Defining Mental Disorder

Jerome Wakefield and His Critics

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8 Quinian Qualms, or Does Psychiatry Really Need the Harmful Dysfunction Analysis? Reply to Harold Kincaid

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I have long been an admirer of Harold Kincaid's wide-ranging and sophisticated contributions to philosophy of psychopathology and philosophy of science more generally. I am grateful to him for his challenging commentary on my harmful dysfunction analysis (HDA) of medical, including mental, disorder. The HDA claims that "disorder" refers to "harmful dysfunction," where dysfunction is the failure of some feature to perform a natural function for which it is biologically designed by evolutionary processes and harm is judged in accordance with social values (HDA; First and Wakefield 2010, 2013; Spitzer 1997, 1999; Wakefield 1992a, 1992b 1993, 1995, 1997a, 1997b, 1997c, 1997d, 1998, 1999a, 1999b, 2000a, 2000b, 2001, 2006, 2007, 2009, 2011, 2014, 2016a, 2016b; Wakefield and First 2003, 2012).

In his chapter, Kincaid enumerates some of the contributions of my work but nevertheless wonders how much influence they have actually had given that the *Diagnostic and Statistical Manual of Mental Disorders* (*DSM-5*) (American Psychiatric Association 2013) eliminated the bereavement exclusion despite my vigorous objections. That was indeed a disappointment and an ill-advised change. However, even there a newly introduced "Note" to the major depression diagnostic criteria that was added in response to the debate opens a back door to many of the conclusions I have argued for. It acknowledges that major depression-mimicking normal sadness is not limited to bereavement but can occur in response to a wide range of losses and that earlier criteria were mistaken to use duration thresholds on the order of a few months to mark a shift from normal to pathological sadness, and it allows for clinical judgment in making the discrimination between normal versus pathological depression. Elsewhere, the HDA remains influential and continues to be regularly applied by researchers to nosological disputes on topics ranging from psychopathy (see my reply to Cooper in this volume) to hebephilia (Rind and Yuill 2012).

Psychiatry's Need for a Conceptual Analysis of Mental Disorder

Kincaid proposes "doing without 'disorder' in the study of psychopathology" and offers a variety of arguments against the usefulness of the HDA for psychiatric nosology. (One

is of course sorely tempted to ask: the study of *what*? "Psychopathology" is a synonym for "mental disorder," so Kincaid seems to beg the question.) Until I read Kincaid's chapter, I had not realized that he and I are coming from such different philosophical perspectives that for him, even my attempt to conceptually analyze "mental disorder" "relies on dubious assumptions about the methods, aims, and abilities of philosophy." Kincaid thus joins the chorus of philosophers of psychiatry attempting to delegitimize conceptual analysis as a way to explain classificatory judgments. In Kincaid's case, this harsh judgment emerges from his Quinian "doubts that there are conceptual truths that can be tested by intuition about possible counterexamples and by reports on what we would say … grounded in skepticism about the analytic/synthetic distinction and the coherence of substantial a priori knowledge." At this point in post-Quinian philosophical history, deploying all that unnecessary philosophical baggage to preempt a proposed conceptual analysis seems "dubious" to me. In the course of his commentary, Kincaid praises the concrete evidential and differentiating points that I make about specific psychological conditions and suggests those points stand independent of the HDA framework that inspired them. I would suggest that a similar "proof is in the pudding" attitude is appropriate to the HDA itself. Rather than summarily extirpating the HDA in the hallowed name of "Two Dogmas," consider it a hypothesis about shared mental representations underlying classificatory judgments within a target linguistic community, to be tested like all such hypotheses by explanatory power and evidential support.

Beyond Quinian doctrine, Kincaid attempts to support his rejection of the HDA's conceptual analytic methodology with the historical argument that studies of science show that "successful science does not first start with getting a definition in terms of necessary and sufficient conditions and then proceeding; instead, science often muddles along with concepts that are undefined." This is quite true as a broad generalization but irrelevant to the present case. The (absurd) "definitions first, science second" doctrine cannot apply to current attempts to analyze "disorder" because we are hardly at the "start" of psychiatry. Medicine in the Western tradition has been pursuing prescientific and scientific theories and research about mental disorder for going on 2,500 years.

The analysis of "disorder" has instead come into prominence as a way of addressing very specific issues that have arisen from ongoing psychiatric practice and science. For example, Robert Spitzer said that he never thought it was important to define "mental disorder" until the dispute over the diagnostic status of homosexuality broke out precisely on the question of whether homosexuality fits the concept of mental disorder. Similarly, the HDA was undertaken as a response to antipsychiatric attacks on the discipline of psychiatry that challenged psychiatry's coherence and legitimacy as a medical profession. As well, the abuses of psychiatric diagnosis in the Soviet Union, the *DSM-5* quagmire over the issue of whether psychiatric expansiveness is leading to medicalization of normal variation, and accusations that big pharma is redefining normal conditions as disorders are all recent challenges that call for attention to the nature of the

concept of mental disorder as a claimed subconcept of medical disorder. Kincaid himself admits elsewhere that his anticonceptual analytic bias does not apply to such situations in which conceptual clarification can be a route to moving a discipline forward: "Philosophical analysis can be useful when it is part of clarifying scientific controversies, but then that is something scientists do all the time" (2008, 678). Scientists "do it all the time" because, far from being some historical or ontological oddity, conceptual analytic exploration is continuous with creative empirical and theoretical exploration. Numerous first-rank scientists have taken on conceptual questions when faced with obstacles to progress that demanded such analysis, from Einstein analyzing the concept of simultaneity and Mach analyzing the nature of the evidence in physics to Hilbert analyzing the nature of mathematical proof and Freud rethinking the nature of mental states.

Kincaid further defends his rejection of conceptual analysis as follows: "So my argument contra Wakefield is that the disciplines that study and treat psychopathology do not need and can get along without a philosophically satisfying (i.e., necessary and sufficient condition) definition of mental disorder." I am not sure what the argument here is supposed to be. "Analysis X is unneeded for discipline Y to get along" does not imply "analysis X is incorrect" or even "analysis X is not valuable and illuminating for discipline Y." For example, science has not needed and gotten along just fine without Quinian philosophy (as Kincaid elsewhere observes, "the full implications [of Quine's work] are still often ignored" [2008, 368]), yet Kincaid nonetheless finds Quinianism valuable and illuminating in understanding the nature of science. Kincaid also argues that conceptual analysis is futile because whether we classify conditions as disorder versus nondisorder does not really matter scientifically; it is merely a matter of *what we say* and has no substantive scientific content, only ethical and social implications: "Nothing social scientific is at issue in asking these questions and to that extent they can be ignored." The HDA explains why he is wrong. Burying one's head in the Quinian sand doesn't change the fact that judging that people are disordered—dissidents in the Soviet Union, runaway slaves in the antebellum U.S. South, females having clitoral orgasms in Victorian England—is not just verbal behavior but makes implicit claims about causal processes and their relation to biological design that interact in complex scientifically significant ways with the "web of belief." At this point in history, when we judge conditions as mental disorders, we generally are doing so on the basis of circumstantial evidence without a knowledge of the specific underlying etiology—just as Hippocrates and Galen did with judgments about physical disorders. However, according to the HDA, disorder versus nondisorder attributions carry with them different presuppositions about the *general domain* in which the etiological explanation for the superficial symptoms lies—very roughly, dysfunctions for disorders versus biologically designed human functioning for nondisorders. Although our intuitions about disorder versus nondisorder based on indirect evidence are fallible, they have generally been surprisingly good indicators of real-world differences in categories of causation.

Consequently, science done without attention to this distinction is likely to be mixing different categories of etiologies, leaving the meaning of one's results ambiguous. Moreover, disorder intuitions, being suggestive guides to divergent causal etiologies, indicate fertile paths for research foci (e.g., see the discussion of hyperemesis gravidarum in my reply to De Block and Sholl in this volume).

Kincaid questions my claim that we are often capable of (fallibly!) inferring biological design and its failures in the domain of mental mechanisms and thus judging mental disorder versus nondisorder according to the HDA, although he allows that this is perhaps possible for physical mechanisms. However, diagnostic manuals from Hippocrates to *DSM-5* illustrate that it is possible to draw some justified inferences about psychological biological design or its failure despite lack of knowledge of specific etiology. For starters, here are ten prominent diagnostic categories each taken from a different chapter of *DSM-5* and thus a different domain of human psychological functioning, each of which in their prototypical presentations appear intuitively incompatible with the biologically designed functioning of a human being: autism spectrum disorder; schizophrenia; bipolar I disorder with psychotic features; major depression, recurrent, with psychotic features; panic disorder; reactive attachment disorder; conversion disorder; anorexia nervosa; erectile disorder (primary impotence); and pedophilia, exclusive type. I agree that judgments about biological design are sometimes difficult, but nosological categories tend to start from cases in which such judgments, although fallible, can be made with relative plausibility.

Kincaid's Analogy between "Cancer" and "Mental Disorder"

In arguing that psychiatry doesn't need an analysis of the concept of disorder, Kincaid's primary argument is by analogy to oncology that, he has argued elsewhere (Kincaid 2008), neither has nor needs a conceptual analysis of "cancer" and yet gets along fine as a perfectly respectable medical specialty without it: "there are no clear individually necessary and jointly sufficient conditions for being a cancer, but that does not mean oncology is based on a mistake and that its 'status as a medical discipline' is at stake."

Let's be charitable and put obvious disanalogies aside (e.g., for starters, no one is actually challenging oncology's status as a medical discipline based on "cancer" being incoherent, unlike psychiatry!). If one looks closely at Kincaid's analysis in support of this proposition, one finds that he is really arguing that various *theories* and *explanations* and *characterizations* of cancer do not apply in any simple or straightforward way across the hundreds of distinct medical categories that fall within the category "cancer." However, cancer itself does have a standard definition, namely, diseases in which, due to uncontrolled cell division, a tumor spreads to other tissue: "Cancer is a large group of diseases that can start in almost any organ or tissue of the body when abnormal cells grow uncontrollably, go beyond their usual boundaries to invade adjoining parts of the

body and/or spread to other organs" (World Health Organization 2020); "A term for diseases in which abnormal cells divide without control and can invade nearby tissues" (National Cancer Institute 2018); "Cancer is the name given to a collection of related diseases. In all types of cancer, some of the body's cells begin to divide without stopping and spread into surrounding tissues" (National Cancer Institute 2015). Indeed, Kincaid himself seems to agree with this definition that unites the category of cancer: "Finally, I should note that there are over 100 different forms of cancer, and what they at most all have in common is uncontrolled cell division sufficient to cause a health problem" (2008, 373). That's nothing to sneeze at as a useful and theoretically interesting reference fixer.

Kincaid's immersion in fascinating details that vary from cancer to cancer or do not distinguish cancer from noncancer seems a case of missing the forest for the trees. For example, arguing against the importance of the normal/dysfunction distinction, Kincaid observes that "the notion of 'normal' functioning of cells and tissues plays little role in identifying cancer because most tumors cells are normal" (2008, 373). But, first, the point of identifying a disorder is to identify the dysfunction responsible for the symptoms, so the normal cells in a tumor are on a first pass not relevant to that task, whereas a breakdown in regulation of cell growth by other cells is a relevant dysfunction. Second, the "uncontrolled" in Kincaid's own characterization is an implicit marker for "pathological"; obviously, there are endless "controlled" (i.e., biologically designed) rapid or extensive cell divisions, from a fetus's growth into an adult to the formation of a scab in a wound, so, contra Kincaid, the definition must contain an implicit reference to a needed baseline of "normal."

However, fruitful as "uncontrolled cell division that spreads" may be as a current reference fixer, a conceptual stickler has to say that this standard "definition" can't be right as a conceptual analysis. Cancer was known and named in ancient times long before it was known that the body is composed of cells. Hippocrates (460–370 BC) used *carcinoma,* Greek for "crab," to describe the projections of tissue and blood vessels reminiscent of a crab's shape that occurred as a tumor spread; the Roman physician Celsus (28–50 BC) translated *carcinoma* into the Latin word for crab, "cancer"; and the Greek physician Galen (130–200 AD) described the growth of tumors using the term "oncos" (Greek for "swelling"), the root of our term "oncology." Since then, there have been many theories of cancer having nothing to do with uncontrolled cell replication. We could conceivably wake up tomorrow and find out that we were wrong about cell division dysfunction and that one of those other theories of cancer is correct, and that would be a discovery about cancer and not a mere semantic manipulation. Cancer was long thought to be one disorder occurring at different locations in the body but now is thought to be many disorders due to diverse dysfunction etiologies, but such issues are not part of what "cancer" means because those are issues about cancer that are being decided empirically.

So, we have here an essentialist theory of cancer masquerading as a meaning, in the way that "H_2O" might be said to be what we mean by "water" when of course it is a theory of water. Lots of people throughout history and even nowadays understand the meaning of "water" without having any idea about H_2O, and entire technical and scientific hydrological disciplines existed before the discovery that water is H_2O. Similarly, cancer was a medical domain of theory, research, and treatment before the cell theory.

"Cancer" does have a meaning that determines its reference, anchored in a certain history. That meaning is more abstract than various theories about cancer and various proposals for how to differentiate cancer from other disorders. Very roughly, one might describe the meaning this way: if one looks back to the kinds of lesions and tumors that caught the eye of physicians from at least ancient Egyptian and Greek times on, they shared the property of inexorable tissue expansion of a tumor; even the Egyptians, who cauterized tumorous breast lesions, already observed that there is no curative treatment that stops the swelling of these masses. Let's call that recognized base set "*those tumors*." "Cancer" means "tissue that pathologically expands into other tissues due to the same kind of dysfunction as the ones underlying *those tumors*." Of course, this is rough and subject to revision in various ways. However, medical science has since established to our satisfaction that the processes underlying *those tumors* crucially involve various forms of dysfunction in which there is pathologically unregulated cell replication, and so as a general essentialist characterization of a pathological natural kind, cancer is "defined" as pathologically unregulated cell division that threatens harm through invasion of tissues. That there are over 100 known forms of this category of pathology does not diminish the importance of this "definition." Indeed, this very definition requiring an invasive nature is cited and deployed when borderline conditions come up for debate among oncologists, as in the recent debates over the diagnostic status of ductal carcinoma in situ, with the possibility raised of there being unknown differentiators between potentially invasive (and thus cancerous) forms and noninvasive (thus not really cancer) forms.

The "uncontrolled cell division" essentialist characterization of cancer, on which the World Health Organization (2020), National Institutes of Health (2015), and Kincaid all agree, delineates a broad natural kind that is the target of a medical specialty. Nonetheless, Kincaid insists that disorders are not natural kinds (see my reply to Lemoine in this volume for further discussion of essences and natural kinds). In fact, many categories of disorder are not initially natural kinds, but nosology tends to get reassembled to preserve natural kinds of etiologies. Once breast cancer is understood to be due to several different sets of mutations or multiple sclerosis as several different pathways to autoimmune neurological dysfunction, those distinct dysfunctions are gradually seen as separate diseases falling under the locational category "breast cancer" or the broader descriptive category "multiple sclerosis," respectively. Disease categories do not generally spring full blown as natural kinds, but they are by nature in search of

natural kinds. Thus, the seemingly incoherent locution that a category of disorder is in fact several disorders.

Whose Intuitions Is the HDA Trying to Explain?

Kincaid, like some other critics, raises the question of who is to be included in the target community of those whose judgments are to be explained by the HDA's conceptual analysis: "We should first ask who gets to vote in the game of definition and counterexample that is to define 'mental disorder.'" Well, the initial community of clinicians, nosologists, and other professionals is pretty obvious. In any event, Kincaid knows well that a scientific theory and its target domain and boundary conditions tend to evolve together to optimize explanatory power and evidential support. Thus, precisely specifying a preset target community is not necessary. However, the intuitions that are used in the HDA's analyses are remarkably widespread, as nosologists like Spitzer (1999) observe, and there is no deep problem about identifying a target community of the analysis. Actually, I think the notion of the target community is a bit more complicated and interactive than that, analogous to the target domain of a scientific theory. I am after one particular meaning of "disorder," and I often write as if the target community has only that one meaning, but that is of course an idealization and a methodological artifact. The "community" is in effect a construct of those dispersed throughout the professional and lay communities who share a certain widespread concept of disorder that is a salient one among the many in circulation and has certain properties that make it important in scientific research and lay debate. Critics in this book, like many others, raise the obvious objection that there are many different meanings for "disorder" depending on the context (see my reply to Murphy on multiple meanings). That is a great point in general, but it is not germane here because in this case, the context of the analysis of mental disorder as I approach it is specified, and the resulting target concept is relatively determinate. Although the HDA is a conceptual analysis, the analysis of mental disorder in the context of our time is also what might be thought of as a transcendental argument that tries to identify whether there is a widespread concept of medical disorder that makes it possible to assert certain conclusions about mental disorder that are widely believed in the psychiatric and lay communities. These transcendental considerations include, most importantly, the following: (1) psychiatry is (in part, or at its core) a branch of medicine; correspondingly, (2) at the conceptual level, mental disorders are disorders in the same sense in which physical disorders are disorders; (3) there is an in-principle distinction between mental disorder versus social deviance and socially disvalued traits; and (4) mental disorder is at least in part a factual scientific concept that allows scientific research to bear on whether or not a psychological condition is a disorder. If an analysis of a widespread understanding of the concept of mental disorder satisfies these criteria and thus explains how it is possible for

someone to believe these theses, it has met the antipsychiatric challenge and justified the beliefs at the heart of modern psychiatry.

This agenda is part of the reason I have not pursued the "harm" component as thoroughly as the "dysfunction" component (for other reasons, see my reply to Cooper in this volume). The value component presents urgent and important challenges. However, differences over nuances about the value component of "disorder" do not present an existential challenge to psychiatry, whereas in light of antipsychiatry, the "dysfunction" component does present what amounts to an existential issue for the standard view of psychiatry as a nonoppressive medical discipline. Many critics argue that "dysfunction" itself in fact harbors value assumptions. I don't think that evolutionary dysfunction hides any ineliminable value assumptions, but one can see why in the end this does not really matter as long as a factual component that can be studied scientifically apart from value assumptions can be extricated from "dysfunction." In effect, "dysfunction" is a placeholder for the factual element in "disorder," whatever it may be, that explains how we can scientifically show that some negatively valued conditions are disorders and some are not. In effect, all value considerations simply can be relocated to the "harm" component. But, again, I think a sheerly factual component is adequately identified as evolutionary dysfunction.

Kincaid also asks, "Do the intuitions of those outside the psychiatric professions count?" This is a point on which Christopher Boorse and I diverge; he limits the relevant community to pathologists, whereas I include professionals and laypeople. Obviously, professionals know much more than do laypeople about disorders, but do they have a different concept from laypeople? The continuity of the recent debates about the validity of proposed *DSM-5* diagnostic criteria across professional and lay contexts suggests that there is a shared concept. Here, my essentialist perspective plus the aforementioned evidence of continuity of discussion inclines me to follow other essentialist writers and to see ordinary concepts as continuous with scientific concepts. Here is Hilary Putnam on this issue:

> Ordinary language philosophers … tend to compartmentalize the language; the presence of water in the physical theory ('Water is H_2O') is held to involve a different use (i.e., a different sense) from the 'ordinary use'. … This compartmentalization theory seems to me to be simply wrong. Our language is a cooperative venture; and it would be a foolish layman who would be unwilling to ever accept correction from an expert on what was or was not water, or gold, or a mosquito, or whatever. … Ordinary language and scientific language are different but interdependent. (Putnam 2015, 361–362)

And here is Keith Donellan, in what is in some ways a critique of Putnam's account, nonetheless agreeing with him and elaborating this point:

> The Kripke-Putnam theory offers an answer to an important puzzle about the relationship of vernacular kind terms and scientific discovery. We seem willing to tailor the application of

> many of our vernacular terms for kinds to the results of science and if necessary to allow our usual means of determining the extension of these terms to be overridden. There is, for example, a product on the market composed half of sodium chloride and half of potassium chloride. It looks like and tastes like ordinary salt. In most ordinary circumstances—in talking about how much to put in the stew, for example—we would be happy to call this product "salt" even if we knew its chemical composition. But if pressed to say whether this product is "really" salt, I think we would, if we know some elementary chemistry and the chemical composition of the product, concede that it is only half salt. To take a couple of more examples, I would give up calling a stone purchased as a diamond a 'diamond' if assured by experts that it did not possess a certain crystalline structure of carbon and I am prepared to be corrected when what I take to be a wolf in a cage at the zoo turns out to be identified by zoologists as being of a quite distinct species. (Donnellan 2014, 180–181)

The judgments of disorder versus nondisorder and the considerations in defending one or the other judgment in a controversial instance manifest equally impressive continuity across lay and professional communities, justifying the assumption that one analysis is likely to explain the conceptual underpinnings of both professional and lay disorder judgments. As with many other essentialist categories subject to scientific exploration, disorder and nondisorder appear to retain the same meaning across lay and professional judgments.

Kincaid's Approach to Mental Disorder

Kincaid says, "The alternative approach I favor denies that we must have a clear conceptual analysis of disorder in order to understand the practice of psychopathology research. … Instead, I think that the ideal for the sciences of psychopathology is to establish the existence of objective, explanatory classifications." The "instead" here is mystifyingly general. Every science attempts to establish objective, explanatory classifications. So, we are left with the question, what makes it the case that a system of objective, explanatory classifications is a system of nosology, that is, a system of mental disorder classifications, versus some other kind of classification? Remarkably, search as one might, Kincaid never answers this question.

Despite eschewing any conceptual analysis of "mental disorder," in order to argue that the HDA fails, Kincaid is forced to offer his own account of the distinction between disorder and nondisorder. He acknowledges the usefulness of some of my analyses that distinguish specific disorders from superficially similar normal reactions, yet wants to deny that those contributions are tied to the HDA's account of the concept of disorder and so attempts to provide an alternative account of the usefulness of the distinctions I have made. He argues that rather than considering my analyses to identify harmful dysfunctions versus biological design, "the question is whether these conditions allow for objective, explanatory classifications, a question that makes no essential judgment

about evolutionary considerations." The problem, of course, is that objective ways of classifying things are a dime a dozen, and most of them don't impact the disorder-nondisorder distinction. For example, when writing about cancer, Kincaid explains, "Oncology, like the rest of medicine, studies and treats conditions that it can objectively and reliably identify and conditions where it can predict and alter the course of development in ways that people perceive to improve their lives." Agreed, but as alluded to by the phrase "like the rest of medicine," this characterization is a non sequitur if one is asked to characterize oncology because it does not differentiate oncology from proctology or, we now see, from psychiatry, for which Kincaid gives a more or less identical account.

One expects to get such an account in the final section of Kincaid's paper, in which he "sketches an alternative pluralist view of psychopathology that makes the search for objective explanatory classifications of psychopathology paramount, a goal inspired by and consistent with Wakefield's insightful critique of psychiatric practice." It turns out that the sketch just repeats what he has already said, namely, that psychopathology is distinguished from normality by being objectively characterized, using any theoretical means available: "I would argue that finding malfunctions in evolutionary mechanisms or breakdown of roles in a complex system are just valuable means to the end of getting objective, explanatory classifications of behavior that psychiatry and related disciplines study and treat."

Kincaid's most detailed statement of his view is the following: "the ideal for the sciences of psychopathology is to establish the existence of objective, explanatory classifications. … *Objective* classifications as I use the term are ones that put individuals into classes based on real differences in facts about those individuals. … *Explanatory* classifications as I am using the term are those that ground regularities and causal relations."

However, both normal and disordered categories can be objective and explanatory, and distinctions between two normal properties or between two disorders can be as objective and explanatory as a distinction between a disorder and a normal-range condition. So, the fact that a classification is objective and explanatory does not remotely make it an adequate classification of normal versus disordered conditions. Kincaid, avoiding any mention of biological design, has nothing at all to say about how that specific distinction is to be made.

A natural question is, if we take this substantively vacuous route, how do we fend off the antipsychiatric critique that all we are doing is creating spurious medical disorder categories to justify intervention into socially undesirable psychological features? Kincaid's answer is that we can refute social constructivism without any reference to evolutionary theory of functions and dysfunctions simply by using objectively characterizable differences to distinguish between disorder and normality: "What we need to show is that the psychiatric-related disciplines can produce objective and explanatory categorizations of behavior, ideally ones that lead to successful treatments. … For

example, the Big Five personality classification system relies on reliable and psychometrically validated measures; scores on those measures predict differences in behavior. Here psychological phenomena are classified in objectively grounded ways that refute pure social constructivist stories."

This response reveals some serious confusions. The objective evidence for the Big Five personality traits might be used to refute social constructivism about personality traits, but it has no bearing on social constructivism about disorder. More generally, the point of the social constructivist attack on psychiatry is not that there is no objective difference whatever between the people psychiatry places in categories of disorder versus nondisorder—there could be lots of objective differences, such as those associated with social deviance—but rather that whatever differences there are do not actually imply medical disorder versus nondisorder. It's the medicalization that is claimed to be spurious, not necessarily the group itself that is being medicalized. Thus, contra Kincaid, to refute social constructivist antipsychiatric claims, one must have an understanding of what it is to be a disorder, and this is one reason why the conceptual analysis of disorder became so important in the wake of the antipsychiatric movement.

As part of his "objective differences" approach to the distinction between disorder and nondisorder, Kincaid suggests that evolutionary dysfunction is just one objective indicator that might be used and neurobiological typical functioning another: "psychiatric research should get on without the concept of a disorder. If we can find compelling evidence of malfunctioning evolutionarily selected psychological mechanisms, then it would have a role in those cases. I also find it plausible that some behavior that gets labeled pathological involves a breakdown in the normal operation of cognitive and neurobiological systems, the idea promoted by competitors to Wakefield's analysis of function." Kincaid here begs the question by not explaining what it means to have a "breakdown in the normal operation of cognitive and neurobiological systems" as contrasted with "malfunctioning evolutionarily selected psychological mechanisms." We are learning that neurobiological mechanisms operate in highly idiosyncratic ways so that a sheerly statistical notion of "normal" neurological functioning won't do (Paulus and Thompson 2019). Kincaid appears to accept a statistical definition of pathological neurobiological performance, but generally, "something has gone wrong" is not be the same as "statistically unusual"; gum disease afflicts the vast majority of humans around the world, yet is considered a disorder, whereas lactose tolerance is statistically unusual among the human race, yet no one thinks it is a disorder because it is believed to be an adaptation in some groups to the availability of milk that resulted from the domestication of animals. As far as I can tell, the only way to distinguish normal neurobiological functioning from a breakdown in such functioning in the sense relevant to disorder judgments is by whether neurobiological systems are performing as they were biologically designed to perform. Thus, once placed within an HDA framework, I see no tension whatever between the evolutionary and neurobiological levels of explanation

of function and dysfunction and would argue that they are complementary (for this argument, see my reply to Gerrans in this volume).

Are There Naturally Selected Disorders?

Like many other critics (see my replies to Cooper and to Garson in this volume), Kincaid argues that some conditions we are inclined to label as disorders could be naturally selected, falsifying the HDA. He thus claims there are "reasons to doubt that evolutionarily based dysfunction accounts fit with the kind of (reasonable) intuitive judgments that Wakefield wants to make about which symptoms constitute disorders and which do not. The problem is that there are plausible evolutionary stories where a wide range of behaviors that we are inclined to call disorders turn out to be the products of evolutionarily selected mechanisms." Focusing on depression and anxiety, he offers three scenarios in which this might occur.

Before examining Kincaid's three scenarios, it is important to keep in mind that what we are inclined initially to say is not always what we are inclined to say once we discover some condition is biologically designed. For example, the long-time general inclination to consider fever a disorder was based on the view that fever is a dysfunction caused by the toxic effects of illness, but when it was established that fever is a biologically designed defensive reaction, this inclination changed and we were no longer inclined to call fever itself a disorder. (Only when we think the fever mechanism itself has gone out of control and beyond its biologically designed parameters do we then consider it a disorder.) So, in considering Kincaid's stories, we have to consider what we are inclined to say about the diagnostic status of the condition in question if we believe one of Kincaid's stories to be true. In fact, I will argue, his three stories are explanations for what we would take to be normal variation, not disorder.

Disorders as Disproportionate Responses

The first of Kincaid's three stories that are supposed to show that disorders can be biologically designed is: "Success under natural selection requires a trade-off between costly unnecessary responses to threats to reproduction and between costly failures to detect such threats. Where the threat to reproductive success is death, it is not hard to imagine that natural selection would err on the side of false positives. A one strike and you are out threat would seemingly produce traits that produce lots of false positives in reacting to such threats, given the extreme consequences of a false negative." That is, Kincaid is arguing that there are cases in which "psychological mechanisms producing false positives would be fitness improving." With all of this, one can agree: to avoid disaster, we do seem to be biologically designed to be a vigilant, anxious, sadness-prone species.

However, is this aspect of our biologically designed nature really considered a disorder? As he notes, I argued that proportionate responses are generally normal in the face of *DSM*'s misclassification of such responses as disordered. However, the additional fact that disproportionate reactions often can be normal has long been embraced by evolutionary-minded nosological theorists and is consistent with common intuitions. Randolph Nesse (2001) has even dubbed this kind of defensive "overkill" due to the need to avoid a fatal mistake the "smoke detector principle." The name derives from the fact that, although having the purpose of warning of fires, one's smoke detector alarm often all-too-irritatingly goes off when there is no fire due to all sorts of other smoky stimuli (e.g., in my apartment, when we are cooking fish). This nuisance is not considered a malfunction of the smoke detector but rather a reflection of the fact that the detector is set to be on the sensitive side to detect smoke in order to avoid false negatives because just one real fire that is missed can be fatal. Kincaid is quite right that the same logic applies to the natural selection of many of our defensive mechanisms that are triggered in circumstances in which the target threat is not in fact present. This is true as well of physical defenses such as fever, which is one reason we often can treat fever and lessen its discomforts without substantially worsening the illness it is fighting, because fever is designed for a worst-case scenario and generally is an overreaction to what is actually needed in a given case of illness.

In mounting this argument, Kincaid ignores the single question that matters. To put it in smoke detector terms: of course, smoke detectors do break (they also run out of battery power, which I leave aside but has some of the same consequences), either going off incessantly or randomly without a relationship to ambient smoke of the kind and level they are designed to detect or not going off at all despite smoke. So, the crucial question that Kincaid fails to address is: what is the distinction that virtually everyone intuitively makes between a normally functioning but overreacting-to-fish-cooking smoke detector and a broken smoke detector? Clearly, it has to do with how the smoke detector is designed to function, and it includes the designed disproportionality within normality, not within malfunction. Perhaps a person unfamiliar with smoke detectors might ask whether its alarm going off while cooking fish means it is broken, but the answer would surely be "no." The smoke detector is working as designed, even though that leads to false positives relative to the detection of threatening fire. Yet, if the smoke detector was chronically triggered by nonsmoke events or the slight smokiness of local air pollution, I would start to suspect that it might be broken because it is not performing as it was designed to perform. It is no longer performing the function of sensitively warning of even a remotely possible fire based on an alarm being triggered by a certain level of ambient smoke particles but simply causing senseless disruption in response to no meaningful stimulus at all in terms of detection of fire.

Analogous to the role of inferences about human design in distinguishing broken versus mistaken smoke detector alarms, inferences about biological design play a

similar role in drawing the distinction between disorder and nondisorder in anxiety and depression. For example, in the case of anxiety, the malfunctions I mentioned above in the smoke detector—random or incessant alarms unrelated to significant smoke or lack of any alarm at all—are analogous, in anxiety disorders, to generalized anxiety disorder, panic disorder, and forms of personality disorder, respectively. Anxiety disorders such as generalized anxiety disorder generally go well beyond any kind of "vigilance" explanation that would fall under the smoke detector principle and have no use at all given that they do not distinguish any threatening situations from others.

Regarding depression, it is for these precise reasons of the logic of disorder that the classic "prototype" cases of pathological depression are, first, "out of the blue" sadness unrelated to any actual loss or, second, depression that is so deep with such severe pathoindicative symptoms (e.g., psychotic ideation, psychomotor retardation), or so enduring (e.g., continuing well beyond the end of any possible threat and unresponsive to positive changes in the triggering situation), that no plausible "false-positive protective" explanation seems capable of explaining the relationship between the trigger and the reaction. These fuzzy cut-points are where the hypothesis of the occurrence of a dysfunction becomes plausible.

In other words, Kincaid's objection is based on a set of incorrect assumptions about what we are inclined to call disordered. Just like my smoke detector, we understand biologically designed reactions to have a wide range and multiple biological-design (i.e., natural-selective) rationales for how they are designed. We can understand that for obvious reasons, we might be biologically designed to react with immediate fear to all snakes, including (disproportionately) harmless ones. Indeed, in my book with Allan Horwitz on anxiety disorders (2012), we argue that anxiety is biologically designed to be disproportionate in many instances precisely due to the dangers of missing a threat to one's life and that the proportionality principle has less applicability than in depression. It is when reactions go beyond any such explanations that we are inclined to call the underlying mechanisms dysfunctional and, if harmful, the condition a disorder.

Disorders as Mismatches between Organism and Environment

Kincaid's second story in defense of biologically designed disorders is: "The prospect that fitness-enhancing psychological mechanisms in the Pleistocene may be invoked in maladaptive ways in complex industrial societies in which they did not evolve." That is, Kincaid invokes the "mismatch" approach to disorder according to which one "finds the roots of disorder in past evolved mechanisms that have to deal with the complexities of modern society. … The idea is that modern society provokes in abundance naturally selected mechanisms producing depressive and anxious systems."

Kincaid is here again missing a basic distinction between what we are and are not inclined to call a disorder. It is of course true that problematic environments, especially

those with chronic or traumatic stress, can cause disorders. However, that is because they create internal dysfunctions that become independent of the toxic environment. The fact that an individual has a negative reaction to a problematic environment mismatched to her biological design is not in itself considered a mental disorder. To take an extreme case of design-environment mismatch, it is not a disorder to be unable to breathe underwater, even though this limitation can kill you. It is not a mental disorder to desire infidelity in an environment that heavily punishes such desires, or to desire to eat fat and sugar in an environment where the novel easy availability of these high-calorie treats is problematic for long-term health, or to have a bothersome fight-or-flight reaction to the many anxiety-provoking situations that occur daily in a mass society. I do understand that it is very tempting to add chronic individual-environment mismatches that cause chronic misery to harmful dysfunctions under the disorder category. However, aside from the fact that that is not how intuitions about disorder work (as the above examples illustrate), there is the problematic outcome that if one locates mismatches under mental disorder, then psychiatry engulfs control of social deviation in which individuals' natures are mismatched to social demands (e.g., Soviet dissidents' longing for freedom was mismatched to their social environment). As the *DSM-5* definition of mental disorder indicates, "Socially deviant behavior (e.g., political, religious, or sexual) and conflicts that are primarily between the individual and society are not mental disorders unless the deviance or conflict results from a dysfunction in the individual" (American Psychiatric Association 2013, 20). Adding mismatches to the disorder category undermines the integrity of the concept in a way that legitimizes antipsychiatric objections.

Kincaid presents me with the following question: "A few miles from where I live, there are over a million people cramped into wall-to-wall corrugated metal shacks without running water and sanitation. Most live on less than $2 a day. Probably 50% are unemployed. … Wakefield argues that depressive symptoms in response to a job loss should not be counted as a disorder. However, what if the job loss is a repeated or a permanent part of life as it is for the residents of these townships and the response is permanent depressive symptoms? Should we deny that they have a depressive disorder? … Such examples should be a chance for clarification of his views."

I believe that I have always been crystal clear on this point: being chronically depressed due to chronic losses is not a mental disorder but a terrible misfortune, just as being chronically anxious due to chronic real threat is not a disorder. In some attenuated sense, such chronic negative responses to real environmental situations may be considered a "mental health problem" but not a disorder. I say this not because this is what I think but because I believe this is how laypeople and professionals tend to judge disorder under such circumstances, I have done empirical research on clinical judgment of conduct disorder in relation to chronic environmental triggering situations that suggests reactions to chronic stressors are not seen as disorders (Wakefield, Pottick, and Kirk 2002). Moreover, Kincaid's suggestion that the individuals in the horrifyingly

afflicted community he describes should be understood as having depressive disorders due to their chronic reactions to chronic deprivations is in its own way horrifying, because it implies that the place where things are "going wrong" in creating this disaster is in the individuals, and the likely conclusion is that what is needed is a massive medical intervention such as provision of antidepressants, whereas what he describes is clearly a social problem inflicted on presumptively normal individuals that requires first and foremost a social response. (Drawing this sort of distinction is not merely a theoretical exercise; for example, United Nations relief agencies often must decide after a disaster whether the priority for limited resources in dealing with stress reactions is psychiatric treatment or environmental intervention [World Health Organization 2015].) Of course, some individuals in such circumstances do develop mental disorders and do need medical treatment. However, the crucial implication of the nondisorder judgment for the majority is that if improved circumstances and jobs are provided to these individuals, it is likely the distress will recede because there is no dysfunction in sadness-generating mechanisms. No such implication follows from the disorder attribution.

These judgments about disorder, the HDA emphasizes, are surrogates for causal hypotheses and consequently are important to scientific research. Although the experience of sadness and at one level the mechanisms involved in generating those experiences may be quite similar in the individuals in the community Kincaid describes and in those with out-of-the-blue chronic melancholic depression (as magnetic resonance imaging studies of disordered and normal sadness suggest [Mayberg et al. 1999]), one would expect to see divergent causal pathways at a deeper level. I have argued similarly that *DSM*'s invalidly lumping adolescent delinquency with true conduct disorder led to confused research outcomes as well as misplaced social priorities (Kirk et al. 1999; Wakefield et al. 2002; Wakefield et al. 2006).

Disorders as Extremes on Dimensions

Kincaid's third story of purported biologically designed disorder is: "The general fact that evolution produces traits with a wide reaction norm, raising the prospect that extremes of human behavior that we call pathological are the distributional tails of normal traits." Kincaid elaborates: "A third evolutionary approach arguing that psychopathology is the standard functioning of naturally selected traits points out that biological traits can have a wide reaction norm. Seemingly normal traits in common environments can exhibit extreme deviations from the average, given subtle changes in the developmental environment. The claim thus would be that major depression is just the tail of the expression of normal, presumably adaptive, trait of sadness. This is the kind of view advocated by the psychometric tradition that wants to treat psychopathology as a continuous trait and replace talk of disorders and psychopathology with talk of abnormal behavior."

The psychometrician's idea, popular in nosology at the moment, is that one scales symptoms or traits on a severity dimension, and we just label the extreme as disorder. Regarding evolution, the idea here is that naturally selected mechanisms generally yield not singular categorical traits but dimensions of symptoms or traits with continuously distributed variations in severity for a variety of reasons (such as interaction with other mechanisms and genes in the individual and with the environment), and thus all the points along the dimension are naturally selected, yet the extreme ones are labeled disorders. (There are also interactions between the fetus and the prenatal environment that alter later outcomes; see Garson's paper in this volume and my response.) If indeed some of the more extreme of the outcomes of a selected mechanism are classified as disorders, the argument goes, we have here disorders that are naturally selected.

This story is based on a manifestly invalid form of reasoning. To see why, consider instances of heterozygous advantage in which one copy of a gene is advantageous and selected for, whereas two copies cause a genetic disease. This is known to occur in single-gene mediated physical disorders such as cystic fibrosis and sickle cell anemia, but it is looking like this sort of situation could be quite a widespread phenomenon among polygenic disorders and even among mental disorders. For example, it appears that the risk genes for autism show evidence of positive selection for various cognitive capacities, yet in certain combinations of these, risk genes yield a devastating disorder (Polimanti et al. 2017). Phenomena such as heterozygote advantage explain why the genes conferring risk for mental disorders are so common and have not been eliminated from the population.

The clearest model of this situation is sickle cell anemia. Consider two individuals with sickle cell trait—a combination of one sickle cell gene and one standard gene—who are considered normal in a malaria-endemic environment in which sickle cell trait protects against malaria. Consider further that this trait that has been *selected for* (and that the sickle cell trait is in fact somewhat more fit than two standard genes). Let's imagine that these two individuals have children. Normal genetic distributional mechanisms operating on their genes will yield a "continuous" distribution along the genetic dimension of number of sickle cell genes and along the phenotypic dimension of degree of red blood cell sickling. (Actually, of course, the genetic distribution is not literally continuous, but neither are any other genetic distributions; they only have a lot more discrete steps, so the principle is the same.) So, this is an instance satisfying Kincaid's premises—namely, a naturally selected gene and a normal mechanism distributing intensities of that gene. Yet, his conclusion does not follow; the various outcomes are not considered equally naturally selected in their own right, and in fact, the tail end of the distribution that is considered a clear disorder—sickle cell anemia—is considered anything but naturally selected in itself. (This is the same error made by De Block and Sholl in their chapter in this volume; for a different worked-out example concerning hyperemesis gravidarum, see my reply to them in this volume.) This situation with regard to sickle cell anemia is of course due to the phenomenon of heterozygote advantage.

The moral of the sickle cell anemia story is that from normal naturally selected mechanisms for distribution of genotypes and consequent phenotypes, some parts of the distribution may be the ones that are responsible for the overall mechanism's natural selection, whereas other parts—due to processes of, or analogous to, heterozygote advantage—may be individually nonselected disordered variants, like sickle cell disease. The two-standard-gene configuration has a positive natural selection history, and sickle cell trait—one standard gene and one sickle cell gene—also has a positive selection history in malarial environments in which sickle cell trait arose, so both can be considered biologically designed, whereas sickle cell anemia—two sickle cell genes—came about as a nonnaturally selected and severely disordered side effect of those positive selection processes. One can consider sickle cell anemia a population-level trade-off for the enhanced fitness of sickle cell trait, but disorders are judged at the individual organism level, and for those with sickle cell anemia, there is no fruitful trade-off for the failure of multiple bodily systems and frequent early death.

Although the situation is much more complex in mental disorders such as depression, the critical point is the same. Depression, I agree, is a naturally selected defensive response that appears in varying degrees. However, the extreme of depressive feelings, which we label major depressive disorder, is judged a disorder because the nature, length, and independence from context of the symptoms (described earlier) suggest not an extreme of a naturally selected reaction but a breakdown in that naturally selected reaction. As in the distinction between pathological depression versus normal sadness, the HDA predicts that we judge disorder at the extreme of a dimension only if we believe that the extreme involves a dysfunction, either by being so severe as to counteract or override whatever effect the trait was selected for or by otherwise causing a dysfunction as collateral damage. Given how we think about disorder, if one really believed the evolutionary scenario as Kincaid presents it, the conclusion would be not that there are naturally selected disorders at the extremes of naturally selected dimensions but Plomin's (2003, 2018) much more radical and implausible conclusion that there are in fact no disorders at all, only dimensions (for more on this, see my response to De Block and Sholl in this volume).

References

American Psychiatric Association. 2013. *Diagnostic and Statistical Manual of Mental Disorders*. 5th ed. American Psychiatric Association.

Baller, E. B., S.-M. Wei, P. D. Kohn, D. R. Rubinow, G. Alarcón, P. J. Schmidt, and K. F. Berman. 2013. Abnormalities of dorsolateral prefrontal function in women with premenstrual dysphoric disorder: A multimodal neuroimaging study. *American Journal of Psychiatry* 170: 305–314.

Donnellan, K. (2014). Kripke and Putnam on natural kind terms. In *Essays on Reference, Language, and Mind*, J. Almog and P. Leonardi (eds.),179–203. Oxford University Press.

Dubey, N., J. F. Hoffman, K. Schuebel, Q. Yuan, P. E. Martinez, L. K. Nieman, et al. 2017. The ESC/E(Z) complex, an effector of response to ovarian steroids, manifests an intrinsic difference in cells from women with premenstrual dysphoric disorder. *Molecular Psychiatry* 22(8): 1172–1184.

First, M. B., and J. C. Wakefield. 2010. Defining 'mental disorder' in *DSM-V*. *Psychological Medicine* 40(11): 1779–1782.

First, M. B., and J. C. Wakefield. 2013. Diagnostic criteria as dysfunction indicators: Bridging the chasm between the definition of mental disorder and diagnostic criteria for specific disorders. *Canadian Journal of Psychiatry* 58(12): 663–669.

Horwitz, A. V., and J. C. Wakefield. 2012. *All We Have to Fear: Psychiatry's Transformation of Natural Anxieties into Mental Disorders*. Oxford University Press.

Kincaid, H. 2008. Do we need theory to study disease? Lessons from cancer research and their implications for mental illness. *Perspectives in Biology and Medicine* 51(3): 367–378.

Kirk, S. A., J. C. Wakefield, D. Hseih, and K. Pottick. 1999. Social context and social workers' judgment of mental disorder. *Social Service Review* 73: 82–104.

Mayberg, H. S., M. Liotti, S. K. Brannan, S. McGinnis, R. K. Mahurin, P. A. Jerabek, et al. 1999. Reciprocal limbic-cortical function and negative mood: Converging PET findings in depression and normal sadness. *American Journal of Psychiatry* 156(5): 675–682.

National Cancer Institute. 2015. What is cancer? February 9. https://www.cancer.gov/about-cancer/understanding/what-is-cancer.

National Cancer Institute. 2018. NCI dictionary of cancer terms. April 3. https://www.cancer.gov/publications/dictionaries/cancer-terms/def/cancer.

Nesse, R. M. 2001. The smoke detector principle: Natural selection and the regulation of defensive responses. *Annals of the New York Academy of Science* 935: 75–85.

Paulus, M. P., and W. K. Thompson. 2019. The challenges and opportunities of small effects: The new normal in academic psychiatry. *JAMA Psychiatry* 76(4): 353–354.

Plomin, R. 2003. Genes and behavior: Cognitive abilities and disabilities in normal populations. In *Disorders of Brain and Mind,* M. Ron and T. Robbins (eds.), vol. 2, 3–29. Cambridge University Press.

Plomin, R. 2018. *Blueprint: How DNA Makes Us Who We Are*. MIT Press.

Polimanti, R., and J. Gelerner. 2017. Widespread signatures of positive selection in common risk alleles associated to autism spectrum disorder. *PLoS Genetics* 13(2): e1006618.

Putnam, H. 2015. Reply to Ian Hacking. In *The Philosophy of Hilary Putnam*, R. E. Auxier, D. R. Anderson, and L. E. Hahn (eds.), 358–364. Open Court.

Quine, W. V. O. 1960. *Word and Object*. MIT Press.

Rind, B., and R. Yuill. 2012. Hebephilia as mental disorder? A historical, cross-cultural, sociological, cross-species, non-clinical empirical, and evolutionary review. *Archives of Sexual Behavior* 41(4): 797–829.

Schmidt, P. J., L. K. Nieman, M. A. Danaceau, L. F. Adams, and D. R. Rubinow. 1998. Differential behavioral effects of gonadal steroids in women with and in those without premenstrual syndrome. *New England Journal of Medicine* 338: 209–216.

Spitzer, R. L. 1997. Brief comments from a psychiatric nosologist weary from his own attempts to define mental disorder: Why Ossorio's definition muddles and Wakefield's "harmful dysfunction" illuminates the issues. *Clinical Psychology: Science and Practice* 4(3): 259–261.

Spitzer, R. L. 1999. Harmful dysfunction and the *DSM* definition of mental disorder. *Journal of Abnormal Psychology* 108(3): 430–432.

Vargas-Cooper, N. 2012. The billion dollar battle over premenstrual disorder. *Salon*. https://www.salon.com/2012/02/26/the_billion_dollar_battle_over_premenstrual_disorder/.

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

Wakefield, J. C. 1992b. Disorder as harmful dysfunction: A conceptual critique of *DSM-III-R*'s definition of mental disorder. *Psychological Review* 99: 232–247.

Wakefield, J. C. 1993. Limits of operationalization: A critique of Spitzer and Endicott's (1978) proposed operational criteria of mental disorder. *Journal of Abnormal Psychology* 102: 160–172.

Wakefield, J. C. 1995. Dysfunction as a value-free concept: A reply to Sadler and Agich. *Philosophy, Psychiatry, and Psychology* 2: 233–46.

Wakefield, J. C. 1997a. Diagnosing *DSM-IV*, part 1: *DSM-IV* and the concept of mental disorder. *Behaviour Research and Therapy* 35: 633–650.

Wakefield, J. C. 1997b. Diagnosing *DSM-IV*, part 2: Eysenck (1986) and the essentialist fallacy. *Behaviour Research and Therapy* 35: 651–666.

Wakefield, J. C. 1997c. Normal inability versus pathological disability: Why Ossorio's (1985) definition of mental disorder is not sufficient. *Clinical Psychology: Science and Practice* 4: 249–258.

Wakefield, J. C. 1997d. When is development disordered? Developmental psychopathology and the harmful dysfunction analysis of mental disorder. *Development and Psychopathology* 9: 269–290.

Wakefield, J. C. 1998. The *DSM*'s theory-neutral nosology is scientifically progressive: Response to Follette and Houts. *Journal of Consulting and Clinical Psychology* 66: 846–852.

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

Wakefield, J. C. 1999b. Mental disorder as a black box essentialist concept. *Journal of Abnormal Psychology* 108: 465–472.

Wakefield, J. C. 2000a. Aristotle as sociobiologist: The "function of a human being" argument, black box essentialism, and the concept of mental disorder. *Philosophy, Psychiatry, and Psychology* 7: 17–44.

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

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

Wakefield, J. C. 2006. What makes a mental disorder mental? *Philosophy, Psychiatry, and Psychology* 13: 123–131.

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

Wakefield, J. C. 2009. Mental disorder and moral responsibility: Disorders of personhood as harmful dysfunctions, with special reference to alcoholism. *Philosophy, Psychiatry, and Psychology* 16: 91–99.

Wakefield, J. C. 2011. Darwin, functional explanation, and the philosophy of psychiatry. In *Maladapting Minds: Philosophy, Psychiatry, and Evolutionary Theory*, P. R. Andriaens and A. De Block (eds.), 143–172. Oxford University Press.

Wakefield, J. C. 2014. The biostatistical theory versus the harmful dysfunction analysis, part 1: Is part-dysfunction a sufficient condition for medical disorder? *Journal of Medicine and Philosophy* 39: 648–682.

Wakefield, J. C. 2016a. The concepts of biological function and dysfunction: Toward a conceptual foundation for evolutionary psychopathology. In *Handbook of Evolutionary Psychology*, D. Buss (ed.), 2nd ed., vol. 2, 988–1006. Oxford University Press.

Wakefield, J. C. 2016b. Diagnostic issues and controversies in *DSM-5*: Return of the false positives problem. *Annual Review of Clinical Psychology* 12: 105–132.

Wakefield, J. C., and M. B. First. 2003. Clarifying the distinction between disorder and nondisorder: Confronting the overdiagnosis ("false positives") problem in *DSM-V*. In *Advancing DSM: Dilemmas in Psychiatric Diagnosis*, K. A. Phillips, M. B. First, and H. A. Pincus (eds.), 23–56. American Psychiatric Press.

Wakefield, J. C., and M. B. First. 2012. Placing symptoms in context: The role of contextual criteria in reducing false positives in *DSM* diagnosis. *Comprehensive Psychiatry* 53: 130–139.

Wakefield, J. C., K. J. Pottick, and S. A. Kirk. 2002. Should the *DSM-IV* diagnostic criteria for conduct disorder consider social context? *American Journal of Psychiatry* 159: 380–386.

World Health Organization. 2015. *mhGAP Humanitarian Intervention Guide (mhGAP-HIG): Clinical Management of Mental, Neurological and Substance Use Conditions in Humanitarian Emergencies*. WHO.

World Health Organization. 2020. Health topics: Cancer. https://www.who.int/health-topics/cancer#tab=tab_1.