AGAINST THE GENERALISED THEORY OF FUNCTION

Abstract

Justin Garson has recently advanced a Generalised Selected Effects Theory of biological proper function. According to Garson, his theory spells trouble for the Dysfunction Account of Disorder. This paper argues that Garson's critique of the Dysfunction Account from the Generalised Theory fails, and that we should reject the Generalised Theory outright. I first show that the Generalised Theory does not, as Garson asserts, imply that neurally selected disorders are not dysfunctional. Rather, it implies that they are *both* functional *and* dysfunctional. The Generalised Theory yields conflicting functional norms, and we should reject it on these grounds.

1. Introduction

This paper defends the Dysfunction Account of Disorder against Justin Garson's Generalised Selected Effects Theory of function.

My argument has two parts. First, I contest Garson's claim that synaptically selected neuropsychiatric disorders are not dysfunctional. This does not follow from his theory. What in fact follows is that neurally selected disorders are *both* functional *and* dysfunctional. I proceed to argue that this contradictory consequence of Garson's theory is sufficient grounds to reject it outright.

2. The Traditional Theory

In his 2019 book 'What biological functions are and why they matter', Garson contrasts his 'Generalised Selected Effects Theory' with the standard evolutionary account, which he terms the 'Traditional Selected Effects Theory' (from here, the 'Generalised Theory' and the 'Traditional Theory').

The Traditional Theory states:

> A *function* (F) of a trait (T) in an organism (O) is an effect of T that increased the inclusive fitness of O's ancestors, in the environment of evolutionary adaptedness, such that T was naturally selected.
>
> (See e.g. Neander, 1991; Millikan, 1989; Godfrey-Smith, 1994)[1]

So, for example, it is a function of my thyroid gland to release appropriate levels of triiodothyronine into my bloodstream, because that is the thing which thyroid glands did in the past which led them to be naturally selected.

[1] Exact formulations of the Traditional Theory actually vary significantly in the literature. For example, Millikan requires some form of reproductive mechanism, while Neander emphasises natural selection at the level of the genotype (Millikan, 1989; Neander, 1991). The important point for our present purposes it that the Traditional Theory excludes attributing direct, proper biological functions to most ontogenetic functions, such as neurally selected effects (see Garson, 2019, for a supporting argument). My definition above merely seeks to accentuate this distinction.

3. The Generalised Theory

The Generalised Theory states:

> A *function* (F) of a trait (T) is an effect that led to T's differential reproduction, or differential retention, in a population.
>
> (Adapted from Garson, 2019, p. 93)

While the Traditional Theory covers only evolutionary functions, Garson's theory manages also to encompass a range of developmentally selected functions by appealing to effects selected by differential retention through ontogeny, as well as by differential reproduction through evolution.

(From here, I use 'ontogenetic function' to refer to effects selected intra-organismically on a developmental timeline, and 'evolutionary function' for effects selected intergenerationally on an evolutionary timeline.)

The most appealing implication of this move is that it allows Garson to attribute direct, proper biological functions to synaptically selected traits in the brain.[2][3] Synapse selection is a neuroplastic mechanism by which the brain overproduces synaptic connections between neurons, and then proceeds to shed the 'useless' ones. According to Garson, the surviving synapses thus acquire the function of doing whatever it was that caused them to be selectively retained relative to the others through ontogeny – just as the thyroid gland acquired the function of releasing hormone by increasing the fitness of those carrying its genotype in the evolutionary past (Garson, 2019). Garson thus expands the domain of direct, proper function into the neuroplastic realm.

4. Garson's Argument for the Generalised Theory

Garson contends that the same argument which supports the Traditional Theory also supports his Generalised Theory. According to Garson, we were motivated to accept the Traditional Theory in the first place because it made sense of three *prima facie* puzzling features of proper functions: (1) the function/accident distinction, (2) their normativity and (3) their explanatory depth.

(1) The Traditional Theory distinguishes between functions, such as the capacity of the nose to detect odours, and accidental effects, such as its capacity to hold up spectacles. Because the nose evolved to smell (and not to hold up spectacles) that is its function. (2) The Traditional Theory also successfully accounts for the 'normativity' of functions. Because a trait's function is determined by its history of selection (as opposed to any current activity) it can fail to perform a function that it is 'supposed' to perform. (3) Finally, we sometimes cite functions as explanations for traits. "Why do Zebras have stripes? For camouflage." The Traditional Theory tells us that a function is an effect which caused its corresponding trait to

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[2] For the those unfamiliar with the function literature, *proper* functions are functions in the sense of 'function of' rather than 'functions as'. Proper functions are often contrasted with causal role functions (Cummins, 1975). Many philosophers are 'pluralists' in that they accept both of these senses of function are legitimate (see e.g. Garson, 2018; Godfrey-Smith, 1993).

[3] *Direct* proper functions have their functions inherently. *Derived* proper functions only have their functions in virtue of the direct proper function of the mechanisms which produce the derived proper functions. This is Millikan's terminology (for more on this see Millikan, 1984; 1989). Nothing in particular hinges on this distinction here.

be naturally selected. Thus, the Traditional Theory can account for functions' apparent explanatory depth (Garson, 2019).

From here, Garson argues as follows:

> The core argument for the [Generalised Theory] is simply a parity of reasoning argument. Consider why we accepted the traditional selected effects view. We did so because it made sense of three big puzzles of function … Since [the Generalised Theory] solves all the same problems, minus an arbitrary distinction, we should accept it.
>
> (Garson, 2019, p. 94)

In other words, the argument which supports the Traditional Theory also, by parity of reasoning, supports the Generalised Theory, and we have no good reason to restrict ourselves to the Traditional Theory's 'arbitrarily' narrow evolutionary scope. We are accordingly forced to accept the Generalised Theory in its place "on pain of inconsistency" (Garson, p. 93, 2019).

But what does it mean to accept a 'generalised' theory of function, as Garson contends we must? It is not simply the claim that applying the term 'function' to ontogenetic functions would be correct. Philosophers and biologists already do this in various contexts, so this would be trivial. Rather, Garson is committed to something like the following: evolutionary and ontogenetic selection processes yield functions of *the same kind*. Ontogenetic and evolutionary functions play – or should play – essentially the same theoretical role. Restricting one's concept of direct proper function to include only evolutionary functions, even for specific purposes or in specific domains, is theoretically unprincipled[4].

Garson then goes on to argue that acceptance of the Generalised Theory spells trouble for the Dysfunction Account of Disorder.

5. The Dysfunction Account

The 'Dysfunction Account of Disorder' (from here, the 'Dysfunction Account') theorises that medical disorder necessitates dysfunction.

It states:

> A *dysfunction* is the inability of some trait T to perform one of its biological functions.
>
> *Medical disorder* necessarily entails dysfunction.
>
> (See e.g. Wakefield, 1992; 2014; Neander, 1983; 1998)[5]

[4] If you are in any doubt, at this stage, that Garson is committed to a form of generality which demands that evolutionary and ontogenetic functions play the same theoretical role, simply consider his critique of the Dysfunction Account (section 6). If Garson allowed that one might distinguish evolutionary and ontogenetic functions theoretically for specific purposes (such as for defining medical disorder) then the problematic impacts he infers for the Dysfunction Account wouldn't follow.

[5] The Dysfunction Account of Disorder is often associated with Jerome Wakefield and his so-called 'harmful dysfunction' analysis of mental disorder, as first laid out in his seminal 1992 paper, but is perhaps more accurately attributed to Karen Neander who advocated this view as early as in her unpublished 1983 doctoral thesis. The Dysfunction Account also owes a debt to Christopher Boorse's 'biostatistical theory' of disease,

So, for example, hormone excretion is the biological function of my thyroid gland, so its failing to excrete sufficient hormone would constitute a dysfunction, and thus qualify as a medical disorder.

6. Garson's Critique of the Dysfunction Account

In what follows, I lay out Garson's critique of the Dysfunction Account from the Generalised Theory. I focus on the case (offered by Garson) of Substance Use Disorder, however any challenge to the Dysfunction Account based on the Generalised Theory will take a similar form.[6]

A. We should accept the Generalised Theory in place of the Traditional Theory.

B. Following the Generalised Theory, Substance Use Disorder is functional and not dysfunctional.

According to Garson, the Generalised Theory "expands the domain of entities that can possess direct proper functions, thereby increasing the likelihood that a given condition is functional, not dysfunctional." (Garson, p. 178, 2019).

On Substance Use Disorder in particular, he writes:

> Though the exact mechanisms [underlying drug addiction] are still a matter of controversy, it's likely that synapse selection is involved. ... Once a synapse is selectively strengthened this way, it comes to acquire a new function. It has the function of causing the behaviour of that led to its differential retention. If that behaviour included seeking out and using drugs, then that becomes its direct proper function.
>
> (p. 180, Garson, 2019; see also Garson & Papineau, 2019)

Precisely what is it that has acquired the proper ontogenetic function of Substance Use Disorder, on Garson's account? There is some ambiguity here, so let us briefly clarify before moving on.

According to Garson, proper functions are proximal: that is, it is the proper function of trait T to yield that effect which is most specific to T or, if you will, that which T can do 'more or less on its own'. So, for example, it is the proper function of the heart to pump; not to circulate blood around the body. According to Garson, "when we identify a trait's function with its distal effect, we're committing a fallacy of division. The function of circulating blood does not belong to the heart but to a bigger system that includes the heart among its parts" (p. 119, 2019).

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however Boorse vigorously rejects the selected effects theory of function and appeals instead to statistically typical contributions to goals (1976, 1977)

[6] Garson also contends that the Dysfunction Account cannot account for cases of developmental mismatch – that is, when a mechanism is performing its evolutionary function of yielding a certain phenotype given certain signals or circumstances, but that phenotype turns out to be disadvantageous to the organism at a later stage of ontogeny (due for example to a change in environment) (Garson, 2019; see also Matthewson & Griffiths, 2017). This case is equally problematic on the Traditional Theory, and I will restrict my scope here to objections that follow from the Generalised Theory specifically.

It follows from this that the trait T which has the proper function 'Substance Use Disorder' is the overall neural system which in fact performs this function. Unless some particular synapse is performing Substance Use Disorder all on its own, it would be fallacious to attribute to this single synapse this system-level effect. Rather, parts of this system have as their proper ontogenetic functions to contribute (howsoever they in fact contribute) to the overall effect – just as the heart has as its proper function to contribute to blood circulation by pumping.

C. From A and B, we should accept that Substance Use Disorder is functional and not dysfunctional.

D. Substance Use Disorder is a case of mental disorder.

Drug addiction, or Substance Use Disorder as it is termed in the DSM, is a commonly accepted disorder and features in official classifications, so we should *prima facie* accept that it is indeed a case of mental disorder.[7]

E. From C and D, we should accept that some disorders are functional and not dysfunctional.

F. The Dysfunction Account states that all disorders necessarily involve dysfunction.

G. From E and F, we should reject the Dysfunction Account.

7. Rebutting Garson's Critique

In what follows, I will show that Garson's premise B is false. I will then argue that premise B being false also leads us to reject premise A. In other words, Garson's critique of the Dysfunction Account fails, and this failure exposes an underlying flaw in the Generalised Theory.

7.1. We Should Reject Premise B

It follows from Garson's Generalised Theory that Substance Use Disorder is functional. So far, he is correct. However, it does *not* follow from Garson's Generalised Theory that Substance Use Disorder is *therefore* not dysfunctional. In concluding this, Garson is simply wrong. To unpack why, we need to know what it is in the brain that performs Substance Use Disorder.

Substance Use Disorder is neurobiologically complex. There are multiple, diverse and to some extent independent structures and circuits implicated in the production of the psychological states and behaviours involved in substance dependence. One neuropsychological theory is that substance abuse is caused and maintained by 'feedback loops' between the reward system, the anti-reward/stress system and the executive systems in the brain (Koob & Simon, 2009). This is supported by functional neuroimaging studies showing associations with neuroplastic changes in several neurological sub-systems of the

[7] I grant Garson this for present purposes, but there is considerable debate in the literature as to whether inclusion in official classification should in fact be taken as conclusive evidence that the case falls within the extension of the concept of disorder (see e.g. Schwartz, 2014; Lemoine, 2013; Matthewson and Griffiths, 2018; Cooper, 2020).

basal ganglia, the extended amygdala and the orbitofrontal/prefrontal cortex (Koob & Simon, 2009; Koob & Volkow, 2016).

As Garson acknowledges, it is not entirely clear from an empirical neuroscientific perspective precisely what role synapse selection plays in the neuroplastic changes associated with Substance Use Disorder. However, let us suppose that the relevant neural systems and sub-systems have indeed undergone dopamine-mediated synaptic selection such that, in accordance with Garson's analysis, these traits have acquired the overall ontogenetic function of performing Substance Use Disorder.

If so then, in accordance with the principle that proper functions are proximal, we can conclude as follows: a complex system of neural traits spearheaded by the basal ganglia, the extended amygdala and the orbitofrontal/prefrontal cortex has acquired the overall proper ontogenetic function of Substance Use Disorder. This system is the trait T which has Substance Use Disorder as its ontogenetic function. Its constituent parts have as their proper ontogenetic functions to contribute (howsoever they in fact contribute) to the yielding of this overall effect[8].

If so, then when constituent traits of this system – such as the basal ganglia or the orbitofrontal cortex – contribute to the overall system-level effect 'Substance Use Disorder', they are performing their ontogenetic function. This follows from the Generalised Theory, and thus far Garson is correct. It does *not* follow however from the Generalised Theory that these neural items and structures are *therefore* not dysfunctional. This is because neural traits have evolutionary functions too, also on Garson's view. Recall that Garson's Generalised Theory encompasses all of the evolutionary functions already covered by the Traditional Theory, in addition to Garson's novel ontogenetic functions. Accordingly, if Substance Use Disorder interferes with an evolutionarily selected effect of one of the implicated neural traits, then Substance Use Disorder is still a dysfunction on the Generalised Theory – whether or not Substance Use Disorder results from synapse selection. So, does contributing to Substance Use Disorder interfere with an evolutionary function of one or more of the implicated neural traits?

For the sake of simplicity, I will only consider the orbitofrontal cortex – a region of the prefrontal cortex located near the frontal lobes – in what follows. However, in principle, one could run the same argument with the amygdalae, the basal ganglia or the prefrontal cortex as a whole.[9]

The precise evolutionary function of the orbitofrontal cortex is naturally a matter of some scientific controversy, but one influential theory, strongly supported by lesion studies, is that the orbitofrontal cortex has an important control function in certain types of decision-making processes. More specifically, the orbitofrontal cortex is hypothesised as playing a role in the suppression of inappropriate actions and impulses (Bechara & Van Der Linded, 2005; Ouellet et al., 2015). As such, if Substance Use Disorder interferes with evolved decision-making capacities, then Substance Use Disorder still constitutes a dysfunction on Garson's view.

The notion that substance dependence adversely affects 'normal' human impulse control and response inhabitation is not only intuitive, it is borne out by the empirical evidence. Psychological research has shown associations between substance dependence and impairments in decision-making processes (Grant et al, 2000; Zhang et al, 2011). Indeed, this finding forms part of the evidence base for neuroscientific theorising around Substance Use Disorder and the role of the orbitofrontal cortex (Bolla et al., 2003; Torregrossa et al., 2008).

[8] Assuming, as we do, that they have undergone relevant selection.

[9] Assuming that their contribution to Substance Use Disorder likewise infringes on their evolutionary functions.

So it would seem that Substance Use Disorder indeed interferes with an evolutionary function of the orbitofrontal cortex.

Accordingly, this is what follows from the Generalised Theory: contributing to Substance Use Disorder (the effect of interest, E) is *both* a function *and* a dysfunction of the orbitofrontal cortex (the trait, T). This outcome is insufficient to reject the hypothesis that disorder entails dysfunction, as the Dysfunction Account states. Premise B is false, and the Dysfunction Account stands.

7.2. We Should Reject Premise A

As established in the previous section of this paper, following Garson's Generalised Theory, causing Substance Use Disorder (E) is both a function and a dysfunction of the orbitofrontal cortex (T).

In other words,

T is doing E
E is a function of T
E is a dysfunction of T

So Garson's Generalised Theory of function appears to imply that some single effect E of some single trait T can simultaneously be both a case of function and a case of dysfunction – in precisely the same sense.

Note first that this contradictory implication has no analogue on the Traditional Theory. The problem here is not that some single trait T can have more than one biological function. The enlarged claw of the male fiddler crab has both a sexually selected aesthetic signalling function and a function as a weapon in confrontation with other males (Dennenmoser & Christy, 2013), with both functions being the result of genetic selection. Of course, the claw of some particular crab may be an effective signal, being large and ominous looking, without having any efficacy in a fight. In case such as this, some single trait T can simultaneously be both functional and dysfunctional even on the Traditional Theory of function. But in the case of the fiddler crab, there are two selected effects in play (E1, E2) – one which the trait *is* performing, and another which the trait *is not*. In the case of Substance Use Disorder on the Generalised Theory, there is a single effect E (Substance abuse Disorder) which *is* being performed, and which is both a function and a dysfunction. And this simply cannot occur on the Traditional Theory[10].

These kinds of contradictory function/dysfunction attributions are only possibly because Garson's Generalised Theory yields conflicting functional norms. Conflicting functional norms arise because Garson is forcing two distinct sources of functional normativity to operate theoretically as one. It is a good indicator that ontogenetic and evolutionary functions are in fact different in kind and should not be yoked together for theoretical purposes.

By way of analogy, consider how an effect E may violate a social or societal norm whilst fulfilling a biological one. Teenage pregnancy, for example, is perfectly functional and normative from an evolutionary perspective, yet perhaps not ideal from a societal one. Because we acknowledge that there are two distinct sources of normativity at play in the case

[10] I shall refrain from arguing this point in any depth here, but on Traditional Theory some single effect E of some single trait T either is an effect which conferred fitness advantage on its bearers in the environment of evolutionary adaptedness, or it isn't. That is, if effect E of trait T either provided a net benefit to the inclusive fitness of organisms possessing trait T in recent evolutionary history, or it did not. If it did, then it is a function, and if it did not, then it isn't.

of teenage pregnancy – social norms and biological norms – we can coherently agree that teenage pregnancy is functional (in a biological sense) and dysfunctional (in a social sense) without any conflict. However, if we insisted that social and evolutionary norms were the same kind of thing and must operate theoretically as one, then our theory would yield seemingly inexplicable contradictions – just as the Generalised Theory does in the case of Substance Use Disorder.

As I shall argue from here, given Garson's relatively weak argument for accepting the Generalised Theory in the first place, the possibility of conflicting functional norms and contradictory function/dysfunction attributions is sufficient grounds to reject the Generalised Theory outright.

Recall Garson's argument for the Generalised Theory. We must accept it because parity of reasoning demands it:

> Since [the Generalised Theory] solves all the same problems, *minus an arbitrary distinction*, we should accept it.
>
> (Garson, 2019, p. 94, emphasis mine)

Elsewhere:

> My main argument for the [the Generalised Theory] is that is solves all the puzzles of function, without *pointless restrictions*.
>
> (Garson, 2019, p. 101, emphasis mine)

Garson maintains that his argument for the Generalised Theory hinges on parity of reasoning, but, in fact, he is leaning extremely heavily on this unargued, unsupported contention that distinguishing evolutionary and ontogenetic functions theoretically would be 'arbitrary', 'unprincipled' and/or 'pointless'.

Garson's flippant dismissal of a more restricted domain as 'arbitrary' covers up a weakness in his argument. Parity of reasoning does not establish that evolutionary and ontogenetic functions must be theorised of and treated as one. In order to establish this, Garson would have to show that that the distinction between evolutionary and ontogenetic functions is in fact, as he maintains, pointless and unprincipled. It is unclear how he could defend this however and moreover what would be motivate it. What does a Generalised Theory do that a specific one cannot?

Garson fails to seriously consider that we may simply have good, independent grounds to favour a restricted, 'un-generalised' theory of biological function. Parity of reasoning aside, we may have a non-arbitrary rationale for thinking that evolutionary and ontogenetic functions are to some extent distinct and should not be forced to operate theoretically as one.

And of course, we have precisely such a rationale. Garson's Generalised Theory of yields conflicting functional norms and seemingly contradictory function/dysfunction attributions. This is theoretically undesirable and good *prima facie* reason to think that evolutionary and ontogenetic functions are different in kind and should be distinguished theoretically.

To further substantiate this, it is instructive to consider that Garson is not entirely opposed to principled theoretical distinctions among proper functions. Garson readily rejects the prospects of a generalised theory across artefact and biological functions on the following grounds:

> I don't think of artifact functions and biological functions as two species of the same genus, like lions and tigers are two species of *panthera* … artifact and biological functions are different sorts of things…
>
> (p. 30, Garson, 2019)

What does this tell us?

Well, it seems that, per Garson, if one has some principled reason to distinguish between two sorts of function – such as reason to think that they are different in kind or do not play the same theoretical role – then one is justified in rejecting generality. And, as established, we do. So, by Garson's own standards, we are justified in rejecting a Generalised Theory of biological function. Garson must accept this, to use his own words, "on pain of inconsistency" (Garson, p. 93, 2019.

In conclusion, the Generalised Theory yields theoretical contradictions. They are however easily resolved by simply abandoning generality about evolutionary and ontogenetic functions:

> T is doing E
> E is a (direct proper) *ontogenetic* function of T, and
> E is a (direct proper) *evolutionary* dysfunction of T

Making such a distinction is principled, reasoned and non-arbitrary, and Garson fails to demonstrate otherwise. Garson's argument for the Generalised Theory is undermotivated and we should reject premise A.

8. What remains?

I have argued that Justin Garson's critique of the Dysfunction Account of Disorder from his Generalised Selected Effect Theory of biological functions is unsuccessful. Firstly, Garson does not show that Substance Use Disorder is functional and *therefore* not dysfunctional. At best, he shows that it is both, which is insufficient to reject the thesis that disorder entails dysfunction. Secondly, as revealed by the case of Substance Use Disorder, the Generalised Theory yields contradictions. This is sufficient grounds to reject the Generalised Theory of function outright.

What remains for Garson? I shall close by suggesting that abandoning a *generalised* theory of evolutionary and ontogenetic functions may bring benefits for Garson's own philosophical objectives. Conflicting functional norms are not the only problem plaguing the Generalised Theory, and it may be that some tricky conceptual stumbling blocks could be circumvented by simply abandoning generality.[11] Doing this could also help facilitate the development of a more thorough, in-depth and specific theory of ontogenetic neural selection in the brain, drawing on Garson's many existing insights and contributions in this area of philosophy and neuroscience. This would no doubt be a valuable contribution to the literature

[11] Because of generality, Garson has to give an analysis of 'population' which is broad enough to apply both to retained synapses in the brain and reproducing organisms (see Garson, 2019). He attempts this by requiring a high degree of fitness relevant interactions among members which, concerningly, would seem to make members of distinct species which interact through predator prey relations (in some eco-system) a paradigmatic biological population. If he gave up on providing a general account, he could simply employ the notion of population which best fit each case.

in and of itself, more so, perhaps, than a Generalised Selected Effects Theory of biological function.

References

Bolla, K. I., Eldreth, D. A., London, E. D., Kiehl, K. A., Mouratidis, M., Contoreggi, C. E. E. A., ... & Funderburk, F. R. (2003). Orbitofrontal cortex dysfunction in abstinent cocaine abusers performing a decision-making task. *Neuroimage*, *19*(3), 1085-1094.

Boorse, C. (1977). Health as a theoretical concept. *Philosophy of science*, 44(4), 542-573.

Boorse, C. (1976). Wright on functions. *The Philosophical Review*, *85*(1), 70-86.

Cummins, R. (1975). Functional Analysis. *The Journal of Philosophy, 72*(20), 741-765. doi:10.2307/2024640

Dennenmoser, S., & Christy, J. H. (2013). The design of a beautiful weapon: compensation for opposing sexual selection on a trait with two functions. *Evolution: International Journal of Organic Evolution*, *67*(4), 1181-1188.

Garson, J. (2019). *What biological functions are and why they matter*. Cambridge University Press.

Garson, J., & Papineau, D. (2019). Teleosemantics, selection and novel contents. *Biology & Philosophy*, *34*(3), 36.

Garson, J. (2018). How to be a function pluralist. *The British Journal for the Philosophy of Science*, 69(4), 1101-1122.

Godfrey-Smith, P. (1994). A modern history theory of functions. *Noûs*, *28*(3), 344-362.

Godfrey-Smith, P. (1993). Functions: Consensus without Unity. *Pacific Philosophical Quarterly*, 196–208.

Grant, S., Contoreggi, C., & London, E. D. (2000). Drug abusers show impaired performance in a laboratory test of decision making. *Neuropsychologia*, *38*(8), 1180-1187.

Griffiths, P. E., & Matthewson, J. (2018). Evolution, dysfunction, and disease: A reappraisal. *The British Journal for the Philosophy of Science*, *69*(2), 301-327.

Matthewson, J., & Griffiths, P. E. (2017). Biological criteria of disease: Four ways of going wrong. *Journal of Medicine and Philosophy*, 42(4), 447-466.

Koob, G. F., & Simon, E. J. (2009). The neurobiology of addiction: where we have been and where we are going. *Journal of Drug Issues*, *39*(1), 115-132.

Koob, G. F., & Volkow, N. D. (2016). Neurobiology of addiction: a neurocircuitry analysis. *The Lancet Psychiatry*, *3*(8), 760-773.

Lemoine, M. (2013). Defining disease beyond conceptual analysis: An analysis of conceptual analysis in philosophy of medicine. *Theoretical Medicine and Bioethics*, *34*(4), 309-325.

Millikan, R. G. (1989). In defense of proper functions. *Philosophy of Science*, *56*(2), 288-302.

Neander, K. (1991). Functions as selected effects: The conceptual analyst's defense. *Philosophy of Sscience*, *58*(2), 168-184.

Neander, K. (1995). Misrepresenting & malfunctioning. *Philosophical Studies*, *79*(2), 109-141.

Neander, K. L. (1983). *Abnormal Psychobiology: A Thesis on the 'anti-psychiatry Debate' and the Relationship Between Psychology and Biology* (Doctoral dissertation, La Trobe University).

Neander, K. (1998). Mental illness, concept of. *Routledge Encyclopaedia of Philosophy*.

Schwartz, P. H. (2014). Reframing the disease debate and defending the biostatistical theory. *Journal of Medicine and Philosophy*, *39*(6), 572-589.

Torregrossa, M. M., Quinn, J. J., & Taylor, J. R. (2008). Impulsivity, compulsivity, and habit: the role of orbitofrontal cortex revisited. *Biological Psychiatry*, *63*(3), 253-255.

Wakefield, J. C. (1992). The concept of mental disorder: on the boundary between biological facts and social values. *American Psychologist*, *47*(3), 373.

Wakefield, J. C. (2014). The biostatistical theory versus the harmful dysfunction analysis, part 1: is part-dysfunction a sufficient condition for medical disorder? *The Journal of Medicine and Philosophy: A Forum for Bioethics and Philosophy of Medicine* (Vol. 39, No. 6, pp. 648-682). Journal of Medicine and Philosophy Inc.

Zhang, X. L., Shi, J., Zhao, L. Y., Sun, L. L., Wang, J., Wang, G. B., ... & Lu, L. (2011). Effects of stress on decision-making deficits in formerly heroin-dependent patients after different durations of abstinence. *American Journal of Psychiatry*, *168*(6), 610-616.