Title: A Generalized Selected Effects Theory of Function

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1. Introduction.

The selected effects (SE) theory of function holds, roughly, that the function of a trait is whatever it was selected for by natural selection or some natural process of selection. For example (if certain biologists are correct), a function of zebra stripes is to ward off biting flies, because that is what they were selected for (Caro et al. 2014). Of course, SE allows a trait to have more than one function, since a trait can be selected for more than one feature. The strongest rationale for SE is that it makes sense of the distinction between function and (lucky) accident, and helps us understand the explanatory and normative dimensions of functions. I will explain these in Section 2.

Natural selection is not the only process that creates new functions. Some SE theorists think there are other selection processes in the natural world. For example, some have suggested that learning by "trial-and-error" (instrumental learning) creates new functions (Millikan 1984, 28; also see Wimsatt 1972, 15; Papineau 1987, 65; Godfrey-Smith 1992, 292; and Griffiths 1993, 419 for similar thoughts). Here, an organism is in a situation where there are a number of behaviors available to it (e.g., run, jump, or pull a lever). It tries out these behaviors in a somewhat random fashion and one of them (say, lever-pulling) results in a reward, such as a food pellet. The behavior is "differentially reproduced," that is, reproduced *over* other behaviors available to it. As a result of this process, the behavior acquires a new function, one that might be evolutionary unprecedented. Trial-and-error is not the only way organisms learn about their world. The point is that it constitutes a kind of function-bestowing selection process when it occurs.

I wish to push this line of reasoning even further, in ways that previous SE theorists have not explored. I call this the "generalized selected effects" theory of function (GSE) (see Garson 2011; 2012; 2015; 2016). In my view, in order to acquire a new function, a trait need not have contributed to its own reproduction (that is, making copies of itself). It need only have done something that allowed it to persist better (longer, more effectively) than some *alternative* trait, within a population. There must be a kind of selection process but it need not act over reproducing entities. My view of function is historical and disjunctive: a function of a trait is any activity that either caused the trait to be differentially retained, *or* differentially reproduced, within a population. Therefore, any trait that has an SE function also has a GSE function, but not conversely. (In the following, I will use "SE" to designate the traditional theory that restricts functions to entities that reproduce, and "GSE" for the disjunctive theory.)

Why would anyone accept GSE? The same rationale that supports SE also supports GSE, but GSE does away with an unprincipled restriction. The restriction is that, in order to have a function, an entity must be part of a lineage of entities that are related to one

another by reproduction or "copying."[1] This restriction has never been convincingly argued for; rather, it has simply been taken for granted as part of the parcel of ideas constituting SE. To be sure, SE theorists have been willing to extend the idea of reproduction or copying well beyond natural selection in the evolutionary context, to include things like learning by trial-and-error, where certain behaviors (loosely speaking) are copied, or repeated, more frequently than others. Yet why restrict functions to things that reproduce?

What are the benefits of extending the theory in the proposed way? One benefit is that it allows a process called "neural selection" to create new functions – that is, new *direct proper* functions - during an individual's lifetime (below I will return to this distinction between direct and derived proper functions). There are different sorts of neural selection processes, including synapse selection, whole-neuron selection, and neural group selection, though synapse selection is the most well-documented of these (e.g., see Edelman 1987; Changeux 1997; Wong and Lichtman 2002; Innocenti and Price 2005; Garson 2012). Synapse selection can sculpt highly sophisticated and adaptive neural structures, such as abnormal ocular dominance columns in the visual cortex. It may even play a role in certain forms of damage-induced neural plasticity, such as cross-modal reassignment (when part of the brain that is specialized for processing information from one sensory organ becomes recruited, through loss or damage, to serve another). But synapses do not, in any obvious sense, reproduce. They are just retained more or less successfully. If GSE is correct, the brain creates new functions throughout life. It seems unmotivated, and even bizarre, to allow natural selection and trial-and-error to create new (direct proper) functions, but not to allow neural selection to create new functions, too.

Extending the theory in this way has far-reaching implications for other areas of philosophy, including philosophy of mind and medicine. These include the attempt to make sense of mental representation in terms of biological function, and the attempt to understand health, disease, and mental disorder in terms of the failure of function. I will sketch some of those implications in the final section.

I have presented the core of this account elsewhere (Garson 2011; 2012; 2015; 2016). In those other contexts, I was mainly concerned with applying the theory to related problems in the philosophy of science, particularly those pertaining to neuroscience and function pluralism. I did not devote significant space to arguing for the core account and defending it from objections. Here, I seek to remedy that shortcoming in two ways. First, I provide a novel argument for the theory, based on parity of reasoning. In short, I

[1] In Millikan's (1984, 18) terms, a functional trait token must be a member of a "reproductively-established family." In Neander and Rosenberg's (2012) more recent parlance, it must be part of a "lineage of trait tokens parsed by changes in selection pressures."

maintain that anyone who accepts SE (where functions are limited to entities that reproduce) should, by parity of reasoning, accept GSE instead. Second, I defend the account from a recent version of the liberality objection, one I have not previously responded to at length.

Section 2 will outline the basic rationale for the traditional SE theory. Section 3 will show how theorists extended SE to include other sorts of selection processes, such as trial-and-error learning and antibody selection. Yet even this broadened construal of SE contains an unnecessary limitation. Section 4 will show how the exact same rationale supports GSE. It will also point to one benefit of GSE, namely, it allows neural selection to create new functions. Section 5 will respond to the charge that GSE assigns functions too liberally. The final section will sketch implications for thinking about representation, health and disease.

Two qualifications are in order. The question I am pursuing here is different from, and independent of, the question of how to define "natural selection," per se. My claim is that, as far as our theory of function goes, we should interpret the notion of selection very liberally to include processes like trial-and-error learning and neural selection, in addition to natural selection in the evolutionary sense. I am not, however, arguing that we should accept a correspondingly liberal definition of "natural selection" itself; I remain agnostic on that question. (See Bouchard (2008) and Godfrey-Smith (2009) for two contrasting approaches to defining "natural selection.")

Along with that, I will not pursue the question, which some authors have discussed, of whether *neural* selection is a legitimate subtype of *natural* selection. Darden and Cain (1989, 123) define natural selection generally enough to include neural selection as one subtype; Reeke (2001, 553) favors this inclusion. Hull et al. (2001, 513) define natural selection more restrictively, in such a way that excludes neural selection; Fernando et al. (2012, 4) favor this more restrictive definition, and argue that Edelman's "neural group selection" is not really a Darwinian process because neural groups do not reproduce. I am not, here, interested in the question of whether neural selection is a subtype of natural selection. My interest here is in explaining what functions are. In order for a trait to possess a function, I claim, mere differential retention is sufficient, regardless of whether differential retention suffices for natural selection.

2. The Rationale for SE.

Why would anyone accept SE? The rationale is simple: SE satisfies three traditional, and widely-recognized, desiderata for a theory of function. It also does so in a way that is biologically plausible and that avoids a host of counterexamples that plague related theories.

The three desiderata for a theory of function are as follows (though different theorists disagree slightly on the exact make-up of this list). First, the theory should make sense of the distinction between function and (lucky) accident. My nose helps me breathe, and it holds up my glasses, but only the former is a function. Why?

Second, some function ascriptions purport to be explanatory. Specifically, in some contexts, when a biologist attributes a function to a trait, he or she purports to explain why the trait exists. When Tim Caro and his colleagues (Caro et al. 2014) argued that a function of zebra stripes is to deter biting flies, they purported to explain, in some causal-historical sense, why zebras are striped, rather than, say, mono-colored. Biologists do not always use function in this explanatory, "why-it-is-there" sense. But sometimes they do, and when they do, we should take it seriously. Philosophers have long been puzzled by the explanatory import of function statements. How can the effect of a trait explain the existence of that very trait? (This is the problem of "backwards causation" – see Ruse 1973.)

Third, functions are "normative," in a special sense that I will explain here. To say that functions are "normative" just means it is possible for a trait token to possess a function it cannot perform (that is, something like malfunction is possible). If I break my arm in a skiing accident, my arm cannot perform its function, or at least not as well. It is malfunctioning or dysfunctional. Normativity, in the special sense that I use the term, has nothing to do with values or ethics. But there is still a question about how a function can linger, as it were, in the absence of the corresponding capacity. How can a trait have a function it is not capable of performing? This question has major theoretical significance for biomedicine and psychiatry, which try to explain the ways that functions can fail.

The main strength of SE is that it neatly satisfies these three desiderata, and it does so in a way that coheres well with real biological usage. First, the reason the zebra's stripes have the function of deterring biting flies, rather than entertaining guests on safaris, is because that is why stripes evolved by natural selection. Second, if the function of the trait is what it was selected for in the past, then when we attribute a function to a trait we offer a causal explanation for why the trait exists, an explanation that cites an effect of that very type of trait (e.g., why zebras generally have stripes, rather than being, say, mono-colored). Third, SE makes the function of a trait depend on its history, rather than its current-day capacities. So, it is easy to see how a trait can possess a function it can no longer perform. SE makes something like dysfunction easy to understand.

Of course, SE has its detractors. Unfortunately, I do not have the space here to discuss the major objections that have been leveled against the theory over the last four decades. Nor

do I have the space to consider how well, or how poorly, other theories of function can satisfy these three desiderata, such as fitness-contribution theories or causal role theories.[2] Nor do I defend, here, why those desiderata are the right ones to consider when assessing a theory of function. I deal with those issues extensively in another place (see Garson 2016). Fortunately, my primary goal here is fairly limited in scope. Instead of defending SE extensively, I want to argue for the following conditional: if one accepts SE, then, by parity of reasoning, one ought to accept GSE instead.

An anonymous referee raised an objection: if the best argument for SE is that it satisfies the three desiderata outlined above, and if GSE satisfies those desiderata with fewer unnecessary assumptions, then GSE might be preferable to SE. However, there might be other, independent, arguments for SE that have nothing to do with its satisfying those three desiderata. For example, one might argue that sometimes, when evolutionary biologists say that a trait has a function, all they *mean* is that it evolved by natural selection. If that is correct, then a good argument for SE is that it mirrors explicit biological usage, perhaps even better than GSE does.

I agree with that assessment (namely, *if* there is a good argument for SE that has nothing to do with its ability to satisfy those three desiderata, then SE might be preferable to GSE). However, I would make two points in response. First, many SE theorists do, in fact, say that the best argument for SE is that it satisfies one or more of those three desiderata. For example, Wright (1973, 159) defends his precursor to SE by pointing to the explanatory role of functions; Millikan (1989, 296) and Neander (1991, 180) defend SE by appeal to the normativity of functions; Lewens (2004, 129) defends SE by pointing out that it satisfies all three desiderata (despite his reservations about the theory which I will indicate in Section 5). None of those authors argue that SE can be directly "read off" surface features of biological usage. To the extent that one is an SE theorist of that stripe, one ought to accept GSE instead of SE. Second, the claim that SE accurately mirrors explicit biological usage is quite controversial. Some philosophers of biology reject SE because they think evolutionary biologists do not, generally, explicitly appeal to selection history when they attribute functions to traits (e.g., Schlosser 1998, 304; Wouters 2013, 480). So I still think that the best argument for SE is that it satisfies these three desiderata. That does not mean that SE is not grounded in biological usage; rather, *these desiderata* themselves are ultimately justified by appeal to explicit biological usage.

[2] For example, some theorists believe that any process that exhibits the right sort of feedback loop, where the past effect of a trait somehow contributes to the continued existence of the trait itself, can generate new functions, even if it does not involve selection (for example, Schlosser 1998; McLaughlin 2001; Sarkar 2005, 18; Weber 2005, 39; Mossio et al. 2009; see Garson forthcoming a for criticism).

To avoid potential misunderstandings, I will make three more qualifications before continuing. First, SE is typically understood as a theory of biological function, rather than a theory of artifact function. This is also how I understand GSE. So, even if it does not capture artifact functions, that is not a strike against it. Second, many SE theorists accept a certain form of pluralism about functions. They believe that sometimes, when biologists attribute functions to traits, they are implicitly appealing to SE, and sometimes they are implicitly appealing to the causal role (CR) theory (see, e.g., Godfrey-Smith 1993, 200; Griffiths 2006, 3). I accept this somewhat restrictive sort of pluralism though I would replace SE with GSE (see Garson forthcoming b). I do not accept a sort of pluralism that acknowledges both SE and GSE as legitimate theories of function in their own right. Third, SE is probably best understood either as a theoretical definition of "function" (as in "water is H2O"), or as a conceptual analysis of the way modern biologists use the term, rather than a conceptual analysis of lay usage. So, the fact that many ordinary people do not use the word "function" in this sense should not count against SE. The same holds for GSE.

3. Selection Processes in Nature.

Some SE theorists have been careful to note that there are other sorts of selection processes in the natural world, in addition to natural selection, and that these other selection processes can create new functions. I am not just referring to multi-level selection processes, for example, at the level of the gene or the group (Lewontin 1970; Sober and Wilson 1998). Other such processes may include learning by trial-and-error, and the selection of antibodies in the immune system. I suspect that this selectionist picture can even be extended to the differential replication of transposable elements in the genome, that is, to bits of selfish DNA that duplicate and re-insert themselves along the chromosome (Elliott, Linquist, and Gregory 2014). New SE functions can arise over an individual's lifetime.

What do these various processes – natural selection, trial-and-error learning, and antibody selection – have in common that makes them selection processes? Very abstractly, the three processes share the same general features. There is a population of entities that differ from one another in certain ways (see Section 5 for a careful explication of what a population is). Because of those differences, some of the entities reproduce, or "get copied," more effectively than others. When a group of entities exhibits this sort of pattern, it can generate new functions. There is nothing special about the connection between natural selection and function, except that natural selection is an instance of this pattern.[3]

[3] Many theorists have tried to give a precise account of what this "general selection" process amounts to, such as Darden and Cain 1989 and Hull et al. 2001. However, they did not attempt to form a connection between this generalized notion of selection and the debate about biological function.

I will begin with trial-and-error, not because it is the most common kind of learning, but because it is the most familiar. By "trial-and-error," I do not refer to any highly specific mechanism, but rather, any learning process that has the following general characteristics. An organism is in a situation where there are different behaviors it can perform. It tries out these behaviors, and one of them is correlated with a reward. That fact that the behavior is correlated with a reward causes that behavior to recur more frequently, in that situation, than the others. This is a sort of "differential replication" of behaviors in the animal's behavioral set (McDowell 2009).

Trial-and-error strikes me as a genuine function-bestowing process because it satisfies the three desiderata described above. Most importantly, it captures the explanatory dimension of functions. Specifically, it allows us to cite an effect of a kind of behavior to explain why that behavior currently exists. For suppose one asked, "why does that animal behave that way?" One correct answer is, "because that behavior results in a reward." Another way to answer is to say that the function of the behavior is to produce the reward.[4]

Now, there is a potential wrinkle here with learning by trial-and-error. In trial-and-error, is there anything like *reproduction* or copying taking place? Millikan (1984, 27) thinks that trial-and-error learning creates new functions, because she thinks that one behavior token is "copied" from another, in the same sort of way that one stretch of DNA is copied from another. Yet we might hesitate to accept this way of putting things. Arguably, trial-and-error is best described as the differential *retention* of one behavioral disposition over *another* behavioral disposition. But if that is correct, then trial-and-error would not, on her view, give rise to new, direct proper functions. (Interestingly, and this is a possibility that I will explore in the next section, Millikan could say that even if trial-and-error learning does not create *direct proper functions*, because it does not involve copying, it still creates *derived proper* functions. I will return to this complication below.) One benefit of accepting GSE over SE is that GSE only requires differential retention, rather than differential reproduction, so it easily explains how trial-and-error can create new (direct proper) functions, without assuming that some behavior tokens are copied from others.

[4] I am neither claiming that trial-and-error, in the sense that I use the term here, is the only way that new behaviors are acquired, nor that it is the most important way. An organism can learn by modeling the behavior of another, even if there is nothing like selection taking place. Kingsbury (2008) offers such reasons in her criticism of SE theorists' appeal to trial-and-error learning. Moreover, there may be other ways that a behavior can acquire a novel function (for example, because it is an adaptation shaped by natural selection). I appeal to trial-and-error because it illustrates the principle that SE can be extended well beyond the domain of natural selection alone.

Another example of a selection process is antibody selection in the immune system (see Garson 2012). At birth, a mechanism of genetic recombination produces a vast number of different antibodies. Each antibody has a distinctive "shape," which corresponds to a real or possible antigen (foreign body). When the antibody makes contact with its corresponding antigen, that antibody is multiplied throughout the bloodstream. This process ensures that we have the antibodies we need to fight off common infections in our surroundings.

My argument is *not* that trial-and-error creates new functions because it is "similar enough" to natural selection in the evolutionary sense. Nor am I claiming that antibody selection creates functions because it is "just like" natural selection in the evolutionary sense. My argument does not hinge on whether there are deep similarities between trial-and-error and natural selection, or merely superficial ones. My argument appeals to parity of reasoning. The reason that natural selection is a function-bestowing process is because it accounts for the explanatory and normative features of function, and the function/accident distinction. But by that reasoning, antibody selection and trial-and-error count as function-bestowing processes, too.[5]

4. A Generalized SE Theory of Function.

In the last section, I showed that there are multiple function-bestowing selection processes in the natural world, and that SE theorists have long recognized this fact. I wish to push this idea even further, to include the differential retention of entities that do not undergo anything like direct reproduction. The most important example is neural selection. As I noted in the introduction, there are three sorts of neural selection, but I will focus on the selection of synapses, because it is the most well-documented. But synapses are not the sorts of things that reproduce, though they can be strengthened or weakened. So SE would not assign functions to them.

I realize that Millikan would recognize neural selection as a function-bestowing process, but only in a very indirect manner. Neural selection is a general capacity of the human brain that, presumably, evolved by natural selection because it helped the organism adapt to the contingencies of its environment (Innocenti and Price 2005, 958). So neural selection, as a general capacity of the brain, has, in her view, the "direct proper function" of helping the organism adapt to those contingencies. Neural selection typically carries out this process by creating novel configurations of synapses. Those novel configurations

[5] I am assuming, as I noted in Section 2, that the best argument for SE is that it satisfies those three desiderata, rather than that it can be explicitly "read off" biological usage. If I am mistaken about that, then it *does* matter quite a bit whether trial-and-error and antibody selection are highly analogous to natural selection. I thank an anonymous referee for pointing this out.

of synapses, in her view, come to have the "derived proper function" of adapting the organism to those contingencies, because those configurations are produced, in the right sort of way, by a mechanism that has the "direct proper function" of so adapting the organism, and which normally carries out said function by creating such configurations – see Millikan (1989, 288). I accept Millikan's distinction between direct and derived proper functions, but I do not see why novel brain functions must arise in this somewhat convoluted manner. In some cases - for example, in the formation of abnormal ocular dominance columns to be described below - it strikes me as an unnecessary complication for our account of novel functions.

Let me put the point against Millikan somewhat differently, and more rigorously. Suppose we maintain, consistently with her published views, that natural selection and trial-and-error learning create new functions (in the sense of "direct proper functions"), but neural selection does not, because it does not involve copying. Suppose we maintain, instead, that unique neural structures generated by neural selection merely have derived proper functions. That seems like a tidy solution if one's only goal is to make sure the theory manages to attribute functions to all of the biological items we think it should attribute functions to. Yet there is a deeper objection here, namely, that the theory is based on an unprincipled restriction. Why impose the restriction in the first place? GSE delivers similar results, without the unprincipled restriction, so it strikes me as preferable.

Synapse selection takes place when two or more neurons synapse onto the same target, for example, another neuron or even a muscle fiber. These synapses behave differently (say, one of them is more active than the other). Because of these differences, one synapse is retained, and the other eliminated. Crucially, these two events (the retention of one and elimination of another) are not causally independent. Instead, there is a competitive process that takes place between them. One is eliminated because the other is retained; this is a sort of "zero-sum" game. Synapse selection has been implicated in the formation of abnormal ocular dominance columns in mammals, the formation of the neuromuscular junction, and even normal cognitive development in humans (see Wong and Lichtman 2002; Turney and Lichtman 2012; Sekar et al. 2016).

Neuroscientists disagree with each another about how frequently neural selection happens. For example, Purves et al. (1996) and Quartz and Sejnowski (1997) downplay the significance of neural selection over other mechanisms of synapse formation. However, I believe that the critics of neural selection tend to misconstrue what the theory actually holds (see Changeux 1997; Dehaene-Lamberts and Dehaene 1997; Garson 2012 for further discussion). Those critics describe neural selection as a "two-step" process where, at birth there are a large number of synapses that get progressively whittled down in early development (e.g., Purves et al. 1996, 461; Quartz and Sejnowski 1997, 539). However, neural selectionists tend to think of neural selection as an iterated process, with multiple rounds of proliferation and reduction, so some of the traditional objections are not valid. Moreover, as noted above, very recent work suggests that synapse selection

plays a fundamental role in the development of normal cognition (Sekar et al. 2016), so it should not be marginalized as an insignificant feature of brain development.

To give a simple illustration, synapse selection is involved in the formation of abnormal ocular dominance columns. In normal mammals, most neurons in layer IV of the visual cortex are "binocularly driven," that is, they are responsive to information associated with either eye. A small number of neurons are "monocularly driven," that is, they are only responsive to information from one eye or the other. Experiments conducted in the 1960s showed that, if a kitten is blinded in one eye at birth (say, one places a patch over the eye), then over the next few months, most of the neurons in its visual cortex become monocularly-driven (Wiesel and Hubel 1963). They are only responsive to information from the non-deprived eye. Even if one removes the patch, the neurons will no longer respond to that eye. This is good for the kitten because it maximizes visual acuity in the non-deprived eye.

The neuroscientists that carried out the research inferred that the underlying process must be a competitive one (Ibid., 1015; also see Kandel et al 2013, 1265). That is, there must be something like a competition between the synapses associated with the non-deprived eye and those associated with the deprived eye. This is because, if one blinds a kitten in both eyes at birth, it retains the same degree of binocularity throughout life. So, the results of monocular occlusion cannot be explained merely by invoking disuse-related atrophy. Rather, the activity of the synapses associated with the non-deprived eye somehow *causes* the elimination of the synapses associated with the deprived eye; the former drive out the latter. (There is still some controversy about whether synapse selection is involved in the formation of *normal* ocular dominance columns.)

When this process takes place, the retained synapse acquires a novel function (in addition to functions it may have had previously). Its function is to do whatever it did that caused it to be retained over the other synapse. The reason, again, is that it satisfies the three desiderata described above: it makes sense of the distinction between function and accident, as well as the explanatory and normative dimensions of function. This is not an argument by analogy. It is a parity of reasoning argument.

Consider the most puzzling of these: the explanatory dimension of function. Suppose a neuroscientist, studying the kitten's visual cortex, were to ask, "why does this visual neuron form synapses only with neurons associated with the non-deprived eye?" A correct answer would be, "because those synapses carry visual information to the rest of the brain." Another way to put the point would be to say, "the function of that synapse is to carry visual information to the rest of the brain." For that is the activity that explains why the synapse was retained over others.

I am not claiming that, when a neuroscientist attributes a function to a synapse, he or she *means* that the synapse arose by neural selection. I am claiming that, to the extent that neuroscientists use the term "function" with explanatory and normative implications, they are implicitly committed to GSE, since GSE makes sense of those explanatory and normative implications better than rival theories of function. The neuroscientific literature makes it clear that *sometimes*, when neuroscientists attribute functions to traits, they do so with explanatory implications (i.e., they purport to answer "why-it-is-there" questions), they do so with normative implications (the functions so identified are capable of malfunctioning), and they acknowledge a distinction between the function of a synapse (or other neural entity) and an accident. For example, electrical stimulation of dopamine neurons in the ventral tegmental area (VTA) of the midbrain can cause rats to vigorously self-stimulate (Witten et al. 2016), but to my knowledge, neuroscientists would not generally consider self-stimulation to be one of the VTA's functions; rather, it would be considered a by-product or accident. Moreover, neuroscientists often use "function" with normative implications; for example, synapses can *malfunction* or be *dysfunctional*, as in Alzheimer's disease (e.g., Rowan et al. 2003). While it is a bit more difficult to show that neuroscientists use the term "function" with explanatory implications, it is not entirely absent: for example, the neuroscientist Dale Purves (1994, 30) uses the phrases "function," "purpose," "role," and "why they are there," synonymously, which suggests that he thinks functions have something to do with explaining the existence of traits.

Neural selection is not the only process that creates, or reinforces, synapses. Some synapses may be genetically "hard-wired." If so, they may acquire functions by virtue of natural selection, not neural selection. Other synapses result from "Hebb's rule," namely, that if one neuron frequently activates another, both neurons are changed in such a way that joint activation is more likely to occur, even if there is nothing like a competition or zero-sum game. In my view, Hebb's rule alone does not create new functions, since selection is not taking place. At most, it amplifies existing functions (see Garson 2012 for discussion).

My goal here is not merely to add one more process (neural selection) to the catalog of processes (natural selection, trial-and-error, antibody selection) recognized by the SE theorist. For, when we add neural selection to that catalog, we have taken SE functions beyond the realm of entities that are capable of reproducing, and into the realm of entities that merely undergo something like differential retention. So, adding neural selection to our catalog forces us to revise substantially our underlying theory of function.

I recommend that we accept the following, quite general, characterization of function: the function of a trait consists in the activity that contributed to its differential reproduction, or to its differential retention, within a population. This definition is historical and

disjunctive. The first part ("differential reproduction") applies to entities that the traditional SE theorist recognizes (natural selection, trial-and-error, and antibody selection). The second part ("differential retention") applies to neural selection, and perhaps to the differential retention of behavioral dispositions. The third part, "within a population," will be explained in the next section.

Some theorists have recognized the importance of "differential persistence" for thinking about the biological world, but have not incorporated it into a theory of function (e.g., Doolittle 2014). Perhaps the view of function that is closest in spirit to my own is Bouchard's (2013). Bouchard argues that, in the context of ecology, the function of an ecosystem's component has to do with the way it contributes to the "differential persistence" of the ecosystem as a whole (that is, the way it helps the ecosystem persist *over* other ecosystems). In earlier work, Bouchard (2008) recommended that we think about natural selection in terms of "differential persistence" but he had not applied this lesson to the functions debate. The most important difference is that his view is an ahistorical, forward-looking account and mine is an etiological, backwards-looking account. I think we need the etiological component in order to make sense of the explanatory and normative dimensions of function. An additional difference is that his main concern is ecology and mine is neuroscience. I also suspect that it may be misguided to treat entire ecosystems as undergoing differential persistence, as Bouchard does, because entire ecosystems do not, in any obvious sense, form populations. Lewens (2004, 129) also considers the prospect that one can generalize SE to encompass a process called "sorting," which is a kind of differential persistence, but he actually uses this consideration to undermine SE because he thinks it leads to an unsolvable liberality problem. I will return to Lewens' argument in the next section.

5. Too Many Functions?

One might worry that GSE opens the floodgates to a host of counterexamples. Consider a bunch of large rocks scattered on a beach, which vary in hardness. The harder rocks better withstand the elements, such as waves crashing in, than others, which quickly erode. Is this a case of "differential retention?" If so, is hardness in rocks a function (see Kingsbury 2008)? Similar examples can be multiplied indefinitely. Consider a group of stars that vary in their masses. Larger stars have shorter lifespans because of a greater likelihood of gravitational collapse. Is small mass a "function" of stars (Wimsatt 1972, 15-16)?

Interestingly, critics of SE have long devised similar sorts of counterexamples against it. Bedau (1991) argued that clay crystals exhibit all of the traditional ingredients of natural selection, namely, differential replication with something like inheritance. But clay crystals do not, intuitively, have functions. Schaffner (1993, 383) devised a clever counterexample involving a "cloner machine" that causes ball bearings to be

differentially replicated on account of their smoothness. But ball bearings do not have functions for that reason.

As noted above, Lewens (2004, 127) considers the idea that the selected effects theory can be generalized to include a process called "sorting." Sorting takes place when, "there is variation across a collection of items, and differential propensities among the items to survive some kind of test, but no reproduction." Sorting processes are ubiquitous in nature. For example, a phenomenon called "longshore drift" can cause the accumulation of small pebbles at one end of a beach, and large ones elsewhere. Does sorting suffice to create new functions? Lewens points out that the same considerations that lead us to think natural selection creates new functions also imply that sorting creates new functions. This leads him to the pessimistic conclusion that it is a "waste of time" (128) to try to distinguish, in any principled way, "genuine" biological functions from these "as-if" functions, and this merits a "deflationary" (18) attitude about functions.

Fortunately, I think all of these counterexamples, from clay crystals to rocks on a beach, can be resolved in the exact same way. The core idea behind this response is that selection always takes place within a *population* of like entities. But rocks scattered on a beach – even those that are sorted into large and small, as in Lewens' example – do not constitute a population. They constitute a mere aggregate. (The same is true for the other counterexamples.) So they do not have functions.

So, what is a population? And how does a population differ from a mere aggregate? My discussion here is somewhat limited by the fact that philosophers of biology, with few exceptions, have not devoted sustained attention to the idea of a population (but see Millstein 2009; Godfrey-Smith 2009; Matthewson 2015). One theme that runs throughout the sparse literature, however, is that in order for a group of entities to constitute a population, it is not enough that the members have a shared history or that they are close to one another in space. Rather, they must exhibit the right sorts of interactions. So, what sorts of interactions make a collection of individuals a population? Members of a population must engage in fitness-relevant interactions, whether competitive or cooperative. My behavior must have some effect on your chances of survival or reproduction, and vice versa. Clearly, we can place further restrictions on this idea, but this suffices for my purposes.[6]

[6] Godfrey-Smith (2009, 51) says that paradigm Darwinian populations must exhibit competitive interactions; cooperative interactions alone do not suffice. As he colorfully puts it, in a case of reproductive competition, "…a slot I fill in the next generation is a slot that you do not fill." However, in this context I will simply restrict populations to entities that exhibit some sort of fitness-relevant interactions, whether competitive or cooperative.

Philosophers of biology who have discussed the notion of a population have restricted their attention to entities that reproduce. Godfrey-Smith (2009), for example, would not consider a group of competing neurons to constitute a paradigm Darwinian population because neurons do not reproduce. So, I am not claiming that in order for an entity to have a function, it must be a member of a population in this richer sense, which requires reproduction. Rather, I am borrowing certain conceptual resources from that discussion in order to tackle this liberality objection.

Once equipped with the notion of fitness-relevant interactions, we can see why a bunch of rocks on a beach does not form a population. For the relative "success" of one rock - that is, its chances of persistence - are independent of the chances of persistence of the others. The same goes for a bunch of stars that differentially persist in a galaxy. They do not affect each others' chances of survival. Incidentally, the same point can be made about the ball bearings in Schaffner's cloner example, or Bedau's clay crystals. Contrary to Lewens (2004), sorting alone does not create new functions.

One might accept that a group of rocks on a beach does not constitute a population, and hence that the rocks do not have functions. But perhaps one could modify the counterexample slightly. (I thank Karen Neander for this interesting objection.) Imagine, now, that some of the rocks are piled up on top of one another. Imagine, moreover, that as waves crash in, they rub against each another. The harder rocks contribute to the gradual erosion of the softer rocks. Here, they have the right sorts of interactions to constitute a "population." In this case, wouldn't I be forced to say that hardness is a function of rocks? I think this is a much trickier case, and one that I have not responded to at any length (though see Garson 2016, Chapter 3, for some suggestions).

We can avoid the force of this counterexample by reflecting more deeply on what populations are. Fortunately, Matthewson (2015) provides a tool to do just that. He argues that fitness-relevant interactions, even competitive ones, are not sufficient for distinguishing paradigm Darwinian populations from others. He claims that paradigm populations must exhibit high degrees of "linkage," too. Roughly, this means that, on average, each individual has fitness-relevant interactions with a large number of other individuals within the group, rather than a handful of its immediate neighbors. For example, suppose we restrict our attention to competitive interactions. If I am a member of a group with high linkage, then my relative "success" has negative repercussions for the fitness of most other members of my group, and not just a few others.

As Matthewson points out, we can use graph theory to make the idea more precise. Suppose we use a graph to represent a given collection of individuals. A node in this graph represents an individual in the population. An edge between two nodes represents a

fitness-relevant interaction between the two individuals. Here is one simple way of measuring the degree of linkage in this population: calculate the ratio of the actual number of edges (in this model) to the number of edges there would be if each node were connected with every other node (with no loops).

Once we carry out this simple linkage calculation, how do we decide whether the group is, or is not, a population? There are two directions we could go here. First, we could treat the notion of a population as a categorical notion, and stipulate some threshold degree of linkage that the group must have in order to count as a population. For example, we might stipulate that, in order for a group of individuals to constitute a population, this ratio must be significantly higher than .5. The main problem with this approach is that there is some arbitrariness about how we set the threshold. Second, we could treat the notion as a graded notion, and simply say that the higher a group's linkage score, the more population-like it is.[7]

If we accept this principle, then we can see why a pile of rocks does not constitute a population – or, at best, why it is not very population-like. Each rock exerts a significant force only on those rocks that are adjacent to it, and it exerts a negligible force on those rocks that are separated from it by other rocks. To make the example more precise, suppose there are ten rocks piled up on each other in a pyramid shape (four in layer one, three in layer two, two in layer three, and one at the top). When we represent this using a graph, we have ten nodes. Suppose each node is only connected to those nodes that are adjacent to it in the pyramid, as in figure 1. The total number of edges is 18. The maximum number of edges (if each node were connected to every other and with no loops) is 45.[8] According to our measure, the pile of rocks would have a linkage of 18/45 or .4. If we use the threshold approach, and we stipulate a threshold of .5, we would have to say it is not a population. If we use the graded approach, we would have to say it is not very population-like. Following out this latter course of reasoning, we could say that the rocks that persist longer have, at best, a very low degree of functionality.

[7] I thank John Matthewson for suggesting to me these two different ways of thinking about populations, that is, graded and categorical.

[8] Where n is the number of nodes, the maximum number of edges, with no loops, is $\frac{n(n+1)}{2} - n$.

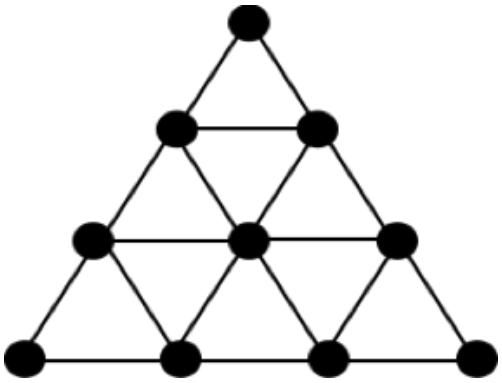

Figure 1. A pile of rocks represented as an undirected graph.

Alternatively, consider the neuromuscular junction in an infant rat. At birth, each muscle fiber is innervated by several different motor neurons. By two weeks after birth, each muscle fiber is innervated by only one neuron. This process is mediated by synapse selection, that is, there are competitive interactions between the synapses that cause some motor axons to retract and others to remain (e.g., Turney and Lichtman 2012; see Personius et al 2016 for recent work on its mechanistic basis). Here, since there are multiple synapses but only one "slot," there is a near-perfect degree of linkage between the synapses – the "success" of any one synapse entails the "failure" of all others.

One anonymous referee pointed out, quite correctly, that if we consider the linkage of the neurons of the brain as a whole, or even one region such as area CA1 of the hippocampus, the linkage value would be extremely low. For example, CA1 is estimated to have about 250,000 pyramidal neurons. Suppose, for the sake of illustration, that each pyramidal neuron makes contact with 10,000 other neurons. The total *possible* number of edges is around 31 billion; the maximum actual number would be about 2.5 billion, yielding a (quite rough) linkage estimate of about 0.08. This is far smaller than the linkage of our pile of rocks. However, this is precisely in line with my viewpoint, since I do not consider the collection of neurons that make up the brain as a whole (or even an area such as CA1) to count as a population for the purpose of assigning functions. In the case of synapse selection in the neuromuscular junction, for example, the relevant population is much smaller: it consists of the group of synapses (that is, the collection of pre-synaptic axon terminals) competing to innervate the same target. Even in the case of Edelman's neural group selection (see Edelman 1987) the relevant population consists of large collections of neural groups, each of which is responsive, in varying degrees, to the same stimulus.

6. Implications for Future Research.

GSE has interesting implications not only for philosophy of biology, but also philosophy of mind and medicine. In philosophy of mind, it has implications for thinking about

mental representations. In philosophy of medicine, it has implications for ongoing debates about health, disease, and mental illness. I will briefly sketch these implications.

Consider mental representations (see Garson 2015, Chapter 7, for more extensive discussion). In particular, teleosemantics generally pairs two separate ideas. The first is that representation should somehow be explicated in terms of biological function. The second is that biological function should be explicated in terms of natural selection.[9] These two ideas suggest that evolution by natural selection is required for generating a basic (or "simple") set of representations, and more complex representations are formed by some sort of manipulation of those simple ideas (as suggested in Dretske 1986, 335, and Neander 1999, 22). One problem here is that, though teleosemantics makes it relatively easy to see how individuals could come to form representations of things such as fire or predators, it is harder to see how creatures could come to represent things that did not play a salient role in their evolutionary histories, such as postmodernism or celebrities.

Suppose, however, that we agree with the first premise (that representation should be understood in terms of biological function), but we also accept that there are many function-bestowing selection processes in the natural world (most importantly, neural selection) and not just natural selection. Not only would we expand the range of functions in the natural world, but we would also expand the range of "simple ideas" that a human being could possess. For example, there is some evidence that the ability to recognize written words of the English language is a result of neural selection (Garson 2011). If that is correct, then teleosemantics could explain how humans can entertain thoughts like, *that is a written word of the English language*.

Papineau (1984; 1987) considered a similar idea in relation to beliefs, that is, that beliefs could undergo a sort of competitive process that yields new functions, and hence new contents, but he did not apply the point to neural selection. Dretske (1988, Chapter 5) also explored this sort of approach when he suggested that a neural mechanism can be "recruited," as a result of learning, to constitute a sign for some external stimulus. In other words, Dretske was also interested in how new representations might emerge over an ontogenetic timescale and not merely an evolutionary one. However, to my knowledge, Dretske did not clearly define this crucial notion of "recruitment" or identify the mechanisms that might underpin it. My view is that neural selection is a mechanism by which this Dretskean recruitment could realistically happen.

Finally, an ongoing debate in the philosophy of medicine (including psychiatry) has to do with the very concept of disease (or disorder). One controversial idea is that we should

[9] See Ryder forthcoming and Neander forthcoming for book-length defenses of this idea.

explicate the idea of disease in terms of the idea of an underlying dysfunction, and we should explicate dysfunction, in turn, in terms of the failure of a trait to perform its evolved function (e.g., Wakefield 1991). Of course, there may be other ways of explicating this notion of dysfunction, other than the way SE does it (see, e.g., Kingma 2010, Hausman 2011, and Garson and Piccinini 2014 for discussion).

Suppose, however, that we accept GSE, that is, that there are numerous function-bestowing processes, independent of natural selection, including neural selection. That opens the possibility that something that may appear dysfunctional from the standpoint of natural selection is actually functional from the standpoint of, say, learning or neuroscience. That is, there may be conflicts between function ascriptions. This implies that we must be particularly cautious when we judge something to be strictly dysfunctional, because it is possible that the item is, in fact, performing a function that is not readily apparent to us (Garson forthcoming c). The point is that, if we accept that there are multiple function-bestowing processes, we must be cautious when we deem something to have no functional significance at all.

In summary, I have tried to show that if one accepts SE, then one ought to accept GSE instead. The argument appeals to parity of reasoning: GSE satisfies the same desiderata as SE, but without an arbitrary restriction. I addressed the concern that GSE attributes functions in an overly liberal way by emphasizing the idea that selection (broadly construed) always takes place within a population. I also tackled a novel version of this liberality objection, namely, that GSE attributes functions to rocks in a pile, and I extended this defense by saying that populations must have high degrees of linkage. Finally, I touched upon implications for philosophy of mind and medicine.

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