Garson, J. (forthcoming). There are no ahistorical theories of function. *Philosophy of Science*

**Title:** There Are No Ahistorical Theories of Function

**Author:** Justin Garson

**Abstract:** Theories of function are conventionally divided up into historical and ahistorical ones. Proponents of ahistorical theories often cite the *ahistoricity* of their accounts as a major virtue. Here, I argue that none of the mainstream “ahistorical” accounts are actually ahistorical. All of them embed, implicitly or explicitly, an appeal to history. In Boorse’s goal-contribution account, history is latent in the idea of statistical-typicality. In the propensity theory, history is implicit in the idea of a species’ natural habitat. In the causal role theory, history is required for making sense of dysfunction. I elaborate some consequences for the functions debate.

**Acknowledgments:** I’m grateful to audience members at PSA 2018, where I presented this material.
1. Introduction

Theories of function are conventionally divided up into two main categories, historical and ahistorical (or backwards-looking and forwards-looking). The selected effects theory (Neander 1983, 1991; Millikan 1984) is an example of a *historical* theory, but there are other historical theories, including some versions of the organizational theory (McLaughlin 2001). *Ahistorical* theories include Boorse’s goal-contribution account (1976; 1977; 2002), the propensity theory (Bigelow and Pargetter 1987), and the causal role theory (Cummins 1975; Craver 2001; Hardcastle 2002). In the 1970s and 1980s, it was common to see these two sorts of theories as competing with each other, though more recently, philosophers of biology have generally adopted a pluralistic stance, and see them as capturing different aspects of real biological usage (Garson forthcoming a). Still, the validity of the basic distinction has never been seriously challenged.

Many proponents of ahistorical theories have argued that we should accept their theories precisely *on account of* their being ahistorical. In other words, their alleged ahistoricity is often held up as a significant virtue of their theories, and a strong reason to prefer them to historical theories. There are two arguments along these lines. The first argument appeals to bald intuition, and says that it’s just obvious that functions don’t always need history. One fanciful variant of this argument appeals to science fiction cases, like swamp creatures, instant lions, and randomly-generated worlds (e.g., Boorse 1976, 74; Bigelow and Pargetter 1987, 188). But one doesn’t have to go as far as science fiction to find plausible cases of ahistorical functions in biology. Many philosophers have a strong intuition that, the very first time a new biological trait emerges and begins to benefit the organism, it has a *function* even if it was never selected for (e.g., Boorse 2002, 66; Bigelow and Pargetter 1987, 195; Walsh and Ariew 1996, 498). The second argument, which is closely related, appeals to ordinary biological usage, not intuition. It says that historical theories run against the way biologists ordinarily think and talk about functions. At least sometimes, when biologists attribute functions to traits, they neither *cite* nor refer to nor think about history or evolution (e.g., Godfrey-Smith 1993, 200; Amundson and Lauder 1994, 451; Walsh 1996, 558; Boorse 2002, 73). Hence, ahistorical theories capture important strands of real biology.

In light of the above, my thesis might come as a bit of a shock. I claim that there are no *ahistorical theories of function* – or, to put it more precisely, the mainstream versions of the allegedly ahistorical theories on the market aren’t actually ahistorical. If we poke and prod at those theories a bit, a historical element falls out, like contraband stashed in a suitcase. In Boorse’s version of the goal-contribution account, history is explicitly embedded in his notion of a *statistically-typical* contribution to fitness. In the propensity account, history is embedded, a little less explicitly, in the idea of a species’ *natural habitat*. Finally, I claim that the only way the causal-role theorist can hope to make sense of dysfunction is to appeal to history.

Before I move on, there is one big qualification I must get out of the way. One could *invent* a purely ahistorical theory of function. One could assert, for example, that all of a trait’s effects are its functions. In fact, the biologists Bock and von Wahlert (1965, 274)
proposed a theory of function very much along these lines. This theory (pan-functionalism?) would be ahistorical, to be sure, since even if the world were created two seconds ago in pretty much its present form, things would still have effects, and so they’d still have functions. In fact, sometimes scientists actually do use the word “function” synonymously with “effect.” They say things like, “climate change is a function of deforestation,” or “poor academic performance is a function of malnutrition.” But this isn’t the ordinary biological use, which the theories I cite above are trying to capture. I’ll come back to this point in the conclusion.

So, I need to amend my thesis slightly. Instead of saying that there are no ahistorical theories of function, I want to say that any theory of function that satisfies two very minimal, very traditional, and largely uncontroversial, adequacy conditions, is also a historical theory. First, the theory should capture some distinction between functions and accidents (the function of the nose is to help us breathe but not hold up glasses). Second, the theory should capture the possibility of malfunctioning or dysfunction. If my heart seizes up due to cardiac arrest, it’s failing to perform its function or it’s dysfunctional. All of the theorists I engage with in this paper purport to satisfy these two adequacy criteria, or something like them, so I’m not begging any questions by insisting on these conditions.

Here’s the plan for the rest of the paper. After the introduction, I’ll turn to Boorse’s version of the goal-contribution theory, and show how it explicitly contains a historical element (Section 2). Then I’ll turn to the propensity theory and show how it contains a reference to history, buried inside the idea of a trait’s natural habitat (Section 3). I will then show how the causal-role theory, if it is to make any sense of dysfunction, must include a reference to history (Section 4). In the conclusion (Section 5), I’ll draw out the big consequences for thinking about functions and suggest a better way of dividing up theories of function.

2. Boorse’s Goal-Contrition Account

Boorse’s view (1976; 1977; 2002), at the most general level, is a goal-contribution account. It holds that a trait’s function is just its contribution to a goal. Here, I’ll focus on the subclass of functions he calls physiological functions. For Boorse, the physiological function of a trait is its species-typical contribution to the survival and reproductive prospects of an organism (1977, 555; 2002, 72). (To be more precise, Boorse carves up species into subgroups based on age and sex; the function of a trait is its typical contribution to fitness within the members of that subgroup.) Though he doesn’t define a corresponding notion of dysfunction, he defines a closely related notion of disease: a disease is simply a state that “reduces one or more functional abilities below typical efficacy.”

Neander (1991, 182) raised a now-famous objection against Boorse; she pointed out that Boorse’s view, as it stands, can’t make sense of pandemic dysfunction: “dysfunction can become widespread within a population…A statistical definition of biological norms implies that when a trait standardly fails to perform its function, its function ceases to be
its function; so that if enough of us are stricken with disease (roughly, are dysfunctional) we cease to be diseased, which is nonsense.” Pandemic dysfunctions, moreover, don’t just occupy the realm of science fiction, as in P. D. James’ *The Children of Men*. UV radiation poisoning in anurans is a good example of pandemic dysfunction. Sadly, climate change might create many more pandemic dysfunctions very soon. A good theory of function shouldn’t foreclose the possibility that all, or most, tokens of a certain trait in a certain species are dysfunctional (or as Boorse prefers, “diseased”).

Intriguingly, Boorse doesn’t deny the possibility of pandemic disease. Instead, he says that in order to make sense of pandemic disease, one has to appreciate function’s historical depth. Specifically, he says that when we consider what’s “statistically typical” for a trait, we cannot just look at what is typical right now. We have to examine the trait’s behavior over a slice of time that includes the present moment and reaches far back into the past: “Obviously, some of the species’ history must be included in what is species-typical (2002, 99; my emphasis).” He tells us that this time-slice should be longer than “a lifetime or two,” and might include “millennia.”

This is an extraordinary admission, given that much of Boorse’s core argument for his view was propped up on the claim that both biology and intuition need purely ahistorical functions, uncluttered by history. His admission implies that two of his key arguments for the view (cited above), don’t work. First, by his own admission, it’s not the case that biologists don’t refer to history; implicitly, when they talk about what’s statistically-typical, they are talking about history. Second, regardless of whether or not intuition supports ahistorical functions, Boorse’s theory doesn’t. It’s just not true, on Boorse’s account, that if lions popped into being from an unparalleled saltation, their distinctive parts and processes would have functions. They wouldn’t, since they don’t have the right history (or to be more precise, they have no history at all).

### 3. The Propensity Theory

Bigelow and Pargetter (1987) also developed an influential “ahistorical” theory of function, the propensity theory. They reject the selected effects theory (and etiological accounts more generally) because the selected effects theory gets the modality of functions wrong. In other words, the statement, “functions are selected effects,” if true, is contingently true; it might be true on the actual world, but there are possible worlds at which it’s false. To illustrate the point, they ask us to consider a world that is pretty much the same as ours except that it randomly popped into being five minutes ago. On that world, they claim, there would still be functions, just no selected effects (188): “we have the intuition that the concept of biological function…[is] not thus contingent upon the acceptance of the theory of evolution by natural selection.” This consideration prompts the need for an ahistorical theory.

For Bigelow and Pargetter, functions are propensities, or probabilistic dispositions. We might quibble over what exactly dispositions are, but any good definition will cite three parts: structure, environment, and behavior. Consider the solubility of salt. There is a
structure, namely, the polar molecular structure composed of sodium and chloride; there is an environment, namely, water; there is a behavior, namely, dissolving. When we say that salt is disposed to dissolve in water, we’re saying that, if you were to take this structure, and put it in this environment, it would perform this behavior, all things equal.

Functions, too, are dispositions. Consider “the function of the heart is to circulate blood.” For this statement to be true, there must be a structure (the heart, embedded the right way in the circulatory system), an environment (which they call the creature’s natural habitat), and a behavior (conferring a fitness boost on the organism). If one were to put the structure in its natural habitat, it would increase the fitness of the organism (relative, I suppose, to creatures without hearts). The crucial distinction between their view and Boorse’s is that in their view, a trait’s function doesn’t depend on actual frequencies of performance. A trait needn’t have an actual track record of boosting fitness to have a function; a mere propensity will do.

This raises the thorny question of what a creature’s natural habitat is. For they’re clear that a creature’s natural habitat isn’t just any environment the creature happens to find itself in. Unfortunately, they refuse to define this crucial notion; instead, they brush it off as vague, but unproblematically so: “there may be room for disagreement about what counts as a creature’s ‘natural habitat,’ but this sort of variable parameter is a common feature of many useful scientific concepts” (192). But one could at least form the suspicion that if one analyzed this unproblematically vague notion, one would find some reference to history tucked away inside of it.

This suspicion is confirmed in the very next paragraph. There, they tell us that, if a creature’s environment were to change very suddenly, then “natural habitat” will still refer to the old environment, and not the new one (ibid). There’s a time lag built into the very idea of a natural habitat. So, for example, if climate change melts enough Arctic ice, then, at least for a time, the polar bear’s natural habitat (and by extension, the natural habitat of the trait itself, namely, their thick, water-repellent fur) is the icy habitat of yore and not the contemporary, denuded one. They take that as given, and I agree.

But why would this be? What makes it the case that, in cases of rapid habitat change, “natural habitat,” at least for a time, refers to the old environment and not the new one? What makes it true, I suspect, is that the idea of a natural habitat is an intrinsically historical notion. It’s something like the environment within which the species recently survived and thrived. And if that’s not what a natural habitat is, I would like to know what it is such that, if a creature’s actual habitat shifts suddenly, the natural habitat, for a little while, is still the old one. Just because a concept is vague around the edges, that doesn’t exempt one from the obligation to give some sort of analysis.

Hence, I conclude that, contrary to rumor, the propensity theory is not an ahistorical theory, or not demonstrably so. But if that’s right, they lose one of the main virtues of the view, which is to get the modality of functions right. To be fair, there’s still a sense in which their view is ahistorical. What they can do, that the selected effects theorist can’t, is to attribute functions to novel traits – so long as that novel trait belongs to the members
of a species that has been around long enough to have a natural habitat. Suppose a gene mutation confers a benefit on an organism, say, pesticide resistance on a flour beetle. I suppose they can say that, at the very moment at which it first confers that benefit, the gene mutation has a function, namely, to make the beetle withstand a certain pesticide. This result, they claim, is “intuitively comfortable” (195). But they can say that only because flour beetles themselves have a history, and so we can talk meaningfully about their natural habitats. Moreover, I think they’ll still have a very hard time dealing with dysfunction (Neander 1991, 183), as I hope to show in the next section. Finally, I think there are good theory-neutral reasons for saying that beneficial traits, on their very first appearance, don’t have functions, but rather, whatever benefit they bring is a lucky accident. But I won’t argue for that here (see Garson forthcoming b, Chapter 2).

4. The Causal Role Theory

What about the causal role theory of function? This appears to be a purely ahistorical view. The causal role theory says, roughly, that the function of a component of a system consists in its contribution, in tandem with the other components, to a system-level capacity of interest (Cummins 1975; Craver 2001; Hardcastle 2002). Craver (2001) helpfully elaborates this view by specifying that the part in question must be a component of a mechanism. All of the basic ingredients of this theory are ahistorical: capacities, components, organization, hierarchy, interests. Even if the world were created five minutes ago, in pretty much its present form, things would still have causal role functions.

The problem enters when we think about dysfunction. Cummins (1975, 758) insisted that functions are dispositions, or capacities: “…to attribute a function to something is, in part, to attribute a disposition to it.” The function of a trait token, then, consists in its capacity to contribute to a system-level effect. But what if the token in question, through defect or disease, loses the capacity, and so can’t contribute to the system-level effect? Then, by Cummins’ analysis, it doesn’t have the relevant function – so it can’t dysfunction either.

Causal role theorists have, by and large, been silent about how to make sense of dysfunctions from this perspective. Almost everything they’ve had to say on that score, however, is consistent with the following theme: a trait token dysfunctions when it can’t do what other trait tokens generally, or typically, do to contribute to the system-level effect of interest. Consider Godfrey-Smith (1993, 200): “Although it is not always appreciated, the distinction between function and malfunction can be made within Cummins’ framework…If a token of a component of a system is not able to do whatever it is that other tokens generally, or typically, do, then its component is malfunctional.” Craver (2001, 72), offers the same general line: “…the ascription of a function to a malformed or broken part is derivative upon a description of how that type of part (X) fits into a type of higher-level mechanism (S). The malformed and broken part can be identified as an X by the typical properties and activities of Xs....” This is, at root, to rely on a statistical norm for making sense of dysfunction.
This account of dysfunction, like Boorse’s, stumbles when it encounters the problem of pandemic dysfunction (Neander 1991). For the modification suggested above implies that, if everyone’s heart seized up at once, nobody’s heart would have a function anymore, so nobody’s heart would be dysfunctional. The best way to solve this problem, and perhaps the only way, is the way Boorse took, namely, to say that the function of a trait is its typical contribution to some system effect, when what’s typical is assessed over a chunk of time that stretches back into the past, for at least “a lifetime or two,” and perhaps “millennia.” But if causal role theorists take that line, they’d have a historical theory.

Craver (2001) and Hardcastle (2002) suggest, all too fleetingly, a different way of thinking about dysfunction, one that depends not on statistics, but on our values, that is, the values and goals of people who make function attributions. Craver (2001, 72) suggests that traits dysfunction when they cannot do what people want them to do: “the mechanistic role of the broken part only appears against the fixed backdrop of shared assumptions about a type of mechanism within which parts of this type generally (or preferably) make important contributions.” The parenthetical remark alludes to a substantially new doctrine, one that demands our full concentration. It suggests that dysfunction is a mirror of human preferences and goals, of our wishing and wanting. If my heart seizes up, it’s dysfunctional, since it’s not doing what I want it to do.

Hardcastle (2002) makes remarks along similar lines. She first says that the function of a trait - what it’s “supposed to do,” as she puts it - depends on the goals of the scientific discipline that makes the investigation: “The teleological goal for some trait…depends upon the discipline generating the inquiry” (153). The palmomental reflex causes a chin twitch when you stroke an infant’s palm; it’s just an accident of cortical wiring with no deep evolutionary rationale. Still, she says, it has the function of indicating the state of brain development in infants, because that’s how biomedical researchers use it. She then says that something malfunctions just when it cannot do what it’s supposed to do (152). The palmomental reflex malfunctions when it can’t indicate the state of brain development. Simply put, dysfunction happens when a trait can’t do what we want.

But dysfunctions can’t be reduced to mere preferences in any straightforward way; this is a point that’s been taken in the literature for decades (e.g., Boorse 1977, 544; Wakefield 1992, 372), for reasons that scarcely need to be rehearsed. I’d prefer not to need sleep and water; I’d prefer if nobody had to go through the pain of childbirth or teething, either. But none of those things are dysfunctions. For that matter, I’d prefer if my hands were equipped with retractable adamantium claws. The fact that my hands can’t do what I want them to do doesn’t make them dysfunctional. If one really wanted to run with this value-centered line about dysfunction, one would at least have to add that, in order for a trait to dysfunction, it’s not enough that it doesn’t do what I prefer, but I must also have a reasonable expectation that it should act in the way that I prefer. But what could possibly ground a reasonable expectation that my hand (say) work in a certain way? Only this: that hands usually do work in the preferred way. But then we’re back to statistical norms,
and long historical slices of time. This value analysis of dysfunction isn’t a contender to a
statistical analysis; instead, the former presupposes the latter.

I’ve walked through three allegedly ahistorical theories of function, and shown that none
of them are purely ahistorical; they’re tainted with history. The conclusion will say what
we should do next.

5. Conclusion

There are no ahistorical theories of function, at least among the mainstream theories that
are put forward as ahistorical. The first, Boorse’s goal-contribution theory, explicitly
refers to what is statistically typical for a trait, where what’s typical is assessed over a
long historical period of time. The second, the propensity theory, refers to the creature’s
natural habitat, which is implicitly historical. And the third, the causal role theory, can’t
hope to make sense of dysfunction (or so I argue) without appealing to a statistical norm,
and thereby (following Boorse) to history. None of these theories will give functions to
the parts of swamp creatures, instant lions, or anything on worlds that are similar to ours
except for being randomly generated five minutes ago. The propensity theory, at least,
can give functions to novel traits as soon as those traits begin benefitting their bearers, as
long as the population in which the traits emerge has been around for long enough to
have something like a natural habitat. But even that theory will probably encounter
problems when it comes to making sense of dysfunction, though I haven’t pushed that
line in any detail here.

If this thesis is correct – that there are no ahistorical theories of function – three
consequences immediately follow. First, we need to jettison this whole way of dividing
up theories of function. The distinction between etiological and non-etiological theories
serves us much better. An etiological theory holds that function ascriptions either are, or
purport to be, causal explanations for the existence of traits. Non-etiological theories hold
that function ascriptions are not, and they don’t purport to be, causal explanations for
traits. But the crucial point is that being etiological and being non-etiological are just two
ways of being historical.

Second, given that there are no ahistorical views, the two main arguments that have
repeatedly been put forward for those theories – the argument from intuition and the
argument from ordinary biological usage – don’t actually work. If we took those
arguments seriously, they’d count as evidence against these allegedly ahistorical theories.
That doesn’t mean those theories are wrong. It does mean, however, that we’d have to
rethink, from the ground up, the motivation for accepting those theories.

A third consequence is that one popular way of thinking about function pluralism must
fail. This sort of pluralist wishes to sort all biological usage under two main umbrella
theories, the selected effects theory and the causal role theory. An argument for this sort
of pluralism is that it mirrors the two main uses of “function” in biology, the historical
sense and the ahistorical sense. If I’m right, this incarnation of the pluralist project can’t
work either.
True, there are some theories of function I haven’t addressed here, which fall a bit outside of the mainstream. Might those come to our rescue? In particular, one might wonder how the modal theory of function (Nanay 2010) fares with respect to my analysis. The modal theory holds that a function of a trait token depends on that token’s behavior on nearby possible worlds, where what’s “nearby” depends on our explanatory interests. I agree that this is an ahistorical theory through and through, since what function a trait has, and whether or not it’s dysfunctional, depend on what’s going on at other possible worlds, rather than the actual past. But it also yields a deeply implausible construal of dysfunction. As Neander and Rosenberg (2012) point out, if the modal theory is right, then many traits that are not generally considered dysfunctional, like the trait of lactose-intolerance in most Pacific Islanders, would actually be dysfunctional. So, while the modal theory doesn’t violate the letter of my second adequacy condition – namely, that it should allow for the possibility of dysfunction – it violates the spirit of that condition by carving up functions and dysfunctions in a wildly revisionary way.

Nanay (2012) argues that the fact that function ascriptions are relative to our explanatory interests can somehow lessen the sting of this counterintuitive consequence, but I don’t see how this helps. To illustrate the problem, consider Temitope, an evolutionary geneticist who’s interested in how human beings might evolve in the near future. Temitope considers a possible world to be “nearby” if, at that possible world, she has a counterpart, and her counterpart’s genome differs from hers by only a single point mutation (yielding at least 6 billion nearby worlds). She reasons that, on some of those possible worlds, some of her traits would do things that enhance her inclusive fitness. For example, we might suppose that there is a possible world at which her body’s ability to dissolve arterial plaque is substantially enhanced, one at which she has tetrachromatic vision, and one at which she’s resistant to malaria. She realizes, with dismay, that her body’s actual ability to dissolve arterial plaque represents a dysfunction. In fact, she realizes that, relative to her professional interests, she has many more dysfunctions than she ever thought possible. So even when we take into account that explanatory interests must harness function ascriptions, we still get deeply revisionary consequences. In my reckoning, a theory that hangs together pretty well with ordinary biological usage is better than a deeply revisionary one – all things equal (see Garson 2016, 105-7, for further discussion).

There’s a twist to this story, which I alluded to in the introduction. I think there is a prominent sense of “function” that scientists use that is ahistorical. Consider that climate change is a function of deforestation, poor academic performance is a function of malnutrition, and wildlife habitat is a function of soil. This notion is ahistorical through and through. “Function,” in this context, means little more than “effect,” and perhaps (as in the last of the three examples) “helpful effect.” But this tepid sense of function isn’t going to sustain a distinction between function and accident, nor will it give us any sense of dysfunction. This is the sort of “function” that Bock and von Wahlert (1965, 274) were getting at when they equated functions with “all physical and chemical properties arising from [the trait’s] form.” It’s also the sort of “function” that Neander (2017) describes in her recent discussion of “minimal functions.” But the proponents of the allegedly
ahistorical theories want functions to do much more than that. They are trying to capture the ordinary biological sense (or an ordinary biological sense) of “function,” where functions differ from accidents and sometimes things dysfunction. Unfortunately, they can’t have what they want.
References


Garson, J. forthcoming a. How to be a function pluralist. *British Journal for the Philosophy of Science*.


