Proper Functions as Etiological Functions of Predominant Traits

Tianqin Ren, University of Missouri

APA Central Division Meeting 2025

Abstract: This paper proposes a revised definition of etiological proper function, arguing that proper functions should be attributed only to predominant traits — traits that have a close-to-one frequency in the population. This restriction preserves the distinction between normal variations and dysfunctions, while better supporting the desired explanatory power and binary attribution of proper functions.

Etiological functions (also called the selected effect functions) are, roughly, the way a trait type has contributed to its bearers' fitness in evolutionary history. Furthermore, they are often thought to be a trait's "proper function": a standard for normal instantiations of the trait type. However, equating proper function with etiological function leads to several problems. Matthewson (2020) points out a tension between a trait's historical contribution to fitness and its proper function: the former comes in degrees, whereas the latter is binary. Christie et al. (forthcoming) argue that the normative implication of etiological function is based on its ability to explain the existence of its bearer and point out that etiological functions, as currently defined, cannot explain the existence of traits in many cases. These criticisms suggest the need for a different, more restricted account of etiological proper functions.

This paper proposes such an account: Not all etiological functions are proper functions; a trait has a proper function only when it is predominant (i.e. has a close-to-one frequency) in the population, and its proper function is its etiological function. This restriction is consistent with the general understanding of proper functions and can support the explanatory and teleological implications they are expected to have. The first section examines how etiological functions are connected to proper functions and explains why this connection should be more restricted than traditionally thought. The second section tests the restricted account of proper function with challenges posed to the traditional account and argues that it is more immune to the challenges. Finally, I will make a brief note on the often-proposed normative implication of proper functions.

1. Distinguishing Etiological Function and Proper Function

1.1. Explanation, teleology, and normativity

The general idea of etiological functions is that they are the way a trait type has contributed to the organisms' fitness in the evolutionary history. For example, pumping blood is an etiological function of the heart because this is how the heart has contributed to the heart owner's fitness in the past. On the other hand, the general idea of proper functions is that they are what a certain trait normally does. Pumping blood is also a proper function of the heart because any normal heart should be able to pump blood. Proper functions are expected to distinguish function from accidental causal contribution (Walsh and Ariew, 1996) and normal variations from dysfunctions (Matthewson and Griffiths, 2017). They are also used as the standard of "normal" by some accounts in philosophy of mind, epistemology, and philosophy of medicine.

Traditionally, the etiological account of proper function defines proper functions just as etiological functions (Millikan 1989, Neander 1991, Griffiths 1993, Godfrey-Smith 1994). However, some intermediate moves are needed to bridge the two general ideas. Etiological functions are often thought to explain why a trait exists in the current population because they refer to the traits' historical contribution to fitness. For example, the heart's ability to pump blood explains why mammals have hearts: this ability is the reason why the frequency of having a heart has increased in the mammal population. Such explanatory power leads to the idea that etiological functions are teleological: the goal and purpose of a trait is to perform its etiological function, because this is what the trait was selected for. Saying that the etiological function of a heart is to pump blood means that the goal and purpose of a heart is to pump blood. This is because a heart is selected for performing this function. The idea of goal-directedness further seems to support a normative claim: because the etiological function is what the trait is *for*, it is also what it *should* do. We finally reached the other end of the bridge: the normatively loaded idea of proper function.

Three moves are made from etiological function to proper function: from explanation to teleology and finally to normativity. Each of those moves faces challenges. First, etiological functions cannot always explain why a trait exists or has a certain frequency in the population

(Christie et al., 2022). Second, there is a mismatch between the degrees of the etiological function's causal contribution and the binary teleological attribution to proper function. (Matthewson, 2020). Finally, whether normativity can be derived from teleology is also questionable. My view is that doing what something is supposed to do does not necessarily mean it is doing anything good, but I will save this final challenge for future discussion. In what follows, I will argue that etiological functions are not always proper functions – they are proper functions only when the trait predominates the population. I will also show that this restricted account of etiological proper function at least alleviates the first two problems listed above.

1.2. Clarifying the definitions

In this section, I will examine the traditional definition of etiological proper functions and point out some vagueness in the concepts involved. Clarifying the concepts will lead to a dilemma: when understood narrowly, etiological functions can no longer be attributed to some traits as biologists and physiologists usually do; but when understood broadly, etiological functions will not be able to explain the purpose of the trait. This dilemma calls for distinguishing proper functions from mere etiological functions.

Proponents of the etiological account of proper functions usually define proper functions as etiological functions. Take Neander's definition as an example:

> It is a/the proper function of an item (X) of an organism (O) to do that which items of X's type did to contribute to the inclusive fitness of O's ancestors, and which caused the genotype, of which X is the phenotypic expression, to be selected by natural selection (Neander 1991, 74).

This definition only directly refers to a historical evolutionary function, namely, the trait's past causal contribution to its being selected by natural selection. However, as discussed in the previous section, more conceptual assumptions are needed to bridge it to the idea of proper function, which is expected to be explanatory, teleological, and normative. Therefore, I will rephrase this definition as defining only the etiological function, but not the proper function:

> The etiological function of a trait token (X) of an organism (O) is to do what previous tokens of X's type did that contributed to the inclusive fitness of O's ancestors, which caused the genotype of X to be selected by natural selection.

Two crucial concepts in this definition require further specification. The first is "selected by natural selection." A trait being selected by natural selection can have different consequences:

(i) **Persistence:** the trait has had a non-zero frequency for a long period of time. For example, brown eyes and blue eyes are both persistent traits in the human population.

(ii) **Spread:** the frequency of the trait has increased in the past and remains stable in the current population. Take the peppered moth story as an example: in the forest where most trees are dark-colored, the frequency of dark-colored morph increased in the peppered moth population and remained stable as the majority. This process can be called the spread of the dark-colored trait.

(iii) **Predominance:** the trait has a frequency close to 1 in the current population. The opposable thumb has a frequency close to 1 in chimpanzees, gorillas, and human beings. This is an example of a predominant trait.

It seems intuitive that etiological functions should be attributed to a trait as long as its historical contribution to fitness has caused any of the above consequences. The second concept to be specified is "cause." Again, the most plausible interpretation of causes here seems to be a broad one: as long as the trait has made some causal contribution to the consequence of being selected, then the way it makes such contribution is the trait's etiological function.

The concept of "caused the genotype to be selected by natural selection" must be understood in a broad sense to accommodate the etiological functions of traits like heterozygous sickle cell. It is commonly accepted that the etiological function of sickle cell trait (heterozygous with only one sickle cell allele) is to give their carrier resistance to malaria. However, this trait only persists at a relatively low frequency in the population because homozygous sickle cell gene leads to severe anemia. The etiological function of malaria resistance is also not the sole cause of the trait's specific frequency in the current population. For a complete causal story, the negative influence

of homozygous sickle cells and the mechanism of allele inheritance should also be included. If the concepts of "cause" and "being selected" are restricted to narrower senses, traits like heterozygous sickle cell will not be attributed with etiological functions, which seems implausible and inconsistent with biologists and pathological practices.

Nevertheless, the inclusiveness of the definition is in trade with the etiological function's explanatory power and teleological implications. Since etiological functions only need to be one of the causes of the evolution of the traits, it will not be able to fully explain the trait frequency in many cases. Plus, if etiological functions can be attributed to traits with broad degrees of selective consequences, there will be a mismatch with the categorical verdict about the purpose(s) of the trait. A contribution that a trait made to fitness either *is* or *is not* the purpose of the trait, and there is no middle ground or degrees of proper functions. But for etiological functions, there can be a spectrum: the contribution can lead to consequences from persistence at a low frequency to spreading to a predominant frequency. This suggests a dilemma for any account taking etiological functions as equivalent to proper functions: restricting the definition leads to an implausible denial of function attribution, while broadening the definition threatens the explanatory and teleological claims proper functionalists want to make.

The solution I propose is to distinguish between etiological function and proper function. Etiological functions, as a concept directly derived from evolutionary history, do not always have the explanatory power and teleological implication of proper functions. Proper functions, on the other hand, need not be attributed to every trait that has been selected by natural selection. The dilemma is resolved when the two concepts are separated, and their connection is restricted. In what follows, I will propose a promising way to make such restriction: to understand proper functions as etiological functions of predominant traits.

1.3. Proper functions and the predominant traits

Consider the classic examples of proper functions: the proper function of mammals' hearts is to pump blood, the proper function of human eyes is trichromatic vision, etc. They are not attributing proper functions to traits that merely persist in the population, but to traits that predominate the population. In other words, the most plausible cases for proper functions are

about traits that (almost) every individual in the population has. This aligns well with an important motivation for the concept of proper function: distinguishing between normal variations and dysfunctions. Proper function is a function that the lack of it will be considered dysfunction. The heart has the proper function of pumping blood because (except for exceptional cases) every mammal has a heart, and pumping blood is the reason why the heart is present in almost every mammal. A mammal without a heart, or with a heart unable to pump blood, would be considered dysfunctional. By contrast, the sickle cell trait does not have a proper function, since not every human has it. Even if a person does not have heterozygous sickle cells, or their heterozygous sickle cells fail to confer malaria resistance, it seems counterintuitive to say that this person or their red blood cells are dysfunctional. While malaria resistance is the etiological function of the sickle cell trait—it partially explains why the trait was selected—it is not a proper function.

So far, restricting proper functions to only traits with a close to 1 frequency in the population seems promising. In the following sections, I will argue that this restriction can mitigate, if not entirely avoid, the explanatory and teleological issues associated with the concept of proper function.

2. Revisiting the Challenges with the Restricted Account

2.1. Explaining evolutionary consequences

Christie et al. (2022) point out that proper functions, if taken as equivalent to etiological functions, cannot always explain why a trait exists in a population. There are cases where a trait type of an organism has contributed to its ancestor's fitness, but the non-zero frequency of the trait cannot be explained by such historical contribution. In this section, I aim to show that these problematic cases do not threaten the restricted account of proper functions. When proper functions are only attributed to predominant traits, they can offer a stand-alone explanation for the evolutionary outcomes of such traits.

The first kind of situation where etiological functions cannot fully explain the evolution of the trait is when two species coevolve. In such a scenario, there could be fluctuating selection, which

means a trait can contribute to fitness during one time period but decrease fitness during another and go back and forth. Referring to the trait's etiological function, namely the way it has contributed to fitness in history, cannot fully explain why the trait has a certain frequency in the population (10-11). For example, the etiological function of zebra stripes is to deter biting flies (Caro et al., 2014). Although the actual evolutionary process of zebra stripes did not involve coevolution, hypothetically, if flies and zebras coevolved, the stripes' etiological function would fail to explain the proportion of striped individuals in the zebra population. The coevolution story roughly goes like this: the biting behavior of flies is also under selective pressure. When there are more non-striped animals, the non-stripe-biting flies have a selective advantage because they have more food, leading to the result that there are more non-stripe-biting flies. Meanwhile, having stripes gives an animal a selective advantage because they are prey to a lesser number of flies, thus less likely to be bitten by a fly. But as the frequency of stripes increases in the animal population, the selective advantage of stripe-biting flies will also increase, raising the frequency of stripe-biting flies and, in turn, decreasing the fitness of striped animals. In this case, only referring to the trait's etiological function, namely the way it has contributed to fitness, cannot explain why the trait has a certain frequency in the population (10-11).

However, the etiological function's hypothetical failure to explain trait frequency lies squarely in the assumption that the striped trait is not predominant in the population. If zebras and flies coevolve, excluding the effect of drift, the striped trait of zebra would never reach fixation. And if it is not the case that almost all zebras have stripes, stripes would not have any proper functions. It still has an etiological function of deterring flies, but we cannot say that zebra without stripes that can deter flies are dysfunctional. The coevolution counterexample is just inapplicable to proper functions. On the other hand, in the actual story where stripes are predominant in the zebra population, its function of deterring flies does provide a stand-alone explanation for the close-to-one frequency in the population: by deterring flies, the stripe increased the zebra ancestors' fitness to the extent that it reached fixation. In other words, there was a selective sweep: at some point, almost all individuals in the population without stripes died out, and almost all individuals in the population had stripes. This is why it is "normal" for a zebra to have stripes and "normal" for the stripes to deter flies. Deterring flies is not only an etiological function of zebra stripes, but also their proper function: holding external factors fixed,

if the stripes of a zebra fail to deter flies (maybe because the contrast of color is not sharp enough), then there is something wrong with the zebra. In this case, because the striped trait's etiological function caused its predominance in the population, this etiological function also becomes the proper function. It fully explains why it has the predominant frequency in the population, which is also the explanation biologists adopt: "Zebras have stripes because stripes deter biting flies." (10)

The second kind of situation where an etiological function cannot fully explain trait frequency is when there is a negative frequency-dependent selection for two competing traits. These situations can often be characterized as strategic games, and the explanation for one trait's non-zero frequency must refer to the strategic game and its payoffs, which cannot simply be characterized by the etiological function of the trait (11-12). For instance, the Hawk-Dove game is often used to describe the dynamic of different strategies toward conflicts. The Hawkish trait is the tendency to compete aggressively for resources, whereas the Dovish trait is the tendency to retreat when facing conflicts and not compete for resources. When both traits are present in the population, the payoffs are roughly like this: if Hawk meets Dove, Hawk outperforms Dove by taking all the resources at no cost; Dove meeting Dove generates a moderate payoff since they just peacefully divide the resources; Hawk meeting Hawk generates the worst outcome, because they have to divide the resource, but there is also a cost for the aggressive competition. When the frequency of Hawk increases, the chance for a Hawk to encounter another Hawk increases accordingly, and the outcome of being a Hawk gets worse. But when the frequency of Dove increases, the chance for a Hawk to meet a Dove increases, improving the outcome of being a Hawk. This dynamic results in an equilibrium: both Hawk and Dove persist in the population. Such persistence of traits, as Christie et al. point out, cannot be explained by the etiological functions of the traits. To explain why the frequency of Hawk is constantly near the equilibrium point, one needs to cite the payoffs and mechanism of the strategic game, rather than just saying that Hawk increased fitness because it has an etiological function. Sometimes the traits don't even have an etiological function, if the whole actual process is a monotone decreasing section of the possible wave curve.

The reason an etiological function cannot fully explain this phenomenon is, again, because neither trait predominates in the population. The Hawk-Dove dynamic results in an equilibrium, where both traits persist but neither reaches fixation. Consequently, neither trait has a proper function; they are simply normal variations coexisting within the population. Negative frequency-dependent selection is a counterexample of the explanatory power of etiological functions. Nonetheless, this situation does not pose a challenge to the explanatory power of proper functions.

To sum up, when proper functions are limited to the etiological functions of predominant traits, they are more capable of explaining the frequency of these traits. The underlying reason seems to be that when a trait has reached fixation, the causal story will be relatively simple, and the etiological function of the trait is often the main cause of fixation. Of course, how exactly the etiological function caused fixation would contain more details, including the match between the environment and the comparison among competitors in the same population, but those details are all packed in the concept of etiological functions.

2.2. The sufficient degree of selection

Matthewson (2020) presents another challenge to the etiological account of proper function: while natural selection comes in degrees, the attribution of proper functions—along with their teleological and normative implications—is typically binary. This mismatch needs explanation if proper functions are grounded in natural selection. One solution is to set a threshold for the degree of selection that aligns with the binary attribution of proper functions. The other option is to adopt a degreed understanding of proper function. Each approach has its costs. A degreed concept of proper function seems to undermine its teleological and normative implications. The former approach, although being able to keep the downstream philosophical accounts of proper function, faces the difficulty in finding an objective, non-arbitrary "sufficient degree of selection" threshold (5).

Being predominant in the current population seems to be a good candidate for such threshold of sufficient degree of selection. I will review the three ways Matthewson discusses in which

natural selection comes in degrees and argue that if proper functions are etiological functions of predominant traits, they still have teleological implications in those cases.

The first example is when traits are under different forces of selection. There was a selection for nasal hair because it helps keep out particulates. There was also a selection for the CFTR channel in the cell because it transports chloride ions across cell membranes. They are etiological functions of the two traits, respectively. Matthewson argues that in humans' evolutionary history, selection on CFTR cell gates was much stronger than selection on nose hairs (39). But it is unclear what "strength of selection" is supposed to mean here. Both traits have reached fixation in the past and are predominant in the current population. Matthewson suggests that lacking nasal hair does not significantly reduce fitness, which might seem intuitive initially. However, this claim becomes implausible when considering the fact that nasal hair has reached fixation—every individual without it has been selected out. If selection on nasal hair was indeed weak, its fixation must have resulted from either genetic drift or genetic hitchhiking. In either case, keeping out particulates would not be a proper function of nasal hair because it is not the historical contribution to fitness that caused the trait to reach fixation. Matthewson also discussed varied selective strengths on different properties in the same structure. For example, the shape of the human sclera improves visual acuity, while the pale color of it helps cooperative interactions by indicating the direction of one's attention. The former is said to be under stronger selection than the latter (5-6). However, although they are about the same structure, the sclera's shape and color are two different traits, so the previous response also applies here: since both are predominant in the current population, they are either under the same strength of selection, or one of them does not have a proper function.

The second example is when selective forces toward certain traits change over time. In the example of natural selection on peppered moth pigmentation, when most trees are pale-colored, lighter moths have a selective advantage; when most trees are dark-colored, darker moths have a selective advantage (6). But this is another example where neither of the traits predominates the population, thus neither of them has a proper function.

The third example is that populations are suitable for natural selection to different degrees. Some populations are more apt to undergo natural selection or engage in richer kinds of natural

selection than others (7). But no matter what degree of selection is involved in a certain population, a trait is either fixed because of its etiological function or not. If a population is more apt for more kinds of natural selection, then its members may have more traits with proper functions; and vice versa. But for each trait, it is a simple yes/no question, and the implications about proper functions are about single traits, not populations.

The ultimate point is that while selective forces can vary in different ways and are often difficult to quantify, the outcome of selection—whether a trait reaches fixation or not—provides a good measure to the degree of selection. There is a natural distinction between traits that predominate and traits that merely persist in the current population, which can support the binary verdict of proper functions.

3. Conclusion and Further Thoughts

I have proposed a restricted account of etiological proper functions: proper functions are etiological functions of predominant traits. An etiological function, by definition, is the trait's historical contribution to inclusive fitness, which has caused natural selection for the trait. A proper function is a special case of etiological function when the evolutionary consequence of such contribution is the predominance of the trait. If a trait does not have a frequency of nearly 1, it does not have a proper function, even if it has an etiological function. This restricted account aligns with the consensus understanding of proper function. It also seems more suitable to make the desired distinction between normal variations and dysfunctions.

Understood as the etiological functions of predominant traits, proper functions are better suited to uphold the explanatory and teleological implications traditionally attributed to them. For a predominant trait, its etiological function, which is also its proper function, can fully explain its close to 1 frequency because such function is the major cause of its fixation. The distinction between predominate and non-predominate traits also seems to be a reasonable threshold for the sufficient degree of selection to generate proper functions, resolving the tension between the degreed nature of natural selection and the binary nature of proper functions along with their further implications.

What is left, then, is the normative implication of proper functions. I believe this is where the connection breaks: even for a predominant trait, there is no necessary connection between its etiological function and goodness in any sense. But leaving that to future discussion, it seems safe to conclude that the restricted account better preserves the explanatory and teleological elements of the etiological understanding of proper function.

References

Caro, Tim., Amanda Izzo, Robert C. Reiner Jr, Hannah Walker, and Theodore Stankowich. 2014. "The Function of Zebra Stripes." *Nature Communications* 5: 3535.

Christie, J. R., Brusse, C., Bourrat, P., Takacs, P., & Griffiths, P. E. (2022). "Are Biological Traits Explained by Their 'Selected Effect' Functions?" *Australasian Philosophical Review*, *6*(4), 335–359.

Godfrey-Smith, Peter. 1994. "A Modern History Theory of Functions." *Noûs* 28 (3): 344–62.

Griffiths, Paul E. 1993. "Functional Analysis and Proper Functions." *British Journal for Philosophy of Science* 44 (3): 409–22.

Matthewson, John, and Griffiths, Paul E. 2017. "Biological Criteria of Disease: Four Ways of Going Wrong." *Journal of Medicine and Philosophy* 1(4): 447-466.

Matthewson, John. 2020. "Does proper function come in degrees?" *Biology and Philosophy* 35 (4):1-18.

Millikan, Ruth G. 1989. "In Defense of Proper Functions." *Philosophy of Science* 56 (2): 288–302.

Neander, Karen. 1991. "Functions as Selected Effects: The Conceptual Analyst's Defense." *Philosophy of Science* 58 (2): 168–84.

Walsh, Denis M., and André Ariew. 1996. "A Taxonomy of Functions." *Canadian Journal of Philosophy* 26 (4): 493–514.