

10 Can a Nonessentialist Neo-Empiricist Analysis of Mental Disorder Replace the Harmful Dysfunction Analysis? Reply to Peter Zachar

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I have watched with amazement the extensive and diverse contributions Peter Zachar has made to philosophy of psychiatry over time. He has done it in the way that I think is most productive yet rarely pursued, by being immersed in and publishing in both philosophy and psychiatry at once, with close ties to colleagues in both fields. I thank him for his contribution to this volume and his illuminating description of various positions including 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 (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). I especially appreciate Zachar’s recognition that “Wakefield’s model, like many other evolutionary models in psychology, is an interactive and contextual, not a reductionist, model.” Indeed, I have been arguing for years and continue to argue (Wakefield 2017) that mental disorders can occur at the intentional-system level without there being any disorder describable at a purely neurobiological level and that context is critical to evaluating whether a disorder exists (Wakefield and First 2012), points often missed by critics and defenders alike.

However, as with Harold Kincaid’s Quinianism (see Kincaid in this volume and my reply), my reply to Zachar is made more challenging by the fact that he and I have deep differences on broader philosophical issues. Specifically, our differences regarding Zachar’s neo-empiricism versus my essentialist realism go well beyond the present topic of the concept of mental disorder and cannot be fully aired in this interchange. I will comment briefly on a few of the broader philosophical issues Zachar raises in his paper and then focus on his substantive views of the concept of mental disorder and especially on points of divergence between us.

One point of apparent convergence is important to mention at the outset. Judging by his comments early in his paper, Zachar and I agree that a requirement of any successful account of mental disorder—and thus of any account of medical disorder that

encompasses mental disorder—is that it respond to the Szaszian type of antipsychiatric attack on psychiatry’s legitimacy as a medical discipline. We also agree that, whatever else one thinks of the HDA, it does offer such a response. In the course of his paper, in a quest for a nonessentialist account of disorder, Zachar offers several characterizations of mental disorder, and I will apply this test of adequacy when evaluating his various suggestions.

Essentialism versus Neo-Empiricism

I am going to focus mostly on assessing Zachar’s positive suggestions for how to understand disorder from a neo-empiricist perspective. However, before proceeding to those issues, in this first section, I briefly present some background and address some of the comments Zachar makes about the inadequacies of the HDA.

My modest form of essentialism (see my reply to Lemoine in this volume for discussion of modest essentialism) allows for multiple meanings of terms depending on context and on an ontological marker that indicates the type of thing being defined from among the many possible essences identifiable based on any base set. Generally, essentialist definitions take the form “something belongs to this category if it has the same nature as X ,” where X is some base set of observably identifiable instances. The concept then generalizes not along the lines of the observable characteristics of the base set but along the lines of the underlying nature of the base set. In principle, modest essentialism allows that meanings can include neo-empiricist-type observational meanings that don’t refer to any underlying essence. Thus, in some contexts, “depression” can refer to a certain phenomenology and set of experiences that can be normal or disordered, and in others—including typical *Diagnostic and Statistical Manual of Mental Disorders (DSM)* diagnostic contexts—it can refer specifically to a disorder, in which case it has an essentialist loading that requires some inferred dysfunction. If it is eventually established that there are several quite different types of dysfunction that lead to the same symptom syndrome, then there is the option of concluding that depressive disorder as conceptualized fails to be construct valid and is in fact several disorders. “Essentialist” meanings encompass virtually any meaning that refers directly or indirectly to nonobservational properties that explain and unify the members of the category and thus goes beyond neo-empiricist constraints that limit concepts to sets of observational properties. While allowing that terms are sometimes used in neo-empiricist ways, modest essentialism holds that most scientific concepts—and certainly psychiatric concepts such as “mental disorder”—have salient primary essentialist meanings. This is particularly so if one is trying to identify the senses of “function” and “dysfunction” that underlie medical diagnosis.

Zachar uses the standard example of “gold”—eventually identified as the element having atomic number 79—to illustrate an essentialist concept, but this may be misleading

in one respect. “Gold” has an essence that is an actual structural underpinning. Obviously, there is no such substantial real essence shared among disorders such as infections, injuries, poisonings, allergies, and so on. The nature of disorders is endlessly variable, so what unites disorders is not any real-essence commonality but rather the “essential” (nonobservational) property that they are failures of natural functions, and natural functions do have an essential (although not structural) commonality, namely, they are naturally selected effects. Unlike gold, neither natural functions nor their failures share a specific material essence. Their essences are more analogous to the modest quasi-essentialist account of artifact categories such as “chair”; chairs have no material substrate or even physical similarities in common (think of bean bag chairs and tree-stump chairs), but they are chairs roughly because they share the fact that they were designed (or retained, if a natural object) to be a place for someone to sit, which is not an immediately observable property of chairs but nevertheless determines their category. This is quite different from the situation with “gold.”

One must distinguish the concept of medical disorder in general from the concepts of specific disorders. Specific disorders are individuated by the specific dysfunction(s) that cause the symptoms and that constitute the essence of the disorder. Zachar says, “Ancient people also noticed and named behavioral aberrations such as melancholia and mania.” The use here of “behavioral aberrations” to suggest no reference to inferred explanatory properties beyond behavior is tendentious. Ancient people noticed and named lots of behavioral aberrations (e.g., hubris, impiety, erotic love, grief), and they, like us, considered melancholia and mania and other mental disorders to be more than mere “behavioral aberrations.” They even recognized that the very same set of symptoms could represent distinct disorders or even disorder versus nondisorder (e.g., melancholia versus grief or despair over romantic rejection). They already used medical concepts as etiologically inferential theoretical concepts, with disorders sometimes named after the theory popular at the time of the unobservable etiology of the symptom syndrome (e.g., hysteria, melancholia, malaria). Given that a dysfunction might have a unique pathophysiology, this is somewhat closer to the “gold” situation.

Trying to portray the HDA’s essentialism as discordant with *DSM*’s prominent definition of disorder, Zachar claims that “Spitzer’s definition of mental disorder [in *DSM-III*] was a listing of features, not an abstract concept such as *harmful dysfunction*.” In his paper, Zachar reproduces the first few sentences of the definition, and they do indeed focus on the observable harms of distress and impairment of role functioning, and remarkably he then ends the quote, tendentiously leaving out the very next sentence that directly falsifies his claim: “In addition, there is an inference that there is a behavioral, psychological, or biological dysfunction” (American Psychiatric Association 1980, 6).

Zachar cites the epistemological difficulties in knowing what was naturally selected as an objection to the HDA. The HDA is a conceptual analysis aimed at understanding

what we mean by “mental disorder” and has little to do with epistemological issues in identifying mental disorders, other than setting the conditions for something being a mental disorder and thus setting the overall conceptual ground rules for epistemological inquiry. Nonetheless, Zachar objects, “The key problem with the harmful dysfunction model is that it offers limited empirical guidance in distinguishing disorders from nondisorders because identifying objective natural functions depends on conceptual analysis, not factual evidence.” A conceptual analysis gives you the conditions under which a concept applies to an object. Finding out whether the conditions actually apply to a given object is an empirical matter that requires factual claims and cannot be determined by the concept alone.

Zachar argues that the guidance offered by the HDA is not effective because evolutionary function and dysfunction are so difficult to establish: “As argued by Richardson (2007), there is not enough information about the selection pressures that were operating during human evolution, particularly on the evolution of the brain, to support empirically based theories of natural function. Wakefield (2001) contends that careful reasoning can reveal what natural psychological functions exist, but one has to worry that reason unconstrained by evidence can be marshalled to defend many different conclusions.”

Of course, I strongly agree with the last point, and that is why so much of my work is critical of psychiatric diagnosis. However, one has to do more than worry. One has to critically evaluate claims and fight the tendency of societies to interpret their local values as an expression of human nature so as to make social deviance into disorder amenable to interventions using medical power. This is the sort of direct answer to Szaszian antipsychiatry that, we shall see, Zachar’s view fails to muster because, lacking an adequate understanding what it is that society means by “disorder,” he cannot argue persuasively about what society is getting wrong.

Zachar, we saw, cites Richardson’s (2007) critique of evolutionary psychology as showing the flaws in an evolutionary approach to psychology, and there are many good such critiques. I am not a partisan of any particular evolutionary psychological views, other than what I take to be plausible hypotheses about natural selection of psychological features that underlie typical psychopathological categories. Mostly, these critiques, like Richardson’s, deal with issues that do not bear on the broad assumptions about function and dysfunction underlying most major *DSM* or *International Classification of Diseases (ICD)* categories of mental or physical disorder. It is pretty obvious or at least plausible for most of them that something is going wrong relative to biological design. Of course, all such judgments about function and dysfunction are fallible, and we not only can be wrong but often have been wrong about what seems obvious. Nonetheless, looking at Richardson’s book, I could not find even one example in his many critiques of evolutionary psychological hypotheses that would cast doubt on a diagnostic judgment among *DSM*’s categories of disorder.

In any event, what seems epistemically challenging now may not be in the future. For example, there are scientific methods now that did not exist a few decades ago for examining the patterns of genetic loci near a target locus and determining whether it is likely that the target locus was the result of positive selection pressures or not (e.g., Ding et al. 2002; Lind et al. 2019; Polimanti and Gelernter 2017), offering a degree of ability to enter into evolutionary inquiry sans time machine that was unimagined even a few years ago. In philosophy of science, our epistemic position with regard to specific factual questions can change radically and relatively quickly, and this makes it additionally important to keep epistemic and conceptual/ontological issues separate. When the positivists wrote, it was impossible to observe the back of the moon; until very recently, the existence of exoplanets was not empirically establishable; and just a few decades ago, before ultrasound, it was impossible to know the sex of a baby before birth, yet no one thought that these epistemological challenges—all overcome in time—somehow undermined the standard meanings of these concepts or demanded a redefinition pegged to what one could at the time establish.

A problem with Zachar's critique of the HDA's conceptual account of "medical disorder" is that, in the examples he offers, he consistently seems to confuse the speculative theorizing on the basis of known facts with conceptual analysis, which is a wholly different thing. For example: "Horwitz and Wakefield (2012) use a conceptual analysis of what we should and should not be expected to do to identify what lies within our biologically designed, naturally selected range of behaviors. According to them, talking to family members without intense anxiety lies in this range, but handling snakes without intense anxiety does not. ... In this analysis, the distinction between disordered and normal is being made not by discovering an objective dysfunction but by reasoning."

Zachar bewilderingly assumes that the described reasoning must be conceptual analysis, but reasoning is not the same as conceptual analysis. To the contrary, speculative reasoning about theoretical matters based on whatever facts and theoretical assumptions one has at hand occurs in science all the time, and that is not conceptual analysis. It is not the result of a conceptual analysis of "disorder" or "anxiety" that Horwitz and I suggested that intense anxiety about speaking with family members might be a sign that the functioning of social anxiety mechanisms has gone awry; it is a result of pondering what we know about anxiety, how we are biologically designed, how reliant we are on family intimacy and support, and what makes sense in light of what we know about human nature. Given how little evidence we have, admittedly we were forced to judge this issue on flimsy grounds, but we argued that, contra the *DSM*, this is a much more likely indicator of pathology than, say, public speaking anxiety. This sort of "reasoning" is not at all the same as "conceptual analysis."

Zachar says, "The HD analysis cannot, therefore, empirically do what it was proposed to do, *factually* demarcate valid psychiatric disorders from the larger class of problems-in-living." To the contrary, the HDA specifies the kinds of facts (and values)

that demarcate disorders from problems-in-living, which is what it is supposed to do. It is science's, not the HDA's, responsibility to ascertain when the facts apply and thus which conditions actually are disorders.

Zachar treats us to a brief history of the notion of "open concepts." He explains that "open concepts are potentially extendable so that a new measurement *may* also become part of our definition of the concept. An open concept refers to what it is that the different operationalizations of it have in common, but it is not reducible to any of those operationalizations." So, it changes but it is the same. I am not sure this makes sense, but if it does, it can't be right. I (Wakefield 2004) have dealt with this notion elsewhere, showing why it is confused and problematic and why essentialism is a better way to address the problems that open concepts are supposed to address. One problem is that neo-empiricism, even when supplemented by open concepts, yields theory incommensurability, which implies that we can't constructively disagree because, having different beliefs, we don't have the same concepts and so there is no common language in which to conduct the dispute and no common agreed construct about which to have our disagreement. This is the sort of problem that caused philosophers of science to largely abandon neo-empiricism. In the course of his discussion, Zachar illustrates my point. He says, "The discovery that most cases of depression are precipitated by stress in the previous six months may lead us modify the concept of depression one way, and the discovery that cases of depression that lack precipitants are more treatment resistant may lead us to modify the concept (and related concepts such as psychiatric disorder) in another way." He is saying that every change in belief, because it alters how you might empirically test for the construct, alters the meaning of the concept and is thus a change of the concept. Consequently, you never actually discover anything about a construct because the discovery makes it a different construct. But, one can discover something about depression without changing the meaning of "depression," and two people can have different beliefs about the very same entity, depression. Essentialism explained how this is possible and was a breath of fresh air that resolved all these self-inflicted problems of neo-empiricism.

Zachar's "Decline-of-Functioning" Account of Disorder

Zachar expends considerable energy defending a "decline-of-functioning" analysis of disorder. A similar idea was suggested by Lilienfeld and Marino (1995) and rebutted (Wakefield 1999a) by obvious counterexamples to sufficiency (declines that are not disorders) and necessity (disorders that are not declines). Zachar evades such easy rebuttal by asserting that the "decline" criterion is neither necessary nor sufficient, just a strong indicator. When examples don't fit the criterion, he brings in qualifiers in an unsystematic and ad hoc way to fix the problem, such as that the declines must be "unwanted" or that they "represent something being *broken*." However, he ignores

what if any inferential theoretical assumptions, for example, about biological design, might lie behind such qualifiers as “broken.” Later (see below), he even re-describes the criterion as “decline-in-functioning and other statistically abnormal developmental trajectories,” which is a wholly different and much broader notion—many developmental deviations involve no decline at all (see below)—that raises the new problem that a statistical deviation account of disorder opens the door wide to Szaszian anti-psychiatric objections. Zachar emphasizes that the “decline-in-functioning” criterion has the virtue of being an objective fact just like dysfunction (“declines-in-functioning are objective... they are often intersubjectively confirmable”)—but, like the overemphasis on achieving reliability in *DSM-III* at the expense of validity, the fact that one’s criterion is objective is irrelevant if it is not identifying what you are trying to identify.

Of course, in certain contexts, decline in functioning offers persuasive evidence of disorder. However, decline in functioning is common, and it is taken to be a disorder when and only when the decline is taken to indicate harmful dysfunction. There are many forms of decline in function that occur occasionally and are seen as biologically designed parts of life and thus not considered disorders, such as sleep that leaves one unconscious, semi-paralyzed, and periodically hallucinating for about one-third of one’s entire life span; the decline in various areas of physical functioning experienced by women in advanced stages of pregnancy; and the decline in capability due to muscular fatigue after vigorous exercise. Then, there are conditions that are considered disorders and involve no decline in functioning but only a failure to proceed to new biologically designed developmental milestones, such as neurological disorders in which children fail to develop the capacity to sit, walk, or speak or psychiatric disorders such as intellectual disability, classic autism, and schizoid personality disorder. Similarly, the “lifelong generalized” subtype of erectile disorder or orgasmic disorder (which has one specifier, “never experienced an orgasm under any situation”) involves no decline but rather failure to develop biologically designed functioning. Again, lead poisoning need not cause a decline in functioning or even a statistical deviation from the normal range of development but may be a disorder nonetheless by simply preventing full development of potential capacities. And so on. Adding a qualifier like “unexplained” or “unexpected” to “decline in functioning” won’t help because many well-explained and expectable declines in functioning (e.g., when someone breaks an arm or contracts pneumonia) are considered disorders. The very same decline in strength, say, might be a nondisorder with nothing broken in one instance (e.g., due to stopping one’s exercising) and represent something broken because due to a dysfunction in another (e.g., an early stage of a neurological disorder). Zachar says that decline in functioning is not an essence, and of course he is right; it is a descriptive term, and this is why, as he himself observes, it is neither necessary nor sufficient for disorder. It is the type of explanation of the decline that makes a decline of functioning a disorder or nondisorder, and the theoretical notion of dysfunction is necessary to make sense of

the distinctions we routinely make between declines that are pathologies and declines that are normal.

Perhaps most troubling is that Zachar embraces the decline-of-functioning criterion despite the fact that it cannot address the Szaszian challenge. Social deviance and oppressed conditions often involve declines or decrements in functioning as socially defined, and without a theory of natural functions, it is the social perspective that will be relied on for such judgments. Decline of functioning encompasses socially disapproved changes in functioning, opening the door to the pathologization of social deviance. If a teenage student attends a new high school and experiences a steep decline in grades for one reason or another, if someone enters a life of criminality, if a couple begins to have major conflicts that decrease their well-being, or if someone experiences boredom or burnout at their job, these declines in social functioning seem to be candidates for mental disorder according to Zachar's "decline" criterion. This is the Szaszian nightmare realized. The HDA blocks such categorizations because decline in functioning is considered indicative of disorder when and only when it is due to a dysfunction, and such social failings are not necessarily indicative of evolutionary dysfunctions.

A bit later in his paper, Zachar expands on the decline-in-functioning account, listing a series of features that to some extent suggest disorder but presumably are not meant as either necessary or sufficient criteria by themselves or even in combination: "decline-in-functioning and other statistically abnormal developmental trajectories; the presence of reality distortion; suicidal ideation; confusion and other cognitive difficulties; intrusive thoughts; difficult to control impulses and compulsions; agitation, anger, and excitement; excessive anxiety and fear; emptiness and anhedonia; somatic preoccupations; seeming more amenable to the skill set of psychiatry than other medical specialties." This list fails to characterize many disorders, and many conditions that are characterized by these features singly or in combination are not considered disorders, so the list is not explanatorily compelling. My hypothesis is that, singly or in combination, the features on this list will be considered indicative of disorder when and only when they are believed to support an inference to dysfunction. But, because dysfunction is a theoretical concept rooted in an essentialist inference about biological design, neo-empiricist Zachar is unable to test my hypothesis against actual judgments because his philosophical blinders preclude any such theory from being considered. That's too bad, because such tests are easy to do! One might get started by asking questions such as: When is suicidal ideation considered indicative of disorder and when is it not (e.g., Masada)? When is reality distortion considered suggestive of disorder and when is it not (e.g., "love is blind")? When are problematic conditions more amenable to the skill set of psychiatry than other medical specialties considered disorders and when are they not (e.g., see the extensive list of nondisorder Z Code conditions in *DSM-5*)? There is no need to allow an esoteric and outmoded philosophical doctrine that was an overreaction to the metaphysical excesses of the nineteenth

century to stop one from testing the explanatory power of a reasonable alternative conceptual hypothesis.

Moreover, once again, Zachar's amplified list fails to satisfy the sine qua non requirement of an analysis of "disorder" of rebutting Szaszian antipsychiatry. All sorts of socially deviant or socially disapproved nondisorders, from interpersonal conflict to criminality, can possess one or more of Zachar's conditions. Thus, a Szaszian would point out that Zachar's list confirms that psychiatric criteria for disorder go way beyond the bounds of true medical conditions and serve as a means of social control. If Zachar's long list of not-really-necessary and not-really-sufficient features is all there is to the concept of mental disorder, then it is compellingly arguable that the antipsychiatrists are correct and psychiatry has no legitimate foundation as a medical discipline.

At the end of his paper, after explaining the network approach (see below), Zachar takes a last shot at listing "considerations that are relevant in making the depressive disorder attribution." He says these include "(a) ... decline in functioning ... (b) ... locked in rather than transient and flexible ... (c) more severe symptoms and more complex symptom networks ... (d) no compensatory factors that allow the person to continue to function (and flourish)." Perusing this list, it seems to me that one could easily satisfy three or possibly all four criteria in a normal reaction to losing a spouse on whom one relied for one's social network, or in losing a job on which one depended, or if one became involved in criminal activity, or if one were involved in a long legal suit. Zachar's list potentially confuses lengthy normal distress and decreased role functioning with mental disorder, offering no answer to the antipsychiatrist.

"Imperfect Community": Zachar on the History and Heterogeneity of Psychiatric Disorders

To block the notion that one can provide an essentialist or any necessary-and-sufficient conceptual analysis of "disorder," Zachar throughout his paper emphasizes the heterogeneity of psychiatric disorders. In support of his position, Zachar presents a brief history of psychiatry that makes it seem as though all sorts of random accretions of psychological conditions to the disorder category took place discontinuously over time: "The disorders of psychiatry are the result of a gradual addition of variations on the symptom clusters of the alienists ... but because of the way the domain was built (by the addition of variants on variants), no single organization can model all of the overlapping relationships." Zachar adopts the dubious Roschian notion that psychosis is the prototype of mental disorder (I have shown elsewhere that this approach just does not work; conduct disorder, dyslexia, and erectile dysfunction are not disorders because of any family resemblance to psychosis [Wakefield 1999a]) and argues that via similarities to similarities, all sorts of conditions got into the category. Zachar seems here to adopt Frances and Widiger's (2012) view that "historically, conditions have become

mental disorders by accretion and practical necessity, not because they met some independent set of abstract and operationalized definitional criteria. Indeed, the concept of mental disorder is so amorphous, protean, and heterogeneous that it inherently defies definition—creating a hole at the center of psychiatric classification” (111). This is a bizarrely skeptical view given that Frances vigorously argued that many conditions added as disorders to *DSM-5* are not in fact disorders, and he sometimes anchors his arguments about disorder in evolutionary theoretical considerations consistent with the HDA’s essentialist perspective—but, I digress.

Zachar’s historical sketch provides an extraordinarily tendentious picture. He fails to examine how specific added conditions were considered prior to the supposed change, does not consider whether the change was one of terminology or refinement of categories rather than a real change of view of disorder versus nondisorder, and ignores the possible theoretical rationale for each supposed accretion that emerged in the inevitable professional disputes over such nosological adjustments. In my own historical work, I have found enormous continuity in what is considered a disorder and in the rationale for disorder versus nondisorder judgments, but with much recategorization and elaboration in response to shifting theories, frequent refinement of single categories into multiple subcategories, changing of emphasis, and terminological shifts reflecting larger theoretical programs, all of which potentially confuse the picture and make it seem like new disorder categories are appearing out of nowhere when in fact disorder versus nondisorder judgments are surprisingly stable (Horwitz and Wakefield 2007; Wakefield 2001). Zachar’s description of the history of psychiatry as semirandom accretions is about as illuminating as someone arguing that the chemical substance “water” is in reality just a random collection of things, as evidenced by the history; first people labeled clear liquids in lakes and rivers “water,” then people expanded the category to include the totally different materials of ice and steam that happen often to occur near water or transform into water, and then astronomers expanded the category to include, for example, stuff detected by spectrometers floating in the Horsehead Nebula that isn’t anything like any of the other instances and is not near any liquid water; the chemical substance water is certainly a “very imperfect community” with variations upon variations and so no unifying criterion!

This brings me to a central thesis of Zachar’s, that “mental disorder” is what he calls an “imperfect community,” a term derived from Nelson Goodman denoting the phenomenon of a class of objects in which any two bear some features in common but the entire class has no one feature in common. The “imperfect community” view of concepts has strong affinities to Wittgensteinian family-resemblance and Roschian prototype-similarity views of concepts. Let me make clear that I believe that concepts can be defined in myriad ways, ranging from empiricist to Roschian to essentialist. However, the evidence is that an essentialist account best represents the structure of

the concept of mental disorder, at least at one crucial level—the level needed to rebut Szaszian antipsychiatry.

Zachar goes the Roschian route and claims that whether someone has a disorder is a matter of degree depending on how many of a set of criteria he presents are possessed by the condition: “As more of these conceptual criteria are met, the more it makes sense to start thinking of a symptom cluster as disordered. Rather than being absolutely present or absent, disorders are a matter of degree.” This mini-argument is wholly invalid. The second sentence—the conclusion that disorder is a matter of degree—does not follow from the first sentence’s premise, that as more criteria are met, it makes more sense to conclude that there is a disorder. In theory, as more *DSM* criteria are met, the strength of the evidence that there is a disorder increases, but either there is or there isn’t a disorder—leaving aside the inevitable boundary fuzziness and unclear cases that afflict most concepts. However, a condition can satisfy just one of Zachar’s criteria and be a crystal-clear disorder or satisfy many criteria and be a crystal-clear non-disorder. On average, the more criteria that are met, the more secure is an inference to the existence of dysfunction and thus the more likely that there is a disorder, but the degree of strength of the evidence for inferring a disorder is not the same as there being a degree of disorder (First and Wakefield 2013; Wakefield 1999a, 2012).

Zachar argues that the fact that there are so many varieties of disorders supports his conception of an “imperfect community” of conditions, that is, conditions not answering to a single conceptual analysis of “disorder.” Other than his questionable history of psychiatry, Zachar supports this position by observing that “the ‘imperfect’ part of the community of psychiatric disorders has been eloquently described by Allen Frances,” the chair of the *DSM-IV* Task Force. Zachar then quotes the following passage describing the multiplicity of types of things that are disorders as evidence in support of his “imperfect community” position:

Some mental disorders describe short-term states, others life-long personality; some reflect inner misery, others bad behavior; some represent problems rarely or never seen in normals, others are just slight accentuations of the everyday; some reflect too little self-control, others too much; some are intrinsic to the person, others are culturally determined; some begin early in infancy, others emerge only late in life. ... Some are clearly defined, others not; and there are complex permutations of all of these possible differences. (Frances and Widiger 2012, 111)

This is a surprising argument for Zachar to use because as a philosopher, Zachar knows full well that Frances and Widiger’s argument is patently fallacious. The fact that various pairs of mental disorders have some opposite properties shows *nothing whatsoever* about the univocal analyzability of “disorder.” Does Zachar think that the fact that there are red spheres and blue spheres shows the unanalyzability of “sphere,” or the fact that there are large numbers and small numbers shows that numbers form an “imperfect community” with no defining features? There is simply nothing to this argument.

Instead of citing irrelevant contrary properties, we might take Wittgenstein's advice to *look and see* whether the entities with these properties share some further unifying features. In any of the categories mentioned, the vast majority of conditions are not considered disorders, so what determines which of the conditions with that property are disorders and which are not? To approach an answer, one might scrutinize actual examples of psychopathology instantiating each pair of the cited contrary categories: short term versus long term (e.g., brief psychotic reaction versus borderline personality disorder), inner misery versus behavior (e.g., generalized anxiety disorder versus pyromania), accentuation of the common versus distinctive rarity (e.g., dysthymia versus fugue states), too little control versus too much control (e.g., intermittent explosive disorder versus sexual sadism disorder), innate versus culturally shaped (e.g., intellectual disability versus reading disorder), and emerges early versus emerges late (e.g., autism spectrum disorder versus Alzheimer's disease). One finds that the inference that there is a harmful dysfunction is common to all of these disordered conditions whatever their other properties and distinguishes them from the vast number of conditions that have the same contrary properties but are not considered disorders.

The vague and stretchable criteria Zachar proposes in his lists noted earlier of considerations for entering his "imperfect community" of psychiatric disorders can serve to rationalize just about any judgment one wants to make. Zachar's analysis thus leaves us right back where we started when it seemed like an analysis of disorder would be an important and useful endeavor: without a principled difference between disorder and nondisorder. With no clear conceptual firewall, every agreed abuse or mistake of psychiatry from drapetomania and sluggish schizophrenia to Victorian surgery for female clitoral orgasm and even the repathologization of homosexuality could find a place within this spongy set of guidelines. Zachar tries so hard to avoid constraining psychiatry by the supposed bogeyman of essentialism that his criteria would leave us with a psychiatry unclear about its own foundational concepts and unconstrained in labeling whatever clinicians want to treat as a disorder, posing a threat to our civil liberties and offering a legitimate target of antipsychiatry.

Zachar on the Causal Network Approach to Intelligence

I now turn to an examination of the pivotal section of Zachar's chapter in which he attempts to show that "essentialism... is not scientifically necessary" for an account of disorder. The HDA implies that "disorder" is inherently an explanatory-sketch concept that applies only if there is an explanation of a condition in terms of a dysfunction. Zachar argues that no such explanatory loading is implied by disorder attributions. To support his claim that "from a neo-empiricist standpoint, essentialism is an excessive metaphysical elaboration that is needlessly grafted onto this complicated network

of ‘observations,’” Zachar elaborates the currently much-discussed “causal network approach” to psychopathology. This view, he explains, holds that “latent variables do not have to be interpreted as referring to real essences,” thus illustrating that a theory of psychopathology need not be essentialist. The network approach, an offshoot of some traditional behaviorist ways of thinking about psychopathology, has recently been elaborated and championed by Denny Borsboom and his colleagues, and I rely on Borsboom’s (2017) recent summary in examining this approach. Although there is much of interest that one might say about the network approach and its implications in general, I limit my remarks to aspects that bear on evaluating Zachar’s attempt to leverage it into an antiessentialist argument.

Zachar initially uses the example of network theorists’ rethinking of the construct of general intelligence (often referred to as *g*) to explain the network approach. According to this approach, general intelligence, as manifested in high performance across a range of intellectual tests, may not be a general factor or cause underlying all cognitive abilities (e.g., rapid neuronal conduction) but rather simply the expression of how networks of cognitive abilities interact. Zachar explains,

An alternative to a causally potent latent variable (or common cause) model is a model in which cognitive abilities are in direct causal relationships with each other. For example, being able to process information quickly might have positive effects on working memory. Cognitive abilities can enter into mutual interactions in a variety of ways. Some people may naturally have high abilities across the board, whereas others are gifted in one or two areas—such as processing speed and attention capacity—but these skills permeate through the ability network and raise scores on tests of general intelligence.

Without getting too deeply into issues regarding the network analysis of intelligence, I offer a few comments before turning to Borsboom’s approach to psychopathology. At one level, intelligence is just a descriptive concept—close to a neo-empiricist understanding—referring to an individual’s performance on intellectual tests of various kinds. However, like all scientists, psychologists generally construe their concepts in an essentialist manner that goes beyond sheer description and refers to the deeper nature of the initial phenomenon because that is how one reaches perspicuous explanation, prediction, and interventive possibilities. Thus, the question “what is intelligence” has many potential levels of meaning depending on semantic or ontological markers that are part of the meaning of a specific usage. (See my reply to Murphy in this volume for further discussion of the multiple levels of concepts.) Zachar’s claim is that, in formulating a theory of intelligence, one can possibly trade the classic theory that there is a latent as-yet-unknown inferred essential explanatory variable that directly explains the performance of all of the individual domains for a network theory that hypothesizes that only certain domains have intrinsically high performance but they interact with other domains in distinctive patterns so as to confer high test performance on those

other domains as well. If so, then no further across-domain underlying essence of intelligence is explanatorily required.

This network characterization is perfectly sensible as a possible alternative theory of what constitutes intelligence at a certain level. However, the possibility of such a theory does not accomplish Zachar's goal of discounting the need for essentialism. The problem is that Zachar's description of high intelligence in terms of network interactions begs the question of the nature of intelligence by already referring to the presence of either "high abilities across the board" or being "gifted in one or two ways [that] ... permeate through the ability network." The network theory thus assumes that there is some variable of ability or giftedness that applies not generically but to specific psychological modules, and that such intrinsically high-performance modules along with certain forms of modular interaction yield generally high modular test performances. None of this relieves us of the question of the essence of intelligence; it just pushes it back a step and relocates the question in the essence of specific modules' ability or giftedness.

One might of course shift more and more of the explanation to specific interactive patterns rather than intrinsic characteristics of key modules. However, if one identifies specific patterns of modular interaction with intelligence, first, one can then say that intelligence does have an essence—namely, those distinctive patterns of modular interaction—and second, one will want to know for each such pattern what is the essential set of conditions that bring it about. Conceivably, intelligence theory might then split into several essentialist theories of the different patterns of interaction that manifest as high intelligence, in the way that there are essentialist chemical theories of each of the two types of mineral that fall under the concept "jade" or in the way that theorists suggest that *DSM* categories of "major depression" or "schizophrenia" in fact encompass multiple disorders with distinct essential etiologies. In sum, the network account of intelligence does not somehow allow the scientist to escape the scientific necessity of identifying what constitutes the essence of high intelligence.

However, for our purposes, perhaps the most critical point about Zachar's use of the intelligence example as an exemplar of the network account is the obvious one that high intelligence is a prototypical normal-range quality, not a form of psychopathology. This reflects the fact that the applicability of network analysis is independent of whether one is dealing with normal or pathological conditions. Consequently, whatever it is that characterizes psychopathology as opposed to normal-range features must be some property over and above whether the phenomenon can be characterized using network analysis. Consequently, even if network analyses in themselves did not require an essentialist approach, the key question would remain unanswered: must one cite some essentialist (i.e., nonobservational explanatory) properties to distinguish those networks that are disorders from those that are not? To answer this question, I examine Borsboom's account of how network theory is applied to psychopathology.

Borsboom on the Causal Network Approach to Psychopathology

The HDA implies that whatever makes a network pathological involves factors such as etiology and history that go beyond a description of the network itself in observational terms. For example, a network analysis that indicated a statistically average intellectually functioning human brain might indicate normality or, if it was the brain of a genius who suffered lead poisoning as a youth or brain trauma as an adult, it might be the result of pathology. A network analysis that revealed that an individual's intellectual functioning is high but emotional functioning is very low might be a schizoid pathology or someone from an emotionally suppressive culture. The distinction between normality and pathology seems to require information that goes beyond the network's manifest performance.

A persuasive piece of evidence that the network approach is not, as Zachar claims, an inherently nonessentialist approach opposed to the HDA is that Borsboom's presentation implicitly presupposes the HDA's rather than Zachar's view. Consider this abstract of Borsboom's (2017) recent paper setting forth the theoretical foundations of the network approach to psychopathology:

In recent years, the network approach to psychopathology has been advanced as an alternative way of conceptualizing mental disorders. In this approach, mental disorders arise from direct interactions between symptoms. ... At the heart of the theory lies the notion that symptoms of psychopathology are causally connected through myriads of biological, psychological and societal mechanisms. If these causal relations are sufficiently strong, symptoms can generate a level of feedback that renders them self-sustaining. In this case, the network can get stuck in a disorder state. The network theory holds that this is a general feature of mental disorders, which can therefore be understood as alternative stable states of strongly connected symptom networks. This idea naturally leads to a comprehensive model of psychopathology. (5)

What is striking about this statement is that Borsboom directly addresses the problem that Zachar insists on ignoring, namely, precisely how pathological networks differ from nonpathological networks. He proposes that a general feature of mental disorders is that exceedingly strong causal relations between symptoms can trigger a sustained pathological feedback loop that becomes stable and inflexible at the severe level ("If these causal relations [between symptoms] are sufficiently strong, symptoms can generate a level of feedback that renders them self-sustaining. In this case, the network can get stuck in a disorder state"). Borsboom is clearly implying that in the formation of such a self-sustaining loop, something has gone wrong and that getting stuck in such a symptom feedback loop is a failure of how these symptom links were biologically designed to occur (see below). It is the unnatural intensity and stuckness in the self-sustaining inflexible symptom pattern that constitutes the dysfunction that, according to Borsboom, is at the core of every mental disorder, a view consistent with the HDA.

I claim that the process of a normal linkage between experiences getting stuck in an intense self-sustaining symptom cycle is best understood as a harmful dysfunction, and Borsboom seems to agree. Later in his paper, he observes that most symptom linkages involved in pathology start out as normal associations due to various normal biological, psychological, and social processes. The switch to a disordered self-sustaining feedback loop, he notes, “thus may involve harmful dysfunctions in these processes” (11):

As may be clear from the examples given in this paper, connections between symptoms are often prosaic. If you do not sleep, you get tired; if you see things that are not there, you get anxious; if you use too much drugs, you get into legal trouble, etc. It is, in my view, likely that these symptom-symptom connections are rooted in very ordinary biological, psychological and societal processes (and thus may involve harmful dysfunctions in these processes). This is surprising, because it means that disorders are not ill-understood ephemeral entities, the nature of which will have to be uncovered by future psychological, neuroscientific or genetic research (which appears a widespread conviction, if not the received view, among researchers). Rather, the fact that we have the set of basic symptoms, and also understand many of the relations between them, means that we already have a quite reasonable working model of what disorders are and how they work. (11)

In other words—and even according to Borsboom’s own understanding—the network approach’s postulation of how things go wrong does require some understanding of the difference between normality and pathology of the sort provided by the HDA, contrary to Zachar’s interpretation. There must be some such differentiating standard because some tight and inflexible linkages between reactions are part of biologically designed functioning and entirely normal, so such reactions can indicate pathology only when they are dysfunctions. Indeed, the above passage indicates that it is not network links per se, which are omnipresent and prosaic, but, consistent with the HDA, deviations from the natural levels of linkage tightness and feedback looping that suggest pathology. Deeper processes sustaining such a dysfunction is its essence.

Before leaving the network perspective, it is worth observing in passing that it harbors some facile assumptions. Network theory’s standard hypothesis, we have seen, is that pathology often emerges from known linkages between phenomena, and it consists of the development of excessively powerful linkages between those phenomena with circular feedback loops forming between symptoms that keep the network going. However, none of these generalizations are as obvious or generalizable as network theorists suggest. Consider Borsboom’s example of the insomnia-fatigue link in depression. First, fatigue can be caused by insomnia, but it can also be phenomenologically and functionally different from insomnia-induced tiredness, involving not sleepiness per se but low energy levels (ask those who have experienced both), and fatigue independent of insomnia can be an important vegetative symptom of depression. As much as one might enjoy heaping ridicule on *DSM* for not recognizing such a commonsensical connection as that between lack of sleep and fatigue, the reality is that fatigue appears as

a symptom because sometimes depression does include an independent fatigue symptom. (However, Zachar's using the link between insomnia and fatigue as an objection to essentialism because the symptoms are not independently explained by the hypothesized dysfunction is a straw-person argument in the extreme because there is nothing in essentialism that is violated by such a link among observable phenomena.) Second, when severe fatigue does result from insomnia during a depression, this does not imply that the link between insomnia and fatigue has been pathologically strengthened; pathological levels of insomnia can naturally produce correspondingly high levels of fatigue. Finally, the notion that when depressive insomnia causes severe fatigue, a pathological feedback loop occurs in which the fatigue sustains the insomnia, is questionable because in most instances when sleep returns to normal, insomnia-induced fatigue correspondingly recedes rather than triggering renewed insomnia.

Nonetheless, network theory is smarter than symptom lists in several ways. Network theory correctly emphasizes that not just the list of symptoms but the dynamics of the causal network of symptoms—whether the system of symptoms itself comprises the dysfunction as network theory holds or are the effects of some underlying dysfunction—matters enormously to diagnosis and treatment. Mapping symptom causal relations can yield additional insight that is lost in the literal-minded symptom-list approach. Indeed, such analysis can lead, for example, to the insight that more or less the same set of depressive symptoms in fact is generated by insomnia and is a sleep disorder rather than a depressive disorder. As well, I heartily agree with several broader theses defended recently by Borsboom, Cramer, and Kalis (2019), including the irreducibility of some mental disorders to brain disorders and the need to take into account intentional content and not just brain-level descriptions in understanding symptom-symptom linkages.

Returning to the issue of essentialism, the network theory of disorder that Zachar presents as his trump card in demonstrating the possibility of a neo-empiricist account of disorder in fact demonstrates the opposite. Like all serious theories of disorder, network theory distinguishes between the natural functioning of the organism consistent with how it is biologically designed and the ways in which that functioning can go wrong—that is, dysfunctions. These implicit assumptions allude to a theoretical distinction that goes beyond anything in the symptom network itself. There is thus nothing in network theory that supports Zachar's attempt to escape the fact that the distinction between normality and disorder implies a distinction of (inferred) types of causes, which in turn requires an essentialist analysis that goes beyond the conceptual straightjacket of neo-empiricism. The failure of Zachar's earnest series of attempts, from decline in functioning to imperfect community to network theory, to vindicate his neo-empiricist approach both in terms of defending against the antipsychiatric challenge and in terms of simply explaining common intuitions about disorder makes the point manifest that “disorder” is an inherently essentialist concept.

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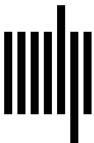
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