Jakob Roloff*

Populations of Neurons and Rocks? Against a Generalization of the Selected Effects Theory of Functions

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Abstract: Millikan's (1984. *Language, Thought, and Other Biological Categories: New Foundations for Realism*. MIT Press) selected effects theory of functions states that functions are effects for which the ancestors of a trait were *selected for*. As the function is an effect a thing's *ancestors* produced, only things that are reproductions in some sense can have functions. Against this reproduction requirement, Garson (2019. *What Biological Functions Are and Why They Matter*. Cambridge University Press) argues that not only processes of differential reproduction but also processes of *differential persistence* can lead to new functions. Since such "persistence functions" have the same explanatory power as traditional selected effects functions, selected effects theorists should include them in their theory. In this paper, I will defend Millikan's theory against this argument. I will show that the proponents of the generalized theory have yet to provide a working notion of populations that avoids a liberality problem. Further, I will argue that persistence functions are at best a marginal case of functions due to their restricted explanatory power.

Keywords: functions; selected effects theory; Millikan; Garson; populations; Philosophy of Biology

1 Introduction

In the traditional selected effects theory of functions, only things that are reproductions or are in some way derived from reproductions can have functions. This central claim of Millikan's (1984) theory of proper functions spelled out in *Language, Thought, and Other Biological Categories* has been put under pressure lately by Justin Garson (2019) in *What Biological Functions Are and Why They Matter*. Garson argues that not only processes of differential reproduction but

***Corresponding author: Jakob Roloff**, Institute of Philosophy, Justus Liebig University Giessen, Rathenaustraße 8, 35394 Giessen, Germany, E-mail: Jakob.S.Roloff@phil.uni-giessen.de. https://orcid.org/0009-0006-7361-761X

also processes of *differential retention or persistence* can lead to new functions. In this paper, I will examine whether proponents of Millikan's theory should accept Garson's argument and adopt his generalized version.

After presenting Millikan's "traditional" (Section 2) and Garson's generalized theory (Section 3), I will discuss several possible ways to defend Millikan's version against Garson's argument (Section 4). I will try to show that the proponents of Garson's generalized theory have yet to provide a working notion of populations (Section 4.2). Further, while reproduction is not necessary to determine the trait types that have the functions (Section 4.1), the way how traits in non-reproducing things should probably be typed leads to further complications, making functions gained by processes of differential persistence at best a marginal case of functions due to their restricted explanatory power (Section 4.3). Those "persistence functions" will differ significantly from functions acquired by differential reproduction. Since they are two different phenomena, it is best not to mix them together.

Before I start, one comment on the methodology. Following Millikan (1984, 38; 2017, Ch. 7), I do not aim to do a conceptual analysis of the concept of a biological function, or selected effects function, or a population. I regard terms like these as theoretical terms, where the aim of debates centering around them should be to stipulate them in a way so that they fulfil a certain explanatory role within a certain theory. Thus, their validity should be measured by their explanatory value and not by comparing them with their use in ordinary language or even in biology. The explanatory role of selected effects functions is to explain why something exists and the explanatory role of populations is (at least here) to be the set of things (or reference class) in which the function-bestowing selective process takes place.

2 Millikan's Selected Effects Theory

Ruth Millikan's theory of proper functions – I will also call it the "traditional theory" – states that having a function does not consist in having a certain disposition, but in the fact that *the ancestors* of the trait were *selected for* because they produced certain effects. Hearts, for example, have the function to pump blood because pumping blood was what our hearts' ancestors did that explains why hearts exist now. Also malformed hearts have the function to pump blood and are thus hearts, even if they actually cannot pump blood at all (cf. Millikan 1984, 17–8). In this section, I will lay out the details of Millikan's theory.

The initial thought behind selected effects theories is that the existence of certain things can be explained by referring to its effects (cf. Wright 1973, 154–61). Millikan's starting idea, parallel to Wright, was "to define a thing's function as what something like it once did that helped cause it to be, to be where it is, or to be as it is" (Millikan

1993, 33). However, this definition would attribute functions to stages of mere cyclical processes like the water cycle since there are effects that earlier stages produced that caused the water cycle to be as it is now.

To exclude such cases, Millikan did two things. *First*, she introduced a reproduction requirement: whether an object has a (direct) proper function depends on whether that object is a member of a reproductively established family (REF). *Second*, she required that there must be some kind of selection process (cf. Millikan 1993, 33–9). I will first spell out what she means by "reproduction" and "reproductively established families" and will then discuss selection.

2.1 Reproductions and Reproductively Established Families[1]

Millikan (1984, 19–20) uses "reproduction" in the sense of copies. Roughly, an object *B* is a reproduction of an object *A* iff aspects of *B* resemble aspects of *A* because they were caused by those aspects of *A*. Had these aspects of *A* been different, the aspects of *B* would have been different accordingly.

For example, a copy of a paper is a reproduction of the copied paper because some properties of the copied paper, i.e., the markings, cause that the copy also has those properties. If the markings on the original paper had been different, they also would have been different on the copy. In contrast, my left arm is not a reproduction of my parents' left arms. If both my parents had lost their left arms in childhood, I would still have been born with two arms, not just one (cf. Millikan 2017, 158; Millikan 1984, 19–23).

Of course, not every property of *A* is reproduced when a copy is made. Markings on the back of a page as well as the paper thickness are usually not copied. The properties "by reference to which *B* is a reproduction of *A*" (Millikan 1984, 20) are the *reproductively established properties*:

Reproductively established properties

(Ch_{RE}) The properties relative to which *B* is a direct reproduction of *A* are the *reproductively established properties* of the REF_F *R* of which *A* and *B* are part.

Reproductively established families (REFs) are then collections of things that are reproductively tied together. There are first-order and higher-order reproductively established families.

1 I have already used parts of this section in Roloff (2022).

First-order reproductively established families

(REF_F) "Any set of entities having the same or similar reproductively established characters derived by repetitive reproductions from the same character of the same model or models form *a first-order reproductively established family*" (Millikan 1984, 23)

Examples of a first-order reproductively established families (REF_F) are the paper copies mentioned above, but also genes. My genes and my parents' genes are members of a REF_F because my genes are reproductions of my parents' genes. My brother's genes and mine are also members of the same REF_F because both are reproductions of the same original, our parents' genes.

Along with first-order REFs, there are also higher-order REFs (REF_H) whose members are not reproductions of each other, but are produced by mechanisms that are members of another REF.

Higher-order reproductively established families

(REF_H) Any set of entities that are produced in (approximately) the same way by members of a prior REF (first or higher order), these mechanisms having produced these products in performing (or trying to perform) the same proper function, form a higher-order REF.[2]

Hearts form a REF_H because they are produced by the prior REF_F of certain genes, and it is a function of these genes to produce hearts that pump blood.

For higher-order REFs, there are no properties relative to which the members of the REF_H are reproductions of another, there are no reproductively established properties. Instead, there is a "Normal character" of the REF_H that is made up by the "properties that are common to all members of a higher-order reproductively established family that have been produced Normally [i.e., in accordance with a Normal explanation]." (Millikan 1984, 25).[3]

Normal character of higher-order REFs

(Ch_{Norm}) The properties that are common to all Normal members of a REF_H *R* form the *Normal character* of *R*.

2 The loosening of the definition by the brackets is supposed to include also malformed items in the higher-order REF. For a more detailed definition cf. Millikan 1984, 24–5.

3 A Normal explanation for the performance of a proper function is a general explanation how a specific proper function has been typically fulfilled in the selection history, cf. Millikan (1984, 33–4, 43–5).

2.2 Direct Proper Functions

If we require that being a member of a reproductively established family is necessary for having a function, the water cycle has no function. The properties of one stage do not reproduce in the sense that if they would be different in one cycle, they would also be different in later cycles. Moreover, it is even difficult if not impossible to separate earlier and later cycles here.[4] Further, they do not form a higher-order REF since what causally produces the water cycle (the water on earth, the sunlight, some physical laws, etc.) has no reproductively gained function to produce the water cycle.

A first characterization of proper functions might look like this:

(F*) The proper function of *m* consists in producing those effects which the ancestors in the REF of *m* produced, which explain why *m* exists.

However, there is still a very similar cyclical process that reproduces in Millikan's sense: "had the earth been in a different place going at a different speed in a different direction last year, this placement and vector velocity would have reproduced itself this year." (Millikan 1993, 35) The more general problem here is that not every effect that the ancestors of something produced that can be mentioned in *any* explanation why it exists now should be its proper functions.

It is further necessary that the ancestors were *selected for* producing this effect, i.e., that the effects are *selected effects*. What does this exactly mean? Saying that something has been *selected for* due to some of its effects goes beyond saying that those effects can be mentioned in *some* explanation of why it exists. It also goes beyond saying that it has been selected in the sense that its frequency increased within the population. Selection *for* something (in contrast to selection *of* something) involves that those kinds of things having a certain character *C* performed some effect *F* more often than other things that do not have *C*, and that this correlation *explains* why it exists. The differences with respect to *C* must be *causally responsible for the differences in reproductive output*, and not only in individual cases but *due to a general explanation*. So, there must be a *non-accidental* correlation between *C* and *F* within a population (cf. Godfrey-Smith 2009, 28–9).

To argue for the importance of such a general explanation, Millikan asks us to imagine a situation where some effect *F* explains why *m* exists. How can the fact

4 I thank an anonymous reviewer for pointing this out. Separating earlier and later cycles is easier in the case of the earth rotating around the sun discussed below, probably because this is a cyclical process in which the different stages reproduce in Millikan's sense, whereas the water cycle is a cyclical process where all stages are constantly and simultaneously taking place.

that this effect was produced by the ancestors of *m* (as opposed to some other cause) ever be relevant to an explanation of the existence of *m*? The relevance of the ancestors in such an explanation comes into play when there is a correlation between a certain reproductively fixed characteristic *C* the ancestors had and the effect *F* (cf. Millikan 1984, 26). This correlation permits us to include the ancestors of *m* in such an explanation.

To give an example for such an explanation, hearts of a certain structure *C* more often pump blood than hearts with a different structure and this explains why the genes that encoded hearts with the structure *C* reproduced more often than genes encoding hearts with another structure.

Thus, a correlation condition (condition (2) below) must be incorporated into the definition of proper functions. Furthermore, the correlation must figure in a legitimate explanation of the existence of *m* (condition (3) below). Millikan's final definition of (direct) proper functions is therefore this:

Direct proper functions (also: traditional selected effects functions)

(PF) "Where *m* is a member of a REF *R* and *R* has the reproductively established or Normal character *C*, *m* has the function *F* as a *direct proper function* iff:

(1) Certain ancestors of *m* performed *F*.
(2) In part because there existed a direct causal connection between having the character *C* and performance of the function *F* in the case of these ancestor of *m*, *C* correlated positively with *F* over a certain set of items *S* which included these ancestors and other things not having *C*.
(3) One among the legitimate explanations that can be given of the fact that *m* exists makes reference to the fact that *C* correlated positively with *F* over *S*, either directly causing reproduction of *m* or explaining why *R* was proliferated and hence why *m* exists." (Millikan 1984, 28)

To summarize, in Millikan's theory there must be a non-accidental causally based correlation between a certain reproductively fixed character *C* and the effect *F* and this correlation must explain the proliferation of the reproductively established family. Reproduction thus plays an important role in her theory and is, according to Millikan, necessary for functions.

3 Garson's Generalized Selected Effects Theory (GSE)

This claim – that reproduction is necessary for functions – has been attacked by Justin Garson (2019) in *What Biological Functions Are and Why They Matter*. Garson proposes that not only selection by differential reproduction, but also selection by

differential retention – I will mainly call it "differential persistence" – can lead to new selected effects functions:

The generalized selected effects theory (short: GSE)

(GSE) "A function of a trait is an activity that led to its differential reproduction, or its differential retention, in a population." (Garson 2019, 93)

The differential reproduction disjunct is meant to capture everything the traditional selected effects theory did. The differential retention disjunct extends the scope of the definition to include *effects that explain how something has managed to persist longer than other things*. The second disjunct is therefore what is at issue here. The clause "in a population" secures that the selection process leading to the differential reproduction or retention occurs within a population. It is supposed to ensure that GSE does not ascribe functions to products of mere sorting processes and hence to avoid a liberality problem. I will get back to this later in § 4.2.

To argue for GSE, Garson discusses the reasons why a selected effects theory in general must be the right theory. According to Garson, we should support some kind of selected effects theory of functions because only such theories can make sense of three puzzles: (1) the explanatory depth of functions, (2) the function/accident distinction, and (3) the function/dysfunction distinction.

First, he argues that function statements are used synonymously with explanations of why a certain trait exists (cf. Garson 2019, 11–6). Functions thus fulfill an explanatory role within biology, they are "just condensed causal explanations" (Garson 2019, 15). Selected effects (or etiological) theories of functions can make sense of this explanatory role.[5] As we have seen above in my discussion of Millikan's notion of proper functions, selected effects functions are those effects that can be mentioned in a specific kind of etiological explanation of why something exists.

With this explanatory depth of functions, also the other two puzzles can be solved (cf. Garson 2019, 15). If functions are those effects that can explain why something exists, then we gain a mean of distinguishing functions from other effects (the function/accident distinction). Further, a thing whose existence can be explained by some past effects can easily fail to perform those effects, either by being in unfavorable conditions or by being malformed itself, thus allowing for the function/dysfunction distinction (or the "normativity" of functions).

5 Garson further argues that functions can have the required explanatory depth *if and only if* they are understood as selected effects, cf. Garson 2019, Ch. 2–3.

Garson's main argument for GSE is then a parity of reasoning argument:

> Consider why we accepted the traditional selected effects view. We did so because it made sense of the three big puzzles of function: the function/accident distinction, the possibility of dysfunction, and function's explanatory depth. Since GSE solves all the same problems, minus an arbitrary restriction, we should accept it. (Garson 2019, 94)

His argument thus depends on whether GSE can indeed solve these three puzzles as well as the traditional theory. To illustrate that it can, he uses an example of neural selection adopted from Young (1964, 156):

> NEURAL SELECTION
>
> [S]uppose there is one neuron, N, that is triggered directly by crabs, a "classification" neuron […]. Suppose N synapses onto two target neurons, N_{T1} and N_{T2}, forming two synapses, S_1 and S_2. N_{T1} causes attack, and N_{T2} causes retreat. Suppose that S_1 and S_2 are weighted the same, so that there is a 50/50 chance that a given response will happen. […] Now a crab appears, which activates N, which activates N_{T1}, which causes the octopus to attack. A moment later, the octopus experiences the searing pain of a counterattack. The pain receptors control a device that inhibits S_1. This makes sure that, when that same crab appears, or others that look like it, the octopus will probably retreat […]. Had the crab made a tasty snack instead, the whole configuration would be flipped the other way around. (Garson 2019, 88)

GSE would, in this case, ascribe the function to cause retreat from crabs to S_2 because that is the activity of S_2 that led to its differential retention. So, how are the three puzzles solved?

First, if we would like to know why the synapse S_2 exists, i.e., why neuron N synapses onto neuron N_{T2} and not onto something else, one correct answer would, according to Garson, be that "'S_2 is there because it causes the octopus to retreat from crabs.' Retreat is the effect that caused the perpetuation of S_2 over S_1." (Garson 2019, 95) It is the effect for which S_2 was selected. GSE functions, or more specifically *functions gained through selection by differential persistence* (short: *persistence functions*), thus have the *same explanatory depth* as traditional selected effects functions.

Secondly, as soon as GSE can say that some effect is what something was selected for, GSE can, of course, easily distinguish between a thing's functions and its other (beneficial) effects or *accidents*. *Thirdly*, as GSE is, like the traditional theory, a historical theory where the function consists in some past effects, it can accommodate the *possibility of dysfunctions*.

As GSE can solve the same puzzles as the traditional theory without introducing a reproduction restriction, requiring reproduction is an arbitrary restriction. Thus, according to Garson, we should prefer GSE to the traditional theory.

4 Selected Effects Functions without Reproduction?

Garson claims that requiring reproduction is an arbitrary restriction within selected effects theories. In this section, I will try to show that this is not so clear. I will discuss several lines of defense a traditional selected effects theorist might take and see whether or not they are successful.

In Section 4.1, I will look upon the *connection between reproduction and the trait type* that has a function. I will concede that trait typing might also work without reproduction. In Section 4.2, I will take a closer look at the *population condition* in GSE and whether it may entail some reference to reproduction. And finally (Section 4.3), I will discuss whether GSE functions acquired by differential persistence and traditional selected effects functions acquired by differential reproduction take up *the same explanatory role*. If not, this would be a good reason to keep them apart, even if GSE functions have some explanatory depth.

4.1 Reproduction and Trait Typing

As we have seen above, the trait type must be causally relevant for the effect *F* and correlate positively with *F* within the population where selection takes place. Moreover, the correlation between the character *C* and the effect *F* within the population then must explain the proliferation of the REF, hence giving rise to a direct proper function. As I have discussed above, this correlation is an important part of both Millikan's (1984, 26) selected effects theory and Godfrey-Smith's (2009, 28–9) understanding of natural selection (cf. 2.2).

In Millikan's picture, reproduction plays a crucial role in typing the traits in the first place as it provides a principled way of typing the traits by emphasizing that there is a certain causal connection between the trait instances. The properties whose instantiation must correlate with the effect must be either reproduced and further reproducible properties (i.e., reproductively established properties of first-order REFs) or some properties than can be produced again and again by the members of another REF performing the same function (i.e., the Normal character of a higher-order REF). This is precisely why there are recurring properties in the first place whose instantiations can correlate with an effect.

How can this be done without reproduction? The only possible solution that I can imagine is that analogously to the reproductively established properties for the first-order REF there are the *persisting properties* for the differential persistence

cases. Those are also properties that recur over time – not in different individuals but in the same individual – whose instantiation can correlate with some effect within a certain population.

Persisting character

(Ch_P) The properties that remain instantiated over time by an individual *R* between t_1 and t_2 form the *persisting character C* of *R* between t_1 and t_2.

Just like the instantiations of a heritable trait form a reproductively established family, the different temporal parts of an individual form a *persisting individual*, which takes the role of a reproductively established family for cases of selection by differential persistence. And the ancestors might just be earlier temporal parts of the same individual. A more detailed definition of persistence gained proper functions might then look like this:

Persistence gained direct proper functions (short: persistence functions)

(PF_{Pers}) Where *m* is a temporal part of a persisting individual *R* and *R* has the persisting character *C*, *m* has the function *F* as a direct proper function iff:

(1) Certain ancestors (i.e., earlier temporal parts) of *m* performed *F*.
(2) In part because there existed a direct causal connection between having the character *C* and performance of the function *F* in the case of these ancestor of *m*, *C* correlated positively with *F* over a certain set of items *S* which included these ancestors and other things not having *C*.
(3) One among the legitimate explanations that can be given of the fact that *m* exists makes reference to the fact that *C* correlated positively with *F* over *S*.

I do not see any immediate problems with this way of typing traits. Note that one striking consequence of this definition is that almost every property of *R* will be part of the persisting character and thus of the trait type of *R*. I will come back to this later in Section 4.3.

4.2 Reproduction and Populations

4.2.1 Garson's Populations

An obvious question about this definition of persistence gained proper functions is how the set of items *S*, the *reference class* of the correlation between the persisting

character and the effect, should be determined.[6] For evolutionarily gained proper functions, it is natural to suppose that this set is the population in which the selection process takes place.[7] For persistence functions, we need a notion of population that plausibly picks out a set of items in which the selection by differential persistence takes place. This must be a *population in which there is a correlation between the persisting character and the persistence function, and this correlation must be part of a good explanation of why the thing still exists*.

To show the necessity of a population condition also for cases of differential persistence, Garson considers the following example, adopted from Kingsbury (2008, 496):

> SCATTERED ROCKS
>
> imagine a bunch of rocks scattered along a beach. Some rocks are harder and some are softer. The softer rocks tend to erode more rapidly, leaving the harder behind. (Garson 2019, 102)

This example makes clear that not every differential persistence in some collection of things can lead to new functions. Garson thus proposes that the differential reproduction or persistence must happen within a *population*.[8] But what exactly determines the population – or the reference class of the correlation between the persisting character *C* and the effect *F* – *especially in cases of differential persistence*?

For this, Garson (2019, 103–8) adopts the consensus from the literature on populations: that populations require "*fitness-relevant* interactions" (Garson 2019, 103).[9]

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6 This has, of course, nothing to do with any special feature of my definition of persistence function. The same applies for Garson's definition of GSE functions.

7 I think that it is plausible to assume that the reference classes here are Darwinian population as conceptualized by Godfrey-Smith (2009).

8 As I said in the introduction, my aim is not to do a conceptual analysis of the concept of a biological function or a selected effects function or a population, etc. However, this (and some of the following) examples might seem like they are intended as counterexamples aiming to show that the here discussed notion of a population is *counterintuitive* and therefore needs adjustments. Even though Garson might have thought of them in that way, I do not wish to use them like this. Instead, I think, *proponents of GSE should develop a notion of a population that plausibly picks out a set of items in which the selection by differential persistence takes place and which can be part of a good selection-based explanation of why something still exists*. Regarding this particular example (Scattered Rocks), there is also a good argument for the same conclusion in an explanation-centered methodology: As I argued above (§ 3), selection *for* depends on there being a correlation between the trait type and a certain effect in a set (or reference class) also including other things, i.e., a population. Thus, a population is necessary for selection *for* and hence must be incorporated in any selected effects theory of functions. I thank Oliver Schütze and Ruth Millikan for urging me to clarify these methodological points.

9 See also Godfrey-Smith (2009, 52), Millstein (2009, 271) and Matthewson (2015, 180) for the relevant contributions from which this consensus is drawn.

It is neither necessary nor sufficient for the members of a population to be spatially close to each other. What is rather important is that they have the right interactions:

> Recursively put, for A to belong to the same population as B, A must affect B's fitness, or A must affect the fitness of some entity C which is part of the same population as B. (Garson 2019, 104)[10]

Just like Garson generalizes the selected effects theory, he must also generalize the notion of fitness-relevant interactions. They include not only those interactions that affect the reproductive fitness, but also those that affect the further persistence of the individual (*persistence-relevant interactions*).

Clearly, the rocks scattered on a beach do not interact in any way relevant to fitness or persistence. However, consider a variation of the rock example given by Karen Neander:

> PILED ROCKS
>
> Imagine a group of rocks piled up on top of one another. Whenever the waves crash in, they jostle each other. The harder rocks not only withstand erosion better than the softer ones, but they contribute to the erosion of the softer ones. Now, there is differential retention of rocks as well as persistence-relevant interactions between them. Even so, it runs against both intuition and ordinary biological usage to give functions to rocks. (Garson 2019, 106)

Garson tries to evade this counterexample by requiring, as proposed by Matthewson (2015, 183–4), that (paradigmatic) populations have a *high degree of linkage*:

> the linkage within a group is the ratio of the actual number of fitness-relevant interactions in that group to the total possible number of fitness-relevant interactions. The closer that ratio is to 1, the more population-like the collection is. (Garson 2019, 106–7)

As the rocks admittedly only affect the further persistence of their immediate neighbors, the degree of linkage within a pile of 10 rocks will be very low, so this pile would not be very population-like.

Although Garson is probably right in requiring a high degree of linkage, this misses the point of the counterexample. His response hinges on his supposition that the pile consists of *ten* rocks. What about a not so large pile of *three* rocks all off which affect each other's persistence? Such a pile would have a high degree of linkage of 1.

A similar counterexample has been given by Conley (2020) in a review of Garson's book:

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10 Those fitness-relevant interactions can be either *competitive interactions* negatively influencing the fitness level of one individual or *cooperative interactions* positively influencing the fitness-level of both individuals. While Matthewson (2015, 193–4) and Schulte (2021, 375–6) view competitive interactions as more important, Garson (2019, 104) takes no position on this.

ROCK SHAKER

> Simply put all the rocks in a spacious container and have a machine shake the container so hard that the rocks are worn away by constant collisions as they fly around inside. As with the original example, harder rocks will be retained while softer rocks wear away to dust.

As a response to such cases, Peter Schulte (2021, 374–6) argues that the counterexamples suggest that we need a refined understanding of what fitness- and persistence-relevant interactions are. For this, he draws on Godfrey-Smith's (2009) work on Darwinian populations.

4.2.2 Competitive Interactions

According to Godfrey-Smith (2009, 51–3), reproductive competition is one factor gluing paradigmatic Darwinian populations together. There is reproductive competition (in the strong sense) when there is "a dependence between my absolute fitness and yours, so that a slot I fill in the next generation is a slot that you do not fill. […] there is a causal dependence between how many offspring each individual has" (Godfrey-Smith 2009, 51). The extent of reproductive competition is "the extent to which adding reproductive success to one individual reduces another's" (Godfrey-Smith 2009, 52).

Peter Schulte generalizes this notion of reproductive competition to use it within the Generalized Selected Effects theory:

> We can say (again, somewhat roughly) that an individual A stands in a relation of competition to B iff the following holds: A exerts a negative influence on B's chances of reproduction or *persistence*, because A behaves in a way that enhances its own chances of reproduction or *persistence*. (Schulte 2021, 374)

Both moves, introducing Godfrey-Smith's notion of reproductive competition and generalizing it for cases of differential persistence, seem pretty reasonable. However, they are not able to solve the problem for GSE. Competitive interactions may well be necessary for the notion of populations required for GSE, but they are still not sufficient. Coming back to the biological realm, there are (at least) three types of counterexamples.

First, just imagine *two species A and B competing within the same niche*. Here, too, a spot that an *A*-individual fills is a spot than a *B*-individual can't fill. *Second*, they don't have to share the whole niche to have a high degree of fitness-relevant interactions, they might only *compete with respect to one resource* that matters to both. For example, "in tidal areas or estuaries, bird species and aquatic species such as crabs or larger fish may compete over prey" (Matthewson 2015, 187). *Third*, *groups of predators and prey* also exhibit a high degree of competitive interactions:

The predator exerts a negative influence on the prey's chances of reproduction or persistence because the predator behaves in a way that enhances its own chances of reproduction or persistence, i.e., by eating the prey (cf. also Matthewson 2015, 185). However, two species competing with respect to the same resource as well as two species being in a predator–prey relationship are nonetheless regarded as forming two distinct populations.

Schulte (2021, 376) acknowledges this and concedes that within evolutionary biology we need a narrower notion of populations to exclude groups of predators and preys. But for cases of differential persistence, he thinks that this broadened notion is quite valid. However, similar problems arise for cases of persistence. Garson assumes without comment that the two neurons in NEURAL SELECTION form the relevant population, surely because they interact in *a* persistence-relevant way. But what about all the other things with which the two neurons compete? What about other neurons (including neurons in other organisms)? What about the neurons' nutrients? What about other structures in the brain?

4.2.3 A Boundary Requirement?

One might think that such cases can be excluded by drawing the right boundaries here:

> The boundaries of the population are the largest grouping for which the rates of interaction are much higher within the grouping than outside. (Millstein 2010, 67, cf. also Matthewson 2015, 192)

However, such a boundary requirement also will not help: Suppose that N_1 and N_2 are neurons of an organism *o* that is part of a (paradigmatic Darwinian) population (in Godfrey-Smith's (2009) sense). Then, effects that other organisms in that population produce that enhance their own chances of persistence will decrease the chances of persistence of the organism *o* and thus also the chances of persistence of N_1 and N_2. As the fate of the two neurons depends on the survival of *o*, they also compete with *o*'s competitors. And if the neuron N_1 detects some food source or a source of danger, for example, its persistence might as well affect the persistence of the other organisms. There is thus also a high degree of linkage. Should the relevant population in NEURAL SELECTION then consist of those two neurons, the neurons of other organisms in the population to which the organism *o* belongs, and maybe even these other organisms?

The plausibility of referring to what N_2 did in an explanation of why N_2 still exists gets lost in such a population. There is suddenly a huge number of other neurons, many of them persisting not so long as N_2, yet many others persisting longer than N_2. Clearly, the level of selection that we are looking for is a selection between the two

neurons. As Garson himself states, the selection is regulated by "a device [inside the brain] that inhibits S_1" (Garson 2019, 88) as a result of the pain. But Garson's proposed and Schulte's modified notions of populations do not allow us to delineate a population here that is suitable for our explanatory purpose. Requiring a high degree of interactions relevant to fitness or persistence and introducing a boundary requirement can exclude differential retention among things that do not interact in a way relevant to fitness or persistence, such as the scattered rocks. But it is unable to draw the lines between interacting things where GSE needs them.

I think that such cases of neural selection are better understood in Millikan's (1984, Ch. 2) terms of relational, adapted, and derived proper functions. Our brain probably has a mechanism with the reproductively acquired *relational* proper function to strengthen neurons (or neuronal connections) if the behavior they caused was successful. Given certain neurons that successfully cause fighting behavior against crabs, the mechanism has the *adapted* proper function to strengthen neurons that caused fighting behavior *against crabs*. And the strengthened neuron then inherits this function as a *derived* proper function: it has the *derived* proper function to cause fighting behavior against crabs. In this way, we can ascribe a proper function to the neuron without requiring that there is a population of which the neuron is part and without also requiring that there are multiple rounds of selection. Unfortunately, I do not have the space here to introduce this idea and Millikan's notions of relational, adapted, and derived proper functions in more detail.[11]

To conclude, these considerations insinuate that the notion of populations in GSE needs to be refined. Further, it shows that Garson's case for GSE is not as clear as he says it is. With respect to this important part of the theory which carries a lot of theoretical weight to avoid a liberality problem, GSE is, in its current form, implausible and needs refinement. Most importantly, the notion of population cannot fulfil the required explanatory purpose to define the set of things (i.e., the population) in which the *selection for by differential persistence* takes place. It is at least questionable whether GSE provides a good explanation of how the underlying mechanisms of neural selection work.

4.3 Different Explanans, Different Explanandum

In this section, I want to show that persistence functions do not *explain the same explanandum in the same way* as traditional selected effects do.

11 For her introduction of relational, adapted, and derived proper functions, see Millikan 1984, Ch. 2. For an example of how these notions can be applied to neuronal mechanisms, see Ryder (2006).

4.3.1 No Possibility of Proliferation

First, unlike traditional selected effects, persistence functions cannot explain how a certain trait (or type of individual) *spread* through the population, they can only explain how it managed to last longer than others. Without reproduction, there is *no possibility of multiplication*, "the only way there can be fitness differences is for the population to get smaller" (Godfrey-Smith 2009, 104).[12]

This can also be illustrated by the fact that the function categories are very different. For traditional selected effects, function categories consist of various individuals connected by their lineage. For persistence functions, if there are function categories at all, they do not consist of various individuals but only of temporal parts of a single individual. For each point in time, it is only possible that one member (one temporal part) of the lineage (the individual) exists. Consequently, even if two very similar things had produced the same effect in the past that explains their present existence, they would not be *homologous*. Rather, they would only be *analogous* due to the lack of common ancestry.

This provides evidence that persistence functions differ from traditional selected effects functions. They might be just borderline cases of selected effects functions.

4.3.2 Selection for a Trait Versus Selection for an Individual

Second, I will come back to a point that I have touched above when discussing the typing of the trait for persistence functions. I proposed that for cases of differential persistence, we could replace the reproductively established or Normal character of an REF with the *persisting character* of an individual *R*, i.e., the properties that remain instantiated over time. However, I noted that almost every property of *R* will

12 An anonymous reviewer suggested that there actually is a possibility for a trait to spread in a population without reproduction, namely by *growth*. For example, crystals of different molecular structure grow faster than others, leading to changes in trait frequencies that do not result from the population getting smaller. However, I am unsure whether we should say in such cases of *differential growth of single individuals* that these are cases of change in trait frequency within the population. After all, the number of individuals with the certain traits stays the same, only their size changes. So, it seems reasonable to uphold that differential growth of single individuals does not change the trait frequency within the population. (Things are different when whole lineages differentially grow by reproducing faster than other lineages, cf. Godfrey-Smith 2009, Ch. 2.2.) I do, however, also find it tempting to say that there are changes in trait frequency here. It seems to come down to the question – about which I remain neutral – how we should count traits instances in such populations (cf. Godfrey-Smith 2009, Ch. 4 for a very interesting discussion of growth and reproduction). But I think that it is important to keep in mind, as the anonymous reviewer also noticed, that there are still no (reproductive) lineages between the crystal molecules.

be part of the persisting character and thus part of the trait type of *R*. This does not pose a direct problem for function ascriptions. For that, we only need some character whose correlation with some effect explains its continuing instantiation.

One consequence of the fact that the persisting character includes almost every property of *R* is that there is no way to select *for* a certain characteristic *without the selection of (almost) every other characteristic* the persisting individual has. In the end, it is not traits that are selected but whole individuals.

Remember what selected effects were initially called upon to explain. The reference to selected effects is supposed to answer a kind of "Why is something there?" question. In the biological sphere, this question is mostly answered by referring to the effects for which this kind of thing was selected during natural selection. Natural selection works because there is a variety of inheritable traits that differ in their reproductive fitness (and, of course, some other factors). Traits that produce the most beneficial results reproduce most often, thus leading to an increased proliferation within the population. What is thus explained is *why a particular trait, rather than another trait is now present, i.e., why an individual has this specific trait*.

The phenomenon that persistence functions explain is quite different. They don't explain why a certain trait proliferated within a population and why a certain individual now has this trait rather than another trait. They rather explain how a certain individual managed to exist longer than others, i.e., why an *individual with its persisting character* exists at a certain point in time. Thus, *it is not the possession of a certain trait by an individual that is explained, but rather why a certain individual still exists at a certain point in time.*

Even if Garson is right that there are selected effects functions gained by differential persistence (i.e., persistence functions), they are different from selected effects functions gained by differential reproduction. That does not mean that traditional selected effects functions and persistence functions, if they exist, have nothing in common. But still, they are different enough that we should keep them apart. This might not be a problem for Garson since he also suggests that they are different by offering a disjunctive definition.[13] What is a problem for Garson, however, is that there are reasons to think that traditional selected effects functions are the paradigm cases whereas persistence functions are only some borderline cases: (1) Selection by differential persistence falls short of the possibility of multiplication, it can only work by making the population smaller. (2) They do not explain why a certain trait rather than another trait proliferated, but rather why the individual still exists at a certain point in time. (3) And the relevant populations – if a sensible notion of population can be upheld at all – are at best marginal cases of

13 See, however, Fagerberg (2022) for a different opinion on this point.

populations, something alike populations of asexually reproducing things, but still marginal compared to them (cf. for the last point also Godfrey-Smith 2009, 103–5). This might cast doubt on whether persistence functions are simply another kind of function and might suggest that they are only borderline cases of traditional selected effects functions.

5 Conclusions

To conclude, even under the assumption that there is a coherent way to define persistence functions and specifically the relevant populations, there are differences to the explanatory roles of traditional proper functions and persistence functions to such an extent that it becomes questionable whether we should treat persistence functions as some paradigm category of functions and not only as some marginal cases. They cannot explain the proliferation of a certain trait within a population, but only explain how a whole individual managed to persist longer than some other individuals with which it competes. But it should be granted that this might still be a non-negligible explanatory power that justifies the postulation of persistence functions.

However, this still has to be shown. The examples of such explanations offered by Garson as well as his and Schulte's conceptions of populations leave it unclear whether persistence functions perform some well-defined explanatory role. We can thus wait for further proposals how to delineate populations of persisting things.[14]

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