# 29 Are There Naturally Selected Disorders? Supplementary Reply to Rachel Cooper

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In this supplement to my reply to Rachel Cooper, I leave the topic of harm on which her chapter focuses and turn to another area of Cooper's critique of the harmful dysfunction analysis (HDA; see my main reply to Cooper in this volume for references) that is not addressed in Cooper's chapter but is prominently cited by another critic in this volume. Some critics, rather than presenting claimed counterarguments directly themselves, "outsource" crucial arguments by simply referring to others' writings as having established that there are counterexamples to the HDA. One such critic is Leen De Vreese, who in her chapter in this volume proclaims that "it cannot be denied that Wakefield's approach has also been refuted in the literature on the basis of counterexamples demonstrating that people's intuitions are not always in accordance with the HDA. . . . These can be found in the literature (see, e.g., Cooper 2007; Schwartz 2007)." I do deny that there is any such refutation of the HDA in Cooper's writings. Thus, although Cooper's paper in this volume does not address the dysfunction component of the HDA, I now consider Cooper's arguments against the dysfunction component that are referred to by De Vreese. For good measure, I will also address the proposed counterexamples to the HDA's evolutionary dysfunction component in the passage De Vreese cites from Schwartz.

In the relevant passage, Cooper (2007), after arguing that evolutionary dysfunction is not by itself sufficient for disorder (I agree; there has to be harm as well), then turns her attention to the HDA and asks, "Could we claim that a condition is only a disorder if it is a *harmful* dysfunction?" (33). She answers that "such an account of disorder cannot be accepted either, as it is not even necessary that a condition be a biological dysfunction for it to be a disorder" (33). She argues for this claim as follows:

> This is because in some cases the genetic bases of disorders may confer a biological advantage and thus be selected. In such a situation, from a biological point of view, there is maybe no dysfunction when cases of the disorder occur. This may well be the case with several types of mental disorder. Conditions including manic-depression, sociopathy, obsessive-compulsivity, anxiety, drug abuse and some personality disorders seem to have a genetic basis and yet occur at prevalence rates that are too high to be solely the result of mutations. This has led

> evolutionary psychologists to suggest that the genetic bases of these mental disorders must be adaptive in some way or other. (33)

This is a manifestly invalid argument based on a common fallacy. From the premise that certain elements of the genetic basis of a disorder were naturally selected, it does not follow that the disorder itself was selected. For example, in the case of genetic disorders such as sickle cell anemia and cystic fibrosis, it is thought that having one copy of certain genes was selected to protect against certain pathogens, but when an individual by chance inherits two doses of that gene, that constitutes a dysfunction and a disorder. Neither the resulting disorder nor its specific genetic basis of two doses of the gene was naturally selected. However, the fact that one dose of the gene confers advantages and was selected for explains the higher-than-expected rate of the disorder. Some have theorized that schizophrenia or bipolar disorder may similarly be partly the result of inheriting combinations of genes that when present individually, in lower frequencies, or in other combinations confer some advantage such as more fluid or creative thought. However, even if some genes underlying a disorder were naturally selected individually or in certain configurations and when they appear in those configurations confer advantages and are not dysfunctions, it may still be the case that there is a specific configuration of the same genes that is a dysfunction and was selected against.

For example, a recent study of the genetics of autism (Polimanti and Gelernter 2017) found that individual genes that confer a risk for autism are associated with cognitive advantages and were positively selected: "Using genome-wide data, we observed that common alleles associated with increased risk for ASD present a signature of positive selection. … ASD risk alleles could positively affect these [cognitive] mechanisms, causing better cognitive ability in carriers as a consequence" (4, 8). However, certain polygenic combinations to the contrary yielded autism: "However, an excessive burden of these risk variants is correlated with the onset of the developmental disorders included in the autism spectrum as the evolutionary cost" (8). Thus, "According to our interpretation of our data, such small-effect alleles were accumulated across the genome (polygenic adaptation) to the benefit of most but to the detriment of some" (9). Cooper's objection is based on a simple misunderstanding of the difference between, for example, single-gene function versus polygenic dysfunction.

Cooper then proceeds to offer some examples that, based on the existence of genetic components, are supposed to show that naturally selected conditions can be disorders:

> The genetic basis of pathological conditions may be selected for a number of reasons. Most obviously, a condition may be selected because it enhances sufferers' biological fitness in some present environment. Linda Mealey (1995a) suggests that the genes for sociopathy are selected for this reason. Sociopaths tend to be more violent and promiscuous than other males, and in tough environments these traits may be adaptive. Other conditions might be of no benefit at

> present but have been biologically beneficial in earlier times. Agoraphobia and other anxiety disorders, for example, may be of no benefit now, but could have been adaptive when human beings lived in more hazardous environments. (Cooper 2007, 33)

Psychopathy, the counterexample Cooper singles out for mention, is one of the most regularly cited proposed counterexamples to the HDA due to Mealey's theory. Psychopathy has long been generally considered a disorder, ever since its distant origins in the concept of "moral insanity," yet here it is being claimed to be naturally selected. So, I will examine in some depth this prototypical example of what critics like Cooper think is wrong with the HDA. (I will address Cooper's other example of anxiety disorders later.)

In response to proposed claims of selected disorders, like Cooper's example of psychopathy, the HDA implies the following counterclaim: because the judgments that a condition is a disorder and that the condition is a biologically designed naturally selected adaptation are incompatible, as one comes to believe that a condition currently considered a disorder is in fact biologically designed, one will also abandon one's belief that it is a disorder. The HDA's prediction of changed disorder intuitions surely qualifies as a bold, novel, and unexpected prediction that has a very low independent prior probability and does not generally follow from other extant accounts of disorder. Consequently, if the prediction is confirmed, it provides strong evidence for the HDA. That is, Cooper's example of Mealey's theory of psychopathy, rather than being a counterexample to the HDA, is a powerful test case for the HDA's counterclaim. (The psychopathy example is also a congenial topic for me to consider because I have examined the related issue of the diagnostic status of youth antisocial behavior [Kirk et al. 1999; Wakefield et al. 1999; Wakefield et al. 2002; Wakefield et al. 2006].)

The story starts a bit before Mealey. In her seminal work on youth antisocial behavior diagnosed as conduct disordered by the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM*), Terrie Moffitt (1993), although not applying an evolutionary perspective, drew a distinction between a pathological form due to brain dysfunction and a nonpathological form that was a strategic response to modern environmental circumstances in which there is a lengthy gap between physical maturity and social independence:

> *Life-Course-Persistent Antisocial Behavior as Psychopathology*. The life-course-persistent antisocial syndrome, as described here, has many characteristics that, taken together, suggest psychopathology. … The syndrome of life-course-persistent antisocial behavior described here has a biological basis in subtle dysfunctions of the nervous system. (Moffitt 1993, 685)

> *Adolescence-Limited Antisocial Behavior Is Not Pathological Behavior.* … Instead of a biological basis in the nervous system, the origins of adolescence-limited delinquency lie in youngsters' best efforts to cope with the widening gap between biological and social maturity. (Moffitt 1993, 692)

Moffitt's analysis illustrated that even a condition long considered a disorder (namely, adolescent conduct disorder as measured by *DSM* antisocial behavioral criteria) is no longer seen as a disorder once no internal dysfunction is inferred.

A couple of years later, Linda Mealey (1995a) published a watershed analysis distinguishing two types of adult psychopathy or sociopathy, one of which consists of largely genetically determined personality traits and the other of which is more environmentally responsive and strategic. She argued, against standard wisdom, that the former "primary" genetic form is in fact a naturally selected adaptation that confers advantages when the psychopath is among mostly nonpsychopathic community members. She thus hypothesized it to be a "frequency-dependent adaptation" that has a potentially successful niche only when it is relatively rare and occurs in the context of a population in which the majority are other naturally selected variants that are not sociopathic. In this case, unlike the cases of cystic fibrosis, sickle cell anemia, and schizophrenia, natural selection is hypothesized to act directly on the condition that has been considered a disorder rather than on a partial genetic basis of the condition that was selected for independent reasons. Thus, this is indeed an ideal test case for Cooper's claim that disorders can be naturally selected adaptations.

The result of this test is that neither Mealey's views nor the views of her colleagues support Cooper's claims. Rather, they confirm the HDA's prediction that disorder and natural section are antithetical hypotheses. Cooper fails to report the fact that Mealey, in her response to comments on the very paper cited by Cooper, dichotomously titles a section "Adaptation or Abnormality?" and poses the straightforward either/or question, "Is sociopathy an adaptation or an abnormality?" (Mealey 1995b, 58). Like Moffitt, Mealey predictably hypothesizes that her category of secondary sociopaths, whose behavior is a strategic response to social circumstances and who are not a genetically shaped personality type, is prima facie a nondisorder. More surprising is what Mealey says about primary sociopaths, who share largely genetically determined personality features and that she recognizes is the condition most likely to be labeled as disordered by others:

> Sociopaths … clearly have both social and psychophysiological "deficits" if the standard we use is the nonsociopath. But in some ways, if sociopathy is indeed a type, using a nonsociopathic standard would be like using a male standard to assess the "normal functioning" of a female, or an adult standard to assess the "normal functioning" of an infant. If sociopaths are not a type designed by natural selection to fill a particular niche, then we could probably agree that they do not function normally; but if they are a type, then … the medical model is no longer appropriate. (Mealey 1995b, 584)

Thus, Mealey, the author Cooper cites to support her claim that a disorder can be an adaptation, in fact directly contradicts Cooper's claim. Instead, consistent with the HDA, Mealey sees the two hypotheses that psychopathy is a medical disorder and that psychopathy is a naturally selected niche adaptation as conflicting hypotheses on

conceptual grounds, such that once one believes primary psychopathy is a naturally selected variation, then despite the history of considering the condition a pathology, "the medical model is no longer appropriate." She pointedly suggests that the distinction between psychopaths and other people is best conceptualized not as psychopathological deviation from normality but as analogous to the often dramatic distinctions between naturally selected variants of normal human beings, such as male versus female and adult versus child.

This reaction is not distinctive to Mealey. Richard Machalek (1995), commenting on Mealey (1995a), similarly expresses the incompatibility between a condition being a medical disorder and being naturally selected:

> As the term itself suggests, the medical model attributes sociopathy to a "pathogen," in this case an emotional deficit that may be genetically rooted and physiologically expressed. … Evolutionary theory takes us beyond mere diagnostic descriptors and prompts us to ask whether such antisocial behaviors may, in some fundamental sense, be advantageous to those who express them. … Framing sociopathy in evolutionary terms accordingly frees us from the explanatory constraints imposed by the medical model that would have us attribute its causes to some "pathogen," when it is not at all clear that the sorts of genetic and physiological processes attributed to sociopathy are necessarily pathological. Rather, we can explore an alternative explanatory possibility. (Machalek 1995, 564)

Here, "pathogen" is a stand-in for "dysfunction." The intuition that the medical disorder hypothesis and the naturally selected adaptation hypothesis are mutually exclusive is widely shared and has been expressed by researchers in subsequent publications on this topic. For example, Kinner (2003) says, "From an evolutionary perspective psychopathy seems to be an adaptation rather than a disease" (67).

Similarly, Lalumière et al. (2001) rely on this basic distinction in formulating their empirical study of developmental trajectories aimed at testing which of the two hypotheses is more likely to be true:

> Psychopaths are manipulative, impulsive, and callous individuals with long histories of antisocial behavior. Two models have guided the study of psychopathy. One suggests that psychopathy is a psychopathology, i.e., the outcome of defective or perturbed development. A second suggests that psychopathy is a life-history strategy of social defection and aggression that was reproductively viable in the environment of evolutionary adaptedness (EEA). These two models make different predictions. … These results provide no support for psychopathological models of psychopathy and partial support for life-history strategy models. (Lalumière et al. 2001, 75)

Reimer (2008) echoed this view: "On any such 'selectionist' model, psychopaths are certainly different than the rest of us, biologically speaking. However, they are not, in any biological sense, disordered" (187). Here, "disordered in a biological sense" is presumably a stand-in for biological dysfunction.

Krupp et al. (2012) further illustrate the way that researchers reconsidered and questioned the pathological status of psychopathy in light of the natural selection analysis and transformed the distinction between selected and disordered into researchable hypotheses:

> Psychopaths routinely disregard social norms by engaging in selfish, antisocial, often violent behavior. Commonly characterized as mentally disordered, recent evidence suggests that psychopaths are executing a well-functioning, if unscrupulous, strategy that historically increased reproductive success at the expense of others. Natural selection ought to have favored strategies that spared close kin from harm, however, because actions affecting the fitness of genetic relatives contribute to an individual's inclusive fitness. Conversely, there is evidence that mental disorders can disrupt psychological mechanisms designed to protect relatives. Thus, mental disorder and adaptation accounts of psychopathy generate opposing hypotheses: psychopathy should be associated with an increase in the victimization of kin in the former account but not in the latter. … These results stand in contrast to models positing psychopathy as a pathology, and provide support for the hypothesis that psychopathy reflects an evolutionary strategy. (1)

The Lalumière et al. (2001) and Krupp et al. (2012) papers illustrate that the HDA allows for the hypothesis of disorder versus natural selection to give rise to testable empirical hypotheses.

In a response to Krupp et al. (2012), Leedom and Almas (2012), although accepting the same overall conceptual distinctions, argued that psychopathy is in fact a disorder after all because it is a spandrel (i.e., a side effect of adaptation) rather than an adaptation per se that was specifically selected for: "Psychopathy may persist because it represents a dominance-related spandrel." The fact that spandrels are not strictly speaking adaptations but side effects of adaptations allows Leedom and Almas to pathologize psychopathy, for they agree that natural selection implies nondisorder.

Krupp et al. (2013), in a paper responding to critics, insisted that their surprising finding of a negative association between psychopathy and violence against genetic relatives "failed to support the hypothesis that psychopathy is a mental disorder, suggesting instead that it supports the hypothesis that psychopathy is an evolved life history strategy" (1), again expressing the assumed opposition between the naturally selected and the disordered. In addition, Krupp et al. lucidly explain why, if psychopathy is a personality type due to an adaptation, then even if brain differences are found between psychopaths and others, the condition should not be pathologized:

> We take it as given that the brains of psychopaths differ from those of nonpsychopaths in systematic ways. Without such differences, psychopaths could not be reliably set apart in their cognition and behavior from nonpsychopaths. But difference is not isomorphic with dysfunction. For instance, although the brains of men and women have much in common, they must also be different on average, as must the brains of young and old, married and single, androphile and gynephile, Anglophone and Francophone, and so on, even if these brain differences are solely the result of differences of experience. While the life sciences have begun

> to recognize that such differences do not inherently reflect disorder, the relationship between difference and disorder nevertheless continues to bedevil the study of mental health.
>
> An argument for dysfunction must marshal supporting evidence, and this must be distinguishable from evidence of difference. (2013, 1)

I conclude from this review that Cooper's own cited counterexample to the HDA not only fails to support her claim that evolutionary dysfunction is not a necessary condition for disorder but, given the unusual occurrence of a major shift in classificatory intuitions by expert researchers, strongly supports the HDA. The literature on psychopathy confirms what I have previously argued primarily on the basis of the history of classificatory judgments about fever, namely, that the HDA correctly predicts that no matter how firmly a condition is initially located within the category of disorder, if it comes to be believed that the condition is biologically designed, then the belief that the condition is a disorder will be challenged and will undergo revision. Of course, such alterations of firm beliefs due to anomalies are likely to be resisted and take place gradually. On the other hand, the examples of fever, adolescent antisocial behavior, and psychopathy illustrate that the change in a condition's disorder status can occur rather rapidly in a research community where new theories of etiology rapidly become known and accepted.

I now consider a further argument in which Cooper adds kin selection to the list of natural-selection evolutionary processes that supposedly can yield disorder:

> Or a condition might be selected through kin-selection processes. As individuals are genetically similar to their kin, an individual can increase the number of copies of their genes by helping their relatives to breed successfully. Thus, through kin selection, a condition that is of no direct benefit to an individual may be selected because it benefits the individual's relatives. (33–34)

Before getting to Cooper's specific example of a possible kin-selected disorder, it is worth pointing out that, consistent with the HDA, it is generally assumed that showing that a feature is due to kin selection demonstrates that it is part of normal variation and not pathological. An example is the ongoing attempt to empirically demonstrate E. O. Wilson's (1975) hypothesis that the prevalence of homosexuality, despite obvious reproductive disadvantages, is due to kin selection. The hypothesis is that although homosexual individuals have not themselves reproduced as much as others, they expended the time liberated from caring for their own children to take care of the children of their kin and thus, by increasing their kin's reproductive success, indirectly caused their own genes to be reproduced. A primary and explicit motive behind this research program is to prove that homosexuality is, via kin selection, a naturally selected normal variant of sexuality and thus, it is inferred, not a disorder.

Cooper's proposed example of a disorder that might be due to kin selection is generalized anxiety disorder (GAD):

> The genetic basis of generalized anxiety disorder might be promoted for this reason. Generalized anxiety disorder causes sufferers to worry a lot, about, among other things, the welfare of their families. While worrying may be of no direct benefit to people with generalized anxiety disorder, it might help their relatives to have someone looking out for them. (34)

The problem with this example is that GAD as defined by recent *DSM*s covers a wide range of conditions, some of which are disputable as instances of disorder, and so the question is whether the solid intuitions about disorder and the plausible explanation of kin selection line up and apply to the same conditions. Often, claims that a category of conditions is both a disorder and naturally selected are due to a failure to distinguish between mild and severe subsets within a category, with the more severe intuitively being disorders while the milder seemingly might be selected but are not persuasive cases of disorder. The argument thus seems to work only because it is based on a subtle equivocation between the two subsets of cases, in which one subset pulls intuitions toward "disorder" while the other subset seems plausibly explainable by natural selection, and both intuitions are then carelessly attributed to the entire category based on a generic label.

GAD offers a good illustration of this fallacy. Anyone who has experienced or treated genuine GAD—I've done both—would scoff at Cooper's argument. When Sigmund Freud initially defined anxiety neurosis (the early name for GAD) as a distinct disorder, separating it off from the wastebasket somatic distress category of neurasthenia, it consisted of continual intense free-floating anxiety not directed at any particular object. If the patient experienced specific worries, they were often primarily inner-directed anxieties about health in reaction to the experience of chronic somatic arousal. There is no imaginable way that this debilitating disorder of undirected anxiety arousal would yield increased safety for kin due to threat monitoring, any more than it would make you safer from fire for your smoke detector to be going off all day even when there is no smoke at all.

However, over time and under the influence of cognitive theoreticians, *DSM* revisions to the diagnostic criteria have expanded the GAD category and refashioned classic GAD to be more "cognitive," requiring that the anxiety take the form of unrealistic but directed worry about multiple concerns such as one's family's welfare. This "worry disorder" category (in fact, an attempt was made during the *DSM-5* revision to rename the category "worry disorder" because it had come so far from the original undirected-anxiety intention) encompasses conditions that are close to normal-range anxieties in our highly vigilant species and are not persuasive cases of disorder, and those are the ones that one might speculate with Cooper might have been a product of kin selection. If this explanation was to be accepted, no doubt these conditions would come to be seen more firmly as nondisorders. In contrast, GAD strictly construed in terms of the kinds of severe anxiety conditions that prompted the formation of the category in the first place confers no conceivable benefit and lacks any plausible kin-related adaptive advantage.

This sort of equivocation is also observed in arguments claiming that depression is a naturally selected disorder. Of course, it is plausible that sadness—even intense sadness, of the kind that occurs in grief—is a naturally selected feature of human life, and the circumstantial evidence is clear cut in favor of natural selection for some range of depressions. However, carefully examined, none of the extant theories of the natural selection of depressive symptoms—whether, for example, that people withdraw to process complex social dilemmas, or withdraw after loss of status to avoid additional harm analogous to primates withdrawing after losing a dominance hierarchy dispute, or withdraw after loss because reduced resources portend danger, or withdraw after a failure to process a redirection of one's actions toward more achievable goals—account for the severe conditions that led to the formation of the category. Depression started as "melancholia" in Greek medicine and was redefined by Kraepelin, and the conditions that fell under the disorder were typically extremely immobilizing, often psychotic, enduring, or recurring over time with no necessary relationship to environmental events such as losses or failures and often involved suicidality. So, while the claim that depressive disorder is naturally selected may seem on first glance to be plausible and in conflict with the HDA, if one is willing to "go into the details," one finds it is generally based on an equivocation between the subset of depression that is nondisordered and naturally selected and the subset of depression that is at this time beyond the explanatory power of any plausible natural selection hypothesis and has always been generally judged as clearly disordered, consistent with the HDA.

A philosopher might attempt to do an end run around the scientific and nosological details and ask: Whatever the actual facts, isn't it *conceivable* that we could tomorrow discover that a prototype mental disorder is in fact a naturally selected condition? This is Cooper's ultimate point, made explicit in her summary statement: "In any event, that it is *conceivable* that some disorders might be biologically adaptive is enough to show that it is not *necessary* for a condition to be a biological dysfunction for it to be a disease. It makes sense to think that some disorders may be evolutionarily beneficial, and this shows that biological dysfunction is not a necessary component of our concept of disorder" (Cooper 2007, 34).

However, Cooper's argument from conceivability is invalid because it is based on an incorrect suppressed premise about what is conceivable. She assumes that a prototype mental disorder must remain a mental disorder no matter what we discover about it (this is the same assumption made by Garson; see his chapter and my reply in this volume)—that is, she assumes that it is inconceivable that there are empirical discoveries that would imply that what we currently consider a prototype disorder is in fact a nondisorder. This, combined with the reasonable claim that it is conceivable that almost any organismic feature could be discovered to be naturally selected, yields her conclusion that it is conceivable that a disorder could be naturally selected. These presuppositions to her thought experiment, in which disorder

status necessarily remains constant while biological design status is allowed to vary, bias its outcome.

However, the thought experiment imagined by Cooper has occurred multiple times as an actual natural experiment, and the results disconfirm her claim. Fever was once considered a prototypical toxin-induced physical disorder until it was discovered to be a biologically designed response to infection, and then it was no longer considered a disorder. Psychopathy was considered a prototypical mental disorder when it was thought to be a failure of biologically designed moral, empathic, impulse-control, or other evolved mechanisms, but experts who became convinced that psychopathy is a naturally selected variant revised their classification and consider psychopathy a nondisorder. Attention-deficit/hyperactivity disorder (ADHD) has been considered a prototype childhood neurodevelopmental disorder, but, despite the fact that in our social environment, ADHD-like behaviors are indisputably harmful in terms of school performance, those who believe that some variants are due to naturally selected novelty-seeking genes that were adaptive in some past environment also have tended to reclassify those variants as nondisorders. That is, the evidence suggests that it is *not conceptually conceivable* that a genuine medical disorder is itself a naturally selected biological adaptation. Yes, we could discover tomorrow that a condition *that we currently consider a clear case of disorder* is in fact is a biologically designed variant. However, we would then question whether it is a disorder. Cooper argues that, because some disorders are biologically selected and therefore not evolutionary dysfunctions, the HDA is thus refuted. However, in every case Cooper cites, the loss of the dysfunction label tracks the loss of the disorder attribution. The results of multiple natural experiments that are actual empirical versions of Cooper's thought experiment strongly support the HDA's account and falsify Cooper's armchair claim.

### Reply to Peter Schwartz's Proposed Counterexamples to the HDA's Dysfunction Requirement

I noted that De Vreese, in addition to citing Cooper's objections to the HDA's dysfunction requirement, also asserts that Peter Schwartz's (2007) proposed counterexamples undeniably refute the HDA. Schwartz himself is confident that he has an endless supply of knock-down counterexamples to the HDA, comparing any attempt by me to defend such an analysis to "the scene in the movie Fantasia where the sorcerer's apprentice is trying to eliminate the magical brooms: crush one, and two spring up" (2007, 56). Recall, however, that the brooms' threatening multiplication was due to the apprentice's ineptitude and his shameful hubris. When the experienced sorcerer returned, the brooms were easily subdued and turned out to pose no real threat at all. Thus encouraged, I consider the objections Schwartz conjures up and examine whether the claimed threat to the HDA is real or illusory.

Schwartz, like Cooper, argues that the HDA fails to adequately explicate "disorder" because it mistakenly requires dysfunction as a necessary condition of disorder. In the passage cited by De Vreese, Schwartz offers two proposed counterexamples that, he claims, are disorders without dysfunctions, which I consider below. (He also presents some concerns regarding the HDA's harm component that I discuss elsewhere in this volume.)

Schwartz's first counterexample to the dysfunction requirement is female anorgasmia: "Female anorgasmia will still not count as disease if orgasm has no function in women" (Schwartz 2007, 56). At another point, when critiquing Boorse's account of disorder, he elaborates,

> For example, it may be that female orgasm makes no specific contribution to survival or reproduction, and thus the mechanisms that bring it about have no biological function. But at the same time, a woman's inability to orgasm may be a serious problem for her, and one which physicians should treat as a disease. (2007, 54)

One must of course agree with Schwartz that when lack of orgasm is a problem for a woman, a physician should try to help. But where does Schwartz get the conclusion that it should be treated "as a disease"? He appears to assume without argument that treatment of a condition must imply that the condition is a disorder. This makes no sense. Physicians treat many nondisorders, from the pain of childbirth to normal grief, and both the *DSM* and the *International Classification of Diseases* (*ICD*) have lengthy lists of "Z Code" categories for commonly treated conditions that are not disorders (*DSM-5* states that these conditions "may be encountered in clinical practice" but "are not mental disorders" [American Psychiatric Association 2013, 715]). As Schwartz himself says, "Doctors have long been involved in inducing sterility and fixing ugly noses, but they do so without claiming that fertility or ugliness are diseases" (2007, 54). As Robert Spitzer, the leading expert on psychiatric diagnosis of the past century, put it in the title to a commentary, "Diagnosis and the need for treatment are not the same" (Spitzer 1998).

In fact, the diagnostic status of anorgasmia—especially during intercourse—is a much disputed question. It is very common for women to have difficulties reaching orgasm during intercourse without some additional clitoral stimulation. Scholarly analyses of the relevant evidence, ranging from Donald Symons's (1979) classic book on the evolution of human sexuality to Elizabeth Lloyd's (2005) recent review, have concluded that female orgasm is likely not a biologically designed feature of female sexuality but rather a variable side effect of other design features, and this is generally taken to imply that orgasm difficulties are not disorders but normal variation.

The psychiatric consensus at this time is represented by *DSM*'s official ambivalence about this diagnosis due to the ambiguity of whether there is a dysfunction. Despite our culture's valuing of the experience of orgasm during intercourse, the evidence

has led most experts to conclude that lack of orgasm is not necessarily a disorder and is instead normal female variation. *DSM-IV* expressed this in the cautionary note to the orgasmic dysfunction criteria that "women exhibit wide variability in the type or intensity of stimulation that triggers orgasm" (1994, 505). *DSM-5* is considerably more explicit: "Many women require clitoral stimulation to reach orgasm, and a relatively small proportion of women report that they always experience orgasm during penile-vaginal intercourse. Thus, a woman's experiencing orgasm through clitoral stimulation but not during intercourse does not meet criteria for a clinical diagnosis of female orgasmic disorder" (2013, 430). Thus, contrary to Schwartz's claims, there is no shared intuition that anorgasmia is a medical disorder even when treated. The example of female anorgasmia shows that even culturally highly undesirable conditions that are often treated are not considered disorders if there is not thought to be a dysfunction.

Note that even if orgasm itself is not a selected function but virtually all women are capable, say, of masturbatory orgasm as a side effect of biological design, then if some dysfunction, such as an inhibition resulting from a psychological trauma, prevents the successful exercise of that capacity in a culture that values sexual pleasure, that would be a harmful dysfunction and a disorder despite the fact that orgasm itself is not a biologically designed effect. This is analogous to reading disorder being a genuine disorder when the harm of inability to be able to learn to read results from some neurological dysfunction, even though reading itself is not a biologically designed function.

Schwartz next argues that a mild case of pneumonia can be a disorder that lacks dysfunction. Before getting to Schwartz's example, it is worth observing that mild cases that fall close to a fuzzy boundary area between disorder and nondisorder are likely to raise perplexing challenges for almost any account of disorder. They may be considered disorders only because, as Spitzer and Endicott (1978) put it, in their "fully developed or extreme form" (18), they are clear disorders. However, Schwartz's "mild pneumonia" example raises issues of a different kind than those raised by boundary fuzziness or early stages of pathology.

Schwartz imagines a case of pneumonia in which the patient "has the bad cough and fever but no problem with his breathing" because there is fluid in only some alveoli so "he has preserved lung function" despite the infection. Schwartz argues that this is a counterexample to the HDA:

> Assume that his doctor properly diagnoses and treats him, and he gets better. But then … it's not clear where the dysfunction was. The lungs were able to carry out their function of gas exchange, and the immune system carried out its function of fighting the infection. Although the cough and fever were unpleasant, they were also important components of the body's response to the microbe. …
>
> So although a serious case of pneumonia involves biological dysfunction, it's not clear that a mild case does too. And we can come up with many cases like this. (55)

Schwartz relies here on the fact that some infectious diseases—such as common colds, the flu, and perhaps in some very mild instances pneumonia—have as their primary symptoms the results of biologically designed defensive mechanisms (e.g., cough, runny nose, fever) that are involved in fighting an infection. Thus, it could conceivably be the case that none of the typically cited symptoms of a specific infectious disease are themselves dysfunctions but rather biologically designed defenses.

Both Christopher Boorse and I have addressed this objection and offered the same response. The symptoms are the result of fighting an infection, and the underlying infection and its effects on the cells and organs—which is what both the body's defenses and the doctor's treatment aim to end—constitute a clear dysfunction of cellular and other processes. Indeed, without certain bodily defenses putting a stop to an infection's advance, even the common cold's destruction of cells could advance deep into the body and pose a threat to the individual's life. So, there is certainly a threatening dysfunction in the form of the infection taking place, and it is that dysfunction to which the various defenses are reacting. The fact that one fends off a serious attack does not mean that no attack took place.

Responding to this reply to the proposed cold- and flu-type counterexamples to the HDA, Schwartz says,

> But this makes the necessary condition so easy to satisfy that it verges on triviality. During menstruation, after all, there is massive cell death as the uterine lining is shed. And during the third-trimester of pregnancy the large uterus interferes with the normal function of the bladder storing urine and of the veins carrying blood back from the legs. But menstruation and pregnancy are normal, healthy conditions. (55)

Taking Schwartz's examples seriously, one might ask: if in both colds and menstruation there is underlying cell death and overt symptoms, and in both cases we treat the condition (yes, Schwartz here contradicts his earlier position on anorgasmia that anything treated is a disorder), yet we consider one and not the other a disorder, what then is the difference that changes our classificatory intuitions? The answer is that menstruation, including the shedding of the uterine lining and the consequent cell deaths that the shedding inevitably entails, is considered an inevitable part of a biologically designed process, whereas the death of mucosal cells due to viral invasion that triggers the symptoms of flu or pneumonia is not. The symptoms of infection pointed to by Schwartz are also biologically designed responses, but they are responses to a nondesigned assault by an infectious pathogen.

Having made a clean sweep of Schwartz's as well as Cooper's supposed counterexamples, I conclude that there is no successful objection to the HDA's dysfunction requirement in the passages cited by De Vreese, and her argument based on the presupposition that such counterexamples are available is left without foundation. More important, the claim by Cooper that the HDA is refuted by the fact that there are or can be prototypical disorders that are naturally selected is falsified by the evidence.

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# Defining Mental Disorder

## Jerome Wakefield and His Critics

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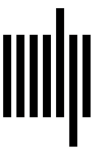

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