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Defining Mental Disorder

Jerome Wakefield and His Critics

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25 Harmless Dysfunctions and the Problem of Normal Variation

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In one of his key publications on the harmful dysfunction analysis of mental disorder (HDA), Jerome Wakefield acknowledged that he has “explored the value element in disorder less thoroughly than the factual element. This is in part because the factual component poses more of a problem for inferences about disorder and in part because the nature of values is such that it requires separate consideration” (Wakefield 1992, 384). More than twenty years have passed since this remark, and yet a thorough consideration of the value component in Wakefield’s HDA is still lacking. Quite a few contributions to this volume promise to change that situation, and ours is one of these. In this contribution, we will analyze the harm or value component and argue that Wakefield’s dealing with it is so problematic that it undermines, at least indirectly, the viability of the HDA.

In the first section, we explore Wakefield’s emphasis on the subjective nature of harm and his exclusive focus on social values. The second section is devoted to an analysis of Wakefield’s examples of conditions that are dysfunctional but harmless (fused toes, albinism, reversal of the heart, dyslexia in illiterate societies, etc.). We argue that these examples are quite problematic because they do not exemplify what they are supposed to exemplify. In the third section, we show how these two problems are connected: Wakefield uses the harmfulness of a condition as an implicit criterion to distinguish normal variation from dysfunction. In doing so, he blurs the distinction between the harm component and the dysfunction component, even though this distinction is central to his HDA.

I. The Downsides of a Hybrid Concept of (Mental) Disorder

Wakefield’s HDA is commonly referred to as the most influential hybrid account of mental and bodily disorder. This account is said to be hybrid because it builds upon the idea that scientific judgments need to be accompanied by value judgments to draw the line between health and disorder. The reasons for proposing such hybrid concepts are easy to understand. First, both pure value accounts and pure objectivist accounts

cannot do the work that objectivists or normativists expect them to do. Second, there is some philosophically attractive kernel in each of these accounts. It is clear that according to Wakefield, the HDA avoids the problems of other nonhybrid accounts (including the pure value account), while preserving the real insights of these accounts (including the pure value account). Because disorder cannot be identified with dysfunction—which is, according to Wakefield, a purely factual component—we need a value component in order to have a full analysis of disorder.

One reason that the factual component does not suffice is because of culture-relative aspects of diagnosis. As Wakefield (1995, 244) himself writes, “I believe it is important to identify both the aspects of diagnosis that are culturally relative and the aspects that remain invariant under cultural transformations.” Obviously, the universal aspects of diagnosis are covered by the dysfunction component, while the culturally relative aspects have to do with value. Wakefield contends that bodily/mental state *S* constitutes a disorder in some cultural environments, while the same bodily/mental state is correctly seen as a healthy, albeit possibly dysfunctional, state in other cultural environments. Dyslexia, for example, is a mental disorder in literate societies, but it is not a mental disorder in illiterate ones. While dyslexia might be a dysfunction in both societies, it is the value component that makes it a disorder in literate ones. So it seems that cultures can be wrong about the dysfunction component, but they cannot be wrong about the value component (Wakefield 2007, 155). “The nature of values themselves plays no role in my analysis,” or so Wakefield has argued (Wakefield 1995, 243). Yet, it does seem that he subscribes to a particular view about the nature of values that are relevant for the value component of the HDA. First, these values are not absolute values, for the values are the culture-relative element in his HDA. Second, these values are social values. This is actually mentioned as the real insight of the “pure value account” of health and disorder: “The value account reveals an important truth: because disorders are negative conditions that justify social concern, social values are involved” (Wakefield 1992, 336). So disorders are harmful according to social norms and not individual norms (Wakefield 2005). We are, after all, a social animal and Wakefield seems to find it awkward to leave out the evaluative responses of others when making judgments concerning how an individual organism functions (Wakefield 1992). What this amounts to is exemplified again by dyslexia: “in a literate society, a person who does not value reading still has a dyslexic disorder if incapable of learning to read due to a brain dysfunction” (Wakefield 2005, 98). In short, Wakefield holds that a dysfunction can only be a disorder if it is considered to be “harmful” (*value relativism*) within a particular cultural framework (*social values*).

This view, however, harbors a lot of problems. Most important, one wonders why Wakefield claims that someone who doesn’t value reading and writing at all should be considered disordered in a literate society. If the individual experiences no harm, why should she be considered disordered? Would Wakefield be willing to bite the bullet and

say that homosexuality is a disorder in cultures that value heterosexuality—assuming of course that homosexuality is dysfunctional as is indeed often assumed by those who defend an etiological account of function (see, e.g., Levin 1984)? One way to avoid this conclusion is by making a distinction between the distress/harm that results directly from the dysfunctional state and distress/harm caused by the social disapproval of the consequences of this condition. For example, one could argue that the harm caused by dyslexia follows directly from the difficulty with reading that is intrinsically tied to dyslexia, whereas the harm caused by homosexuality/bestiality is caused by being ostracized by your peers, a consequence that is not intrinsically tied to bestiality or homosexuality. Yet, “intrinsicity” is a very difficult concept in general (Francescotti 1999; Witmer et al. 2005), and in this case, it is relatively easy to come up with examples that blur the distinction. The examples of dyslexia and homosexuality are, however, closer to each other than one may suspect. After all, dyslexia is harmful in culture A because it is intrinsically tied to not being able to read and because culture A values being able to read, whereas (exclusive) homosexuality is harmful in culture B because it is intrinsically tied to not being able to be attracted to individuals of the other biological sex and because culture B is a heteronormative culture.

This difficulty with social values explains why the *Diagnostic and Statistical Manual of Mental Disorders (DSM)* adopted a different position on the relevant values and emphasized the importance of what Wakefield would call individual values for psychiatric diagnoses. For example, systematic cross-dressing is not considered a mental disorder as long as it is “ego-syntonic.” Only if it becomes ego-dystonic will it qualify as a mental disorder (Gert and Culver 2009).

Now, it certainly is conceivable to develop a version of the HDA that treats individual values as central, rather than social values. One of the reasons Wakefield has put forward to break with an objectivist account like Boorse’s, and to include values in his account of (mental) disorder, is the distinction between “evolutionary harm” (lowered fertility, lowered longevity) and harm to an individual’s well-being. And even though well-being for most members of our species does clearly depend on social factors, it seems reasonable to let the individual judge this, rather than letting the individual’s well-being be judged by cultural standards. In the case discussed above, the fact that a society values reading seems insufficient to judge someone with dyslexia as being disordered since the individual may not value such a skill and therefore experiences no harm for lacking it. There could be various conditions that a society disvalues but that produce no harm in the individual who does not share the same values, partly because social norms are neither understood in the same way by everyone, nor are they universally accepted.

Yet, this “individual value” solution is not without its own problems, as those familiar with pure value accounts of disorder and health know (Reznek 1987; Fulford et al. 2005). First of all, the individual values probably all have—at least in part—a social

origin. So chances are that if transvesticism is ego-dystonic, this is mainly due to the cultural norms of the transvesticist individual. That is why the American Psychiatric Association decided to remove ego-dystonic homosexuality from the *DSM III-R* in 1987, after having removed homosexuality as such already thirteen years earlier from the *DSM-III* (De Block and Adriaens 2013). In 1987, it was generally agreed upon that much—if not all—of the distress homosexual individuals experience has to do with their being stigmatized and discriminated against. Second, adopting individual values as the relevant values to make the distinction between health and disorder is confronted with the problem of anosognosia. Quite a few patients who suffer from severe mental and neurological disorders do not seem to consider their condition problematic, let alone disordered. Yet, few philosophers would argue that they are not disordered, just like few people would defend the view that comatose patients are healthy because they are unable to have value judgments about their condition (Sullivan 2003).

The value component of Wakefield's HDA may result in one advantage of his account over pure objectivist accounts like Boorse's: it embraces an "important truth" of the pure value account. But as we have seen in this first section, the problems or downsides of a pure value account do not seem to be convincingly solved by the HDA, nor is it easy to come up with straightforward modifications of the HDA that do away with these downsides. In the following section, we will explore why Wakefield thinks he needs the value component to take into account the normativist truth that "all disorders are undesirable and harmful" (Wakefield 1992, 376). Does this "fact" really show "that values are part of the concept of disorder" (Wakefield 1992, 376), as he himself is clearly convinced that it does? Or could it be that all dysfunctions are harmful and that therefore we only need a scientific judgment to establish whether or not a condition is disordered?

II. Fused Toes and Albinism

The kind of analysis of disorder that Wakefield proposes is an analysis of the concept "disorder" as it is used by the lay public and by professionals. It aims at analyzing uncontroversial judgments about which conditions are disorders. Examples of these uncontroversial judgments include the judgments that acute leukemia is a disorder and the judgment that schizophrenia is a disorder. If the HD analysis is correct, (1) all uncontroversial disorders—what Boorse (1977, 544) has called "the paradigm objects of medical concern"—are both harmful and dysfunctional, and (2) our intuitions about more controversial cases can change due to discoveries with regard to their harmfulness or their functionality. For example, the HDA "predicts that if what is now considered a disorder is shown to be a selected feature, then our intuitions would change and we would come to consider it a non-disorder, re-conceptualizing it as a normal variation—as has happened with fever" (Wakefield 2011, 152).

This prediction is interesting and raises a series of issues. For example, what would the consequences be for the HDA if it would be shown that schizophrenia—a paradigm object of psychiatric concern—is an adaptation, as some speculatively inclined evolutionary psychiatrists have already hypothesized (Stevens and Price 1996)? Another issue, and one that we will focus on here, has to do with the idea that each of the conjuncts “harmful” and “dysfunctional” is in separation from the other insufficient for a condition to be a disorder. So, if the HDA is correct, (1) some conditions must be both dysfunctional *and* harmless, and (2) our judgment that these conditions are not disorders must also be relatively uncontroversial. It is important to note that the HDA fails if all dysfunctional conditions are harmful or if there are harmless and dysfunctional conditions that are generally considered disorders.

Do we have examples of conditions that are (1) dysfunctional, (2) harmless, and (3) uncontroversially not disordered? Wakefield gives a few examples of physical conditions that are supposed to meet these criteria: fused toes, albinism, and dextrocardia¹ (“reversal of the heart”). We will argue that, contrary to what Wakefield contends, each of these examples does not meet at least one of these criteria.

Let’s start with the fused toes. According to the *International Classification of Diseases (ICD-10)* of the World Health Organization, fused toes and other forms of syndactyly are disorders. Of course, you can always argue that the WHO is wrong here. But does it really seem reasonable to think that one can speak of an uncontroversial medical judgment if this judgment—“fused toes is not a disorder”—goes against the arguably most important health classification system that is published by the arguably most important international health and disease agency? Since all three criteria have to be met, this would be enough to dismiss fused toes as relevant support for Wakefield’s HDA. But let us, for the sake and the pleasure of the argument, check whether the other two criteria are met. Is it really harmless? Suppose for a moment that we could choose to have fused toes or toes that are not webbed. What would most people choose, all else being equal? We think this is pretty much a no-brainer, at least in our culture. Fused toes are often surgically treated, and we assume that this is because they are seen as undesirable. Of course, most people would choose not to have fused toes because of aesthetic reasons and not for more “serious” reasons. Fused toes do not, for example, impair walking or running. But why should we leave concerns over bodily beauty and attractiveness out of the “harm” evaluation? We readily admit that the harm associated with fused toes is mild and that the large majority of individuals with fused toes can live happy lives, but mild harm is harm nonetheless. So *syndactyly* is generally seen as a disorder and as something (mildly) harmful, even though Wakefield contends the opposite. What about Wakefield’s assumption or claim that syndactyly is a dysfunction? The problem here is not so much that there isn’t any account of function and dysfunction available according to which syndactyly clearly qualifies as a dysfunction. The problem is that it is far from obvious that this condition is dysfunctional if one

accepts Wakefield's etiological account of function. Do people with fused toes have fused toes because of a mechanism that was naturally selected because it generated toes that are not fused? There are evolutionary reasons why we have toes, and it is most probably the case that having many digits is an adaptation. It seems far from certain, however, that having five rather than four or six digits is an adaptation. Nobody seems to know for sure why we have five (or ten) toes. We do not think it is very likely that something in the environment of our species (or of species' ancestors) made having ten toes selectively advantageous. In our view, the best supported hypothesis we have right now is that there is a developmental constraint at work (Amundson 2005) that tends to lead to ten toes rather nine or eight. But the loosening of this constraint in individual cases is not—or at least not obviously—a failure of the naturally selected developmental mechanism (Alberch and Gale 1985; Tabin 1992; Galis et al. 2001).

The second example, albinism, is also a disorder according to the *ICD-10* (code E70.3). So it is *certainly* not the kind of example that Wakefield needs to support the need for a value component. Still, it seems somewhat better suited than the fused toes example because everybody seems to agree that albinism in humans is (the result of) a dysfunction in the etiological sense, more precisely a dysfunctional absence of the melanin pigment. Yet, it seems very awkward to claim that this condition is harmless. First, the albino phenotype is so salient that individuals with albinism are often socially stigmatized (Maron 2013). This is the case, for example, in Tanzania, where it is believed that owning albino body parts can make one powerful or wealthy, especially if those parts come from children who screamed while their parts were harvested. Understandably, many parents don't send their albinistic kids to school because they fear for their lives, thus leading to an increased risk of poverty among albinistic people. Second, even if one would dismiss the stigmatization harm as irrelevant for the HDA, one could still point to the increased skin cancer risk of people with the oculocutaneous type of albinism. Individuals with albinism need more skin protection than others to avoid skin cancer (and sunburn). Of course, an increased risk for certain disorders is not a disorder itself. Smoking is not a disease, even though it increases your risk to develop all sorts of diseases. However, it seems to square with Wakefield's account of harm to call the increased risk of developing skin cancer a form of harm, for the same reasons that smoking is generally considered harmful (even though it does not seem to be a dysfunction). Third, both individuals with the oculocutaneous and the ocular type almost invariably suffer from (mild to serious) visual problems. If photophobia and astigmatism in nonalbinistic people are seen as diseases of the eye, then why should we not see them as harmful in individuals with albinism?

In the *ICD-10*, albinism and fused toes can be found alongside Wakefield's third example, dextrocardia (Boorse 2011). Probably, many people would disagree with dextrocardia being classified as a disease (e.g., Boorse 1977). But again, it is really hard to argue that this condition is an uncontroversial nondiseased condition, given its being

listed as a disease in the *ICD-10*. *ICD-10* distinguishes many types of this “disease,” and Wakefield is right that at least one type (*dextrocardia situs inversus*) is quite harmless. People with this condition tend to live as long and as healthily as people without it. With regard to the third criterion, Wakefield himself notes that “a lesion can be a harmless abnormality that is not a disorder, such as when the heart is positioned on the right side of the body but *retains functional integrity*” (Wakefield 1992, 375, emphasis added). So Wakefield himself explicitly acknowledges that in the harmless form of dextrocardia, the heart still does what it was selected for. In his view, the condition is dysfunctional, but the dysfunction can clearly not be a dysfunction of the heart. He is less explicit, though, on what constitutes the dysfunction here, but our guess is that Wakefield considers it to be the result of a dysfunctional development. But why would this development be dysfunctional? The embryonic development of all humans is to some degree the result of selection. However, the issue is whether this development was selected or whether it was not only selected but also selected *for*. If there occurred selection of this development, but not selection for this development, the absence (or the mirroring) of this developmental pattern does not seem to constitute a dysfunction in the etiological sense.

Sober’s (1984) well-known distinction between selection of and selection for has often been used in the literature on etiological accounts of functions (see, e.g., Sterelny 1990; Shapiro 1992). It is generally agreed upon that the difference between a trait/property that is selected for and one that is selected can be established by counterfactual reasoning that tracks the causal role of the target of selection (that which is selected for) in the selection process. For example, the warmth of the polar bear’s coat has been selected for, while there has only been selection of its weight, because the polar bear’s coat would not have been selected if it would have been heavy but not warm, and the coat would have been selected if it had been warm but not heavy (Sober 1984). According to the etiological account of function, the coat of the polar bear would dysfunction if it failed to be warm but not if it failed to be heavy. Likewise, if people with a particular form of dextrocardia tend to live healthy lives, this is reason to believe that the human embryogenetic processes were not selected for generating a heart at the left (and the lungs at the right). In other words, the harmlessness of this condition suggests that this is not a dysfunction. We do not claim to know the eventual selectionist story about the normal position of heart and other thoracic organs, but we do have reasons to doubt that the present biological knowledge renders *dextrocardia situs inversus* an uncontroversial example of a dysfunctional condition.

Albinism, fused toes, and dextrocardia are all physical disorders. Maybe, the HDA is better suited to deal with psychiatric judgments than with other medical judgments. If true, this could entail that the uncontroversially nondisordered conditions that are dysfunctional but harmless should be looked for in the psychiatric literature. And indeed, Wakefield also points to some mental conditions that are dysfunctional and

harmless and are generally not seen as disorders. For instance, a dyslectic condition in illiterate societies wouldn't be a disorder because it is a harmless dysfunction. Likewise, lack of male aggression in current Western societies would be more harmless than the high level of aggression of most males, even though these higher levels of aggression were selected for in our ancestral environment.

The first example—dyslectic conditions in illiterate societies—is highly problematic. After all, the dysfunctional nature of the dyslectic condition is far from established. Most evolutionary social scientists think reading, writing, and dyslexia have no prior history of selection: “The ability to read and write are by-products of adaptations for spoken language, enabled by their causal structure. Random evolutionary noise exists as well, for example, the gene variants that cause dyslexia” (Tooby and Cosmides 2005, 26). Because Wakefield's HDA entails that psychological structures without a selective history cannot genuinely dysfunction (Murphy 2006, 82), dyslexia is not to be thought of as a dysfunction. Of course, one need not take Tooby and Cosmides's view of dyslexia for granted, but there is little reason to prefer an adaptationist account of reading and writing above their account. Tooby and Cosmides are renowned adaptationists (in the Gould-Lewontin sense), and it is telling that they offer the example of reading and writing to show that they are not “panadaptationist.” In other words, if there would be a more plausible adaptationist story to tell about our capacity for reading and writing, adaptationists like Tooby and Cosmides would most likely endorse it.

The second example is probably the most convincing example Wakefield gives of a condition that meets the three criteria. Evolutionary psychiatrists have emphasized over and over again that the absence of a particular emotion and/or attitude can be as dysfunctional as having too much of that emotion or attitude. They argue that extreme jealousy is rightly seen as a mental disorder but that the evolutionary perspective helps us to see that it is equally pathological not to feel any jealousy after finding out that your beloved husband cheated on you with your best friend. So evolutionary psychiatrists would argue that even though this lack of jealousy is currently not seen as a mental disorder, it should be seen as a disorder. The case that Wakefield presents here is different. Whereas the absence of jealousy is still maladaptive in our current social environment and thus a “harmful dysfunction” (although perhaps less so than in the ancestral environment, due to, for instance, modern birth control methods), the absence of aggression is not maladaptive now, even though it is the result of a breakdown of a mechanism that is thought to be a full-fledged adaptation. In this case, the mismatch model of evolutionary medicine is turned on its head: there are lucky individuals who would suffer from a disorder if they would live in the environment of evolutionary adaptedness, but they live happy and good lives because they are lucky enough to find themselves in a world where lack of aggression is highly valued and where the “typically male aggression” is punished severely enough to make it maladaptive. A benign environment makes all the difference here. We fully agree with

Wakefield that this condition is harmless—it's even beneficial—and that most laypeople and medical professionals would not see this as a disorder. It is less clear, however, whether this lack of aggression is really dysfunctional. Conditions like this “lack of aggression” are often used to counter the etiological account of function. For instance, McLaughlin critiques Wright's etiological account of function in the following way:

At its first appearance, a beneficial trait in an organism doesn't have the proper etiology and thus cannot, according to Wright, be said to have a function—although a few generations later it will have acquired one: Organismic mutations are paradigmatically accidental in this sense. But that only disqualifies an organ from functionhood for the first—or the first few—generations. This is a problem that Wright shares with all the other etiological analyses. (McLaughlin 2001, 101)

While Wakefield would be likely to argue that those individuals without aggression are simply lucky accidents until this trait will have been selected,² the problem is that the only convincing example that he gives of a harmless dysfunction constitutes a classic counterexample to the natural selection account of function he considers crucial for his HDA.

Wakefield develops this example primarily as an example of a breakdown of mechanisms that usually result in highly aggressive responses, thereby suggesting that even reduced levels of aggression (and not just the total lack of aggression) would be dysfunctional: “For example, high levels of male aggression might have been useful under primitive conditions, but in present day circumstances such aggressive responses might be harmful. Consequently, even if a disposition to highly aggressive responses is the natural function of some mechanism, the loss of that function might not now be considered a disorder” (Wakefield 1992, 384).

In this example, mild aggression is a dysfunction in current societies and not just an example of normal variation (of aggression or of the underlying psychological mechanisms). Interestingly, as we will see in the next section, this is one of the very few places where he does not seem to use—implicitly—a harm judgment (or a related value judgment) to distinguish normal variation from dysfunction. The fact that this is so unusual could indicate that it is very difficult—and maybe even impracticable—to distinguish between suboptimal normal variation and dysfunction without using value judgments, even though Wakefield is trying to say the inverse.

III. Normal Variation and Harmless Dysfunctions

One of the supposed benefits of the HDA is that it helps to distinguish harmful misfortunes and what are commonly considered to be true disorders (e.g., being illiterate due to circumstances vs. due to a reading disorder or being short vs. having a height disorder). With the former set of conditions, there is harm to the individual and yet there is no disorder, precisely because there is no underlying or accompanying dysfunction.

One interesting consequence of this is that in such instances, “no matter how harmful these conditions may be, they are part of the way we are biologically designed” (Wakefield 2010, 343). Such a claim, however, does not seem to capture what is at stake when looking at how a large majority of health conditions fall along a continuum. This problem should become apparent by looking at the following examples.

It has been argued that the nausea and vomiting experienced by many pregnant women is likely an evolved trait that protects the developing fetus against toxins, implying that while such symptoms are harmful for the mother, they need not indicate a disorder (Nesse and Williams 1994). Being able to determine, however, at what point variations in the intensity and duration of such symptoms shade into hyperemesis gravidarum, or severe morning sickness, which occurs in 0.3% to 2% of pregnant women (Goodwin 2008), is rather difficult. While Wakefield clearly accepts this latter condition to be a disorder (Wakefield 1999, 392), what seems implicit in his account is that because there is normal variation in this naturally selected function, it is actually only when such variation becomes harmful for the individual that it will be considered unhealthy. In making his argument, Wakefield does not refer to the lowered fitness of women suffering from hyperemesis gravidarum. The fact that he considers it disordered (and thus also dysfunctional) is not because he knows that pregnant women with severe morning sickness have on average lower fitness than pregnant women with mild morning sickness but because he implicitly uses harm to distinguish normal variants from dysfunctional ones. This also seems to square with most people’s intuitions: if there are polymorphous traits that are equally well functioning (and with equally high fitness values), the trait that confers substantial harm would probably be seen as a disordered trait.

As Lilienfeld and Marino (1995) have argued, Wakefield’s analysis needs to address the problem of normal variation. Natural selection is a process that filters out rather extreme forms, but while the resulting traits fixate within a population, there still remains much variation. In some cases, the remaining variants do not differ in fitness. Blood types are a good example of this, and this might also be true for severe and mild morning sickness. The existence of normal variation is especially interesting for medicine because not all suboptimal variation is judged as disordered. According to Wakefield’s etiological approach, there is only a dysfunction when a trait within the variation that has been selected for breaks down, having detrimental effects on individual fitness. However, variation in suboptimal traits suggests that not all fitness reductions will be considered disordered, and this needs to be explained.

One example of this problem could be seen in the variation of heart rate-related conditions. The extreme of heart failure is a clear example of where the heart is failing to perform its naturally selected function of pumping blood, resulting in the death of the organism, and is thus disordered. The conclusion that death is unhealthy is rather trivial, though. Wakefield’s HDA should also be able to capture diseases that are

not the result of such drastic breakdowns. For example, within any given population, there will be variations in the level at which hearts perform this function, many of which will be suboptimal, as in the case of hypotension. The main question for Wakefield now becomes when hypotension becomes a dysfunction. Can the cutoff between suboptimal hypotension and dysfunctional hypotension be made without reference to the harm it causes? After all, what will constitute hypotension in any given case will differ between individuals, and while it is potentially harmful to the individual, hypotension is not in itself a disorder. It seems uncontroversial to argue, however, that severe forms of it are. Since the line between healthy and pathological hypotension is unclear (see also Schwartz 2007), in part because it is unclear when it would have negative effects on fitness, he seems caught in the trap of either considering all suboptimal variation as dysfunctional, since they are all fitness reducing (compared to the optimal heart rate), or using harm to determine when the variation should be considered a dysfunction. If Wakefield would opt for the first alternative—all suboptimal variation is dysfunctional—the value judgment is really all that matters for most diseases since most variants of a trait in a population are suboptimal. Furthermore, this would entail that Wakefield's use of "dysfunction" parts from how the concept is used in biology, where not all suboptimal variation is considered dysfunctional. If he opts for the second alternative—harm makes the difference between suboptimal and dysfunctional variants—he would blur the distinction between the harm component and the dysfunction component, a distinction that is absolutely basic for his hybrid account of disorder.

Another example of this problem arises when trying to distinguish shortness from height disorders (Wakefield 1999, 2010). When is normal variation in height to be considered a dysfunction? For Wakefield, there is a clear and commonly accepted divide between those unlucky individuals who are short, and who may experience some harm because of it, and those who are short because there is a hormone deficiency causing their diminished stature (1999, 379). He argues that where this line will be drawn will be a matter of convention and falls outside of conceptual analysis. This is an odd claim since no one disagrees with the fact that there is a difference between being short and having a hormone deficiency. The fruitfulness of such an analysis, however, stands or falls not on the extremes but precisely on these borderline "fuzzy" cases where a decision is needed. At the extreme end of normal variations in shortness, there is clear harm to the individual (according to social values), as witnessed in the frequent complaints and social stigmas attached to being short. Moreover, it is possible that this social harm could be linked to some form of evolutionary harm (and hence dysfunction) in the sense that average-height males have higher reproductive success than shorter and taller males (Stulp et al. 2012). As with the example of hypotension, would he then argue that all suboptimal deviations are dysfunctional? While Wakefield will likely cling to the argument that no matter how harmful being short is, without an

underlying dysfunction there is no disorder, it seems reasonable to argue that extreme cases of shortness, like severe hypotension, cause enough harm/distress to render the individual dysfunctional. In other words, the line between suboptimal height and a dysfunction will be drawn precisely where the individual is sufficiently harmed due to their height. At this point, extreme shortness is just as much a disorder as having a hormone deficiency, even if there will obviously be a different prescription for the latter.

This problem of borderline cases is not an appeal to rare medical conditions but seems to plague all disorders that are a matter of degree (e.g., those with the prefix hyper- or hypo-, or those with dys-).³ As such, Wakefield struggles to account for how the line is drawn between suboptimal variation and disorder within a large suite of common conditions. Is Wakefield led to conclude, then, that suboptimal functioning of the corpus callosum that produces a mild inability to read, or suboptimal developmental variations leading to mild abasia, or suboptimal hepatic regulation of cholesterol engendering mild hypercholesterolemia, or suboptimal loss-coping mechanisms producing mild intense sadness are all considered dysfunctional? If he does not wish to make such a judgment, then what might make Wakefield's account "unworkable," as Lilienfeld and Marino (1995) suggest, is that in order to work, it must assume precisely the opposite of what it claims: the suboptimal is considered dysfunctional when it is harmful.

We have seen, two different problems arising from normal variation. The first, which we explored through variations in morning sickness, suggests that fitness can be held equal across variations, and yet it is reasonable to consider some variations to be disorders precisely because they are harmful to the individual. The second, which we saw in terms of suboptimal variations, suggests that if there will be variations in fitness, and thus the attribution of a dysfunction, this attribution will most likely occur at that point where the variation is sufficiently harmful, in relation to individual or social values. In both cases, the line between dysfunction and harm is blurred. At the limits of the normal range of variation, which is precisely where medical judgment is needed and thus where conceptual analysis is put to the test, Wakefield smuggles the harm component into his account of dysfunction. As we have seen, this is the underlying problem that gets Wakefield into trouble when trying to provide uncontroversial examples of harmless dysfunctions, such as albinism or fused toes. If having fused toes is considered a suboptimal trait, then the point at which it would become a dysfunction is when it would pose enough harm to the individual. It is because he has not adequately accounted for the consequences that the problem of normal variation poses that his account cannot do what it set out to do.⁴ In responding to a similar remark made by Murphy and Woolfolk (2000a) that there is a continuum in nature regarding the intensities and varieties of conditions, Wakefield argues that he is concerned with

explaining what occurs outside of such ranges in the clear-cut cases (i.e., explaining the *intuition* that something is a disorder). The way in which he states this appeal to intuition is rather telling: “Our intuitions tend to attribute dysfunction and disorder in extreme cases where the behavior does not appear to be a useful strategy by any stretch of the imagination but rather seems to *devastate the individual’s social functioning and interfere with other designed functions*” (Wakefield 2000, 260, emphasis added). In other words, the reason we seem to intuit that there is a *dysfunction*, let alone a disorder, is because as one moves into such an extreme, there is clearly harm to the individual. In other words, we also seem to have the “intuition” that conditions that exist on a continuum are only actually pathological if they are harmful to the individual. Prior to that, they are simply functional anomalies. It seems possible to have the intuition, then, that in such borderline cases, some levels of harm are sufficient to render someone disordered. At this point, the usual response of harm without dysfunction simply does not hold water.

As such, not even an appeal to intuitions seems sufficient to prop up the HDA. In the end, Wakefield only seems to describe the obvious or what is commonly accepted. He acknowledges that normal variations and fuzzy boundaries exist but claims that boundaries are not what conceptual analysis is meant to clarify. But if not, then the concepts seem redundant to medical judgments, and he seems to be doing nothing more than trying to find scientific support for some “folk psychiatry.” As Murphy and Woolfolk write, “It is precisely the disagreements and divergent usages that provide the most information about the way the concept is used in the various theories that employ it” (2000b, 289). One would think that putting a concept to the test by appealing to those tough borderline cases would be yet one more chance to strengthen the HDA, but the fact that Wakefield avoids doing so seems to reveal a fundamental dysfunction at the heart of his endeavor.

Notes

1. These examples seem to come from Kendell (1975), who himself seems to take them as emblematic. Interestingly, roughly the same set of conditions (alongside hemophilia and color blindness), likely finding its source in the work of Geoffroy Saint-Hilaire, has been discussed quite some time ago by the philosopher of medicine, Georges Canguilhem (1991, 2008), suggesting that they are prototypical examples of where medical judgments differ. In the case of Canguilhem, he suggests that it is up to the individual to judge whether such conditions are mere anomalies or diseases.
2. In fact, Wakefield makes this argument by suggesting that if there would be a genetic mutation for blue eyes that is also protective of a disease, it could only be said to have this function after having been selected (2001, 357). Until that point, it would simply be a mutation with accidentally beneficial effects.

3. Murphy (2001) provides the example of dysthymia to suggest that there could be normally functioning mechanisms that are still deemed disordered, in this case due to having received deviant information.

4. The fact that one can more easily find examples of harmful conditions that are not dysfunctional has to do with one of the central tenets of error management theory (and evolutionary theory in general): type I errors (false positives) are less fitness undermining than false negatives.

References

Alberch, P., and E. A. Gale. 1985. A developmental analysis of an evolutionary trend: Digital reduction in amphibians. *Evolution* 39(1): 8–23.

Amundson, R. 2005. *The Changing Role of the Embryo in Evolutionary Thought: Roots of Evo-Devo*. Cambridge University Press.

Boorse, C. 1977. Health as a theoretical concept. *Philosophy of Science* 44(4): 542–573.

Boorse, C. 2011. Concepts of health and disease. In *Handbook of the Philosophy of Science Volume 16: Philosophy of Medicine*, F. Gifford (ed.), 13–64. Elsevier.

Canguilhem, G. 1991. *The Normal and the Pathological*. Trans. C. R. Fawcett and R. S. Cohen. Zone Books.

Canguilhem, G. 2008. *Knowledge of Life*. Trans. S. Geroulanos and D. Ginsberg. Fordham University Press.

De Block, A., and P. Adriaens. 2013. Pathologizing sexual deviance: A history. *Journal of Sex Research* 50(3–4): 276–298.

Francescotti, R. 1999. How to define intrinsic properties. *Noûs* 33(4): 590–609.

Fulford, K. W. M., M. Broome, G. Stanghellini, and T. Horton. 2005. Looking with both eyes open: Fact and value in psychiatric diagnosis? *World Psychiatry* 4(2): 78–86.

Galis, F., J. J. M. van Alphen, and J. A. J. Metz. 2001. Why five fingers? Evolutionary constraints on digit numbers. *Trends in Ecology and Evolution* 16(11): 637–646.

Gert, B., and G. Culver. 2009. Sex, immorality, and mental disorders. *Journal of Medicine and Philosophy* 34(5): 487–495.

Goodwin, T. M. 2008. Hyperemesis gravidarum. *Obstetrics and Gynecology Clinics of North America* 35(3): 401–417.

Kendall, R. E. 1975. The concept of disease and its implications for psychiatry. *British Journal of Psychiatry* 127: 305–315.

Levin, M. 1984. Why homosexuality is abnormal. *The Monist* 67(2): 251–283.

Lilienfeld, S. O., and L. Marino. 1995. Mental disorder as a Roschian concept: A critique of Wakefield's "harmful dysfunction" analysis. *Journal of Abnormal Psychology* 104(3): 411–420.

- Maron, D. F. 2013. Witchcraft trade, skin cancer pose serious threats to albinos in Tanzania. *Scientific American*, October 11. <http://www.scientificamerican.com/article.cfm?id=witchcraft-trade-skin>.
- McLaughlin, P. 2001. *What Functions Explain: Functional Explanation and Self-Reproducing Systems*. Cambridge University Press.
- Murphy, D. 2001. Hacking's reconciliation: Putting the biological and sociological together in the explanation of mental illness. *Philosophy of the Social Sciences* 31(2): 139–162.
- Murphy, D. 2006. *Psychiatry in the Scientific Image*. MIT Press.
- Murphy, D., and R. L. Woolfolk. 2000a. The harmful dysfunction analysis of mental disorder. *Philosophy, Psychiatry, & Psychology* 7(4): 241–252.
- Murphy, D., and R. L. Woolfolk. 2000b. Conceptual analysis versus scientific understanding: An assessment of Wakefield's folk psychiatry. *Philosophy, Psychiatry, and Psychology* 7(4): 271–293.
- Nesse, R., and G. C. Williams. 1994. *Why We Get Sick: The New Science of Darwinian Medicine*. Vintage.
- Reznek, L. 1987. *The Nature of Disease*. Routledge & Kegan Paul.
- Schwartz, P. H. 2007. Defining dysfunction: Natural selection, design, and drawing a line. *Philosophy of Science* 74: 364–385.
- Shapiro, L. A. 1992. Darwin and disjunction: Optimal foraging theory and univocal assignments of content. *Proceedings of the Philosophy of Science Association* 1: 469–480.
- Sober, E. 1984. *The Nature of Selection*. MIT Press.
- Sterelny, K. 1990. *The Representational Theory of Mind: An Introduction*. Blackwell.
- Stevens, A., and J. Price. 1996. *Evolutionary Psychiatry: A New Beginning*. Routledge.
- Stulp, G., T. V. Pollet, Verhulst, S., and A. P. Buunk. 2012. A curvilinear effect of height on reproductive success in human males. *Behavioral Ecology and Sociobiology* 66(3): 375–384.
- Sullivan, M. 2003. The new subjective medicine: Taking the patient's point of view on health care and health. *Social Science & Medicine* 56: 1595–1604.
- Tabin, C. J. 1992. Why we have (only) five fingers per hand: Hox genes and the evolution of paired limbs. *Development* 116: 289–296.
- Tooby, J., and L. Cosmides. 2005. Conceptual foundations of evolutionary psychiatry. In *The Handbook of Evolutionary Psychiatry*, D. Buss (ed.), 5–67. John Wiley & Sons.
- Wakefield, J. C. 1992. The concept of mental disorder: On the boundary between biological facts and social values. *American Psychologist* 47(3): 373–388.
- Wakefield, J. C. 1995. Dysfunction as a value-free concept: A reply to Sadler and Agich. *Philosophy, Psychiatry, and Psychology* 2(3): 233–246.

Wakefield, J. C. 1999. Evolutionary versus prototype analyses of the concept of disorder. *Journal of Abnormal Psychology* 108: 374–399.

Wakefield, J. C. 2000. Spandrels, vestigial organs, and such: Reply to Murphy and Woolfolk's "The harmful dysfunction analysis of mental disorder." *Philosophy, Psychiatry, and Psychology* 7(4): 253–269.

Wakefield, J. C. 2001. Evolutionary history versus current causal role in the definition of disorder: Reply to McNally. *Behavior Research and Therapy* 39: 347–366.

Wakefield, J. C. 2005. On winking at the facts, and losing one's hare: Value pluralism and the harmful dysfunction analysis. *World Psychiatry* 4(2): 88–89.

Wakefield, J. C. 2007. The concept of mental disorder: Diagnostic implications of the harmful dysfunction analysis. *World Psychiatry* 6(3): 149–156.

Wakefield, J. C. 2010. Misdiagnosing normality: Psychiatry's failure to address the problem of false positive diagnoses of mental disorder in a changing professional environment. *Journal of Mental Health* 19(4): 337–351.

Wakefield, J. C. 2011. Darwin, functional explanation, and the philosophy of psychiatry. In *Mal-adapting Minds: Philosophy, Psychiatry, and Evolutionary Theory*, P. R. Adriaens and A. De Block (eds.), 43–172. Oxford University Press.

Witmer, D. G., W. Butchard, and K. Trogdon. 2005. Intrinsicity without naturalness. *Philosophy and Phenomenological Research* 70(2): 326–350.