

Garson, J. (2023) "Brain Disorders, Dysfunctions, and Natural Selection: Commentary on Jefferson," *Philosophical Psychology*. [This paper is part of a symposium on Anneli Jefferson's (2002) book, *Are Mental Disorders Brain Disorders?* (London: Routledge).]

Title: Brain Disorders, Dysfunctions, and Natural Selection: Commentary on Jefferson

Author: Justin Garson, Department of Philosophy, Hunter College and The Graduate Center

Abstract: I argue that despite the merits of Jefferson's account of a brain disorder, which are many, the notion of function she deploys is unsuitable to the overall goals of that account. In particular, Jefferson accepts Cummins' causal role theory of function and dysfunction. As the causal role view, in its standard elaborations, is wedded to human interests, goals, and values, it cannot serve as a value-neutral anchor for her hybrid "harm-dysfunction" account of disorder. I argue that the selected effects theory, or some comparably value-neutral account, would serve her purposes better.

Acknowledgements: I'm grateful to two anonymous reviewers for their helpful comments. I'm also grateful for the financial support of the John Templeton Foundation (#62220). The opinions expressed in this paper are those of the author and not those of the John Templeton Foundation.

What is a brain disorder? This is an extremely important topic in psychiatry (and its philosophy), the mental health professions more generally, and mental health service users. That's because, if we wish to say that a certain distressing or disturbing mental phenomenon stems from a disorder (dysfunction, impairment, pathology, defect, etc.) of the brain, or just *is* such a disorder, we must know what brain disorders are. How would one decide, for example, whether the brain of a person with depression, or delusions, or ADHD, or autism, is dysfunctional, or exhibiting normal variation, or is actually functioning exactly as it's supposed to? Different judgments along these lines have different impacts on research, treatment, and stigma. As such, we have a profound ethical obligation to think about them in a serious way.

In Jefferson's (2022) view, *one way* to have a brain disorder is to have a certain (harmful) type of brain state that realizes a psychological dysfunction (39). An important consequence of her view is that it may be impossible to decide, of a given brain state, whether it is or is not a disorder, simply by poking and prodding at the brain enough (9). You might also have to know what psychological state it realizes. For example, she asks us to suppose that we were to discover "differences in the dopamine system in addiction that underlie the cravings for the drug...that we have characterized as dysfunctional psychological processes (40)," and that such dopamine differences can be correctly said to realize those cravings (on some plausible account of the realization relation). Then that brain difference would also constitute a brain dysfunction. On the assumption that it is also harmful, it would constitute a disorder. I think this is an important innovation, and I'm inclined to accept it.

My main concern with her otherwise masterful treatment of the topic is that the notion of dysfunction she deploys is mismatched to her purposes, and as such, it cannot do what she wants it to do: help us make principled decisions about difficult cases, and prevent the overreach of psychiatry into normal (that is non-disordered) forms of thinking, feeling, and acting. I also think

that historical accounts of function, most notably the selected effects theory, can do exactly what she would like the theory to do, and it does not have the shortcomings that often motivate people to reject it.

Jefferson's account starts on relatively firm and well-explored grounds. She adopts what is known as a "hybrid" view of disorder (4). A traditional debate in the philosophy of medicine pits "naturalists" against "normativists." Though people define these terms somewhat differently, here is one standard gloss. "Naturalists" think the notion of disease (pathology, disorder, etc.) can be understood entirely in value-free terms, like "aluminum." Boorse (1975), for example, defines "disease" in terms of the inability of a bodily part or process to render its species-typical contribution to survival or reproduction. In his view, whether a condition counts as a disease doesn't depend, constitutively, on how we feel about it. "Normativists," in contrast, argue that disease terms are essentially value-laden. One approach holds that the term "disease" functions a bit like the term "pest" or "weed:" it gathers together a number of disparate conditions that are joined by the fact that we don't like them. A rather extreme normativist view would be one that holds that disease terms do little more than convey such negative value-judgements. Consider psychologist Peter Sedgwick's (1981, 121) proclamation that "outside the significances that man voluntarily attaches to certain conditions, *there are no illnesses or diseases in nature.*"

In contrast to naturalist and normativist views, "hybrid" views occupy the apparently reasonable middle ground: the concept of disease combines both facts and values. The most well-known and influential normativist view is Wakefield's (1992) harmful dysfunction account. In this view, for something to be a disorder, it must be harmful (as assessed by the predominant values of the society within which the attribution is made) and it must be dysfunctional (that is, it represents a failure of a part or process to carry out its natural function, where the notion of function is understood in objective, value-neutral terms). Jefferson also accepts a hybrid view, in which disorder involves both dysfunction and harmfulness (4), though she does not accept Wakefield's particular construal of function.

The hybrid view has obvious benefits. It explains why people do not consider certain socially-disvalued conditions to be disorders (such as unwanted pregnancy, being unemployed, being a racist, having ordinary jealousy or grief, the pain of teething or labor, minor cosmetic blemishes, having a short temper, being an underachiever, and so on). It also recognizes the ineliminable role of value judgments in medicine: plausibly, syndactyly, or idiopathic guttate hypomelanosis, aren't disorders or diseases, since, even though something might be "going wrong" in a manner that warrants the dysfunction label, they're not sufficiently harmful. When the harmful dysfunction analysis is wedded to a plausible and naturalistic conception of function such as the selected effects theory, it can do exactly what we want such a theory to do: it provides concrete guidance on difficult cases and prevents the overpathologization of non-disordered conditions.

One can accept a hybrid, harm-dysfunction account of disorder without accepting a specific theory of function like the selected effects theory. Presumably, one might accept a harm-dysfunction view and also accept, say, the organizational account of function (Mossio et al. 2009). But there's an important and somewhat subtle proviso here. A hybrid view, as such, will impose certain constraints on the "function" part of the harm-dysfunction formula. The hybrid view only has the benefits enumerated above if the "dysfunction" part of the harm-dysfunction

equation isn't, itself, just another expression of socially-widespread values and preferences. Whether a trait has a function, and what function it has, mustn't depend on our wants, our likes and dislikes, and so on. There should be a value-free "fact of the matter" about what functions are. Failing that, we would be led to the absurd result that the "dysfunction" clause doesn't actually amplify the "harm" clause, but simply restates it in a somewhat roundabout way. It would be redundant – it would effectively be a harm-harm theory of disorder. It would also be misleading, since it would take a value judgment about harm and frame it in the objective-sounding terminology of function. My point isn't to insist that one *must* accept the hybrid view. My point is that *if* one embraces the hybrid view, for the sorts of reasons that typically lead people to embrace it, then one shouldn't also make the question of what function something has, or whether it's performing its function correctly, depend on values.

But this would appear to be precisely the move that Jefferson makes. She seems to think the ideas of function and dysfunction are inherently value-laden. There are two places in the text that lead me to this conclusion. First, she writes, "both the notion of disorder and, to a lesser extent, that of dysfunction presuppose that there is something wrong with the agent. Unless one thinks that we can get the 'something wrong' out of evolutionary history, we will need to locate it in the fact that a condition is harmful to the person who has it" (4). That seems to suggest that the "dysfunction" part of the harmful dysfunction equation is actually a shorthand for "harm," which would make it redundant and misleading. True, her statement is a hypothetical one. She does not say that we *cannot* get the "something wrong" out of evolutionary history. (I happen to think we can and must.) But neither does she denounce this apparently inconsistent set of commitments. Moreover, given her view that "the disorder label performs a useful function for some people suffering from mental distress, by signalling that there is something objectively wrong with them and they are not just 'acting up,'" (14), it seems that she is equally wedded to the view that the notion of dysfunction should be anchored appropriately in the objective facts of the situation and not merely our collective values.

I have a second reason for thinking she embraces a value-laden notion of function. She writes, "I take the broadest possible line here in order to avoid turf wars in philosophy of medicine and biology. According to Cummins (1975) we can define functions as contributions of a constituent part to the activity of a system it is embedded in" (4). Cummins' theory of function holds that the function of a trait is, roughly, some interesting contribution it makes to a system-level capacity, where what's "interesting" depends on the goals and interests of the people doing the ascribing. The function of the heart is to pump because that's how it contributes to an interesting system capacity, namely, the capacity of the circulatory system to move blood around and get oxygen to our cells. Her motive for taking this stance, as the passage indicates, seems to be that Cummins' theory is the most general. Nearly all theories of function hold that a trait's function has something to do with its contribution to a larger system; they just disagree about the nature of that contribution. (To be strict, selected effects functions – in which the function of a trait depends on what it was selected for by natural selection or some comparable selection process – are not, logically speaking, a subtype of Cummins-functions, since a trait can have a selected-effects function without having the corresponding Cummins-function. Cummins-functions are dispositions: for a trait type, like the human eye, to have the Cummins-function of seeing, it must be the case that at least some human eyes are actually capable of seeing [Cummins 1975, 758]. This is not the case for selected effects functions. If a global pandemic rendered everyone

incapable of seeing, the eyes would, at least for a time, have the selected effects function of seeing, since that's what they were designed to do. But as a rule, traits with selected effects functions will also have Cummins-functions, so we can take Jefferson's point as given.)

I see two problems with this line of thought. First, generality isn't, in and of itself, a virtue. If it were, then we should conclude that the "true belief" theory of knowledge, in which knowledge is just true belief, is the best theory of knowledge, or at least a highly plausible one. After all, everyone seems to agree that knowledge requires true beliefs; they just disagree about what exactly the mysterious third ingredient is.

The second, and more serious, problem, is that Cummins-functions are relative to our interests, goals, and values. That means that, for Cummins, whether something has a function, and which function it has, depends partly on how we think about it and what we care about, as I will shortly show. That, in turn, would undermine the point of a hybrid view.

As I noted, Cummins thinks, roughly, that the function of a part of a system consists in its contribution to some interesting capacity of a larger system, where what's interesting depends on our goals, interests, or values. The heart, by pumping, contributes to the body's ability to circulate blood, *and* it contributes to the body's ability to make sounds that one can listen to through a stethoscope. From a purely objective point of view, both could equally be called "functions" of the heart. But we often describe only the first as a "function," and the second as a "side effect." Why? According to Cummins and other causal role theorists, it's because blood circulation is the capacity we're usually interested in. Outside of our interests, neither has greater claim to be the heart's "function" than the other (1975, 762).

This reliance on the goals, values, and interests is even clearer when it comes to dysfunction or malfunction. In addition to pumping, hearts sometimes seize up (cardiac arrest). This activity contributes to the body's capacity to have strokes. But we generally think of cardiac arrest as a malfunction or dysfunction, rather than one among many of the heart's functions. Why? In the nature of things, according to Cummins' view, pumping has no greater claim to be a function than seizing up. Commentators who've pondered the matter in a serious way seem to agree it has something or other to do with our values (Craver 2001, 72; Hardcastle 2002, 153). Put crudely, we don't *like it* when hearts seize up and cause brain damage, so we consider it a "malfunction" rather than a "function." Other theories of function, such as the modal theory (Nanay 2010), also end up averting to the interests and goals of the one doing the ascribing in order to whittle down the plurality of function ascriptions they'd otherwise license.

I don't think Cummins is strictly wrong. Many philosophers of biology are pluralists about function (e.g., Neander 2017; Sterner and Cusimoto 2019). By "pluralism," I mean the rather unremarkable doctrine that biologists and other scientists use the term "function" to mean slightly different things. It's likely that Cummins captured at least one strand of biological usage. Questions about the correctness or incorrectness of invoking Cummins-functions boil down, then, to whether they're suitable or unsuitable for various theoretical tasks. I submit that Cummins-functions are unsuitable for the role that Jefferson demands of them, namely, to account for the "dysfunction" side of a hybrid harm-dysfunction account of disorder.

If Cummins' theory is unsuitable for the task, which theory of function might fit the bill? My own preference is the selected effects theory of function (Garson 2019; forthcoming). As noted above, on this view, a trait's function is, roughly, what it was selected for, by natural selection or some comparable process of selection. The function of the heart is to pump blood because that's what it was selected for, by evolutionary natural selection. The function of my pet rat's standing on his hind legs is to get a piece of celery, because that is what the behavior was "selected for" – in this case by a natural process of trial-and-error. It's impossible to enumerate, here, all the virtues of the theory or how it avoids the charges that are often leveled against it, though I will sketch some of the key virtues below. My point is that it's difficult to see how Jefferson's theory gets off the ground unless she accepts it, or a view with a similarly naturalistic character.

To see how the adoption of something like the selected effects theory would impact her view, it's useful to consider how a selected-effects theorist and a causal-role theorist might tackle the problem of whether or not a specific psychological condition is, or involves, a disorder. It would be particularly useful to apply them to a somewhat controversial category, ADHD, so we can see how they resolve actual diagnostic controversies. Sometimes scientists disagree about whether ADHD stems from a brain disorder, or whether it's just part of our species' natural variation in cognition, on a par with, say, being left-handed, or whether it's a designed feature (see Swainpoel et al. 2017). This is not an idle dispute. It's bound up in complex ways with research, treatment, and stigma. As Jefferson (2022, 74) points out, evidence is accumulating that the notion that mental disorders stem from brain dysfunctions actually promotes certain forms of stigma, rather than alleviates them. Moreover, I suspect our willingness to medicate ADHD is connected in a complex way with whether we think it represents a brain disorder or ordinary variation in cognition. This is not an all-or-nothing claim. One might think ADHD is a brain disorder but choose to forego medication. One might also think it's part of normal variation and choose to medicate it. But it seems to me that if one really thinks that ADHD represents normal cognitive variation that happens to be "mismatched" to our overly rigid educational systems (among other things), then a very natural line of thought is that we ought to devote more efforts to changing those systems (Wakefield 2021, 356; Taylor and Vestergaard 2022, 13-14). The larger point is that seeing something as a brain disorder tends to motivate certain kinds of interventions, and seeing it not as a brain disorder but part of normal cognitive variation tends to motivate other interventions.

Now suppose, as a thought experiment, scientists find a distinctive type of brain state underlying ADHD – call it "S." Suppose there were such a tight correlation between the outward signs of ADHD, and S, that psychiatrists began using S as a biomarker for ADHD. Instead of having kids do complicated cognitive tests as part of diagnosis, the doctor just runs an fMRI and looks for S. Suppose, furthermore, that variations in the *S-ness* of the brain correlate perfectly with variations in the degree of various outward symptoms of ADHD. How would we decide whether ADHD is a disorder? Well, S would have to be dysfunctional. What would have to be the case for S to be dysfunctional? In Jefferson's view, that would depend on whether the symptoms of ADHD are *psychologically* dysfunctional. And what would have to be the case for that to be true?

If function and dysfunction depend partly on human goals, values, and interests, then it would seem that whether or not ADHD is psychologically dysfunctional would rest either on the interests and values that the researchers who study ADHD have, or the interests and values of the

broader society. Arguably, in most contexts, S prevents the brain from contributing to various high-level cognitive abilities that some countries, like the United States, care very deeply about, such as the ability to sit still and pay attention to a teacher for several hours a day. Would that be enough to make it a dysfunction? If so, we're back to the problem that pretty much anything could be a disorder if society finds it sufficiently inconvenient, distressing, or unsettling. It would effectively be a version of the harm-harm theory of disorder and would fail to satisfy the core motives for a harm-dysfunction account of disorder. Perhaps Jefferson would argue that in this case, some other criterion is more relevant for judging its functional status. It's not clear to me how one would choose that criterion.

In contrast, how would that same decision process go on the selected effects theory? You would ask whether there is any evidence at all that our brains, or at least some people's brains, might have been designed for high levels of distractibility, for fluid and shifting attention, and for impulsiveness, whether they're neutral with respect to design (like handedness) or whether these qualities are best construed as a failure of design (like bronchitis). How would you figure that out? Aren't evolutionary theories about the mind merely a bunch of "just-so stories" that can never be confirmed or disconfirmed?

It turns out that there are quite interesting and numerous lines of evidence that ADHD traits are designed features, rather than dysfunctions. I'll list four that, together, provide a strong prima facie case that ADHD, or some of its traits, represents an evolved cognitive style. First, about 15 years ago, the anthropologist Dan Eisenberg and colleagues (2008) studied a largely nomadic group in northern Kenya, the Ariaal. Most Ariaal are nomadic as they need to lead their livestock from place to place. Some have settled into towns and rely on agriculture and the market economy. Eisenberg screened his subjects for a gene associated with ADHD, an allele of the DRD4 gene on chromosome 11 (see Qian et al. 2018). He found that nomadic Ariaal who had ADHD traits were actually healthier and better fed than those who didn't have such traits, as judged by body mass index. But the reverse was true among the sedentary Ariaal: those who had ADHD traits were actually worse off than their non-ADHD peers. That suggests that in environments that reward a certain level of exploration, novelty-seeking, hypervigilance, and movement, ADHD traits can actually be a real boon. Eisenberg's research seems to support the "mismatch" approach.

A second line of evidence for the claim that ADHD is an evolved cognitive style is that ADHD traits are disproportionately represented in populations with long migration histories (Chen et al. 1999; Matthews and Butler 2011). Our conventional understanding of human evolution is that modern humans evolved in Africa around 200,000 years ago and that many began migrating across the globe around 50,000 years ago. Those groups that have a long migration history have a higher incidence of ADHD traits than those who don't. (So, for example, there is a higher frequency of ADHD traits, as assessed by the frequency of the long allele of the DRD4 gene, in South American indigenous communities today, than in East Asian communities.) Perhaps increased exploratory behavior and novelty-seeking conferred an advantage on early migrants.

A third line of evidence in support of this picture comes from neurocognitive studies of people with ADHD. The philosopher and neuroscientist David Barack and collaborators (2022) have gathered preliminary evidence that people with ADHD traits tend to excel at a certain

computerized game that's meant to simulate a foraging environment. In the game, there are various "patches" and the player must decide how long to spend at each one before moving on to another. By spending less time at each patch, they gain a greater reward.

Finally, anthropological data fits together fairly well with what some educators have found quite independently, that when kids with ADHD in the classroom are given more opportunities for movement, and given more opportunities to direct their own education, they tend to not need medication.¹ Presumably that is because they are given environments that more closely resemble the sorts of environments that ADHD traits evolved in.

So, while there's some speculative elements here, and the emerging evolutionary picture might turn out to be wrong, the selected effects theory gives us a rigorous framework for thinking about function and dysfunction and it points us in the direction of what kind of evidence we'd need to gather to make warranted claims about function and dysfunction. The case of ADHD also helps to debunk the popular claim – I should add, *not* a claim that Jefferson makes – that evolutionary approaches to the mind are merely a bunch of "just-so stories," or worse, post-hoc rationalizations of the social status quo. Thirty years ago, that charge might have seemed vaguely plausible, if uncharitable. Today, it would simply be naïve. Evolutionary hypotheses about the mind are often supported through evidence such as cross-cultural studies, archaeology, neuroscience, developmental psychology, genetics, and so on (e.g., Barrett 2015; Garson 2022, Chapter 4). In fact, given that evolutionary claims about physical and psychological traits often rely on the exact same *sorts* of evidential bases, it would be difficult to justify the claim that evolutionary hypotheses about physical traits can be evidentially warranted and evolutionary hypotheses about psychological traits cannot – unless we're arbitrarily and inappropriately ratcheting up the standard of evidence for the latter.

Another possible objection to the selected effects theory of function is that it's tacitly value-laden (Radden 2019; also see Powell and Scarffe 2019 for discussion). One way to run the argument would be as follows: the only reason philosophers of biology define "function" in terms of past contributions to survival and reproduction is that we happen to value surviving and reproducing, and natural selection tends to promote traits that help us survive and reproduce. So, they conclude, functions must have something to do with natural selection. In this view, the selected effects theory simply conceals our value judgments under a thin veneer of objectivity. If we had a completely different value system – say, we loathed surviving and breeding – then we'd have a different account of functions entirely.

If that were true, then I'd agree we should abandon the selected effects theory. It's far better to be overt and explicit about what our values are than to hide them under the guise of scientific objectivity. But that's not the justification for the selected effects theory. The justification for the theory is that *selected effects* are the only kinds of thing we happen to know of in the world that can satisfy all of the core theoretical demands that scientists and lay folks impose on the notion of function. (By the same token, the thirteenth element of the periodic table is the only kind of thing in the world that happens to satisfy all of the demands that modern science imposes on the notion of aluminum.)

¹ See <https://www.psychologytoday.com/us/blog/freedom-learn/201009/experiences-adhd-labeled-kids-who-leave-typical-schooling>, accessed May 5, 2023.

What exactly are those demands? There are at least three. First, we distinguish between functions and accidents (lucky benefits, incidental side effects, and so on). The function of the heart is to beat, not to make sounds, even though it does both and even though both can be helpful to us. Second, we distinguish between function and malfunction (or dysfunction). Beating is functional; cardiac arrest is malfunctional. Third, at least in much of ordinary biological usage, functions are meant to be explanatory. In particular, the effect of a trait is cited in an explanation of the existence of that very trait: for example, in the ordinary biological context, to say that the function of zebra stripes is to deter biting flies is to say, among other things, that zebras have stripes, in part, because stripes deter flies (Garson 2019; forthcoming). If functions are selected effects, they can fulfill all of these core theoretical demands. If functions aren't selected effects, it's unclear how they would fulfill those demands.

In sum, I agree with Jefferson's core thesis – minus the dysfunction part of her view. The concept of a brain disorder, or disorder more generally, is best construed in a hybrid manner that respects a role for both facts and values. The only remaining issue is to develop an account of function that can do the sort of thing that psychiatrists and other mental health professionals seem to want it to do. Of course, it may turn out, on the construal of function advanced here, that some of the things doctors *call* brain disorders aren't *really* brain disorders. It could also turn out that some of the things we don't think of as brain disorders actually are. But to the extent that psychiatry purports to be an actual branch of medicine, rather than an enforcer of widely-shared social norms, then we need to be prepared for such surprises.

References

Barack, D. L., Ludwig, V. U., Parodi, F., Ahmed, N., Brannon, E. M., Ramakrishnan, A., and Platt, M. (2022, September 29). Attention deficits linked with proclivity to explore while foraging. <https://doi.org/10.31234/osf.io/nyvjq>

Barrett, H. C. 2015. *The Shape of Thought: How Mental Adaptations Evolve*. Oxford: Oxford University Press.

Boorse, C. 1975. On the distinction between disease and illness. *Philosophy and Public Affairs* 5: 49-68.

Chen, C., Burton, M., Greenberger, E., and Dmitrieva, J. 1999. Population migration and the variation of dopamine D4 receptor (DRD4) allele frequencies across the globe. *Evolution and Human Behavior* 20: 309–324.

Craver, C. 2001. Role functions, mechanisms, and hierarchy. *Philosophy of Science* 68: 53–74.

Eisenberg, D. T. A., Campbell, B., Gray, P. B., and Sorenson, M. D. 2008. Dopamine receptor genetic polymorphisms and body composition in undernourished pastoralists: An exploration of nutrition indices among nomadic and recently settled Ariaal men of northern Kenya. *BMC Evolutionary Biology* 8:173.

Garson, J. Forthcoming. What are functions good for? *Australasian Philosophical Review*.

Garson, J. 2022. *The Biological Mind: A Philosophical Introduction: Second Edition*. London: Routledge.

Garson, J. 2019. *What Biological Functions Are and Why They Matter*. Cambridge: University of Cambridge Press.

Hardcastle, V.G. 2002. On the normativity of functions. In *Functions: New Essays in the Philosophy of Psychology and Biology*, ed. A. Ariew, R. Cummins, and M Perlman, 144-156. Oxford: Oxford University Press.

Jefferson, A. 2022. *Are Mental Disorders Brain Disorders?* London: Routledge.

Matthews, L., and Butler, P. 2011. Novelty-seeking DRD4 polymorphisms are associated with human migration distance out-of-Africa after controlling for neutral population gene structure. *American Journal of Physical Anthropology* 145(3): 382-89.

Mossio, M., Saborido, C., and Moreno, A. 2009. An organizational account for biological functions. *British Journal for the Philosophy of Science* 60: 813–841.

Nanay, B. 2010. A modal theory of function. *Journal of Philosophy* 107: 412-431.

Neander, K. 2017. Functional analysis and the species design. *Synthese* 194: 1147-1168.

Powell, R., and Scarffe, E. 2019. Rehabilitating “disease:” Function, value, and objectivity in medicine. *Philosophy of Science* 86: 1168-1178.

Qian, A., Tao, J., Wang, X., Liu, H., Ji, L., Yang, C., Ye, Q., Chen, C., Li, J., Cheng, J., Wang, M., Zhao, K. 2018. Effects of the 2-repeat allele of the DRD4 gene on neural networks associated with the prefrontal cortex in children with ADHD. *Frontiers in Human Neuroscience* 12: Article 279.

Radden, J. 2019. Mental disorder (illness). In *The Stanford Encyclopedia of Philosophy* (Winter 2019 ed.), edited by E. N. Zalta, <https://plato.stanford.edu/entries/mental-disorder/> (accessed May 5, 2023).

Sedgwick, P. 1981. Illness – mental and otherwise. In *Concepts of Health and Disease: Interdisciplinary Perspectives*, ed. A. L. Caplan, H. T. Engelhardt, Jr., and J. J. McCartney, 119-29. London: Addison-Wesley.

Sterner, B., and Cusimano, S. 2019. Integrative pluralism for biological function. *Biology and Philosophy* 34(6): 1-21.

Swainpoel A, et al. (2017) How evolutionary thinking can help us to understand ADHD. *BJPsych Advances* **23**(6): 410-418.

Taylor, H., and Vestergaard, M. D. 2022. Developmental dyslexia: Disorder or specialization in exploration? *Frontiers in Psychology* 13: Article 889245.

Wakefield, J. C. 1992. The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist* 47: 373–388.

Wakefield, J. C. 2021 Does developmental plasticity pose a challenge to the harmful dysfunction analysis? Reply to Justin Garson, in Faucher, L., and Forest, D. (Eds.), *Defining Mental Disorder: Jerome Wakefield and His Critics*, 353-383. Cambridge, MA: MIT Press.