, statisticalist view of natural selection (cf. section 3), then SETF based on natural selection is not a fertile ground to root the causal basis of intentionality. Or, in other words, that natural selection does not provide what Brentano's Problem requires. This means, as we will see in section 4 based on the Statisticalist School, that the solution to Kant's Puzzle on which etiological teleosemantics rests is unsuitable for teleosemantics' aims. To see why this is so, I need first to say something more about the solution to Kant's Puzzle that the SETF (in its evolutionary version) appeals to.

2.1. The causal structure of etiological teleosemantics

Before sketching the teleological underpinnings of etiological teleosemantics, let me first highlight what the main *explanandum* of teleological explanation is. This will help me analyze whether such teleological underpinnings really provide proper *explananda* or they need to be refurbished.

Kant's main worries were about the organizational properties of organisms. Organisms are arranged in a highly complex way that allows them to preserve and reproduce life. The complex interactions in which the many parts of an organism participate are suitable for the organism's life conditions and needs. Kant believed that this kind of complex organization, tied to organismal needs and responsible for the organism's activities, requires teleological explanations. I opt to extend the view of Kant and posit that teleological explanations deal with the adaptive dimension of organic systems. This, importantly, is not reduced to inner organizational properties but also to the external relationship that any organism bears with its environment. To use some contemporary jargon (Maturana and Varela 1980, Moreno and Mossio 2015, Kauffman 1993): we can appreciate the adaptive character of organic systems both in their *operational/organizational dimension* and in their *interactive dimension*. In this sense, teleology deploys the role of explaining how the complex organization of an organism works —through inner regulation— to fulfill its own needs, and how the organism confronts its environmental conditions (through behavior or any kind of motility mediated by sensorimotor capacities) adaptively. Shortly, the moral is that *teleology is there to explain aptness*. Kant's Puzzle is to understand such explanations in causal terms. Aptness concerns the organismal capacity to sustain and reproduce one's life according to one's own needs, adequate both to one's inner and outer conditions, or what Darwin pictured as "those exquisite adaptations of

one part of the organization to another part, and to the conditions of life" (Darwin [1859]1996, 114).

Teleosemantics' core is to anchor intentionality in natural teleology; that is, to connect Brentano's Problem with Kant's Puzzle. While the *explanandum* of teleological explanation is the aptness of living beings, the *explanandum* of intentional explanation is one specific kind of adaptive activity that organisms perform: goal-directed behavior. This is crucial for arguing that the analysis of natural teleology incorporates the analysis of intentionality. As both riddles concern causation and goal-directed behavior is one kind of adaptive activity in organisms, the causal grounds of teleology provide a solution to the causal underpinnings of intentional behavior. As remarked, the causal basis of the SETF is natural selection. In the next subsection, I will describe the causalist view of natural selection understood in terms of populational forces.

2.2 Populational causation

The main source of teleology on which teleosemantics rests is natural selection; that is, on explanations of aptness grounded in natural selection. Darwin was motivated by Paley's remarks of organisms' aptness and their design-like character, but he rejected Paley's answer. Henceforth, Darwinian selection became the mainstream answer for understanding the aptness of organisms, overcoming previous and competing views. As Mayr stated, "Darwin had solved Kant's great riddle" (Mayr 1991: 131). Consequently, aligned with teleosemantics' core's strategy, Darwin had provided the key to both Kant's Puzzle and Brentano's Problem. However, the causal basis for intentional causation does not come from Darwin himself but from 20th-century scholars working in the Modern Synthesis (henceforth, MS) framework. Intentional causation eventually rests on what came to be known as the Causalist School on natural selection; i.e., the idea that the causes of aptness lie at the populational level. We will see that, indeed, Darwin's ideas on evolutionary causes cannot be grounded in this view.

The main locus of the Causalist School on natural selection latent in the teleosemantic literature is Elliot Sober's *The Nature of Selection*.[3] The view pictured by Sober consists in looking at populations as *entities on which different causal forces act* —principally natural selection— in such a way that evolutionary biology needs *not put its hands on individual phenomena*. This signifies that *trait types*, not trait tokens, are those natural kinds on which the evolutionary causes rely, hence determining the function of the trait —what is known as *selected-effect* functions. Let's present each piece of this picture starting with the following quote:

> . . . 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, p. 344, emphasis added).

[3] Another figures of the Causalist School are Abrams (2012), Millstein (2006), Pence and Ramsey (2013), Ramsey (2016), Reisman and Forber (2005), and Stephens (2004).

Sober proposes to see that different forces act on populations through evolutionary history, in a parallel way as different forces act on objects in a Newtonian paradigm. Such forces are migration, mutation, drift, and more importantly, insofar as it biases the course of the population in adaptive ways, natural selection. As he notes, the fact that the causes of adaptive evolution lie at the populational level opens up the possibility to ignore individuals; that is, natural selection explanations need not take into account how organisms die, live, and reproduce; they rest on the variation in fitness within groups or populations.

What are those natural kinds that provide the causes of variation on fitness that are relevant for selection explanations? In Sober's proposal, they are trait types. The fitness of a trait in a population is the core element in the *explananda* of evolutionary processes. Evolution is thus explained not by how individuals differ on fitness as a consequence of trait variation, but by how populations possessing such traits do. In section 3 I will discuss whether the notion of fitness involved is a causal or a statistical one, and consequently, whether the selection process present in populational thinking involves causal or statistical explanations.

On these grounds, Sober proposes his well-known distinction between selection-for and selection-of effects (Sober 1984: 97-102). The former concern the causal role that a trait performs that makes it being selected, preserved, and spread in a population: "'Selection for' is the causal concept *par excellence.* Selection for properties causes differences in survival and reproductive success" (Sober 1984: 100, emphasis in the original). *Selection of* regards those properties of the selected trait that do not play any relevant causal role; *selection for* is causally relevant for fitness variation and selection, while the latter is not: "When there is selection for one trait and selection against another, the traits make a causal difference in survival and reproductive success" (Sober 2013: 339). Let's put an example. Imagine you have a salt shaker and you put inside two kinds of salt: one thin and white and the other thick and pink. At the time of seasoning up your dish, only the thin and white salt will go through the holes of the salt shaker, while the thick and pink one will remain inside. In this scenario, thin salt was selected for seasoning up your food because it is its being thin what made it pass through the holes; its whiteness is not a force because in the processes of selection this property did not play any relevant function, it was only selected of the population of salt grains in the salt shaker. What is relevant here is that the notions of selection-for and selection-of regard trait types; that is, whether the presence of a trait in the members of the population does or does not contribute to fitness variation.

Another important defense of the Causalist School can be found in Ernst Mayr's distinction between ultimate and proximate causation (Mayr 1961, 1974). Proximate causation belongs to the individual and ahistorical levels. Ultimate causes correspond to the evolutionary and historical levels. This also means that the difference in causes underlies a difference in the kind of question such causes are invoked to answer. Proximate causation concerns how-questions, that is, how organisms function. Ultimate causes regard why-questions, that is, why organisms function in certain ways. Moreover, such questions are approached from different disciplines within biology. How-questions are addressed by functional biology —e.g. physiology, developmental biology, morphology, while evolutionary biology is devoted to answering why-questions. As Mayr stated, his distinction puts some order within the many roles that each

discipline in biology plays. This introduces a division of explanatory labor. The *explanandum* of each discipline can be determined according to this distinction in a way that the scope of explanatory aims is constrained and specified, both concerning what they can and should explain, and what they cannot and shouldn't attempt to explain.

The distinction between ultimate/proximate causes is central to understanding the teleonomic character of living beings. Striving to stay away from the unpleasant connections that teleology has had in the history of biology, and to defend a purely mechanistic purposiveness (Mayr 1961: 1504), Mayr borrowed the term "teleonomy" from C. Pittendrigh (1958) to speak about the aptness and design-like character of individuals without non-natural connotations. In Mayr's view, a teleonomic system is any system that is the result of a program. This goes from human-made machines to living beings. As it can be appreciated, teleonomy tries to account for the aptness of living beings insofar as we can explain which are the purposes of each part of a programmed system by identifying the designed program that had built the system. The role ascribed to a system by a program, therefore, explains the capacity of the system to be intrinsically and extrinsically apt. The successfulness of the system relies both on the adequacy of the program to the system's conditions of existence and on the implementation of the program. Mayr stresses that natural selection has no teleonomic character but that individuals do. However, he can conclude that the teleonomic character of an individual is caused by ultimate causation and explained by evolution. First, he remarks that the teleonomy of individuals is a consequence of being genetically programmed (Mary 1974: 114). Second, as natural selection belongs to the realm of evolutionary biology and it is the main ultimate cause in biology, ultimate causation and evolutionary biology tackle the teleonomy of individuals.

Even though Mayr's and Sober's views are different, there exist relevant connections concerning the SETF. The first one is that both proposals allow to speak about natural design without involving a designer, as Darwin intended. In other words, both are proposals devoted to understanding, from a naturalistic standpoint, the aptness of living beings. More importantly, in both proposals, the causes of aptness do not lie at the individual level but at the populational one. Selected-effect functions are causal at the populational level, and the teleonomic character of individuals is caused by ultimate causation coming from natural selection. Therefore, why-questions are eventually answered by evolutionary biology. Natural selection understood as a causal process at the populational level is the main tool to explain aptness. These elements constitute the process of adaptation by natural selection, where a trait "A is an *adaptation for* task T in a population P if and only if A became prevalent in P because there was *selection for* A, where the selective advantage of A was due to the fact that A helped perform task T" (Sober, 1984, p. 208, emphasis added). We can summarize the causalist position by saying that *adaptation is the key causal notion responsible for producing aptness* in living beings.

The causalist view on natural selection works for etiological teleosemantics. First, it provides the necessary causal ground. Evolutionary causation allows to understand proper cognitive functions in a naturalist way and to solve the problems underlying causation. Natural teleology is not about future stages causing current ones, but about past selection processes attributing proper functions to traits. Moreover, the mismatch between the intentional object and the

world object may be explained at the level of tokens. The function of a particular representational system is to represent whatever it was designed to represent according to the type it belongs to. However, an error is understood as a deviation from evolutionary design. In other words, by attributing functions to trait types, it is possible to understand error at the token level, as a mistaken instantiation of the type it belongs to. As etiologists argue, this explanation of error does not presuppose prior intentionality. So the intentionality of the mind is rooted in a secure land. Or so it seems.

3. Evolutionary causation: the Statisticalist School

The statisticalist view, whose foundational works are those of Walsh et.al (2002) and Matthen and Ariew (2002), as its name clearly suggests, claims that natural selection and other evolutionary processes involve statistical, not causal explanations.

The key term in natural selection explanations is trait fitness. This, as emphasized by statisticalists (Ariew 2003; Ariew and Lewontin 2004), is different from individual fitness. Trait fitness is a property of traits belonging to a population. Individual fitness concerns individual life successes based on persistence and reproduction. Crucially, while "[t]rait fitness is the average survivability of a group of individuals possessing a type of trait" (Ariew 2003: 562), individual fitness concerns those causal processes that produce the persistence and reproduction of an individual. And here lies the difference. Trait fitness is statistically accessed, while individual fitness is causally accessed.

Populations, in populational explanations, are abstract entities. The parameters involved in such explanations are abstracted from individual-level phenomena. Populational explanations, therefore, are based on an analysis of the statistical properties of populations. The effects on populations are statistical too; they concern the distribution of trait types in a population as a function of variation on trait fitness. This constitutes the core of populational thinking the MS came to defend.

Walsh (2003, 2019) pictures two levels at which the explanation of adaptive evolution rests, the individual level and the populational level. He notes that most of the contemporary discussions around the foundations of evolutionary theory —whether MS requires no modification, an extension, or a revolution— turn around the two-force model. It concerns the idea that beyond natural selection, as an evolutionary cause, we also have individual causes acting on evolution, in a way that discussion turns around the relevance of each level when explaining adaptive evolution. The statisticalist view is not based on the two-force model, but on the two-level one. There are no two competing levels of causes because natural selection is not an evolutionary cause. Rather, there are two types of explanations: individual, causal explanations, and populational, statistical explanations. Once populational causes are removed, the statisticalist view on the causes of adaptive evolution contends, "[t]here is one level of causation; all the causes of evolution are the causes of arrival and departure (the 'struggle for life')...It is 'proximate' causes all the way down" (Walsh 2019, pp. 238, 242).

As I will note later, this provides a non-reducible and indispensable explanatory role for each explanatory level. But, before, it is important to make explicit the connection that exists, according to the Statisticalist School, between the two levels. As remarked, populations, in populational explanations, are abstract entities. This doesn't mean that populations are not composed of individuals. If we put aside the difficulty of specifying the boundaries of a population, populations could be considered sets of individuals. The abstraction of populations lies in the very explanations of populational biology –i.e. how populations are treated in the explanations done in evolutionary biology- not in the ontology of populations. And here is where trait fitness, as a statistical measure based on individual fitness, comes to the fore. Populations need not be abstract to be treated as abstract in populational explanations.

Walsh proposes to see trait fitness as an *analytic consequence* of individual fitness (Walsh 2015, 2019) –cf. Wash (2007) for the related notion of *mere statistical effect*. The idea is that the properties of populational structures are adjudicated on the basis of the mathematical consequences of the arrival and departure of organisms (individual fitness). There are two levels of explanation, the causal-individual and the statistical-populational, where the statistical properties of the latter are a higher-order consequence/effect of the causal properties of the former. Explaining evolution, therefore, requires dealing with the consequences of individual-level causal processes at the populational level to see the changes in population structures through time. Such consequences are analytically assessed from the mathematical theory of populational biology.

As it can be appreciated, this entails a division of explanatory labor and of explanatory scope. We cannot dispense with individual-level causal explanations and population-level statistical explanations. Evolution is after all a historical and populational phenomenon. Populations evolve. Individual-level causal explanations provide individual fitness values, while population-level statistical explanations average them in terms of trait fitness variation in order to predict and explain changes in populational structures. Crucially, individual fitness is a necessary element in the explanation of evolution, yet not a sufficient one to explain evolutionary processes; we cannot explain evolution from the individual level. Trait fitness —as an abstraction of individual fitness- on the contrary, is both sufficient and necessary to explain changes in population structures insofar as it concerns those (statistical) properties of those entities that evolve -populations (Walsh et.al 2002: 460-462). This does not mean that individuals are not relevant for evolution. Individual lifespan provides the causal basis of trait fitness. Without individuals, evolution has no causal roots; without populations, evolution becomes development. As Ariew defends,

> 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).

Statisticalists contend that the Causalist School is not Darwinian (Walsh 2000, 2010, 2015; cf. also Godfrey-Smith (2009)). There are important differences between Darwin's proposal and the view proposed by MS. First, Darwin's insight was that the distribution of form and function deserves populational (and historical) explanations. This is common ground both in the Causalist and the Statistical Schools. Yet, the issue concerns what kinds of explanations are involved. And here lies the difference. Darwin's view is not causalist. According to him, individual causation provides the causal grounds of adaptive evolution. His notion of struggle for life came to encompass individual causation: evolution by natural selection "follow[s] inevitably from the struggle for life" (Darwin 1859 [1968]) (i.e. it is a statistical effect or an analytical consequence of organisms struggling for life). The causes of life, death, and reproduction —the causes from where trait fitness is averaged— are individual causes (I will reframe Darwin's notion of 'struggle' in contemporary terms in section 5). Consequently, Darwinian fitness is not trait fitness but individual fitness. The view of population thinking stressed by Darwin concerns the crucial explanatory role of populational explanations, yet contrary to MS population-thinking, he did not defend a causalist view of natural selection. As Walsh contends, "[t]he source of the error [in the Causalist School], I believe, lies not in the *Origin* itself but in an erroneous metaphysical picture drawn from the Modern Synthesis theory of evolution. That theory explicitly construes selection as a force acting over populations of genes" (Walsh 2000: 137). As Godfrey-Smith (2009) has shown, Darwin's definition of natural selection makes reference to struggle for life, while MS's proposals eliminate all reference to struggle for life and replace it with statistical language. The statistical underpinnings of natural selection were provided some decades after the *Origins*, as expected, by the use of statistics in the theory of natural selection (cf. Walsh (2003) on the statistical underpinnings of Fisher's Fundamental Law and the analogy with thermodynamics).

To summarize, the statisticalist view carefully distinguishes between individual fitness and trait fitness and remarks that trait fitness, which is involved in populational explanations, is statistically accessed from the variation in individual fitness. Individual fitness incorporates the proper causes of adaptive evolution. Evolution is accounted for by citing individual causes in statistical explanations: "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 et.al 2002: 464).

4. Challenges for etiological teleosemantics?

What are the consequences for etiological teleosemantics if the Statisticalist School is right? In this section, I will present two challenges that etiological teleosemantics would face if the statisticalist perspective turns out to be the correct one. In this sense, I will defend the *weak implication*: *if* the Statisticalist School is right, then etiology is challenged. Moreover, by now, I did not say anything about the connection between individual causation and natural teleology. If the solution to Kant's Puzzle rests on those processes that cause the aptness of organisms, and the causes of aptness are individual, we have to see how individual causation involves teleology, and whether it can be naturalized. I leave this for section 5. I will defend that the role

of organismal agency in linking teleology and individual causes provides reasons for defending the *strong implication.*

The challenges presented here concern primarily the etiological theory of mainstream teleosemantics and not teleosemantics *per se*[4]*.* However, the relevance of these challenges for teleosemantics comes to the fore once we recognize that they concern the foundational grounds of teleosemantics, and that, consequently, the challenges are directed to the heart of the proposal of naturalizing intentionality pursued by mainstream teleosemantics. Moreover, the literature discussed in this section comes entirely from teleosemantics, without taking into account other accounts of etiological functions that are not interested in the issue of intentionality.

4.1 Challenge 1: Functions without causation?

The first challenge is that the solution to Brentano's Problem proposed by etiological teleosemantics fails in its biological foundations. First, note again that etiological functions are taken as selected-effect functions based on the idea of selection-for introduced by Sober: "On an etiological theory, functions are what entities were selected *for*. Mere selection *of* a trait is not enough to confer a function on it" (Neander 2017a, 132). This means that etiological functions must play the alleged causal role in evolution. Supposedly, etiological functions cause the existence of a certain trait; it is not merely about the distribution of traits within a population, as the statisticalist would argue: "Selection does more than merely distribute genotypes and phenotypes...: *by* distributing existing genotypes and phenotypes it plays a crucial causal role in determining which new genotypes and phenotypes arise" (Neander, 1995, p. 585, emphasis in original).

There is a *prima facie* problem here. Etiological functions cannot be selected-effect functions just because there are no such things as selected-effect functions *qua* populational forces. If selected-effect functions are defined on the grounds of Sober's work, then selected-effect functions are not referring to any real natural kind. Under a statisticalist view, etiological functions then, on pain of avoiding the same unhappy end, cannot be understood as selected-effect functions.

But more important is the fact that etiological teleosemantics fails to solve Brentano's Problem. This failure lies in the very teleosemantics' core outlined before. As explained, the core idea of any teleological theory of content is to solve Brentano's Problem by solving Kant's Puzzle. Unfortunately, if the statisticalist reading is right, the biological solution to Kant's Puzzle on which etiological teleosemantics rests is wrong. The statisticalist's thesis is universal. All causes of evolution lie at the ontogenetic level. Thus, there is no way that causalists can solve Kant's Puzzle, principally because, under the statisticalist reading, natural selection has no causal weapons to explain the Causal Asymmetry of teleological explanations. Since populational explanations of natural selection processes are not causal, they are not suitable to ground the causal basis of teleology. In this sense, in etiological teleosemantics, the teleosemantics' core itself fails: Brentano's Problem cannot be approached if Kant's Puzzle is not solved first.

[4] I thank an anonymous review for comments on this point.

As explained, the trouble begins when we understand that selected-effect functions are based on trait fitness not on individual fitness. Thus, they are not causal and, as a consequence, neither are etiological functions. Rather, we can claim that etiological functions could be understood as statistical functions based on the change in populational structure as a consequence of the statistical properties of the population –which are a consequence of individual fitness. *I think that this is the proper reading of etiological functions: as statistical functions.* So, one may ask, why etiological teleosemantics doesn't work under a statisticalist view of etiological functions?

The main reason is that statistical functions cannot provide the causal grounds for teleological functions, thus they fail in any attempt to deal with Causal Asymmetry. But there is another important point to highlight which concerns the explanatory role of functions. As Garson and Papineau, defending etiological functions, describe it:

> First, functions are explanatory. One peculiar feature of functions is that, when biologists attribute a function to a trait, they are often trying to give *a causal explanation for why that trait exists*. One virtue of the selected effects theory is that it makes sense of this explanatory aspect of functions (Garson and Papineau 2019: 4, emphasis added).

This is an ontological issue. As these authors remark, the teleological function of a trait must explain why such a trait exists. A teleological function is connected with the adaptiveness of a trait —the adaptiveness of a trait being due to having such function; that is, with the causal role of such function that makes it part of nature. As they highlight, such explanation must be causal, concerning the process that made this trait part of nature. Accordingly, under a causalist view, as Garson and Papineau argue, etiological functions are suitable for this task. In etiological teleosemantics, the proper functions of a representational system concern those causal connections that have produced a goal-directed behavior that made a certain trait type being selected. The problem now is quite expected. Etiological functions, under its statisticalist definition, do not provide a causal explanation for the existence of traits. Thus they cannot accomplish the explanatory role that functions have. Etiological functions, qua statistical functions of trait types, do not explain the existence of a trait in causal terms.

Bickhard (2003) has argued that etiological functions are causal epiphenomenal at the individual level. This is so because etiological functions do not concern how individual systems operate; they are not based on the causal processes within a particular token. This is true independently of one's commitment to statisticalism. The solution of error promoted by etiological teleosemantics rests on the possibility that the causal processes in an individual perform a function that deviates from the functions specified by the evolved trait type; i.e. error is a mismatch between trait tokens and trait types. However, if we accept the statisticalist view, this section concludes that etiological functions are also causal epiphenomenal at the evolutionary level. Statistical, etiological functions are the statistical effect of individual causes. Etiological functions, therefore, if statisticalism is right, are causal epiphenomenal both at the ontogenetic and the phylogenetic levels.

4.2 Challenge 2: Statistical norms for non-statistical explanations?

This second challenge is a direct consequence of the first one, insofar as etiological functions provide the normative dimensions of content needed to solve the Causal Mismatch. Once again, this section is based on the assumption that statisticalism is right. From this standpoint, the problem is, roughly, that the kind of norm provided by etiological functions is not the kind of norm needed for teleosemantics. My argument is structured as follows:

Premise: Statisticalism is right.
1. Teleosemantic norms cannot be statistical.
2. But etiological functions under the statistical view are statistical functions.
3. Then etiological functions provide statistical norms.
4. Therefore, etiological norms are not teleosemantic norms —i.e. they are not the norms that a teleosemantic project can appeal to. Etiological functions are not the kind of function adequate for teleosemantics.

Conclusion: If Statisticalism is right, etiological norms are not teleosemantics norms.

Surely, the critical step is 1. There are two ways of arguing for it. The first one is quite straightforward: to take a look at the literature on etiological teleosemantics to see why etiological norms cannot be statistical. The second way is by providing an argument independent of any specific literature. Let's start by the first one.

The first way to defend point 1 is by noting that etiologists themselves also defend it. A central requisite for a theory of norms is that norms cannot be statistical. This is highlighted by different scholars; thus, Neander: "It might help to note that the normativity of biological functions is neither simply evaluative or statistical" (Neander 1995: 111). A theory of proper functions must give more than statistical generalizations, but "[t]he description of the normal system as the system that functions 'as designed' is thus not merely a generalization but a useful generalization in ways that surpass mere statistical generalization" (Neander 2017b, p. 1161).

The clearest example in the literature is found in the work of Ruth Millikan. She even promotes a typographical distinction in her use of "Normal" instead of "normal" (for instance, in Millikan (1984, chs. 1 and 2) and Millikan (2017, ch. 6)). She "...capitalize[s] *Normal* —to distinguish it from *normal* in the sense of *average*" (Millikan 1984, p. 34, italics in the original). The difference, therefore, entails that Normal denotes a proper function while normal is just a mere average, statistical distribution in a population. "Proper functions do not concern norms in any evaluative or prescriptive sense. They do not concern norms in a statistical sense either. On the contrary, there are many items that usually fail to perform their proper functions" (Millikan 2000: 88).

The conclusion from these quotes is that the norms for teleosemantics cannot be statistical. Etiological functions, under a causalist reading, are not statistical but causal. They do more

than just point out the typicality or high average of traits, they provide a causal criterion to determine when a trait token is functioning properly and when it is not.

This point is usually considered an important advantage of etiological functions over Cummins-functions (Cummins 1975). As Cummins-functions are not teleological functions, the demarcation between proper and improper cannot be based on the natural purposes or the goals of a trait; rather, at most, Cummins-functions can only provide a statistical criterion for function/malfunction demarcations:

> By contrast [with the etiologists], all that the systems account [Cummins Functions] can offer is a statistical criterion: in *most* systems of a certain kind this kind of trait does F, so here the trait is malfunctioning in not doing F. By contrast with the etiological analysis, this statistical systems account seems to lack any normative content: it doesn't seem to show that a trait in any sense *ought* to be doing F; it just says it *isn't* doing F, and so is statistically unusual, but nothing more (McDonald and Papineau 2006; pp.11-12, emphasis in original).

However, there is a way to refute my first defense of points 1. One could refute my argument as follows: the rejection of statistical norms by etiologists aims at demarcating the sense of normal —as high average or typical— from proper normativity. That is, that a trait is frequent or usual does not mean that it was selected for during evolution.[5] As Neander explains in connection to pandemic scenarios:

> there is no incoherence in the idea that functional impairment could become typical in a population for a time, in a pandemic or due to an environmental disaster. The relevant function-dysfunction distinction does not seem to be simply the typical-atypical or expected-unexpected activity distinction. This much is fairly uncontroversial (Neander 2017b: 1152).

Let's call "typical-norms" these kinds of norms based on averages. The central point to rebut my critique is that etiological norms, even if statistical, are not based on the notion of average or typicality. Etiological functions and etiological norms are based on trait fitness. The process of natural selection is explained by the change in populational structure due to its statistical properties. But this doesn't mean that trait fitness and populational change entail that the selected trait is typical or normal. A trait with low frequency could have been selected notwithstanding. Trait fitness is not related to average. The attack to my critique does not deny point 2, but it does reject point 1. *Even if* etiological norms are statistical norms, etiological

[5] Note that Neander's example concerns the current frequency of a trait achieving or not its function. However, this scenario can also be presented in relation to past, historical frequencies. Let's appeal to the case of sperm, used by Millikan. Rightly, she points out that the current frequency of a trait function does not lead us directly to its proper function –sperms perform their proper function at a very low frequency. However, this argument not only applies to current frequencies but also to historical frequencies (its frequency during selection processes): it is not necessary that in selection processes a trait must have a high frequency to be selected for. Sperms could perform their proper function infrequently and nonetheless be selected to perform such function. I thank an anonymous review for comments on this point.

norms are not typical-norms. Point 1* should be: Teleosemantic norms cannot be typical-norms. So my critique does not work. Unless we have more reasons to defend point 1.

To answer this counterargument, there is a second way to defend point 1 besides any specific literature on teleosemantics. I believe that there are good reasons to defend that natural norms for teleosemantics cannot be statistical (i.e. to defend 1). But first, let's consider the following point. In some sense, the presented counterargument is sound. Trait fitness is not just a matter of high average. Yet, etiological theories are based on the bias that Natural Selection introduces that results in a trait type being selected, preserved, and spread. Although trait fitness is not just high-average, trait fitness is related to the increment of the frequency of a trait in a population. The notion of selection for, both under the causalist and the statisticalist readings, concerns the consequences for a population when its individuals possess the selected trait. Such consequences do concern the increment on average in the population. That a trait has been selected is related to what contributions such a trait had provided that make it more frequent and reliable in a population; similarly, if a trait is "selected against" (Sober 1984), it means that natural selection biased it towards a low-frequency in the population. In this sense, trait fitness is related to an increment in average. Besides this preliminary point, there are further and more relevant reasons to discard the alleged refutation.

The central reason lies in the very idea of appealing to selected-effect functions —*qua* populational forces— to solve the Causal Mismatch. The etiological solution to the problem of misrepresentation needs to consider selected-effect functions as causal dispositions. The central idea of this solution lies in understanding error as a mismatch between the evolved trait type and the individual trait token. The normativity of content is not attributed at the ontogenetic level but at the phylogenetic one. Crucially, the processes of normative attribution must be causal. The explanatory target is to understand how representation and reference can be causally linked even if the possibility of error is taken into account. This is what makes teleosemantics a naturalist project. To account for the Causal Mismatch, one must explain how the normative content of a representation is attributed in a way that the reference of such representation is understood in causal terms. Etiology, under a causal reading, can provide such kind of causal explanations, insofar as what specifies the normativity of content is the causal role that a trait type has had that made it part of the population. But, if the normativity of content is explained statistically, the connection between a representation and its reference would lack the causal grounds needed for the naturalization of intentionality.

Here we can appreciate the connection with challenge 1. Teleosemantics' core works for the naturalization of intentionality insofar as it can account for intentional causation from a legitimate scientific viewpoint. The strategy to appeal to etiological functions makes it possible to root the normativity of content in solid causal grounds. However, insofar as such causal grounds are not the alleged ones by etiologists (challenge 1), the causal roots of normativity are cut. Hence, we have a crucial reason to defend point 1 (that teleosemantic norms cannot be statistical). The normativity of content that specifies the extension of any representation must be assessed in causal terms in order to provide a naturalist view of intentionality. If my considerations are correct, point 1 stands, and consequently, my critique concludes that, under

a statisticalist view, etiological norms, and therefore selected-effect functions too, are not suitable for a naturalistic solution to Brentano's Problem.

To conclude, let's appreciate a defense of point 1 —besides the issue of typical-norms—in the teleosemantic literature, particularly in Millikan's emphasis on "Normal explanations". Her emphasis on Normal (capitalized) traits involves not merely to separate them from typical-norms. It also stresses the kinds of natural norms that teleosemantics needs and what kind of explanation specifies the normativity of content. Normal explanations (of a representation) concern the causal role of a trait that had assigned (past tense) a normative content to the representation. Note that a Normal Explanation need not be identified with any specific theory of teleological function. If we look at it with etiological (Millikan's) lenses, such causal role is attributed to *trait types* and concerns the historical conditions that made a trait type being selected. Accordingly, the historical process of natural selection is what attributes normative content to representational systems. However, here we find again the aforementioned problem: under a statisticalist view, etiological functions do not provide Normal explanations; causal roles cannot be attributed to trait types. As a consequence, Normal explanations, if causal, are not etiological explanations of trait function.

5. Agential Teleosemantics: a new solution to Kant's Puzzle, a new solution to Brentano's Problem

5.1 Agential Teleology

My proposal till now states that if we adopt a statisticalist view of natural selection, etiological teleosemantics has a foundational problem in its solution to Brentano's Problem. In this last section, I intend two things. First, I will argue that teleosemantics' core is still a valid and interesting path towards the naturalization of intentionality. However, to the extent that the condition of etiological teleosemantics highlighted here is present in its teleological framework, it is necessary to propose an alternative view of natural teleology in order to reframe teleosemantics on proper causal grounds. In other words, we need to propose an alternative solution to Kant's Puzzle to anchor a new answer to Brentano's Problem. The ideas presented here are just a sketch that requires further elaboration, and thus I do not expect to solve everything but just to propose a different strategy for teleosemantics in the context of the causalism vs. statisticalism debate on the causal basis of natural selection.

Secondly, I will provide some reasons why the statisticalist view is correct. Therefore, while till now I just defended the *weak implication* (i.e. an analysis of the implications for etiological teleosemantics *if* we adopt a statisticalist viewpoint), to point out the main reasons why statisticliasm is true entails a defense of the *strong implication.* As expected, both points are connected: a defense of statisticalism consists in pointing out an alternative locus of evolutionary causes and, consequently, to link such causes with the causal explanation of aptness –i.e. the grounds of natural teleology. In other words, the defense of an individual level account of natural teleology operates as an argument in favor of the statisticalist view, insofar as it means a defense of the causal grounds of the explanation of aptness at the individual level, as statisticalists support. So, now it is necessary to specify what is the locus of

evolutionary causes and how it connects with natural teleology and, consequently, with the causes of aptness.

"The only genuine forces going on in evolution are those taking place at the level of individuals" (Walsh et.al 2002: 453), so the Statisticalist School argues. Individual causes of evolutionary processes concern those that specify the fitness of an individual —its persistence and reproduction; that is, the life, death, and reproduction of an organism. This includes what Darwin referred to as the *struggle for life*: organisms pursuing successful life conditions in such a way that their fitness increases. As it can be appreciated, struggling is an action and an activity that an individual organism performs. Although struggling has a connotation of competitiveness, we can reinterpret it in contemporary terms by claiming that aptness is caused by the *adaptivity* of organisms as *agents* (Sultan et.al. 2022; Walsh 2015).

The core idea is that the organism itself, not its genes, defines and regulates its ontogenetic trajectory. Such trajectory is adaptive insofar as the organism is constantly adjusting it according to both its inner and outer circumstances. Agentivity hinges on the capacity of an organism to self-regulate its life conditions in adaptive ways in order to endure and reproduce. This view posits organismal activity not as the execution of a genetic program but a as goal-oriented process carried out by the organism itself towards an adaptive phenotypic state. The goal-directedness of organismal activities stresses their adaptive dimension. That's why Walsh (2007, p. 195) states that "Evolution is adaptive because ontogeny is adaptive". Therefore, "[t]hose exquisite adaptations of one part of the organization to another part, and to the conditions of life" (Darwin 1859 [1968]) are not caused by genetic programs nor by evolutionary design, but by what agents do during development to stay adapted.

As it can be appreciated, the causes of evolution are proximate causes. Teleology, therefore, lies in the activity of organisms during their lifespan that determines their individual fitness. Importantly, while Mayr posits that functional biology is devoted to analyzing proximate causes, from a contemporary perspective we can locate proximate causes of aptness in two broad frameworks devoted to the study of individuals: organismic biology and developmental biology. The term 'developmental biology', which is concerned with the temporal dimension of individual development, today embraces many frameworks that have challenged certain core tenets of MS, such as Evo-Devo, Eco-Devo, Developmental Systems Theory, Ontogenetic theories of Inheritance and Developmental Psychobiology. Although there is not a unified view, in all cases the task is to transcend the gene-centered view of development and evolution and provide a theory of development where the organism is the proper unit of development. Organismal biology has a philosophical connotation, but it refers to the physiological level: how parts interact in order to produce a certain outcome. Note that this is usually understood in mechanistic terms, thus it seems that there is no place for teleology. The emphasis on organismal biology is because organismal biology does posit organisms as teleological agents while still relying on the physiology and functioning thereof. The connection between these areas requires further exploration. If the revival of teleology at the individual level involves two different temporal scales —the organismic and the developmental one— we need to unify them in order to propose a coherent picture of natural teleology. Let's briefly point out some core processes of both frameworks.

In recent decades a number of mechanisms have been put forward with the aim of constructing a view of ontogeny as a context-sensitive, multi-causal, and complex process. This involves different areas of research, such as niche construction theory (Odling-Smee et al. 2003; Stotz 2017), developmental studies on phenotypic plasticity (Bateson and Gluckman 2011; West-Eberhard 2003), the study of norms of reaction (Schlichting and Pigliucci 1998; Sultan 2015), ecological developmental biology (Gilbert and Epel 2015; Lewontin 2001), epigenetic systems of inheritance (Jablonka and Lamb 2005), developmental approaches to homology (Wagner 2014), molecular epigenetics (Griffiths and Stotz 2013; Keller 2002; Moss 2003), psychobiological development (Gottlieb 1997; Keller 2010; Michel and Moore 1995), and embryology (Amundson 2005; Robert 2006). In all cases, we have organisms understood as subjects self-regulating their development in an adaptive way according to their living conditions, not as the implementation of an evolved developmental program. Within organismic biology, we also find many interesting phenomena that have been investigated during the last decades, such as the self-organizational capacities of organisms (Camazine et al. 2003; Goodwin 2001; Kauffman 1993; Mitchell 2009; Müller and Newman 2003), global dynamics studies by systems theorists (Bertalanffy 1969; Boogerd 2007; Noble 2008), the autopoietic (Di Paolo 2005; Maturana and Varela 1980) and cybernetic (Ashby 1991) properties of organisms, the autonomy of organic systems (Barandiaran et al. 2009; Moreno and Mossio 2015), among others. Here, we also find organisms doing things to stay adaptive, in such a way that their physiology cannot be understood without taking into account the directedness of these processes towards an adaptive way of being alive. This has led many scholars, both within the organismic and the developmental frameworks, to defend the agentive view of the organism and its teleological underpinnings

Precisely, these recent emphases on individual causation give us reasons to support the statisticalist view and, consequently, argue for the *strong implication*. As highlighted in the introduction, I connected the position adopted concerning the role of organisms in evolution with different sides of the statisticalism vs. causalism debate. If organisms are irrelevant in explaining evolution –as Sober argued- it seems that causes cannot lie at the individual level; but if organismal agency is the locus of the solution to Kant's Puzzle, individual causation provides the proper causes of evolution, as statisticalists claim.[6] The rise of developmental and organismal biology has come to challenge the evolutionary view pictured by MS. Recent views in biology, and their theoretical and experimental backup, promote reasons for defending the *strong implication:* statisticalism is true and, therefore, teleosemantics must be challenged and reframed.

From what I have been presenting, I conclude that the causes of evolution are explained by the teleological dimension of organisms as agents. Let's call this Agential Teleology. In the vein of the picture developed here, Agential Teleology explains the aptness of living beings by stressing

[6] Certainly, one can take a mid-term position and argue for both individual and populational causes of evolution. As Walsh noted (2003, 2019), this view defends a two-force model (cf. section 3): evolutionary forces come from two different sources. However, as it was remarked above, this view is not endorsed by statisticalists insofar as it blurs the nature of populational explanations based on trait fitness. There are not two forces but two different levels of explanation (cf. Walsh (2019) for details).

the adaptivity of organic agents. Adaptivity not adaptation, agents not populations, provide the causal bases of natural selection.

5.2 Towards Agential Teleosemantics

Agential Teleosemantics is rooted in Agential Teleology. This means that the proper function of any representational system is established during individual ontogeny. While etiologists claim that a representational system must represent whatever it was selected for during natural evolution, in Agential Teleosemantics the possibility of error and the explanation of Causal Mismatch hinge on what a system must represent according to those proper functions developed during ontogeny and performing a particular causal role in any organismal activity. Insofar as the developmental and organismal dimensions of organisms have a teleological flavor, the teleosemantics' core is preserved from etiological teleosemantics. Agential teleology posits teleological functions, hence a normative valuation on traits according to their individual history in organismal development. This, therefore, accounts for the problem of misrepresentation. As Agential Teleology does not involve prior intentionality, it seems that this new solution to Kant's Puzzle provides an alternative naturalistic position for dealing with Brentano's Problem. Figure 2 locates this proposal in biology by stressing how different biological frameworks provide alternative teleosemantic projects.

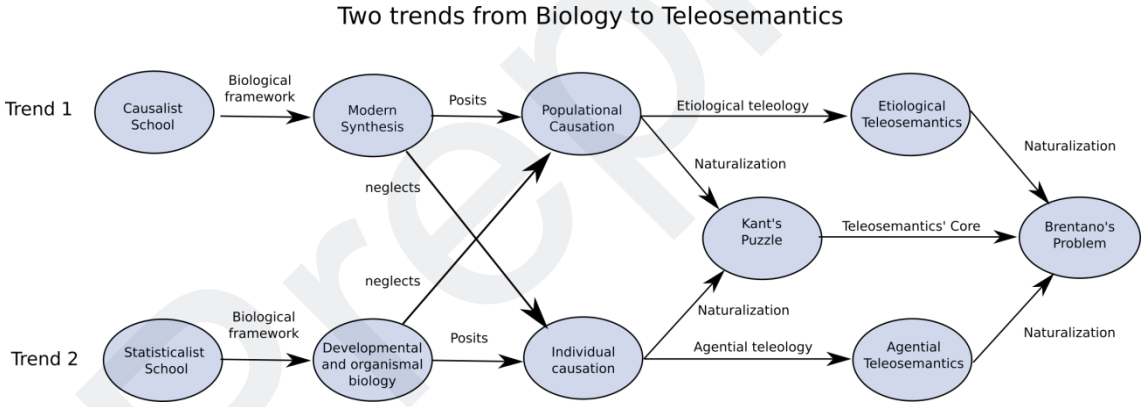

Figure 2: Two trends from Biology to Teleosemantics. Trend 1 is defended by the Causalist School based on the MS framework. The MS posits populational causes to provide a naturalist solution to Kant's Puzzle while neglecting individual causation as an evolutionary force. Populational causation is the central foundational element of etiological teleosemantics and therefore the grounds for the naturalist solution to Brentano's Problem. Trend 2 is defended by the Statistical School based on the biological framework recently pursued by developmental and organismal biology. Such a framework posits individual causes to provide a naturalist solution to Kant's Puzzle while neglecting populational causation. Individual causation is the central foundational element of agential teleosemantics and therefore the grounds for the naturalist solution to Brentano's Problem

Certainly, I didn't say anything specific about the core ingredients of teleosemantics: its *teleo* side, and its *semantic* side. I did not propose a theory of functions based on the adaptive agency of organisms (but see the organizational view of function presented by Mossio et al.

(2009), or the view of function defended by Walsh (2014) in relation to plasticity). Nor I said anything about the nature of representational systems in relation to content determination —for instance, whether Agential Teleosemantics fits with an input-based or output-based account. This is for further work. By now, I limit myself to sketch a different proposal motivated by the discussion about the nature of natural selection.

The view outlined here has many points in contact with different proposals within teleosemantics, particularly with Dretske's account (Dretske 1981, 1988). Even though he did not put up his view with the focus on the agential capacities of organisms recently defended in theoretical biology, nor he directly took part in the discussion around evolutionary causation, he did defend that the processes responsible for attributing functions to representations (more specifically, to type 3 systems of representation; Dretske 1998: ch. 3; 2004) reside in individual development. Moreover, other contemporary proposals, while not anchored to current debates on evolutionary causation, pursue a similar strategy as Agential Teleosemantics, such as Bickhard's Interactivism (Bickhard 2000, 2009), de Prado Salas (2018) on reproduction, or Schroeder's (2004) emphasis on cybernetic properties of cognition. It is expected, then, that *ontogenetic selected functions* do have many points of contact with Agential Teleosemantics. This includes Shea's recent emphasis on the role of persistence in function determination (Shea 2018), Millikan's insight on operational condition and external inheritance systems (Millikan 1984), or Garson and Papineau's proposal on novel content (Garson and Papineau 2019). These remarks help me to constrain the scope of my critics. My proposal here is foundational and it touches on some parts of the Teleosemantic Project, which, although central, does not purport a rejection of teleosemantics at all. On the contrary, my proposal is totally aligned with teleosemantics' aims. Having said so, I do believe that many proposals on ontogenetic function fit with agential teleosemantics. Moreover, I did not discuss the specific proposals concerning the content determination and the variant of teleosemantics accounts (such as informational teleosemantics, consumer-based accounts), and their possible connections with agential teleosemantics (but see the last paragraph).

Let's conclude with some programmatic open questions. Most of the proposals of teleosemantics are devoted to explain the cognitive capacities of animals —bee dance, frogs catching flies, human beliefs, and so on. This is not the case, for instance, with Nicholas Shea's account. Although his 2018 book is devoted to cognitive science, his previous work on teleological functions was not restricted to the cognitive level but pursued a teleosemantic account applied to all living beings. I am not going to discuss his proposal here (cf. Griffiths 2013, Rama 2022, Rama in preparation). I just want to stress that Agential Teleosemantics need not concern animal cognition exclusively. In this sense, it represents a philosophical project both for cognitive science and for theoretical biology. This means that the two core ingredients of Agential Teleosemantics are latent in all individuals. The first one, concerning the teleological side of teleosemantics, is quite simple to defend, together with etiological theories of functions, insofar as it comes from biology itself. Thus, if Agential Teleosemantics is based on the teleological dimension of agents, and all living beings are agents, therefore the teleological notion of function defended here is present beyond animal cognition.

The second ingredient, concerning semantics, promotes an open question for further analysis. Should we extend semantics beyond animal cognition? This is a difficult issue insofar as semantics is usually employed as an umbrella term including different phenomena. Shea accepts it and suggests an informational teleosemantics beyond animal cognition. I propose that the possibility to extend semantics beyond animal cognition concerns the use of informational talk in biology, particularly in developmental and organismic biology, and to the extent that it is in these areas where agential teleology is based on. If we set aside the etiological functions defended by Shea, and endorse a view on function based on agential teleology, there are good reasons to extend Agential Teleosemantics beyond animal cognition. In a nutshell, informational processes are central to the teleological character of agents. As remarked, agential teleology concerns the adaptivity of each organism to its life conditions. This is the common ground that connects different phenomena within developmental and organismic biology. In all these cases, organisms adaptively confront their living conditions in order to persist and reproduce. Crucially, *if such agential capacity is adaptively directed* —that is, directed towards an adaptive way of being a living system, then organismal responses, through regulation, niche construction, self-organization, plasticity, and so on, *must be sensitive to its life conditions* (Rama 2021). Here information enters the scene. Information is central to explain how an agent's activity —from cell development and the physiology of organs to motility and behavior— is sensitive to both its inner and outer conditions in such a way that the performed activity is adaptive to such conditions. In sum, adaptive agents are informed; sensitiveness is a prerequisite for agential teleology. In this sense, a research question within Agential Teleosemantics concerns the possible unification of informational talk between developmental and organismic biology, for instance, as it is suggested by developmental system theorists (Oyama 1985, Griffiths 2016, 2017, Griffiths et.al 2015, Stotz 2006, 2019, Calcott et.al 2020, Griffiths and Stotz 2013), and cognitive science, such as the proposals of Shea (2018), Neander (2017a), Dretske (1981, 1988), or even Fodor (1998).

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