Rethinking disease in psychiatry: Disease models and the medical imaginary

Jennifer Radden D.Phil., Oxon., Professor Emerita of Philosophy

Philosophy Department, University of Massachusetts Boston, Boston, USA

Correspondence
Jennifer Radden, Philosophy Department, University of Massachusetts Boston, 1000 Morrissey Boulevard, Boston, MA 02125, USA.
Email: jennifer.radden@umb.edu

Abstract
The first decades of the 21st century have seen increasing dissatisfaction with the diagnostic psychiatry of the American Psychiatric Association’s Diagnostic and Statistical Manuals (DSMs). The aim of the present discussion is to identify one source of these problems within the history of medicine, using melancholy and syphilis as examples. Coinciding with the 19th-century beginnings of scientific psychiatry, advances that proved transformative and valuable for much of the rest of medicine arguably engendered, and served to entrench, mistaken, and misleading conceptions of psychiatric disorder. Powerful analogical reasoning based on what is assumed, projected, and expected (and thus occupying the realm of the medical imaginary), fostered inappropriate models for psychiatry. Dissatisfaction with DSM systems have given rise to alternative models, exemplified here in (i) network models of disorder calling for revision of ideas about causal explanation, and (ii) the critiques of categorical analyses associated with recently revised domain criteria for research. Such alternatives reflect welcome, if belated, revisions.

KEYWORDS
disorder analogy, Melancholy, networks, RDoC, Syphilis

1 | INTRODUCTION

The first two decades of the 21st century have seen increasing dissatisfaction with the diagnostic psychiatry associated with the American Psychiatric Association Diagnostic and Statistical Manuals (DSMs), and World Health Organization International Classifications of Diseases (ICDs). This is psychiatry, for the most part, as we presently know it, whereby a classification of separate mental disorders is used, each disorder presumed to arise from morbid factors within the person. Its flaws are theoretical and methodological as well as practical, ethical, clinical, and, increasingly, political. One source of these problems, I want to point out, may lie deep within the history of medicine where, coinciding with, and fostering, a more orderly, precise, and purportedly scientific approach to psychiatry, advances useful for much of the rest of medicine may have invited mistaken and misleading conceptions of at least some psychological disorder. Based on powerful analogical reasoning, these models of disease or disorder arise and function, in part, with what is assumed, projected, and expected—in the realm of what might be called the medical imaginary.

To trace these elements of medical history, the present paper makes reference to two features of concern to critics of DSM psychiatry: its presuppositions about causal explanation and its reliance on categorical, rather than dimensional, analyses. By way of support for the case made here, examples of revisions to the understanding of psychiatric disorders are introduced: first, network models of disorder requiring not only dimensional analyses but new ideas about causation and even validity, and second, recent proposals for research domain criteria (RDoC). The contrasts emphasized here include that (i) symptoms may be explained by many causes (as in networks), or (DSM style) by a single cause in the form of a dysfunctional system located in the subject’s brain or body; (ii) disorders will be loose, vaguely bounded items in networks while however vague their symptom profile, the core disease entities of the DSM model are distinct, human-independent, natural kinds; (iii) as networks, disorders are at best relatively stable sign and symptom clusters yet the DSM approach...
assumes that stability across episodes and between individuals is ensured by common anchoring disease processes.

The aspect of the DSM model (iii above) by which separate disorders correspond to sharply bounded categories contrasts with the dimensionality incumbent in network models and illustrated by RDoC. Any model must use categories of some kind. But here, “categorical” implies both that the underlying causal mechanisms giving rise to mental disorder are discrete biological attributes common to all cases of disorder assigned to a given diagnostic category (such as depressive disorder), and so universal. In this presumed universality, it has been argued, lies the central feature of such categorical conceptions of disease.

The history here is contested, and the evidence equivocal. Many would insist that the DSM-based model of diagnostic psychiatry, with its causal presuppositions and categorical nosology, finds a triumphant confirmation in the findings, hypotheses, and dreams of biological, and ultimately genetic, psychiatry, at most rejecting as with its causal presuppositions and categorical nosology, finds a

antibiotics served to prevent progression to the final psychological

errors.

Melancholy and Syphilis were not as apparently different in their

imbalance derived from Galenic and the neo-Galenic humoural

medicine of medieval and early modern thought. The Anatomy of Melancholy is an important record here, representative of most, if not all, that had come before. His approach primarily encyclopedic, Burton’s aim was to preserve and compile classical and renaissance wisdom about Melancholy. Attention was to symptoms, and to the disease’s individual course or natural history in particular sufferers, and its primary treatment approach involved a general attunement, or rebalancing, of the bodily and psychic systems.

The Anatomy cannot be easily summarized. (The summary pro-

vided here is derived from research compiled by the present author.6 Readers should also be encouraged to turn to the Anatomy itself, and to the several valuable historical commentaries.7,8) It is an enor-

mous, partially satirical and literary work, open to a range of interpre-
tations of which the following is only one. Without forgetting that qualification, features can be extracted from its account of Melancholy that constitute an informal model of the mood disorder(s) that is Burton’s subject. First, there is repeated emphasis on the range and diversity of symptoms it produces, symptoms which themselves cause subsequent symptoms, in what resemble nothing so much as the elaborate and multistranded feedback loops of contemporary enactivist cognitive psychology.10 The relation of mind, body, and world is depicted as multidirectional interactions, from mind to body, and body to mind. Among its symptoms, the commonest and most characteristic are feelings of sadness and unwarranted (or “uncaused”) fear. The causes of melancholy are infinite: Aside from religious and astrological elements, they include bodily states, expe-
riences, other feelings, and distorted imaginings. Disorder, often emanating from a troubled imagination, is restored through multi-modal ministrations to the body, mind, and soul, in efforts that are construed as a largely system-wide righting of humoural imbalance. But reference to humours in this account is open to debate: Anti-Galenic medicine in the period rejected the alleged effects of these bodily fluids as outmoded science, and while still adhering to humoural language, Burton’s humouralism often seems more metaphorical than literal. Like his near-contemporary Sydenham (1624-1689), Burton emphasizes not underlying causes so much as the disease’s symptoms and temporal course or natural history. (Making its natural history paramount, Melancholy was a progressive disorder: Neglected, it worsened through time, and was best addressed through early preventive measures.)

The bacterial analysis of disease associated with the 19th cen-
tury arose from the German research on cellular biology by figures like Virchow (1821-1902) and Koch (1821-1910). The features of these findings that are well known have shaped much medical knowledge since that time.11,12 For each distinct disease, a single, specific, cellular-level cause must be sought (Virchow), when any such finding requires independent verification (Koch). Together, these principles, expectations, and demands, laid out a model or picture of disease ontology comprising an underlying core causally responsible for its more readily observable signs and symptoms which, as downstream effects, provided clues to the real, underly-
ing disease entity or process. By identifying underlying agents (eg, particular bacteria), these new approaches could then target them for specific, effective treatment. What has been called the
"aetiological standpoint" that ensued, it has been explained, was made possible only when diseases ceased to be conceived, and defined, as symptoms, and instead, focus was on the search for their universal and necessary causes. The same cause, it came to be accepted, was common to every instance of a given disease, becoming its defining feature, so that without its cause, a disease of that kind was not judged to be present. By the second half of the 19th century, medicine was beginning to emphasize and value causes of a particular kind: universal necessary causes that met recognized criteria, explained disease phenomena, and held out promise of effective remedy. And the greatest success of this effort was the bacterial theory of disease.12

The aetiological standpoint contrasts with Burton's account of Melancholy in several respects: (i) its conception of causes is as singular defining conditions that are both necessary and sufficient; (ii) an orderly and consistent part is played by a single cause rather than many; (iii) its emphasis is on endogenous causes only; (iv) its symptom profile is stable and consistent; (v) its depiction of causes will more likely be specific, local events or morbid states in the body than effects that are holistic and system-wide; and finally (vi) it affords a-temporal analysis, allowing snapshot forms of observation and identification for diagnostic purposes. The case of Syphilis perfectly conformed with those features, as did other bacterial diseases and many nonbacterial diseases, including specific organ-affected conditions such as cancers.

Melancholy and Syphilis are introduced here to highlight the differing conceptions of disease involved in how they were understood. As a disease, Burton's Melancholy is not a discrete entity at all: No categorical analysis (in the sense noted above) could contain it. Lacking any specific cause comparable to the bacteria causing Syphilis, Melancholy was brought about by innumerable factors. It was associated with an unlimited range of symptoms, of which sadness and fear without cause were the most common, but not deemed (at least by Burton) essential. Remedial responses were comparably diverse, in contrast to the neat, targeted antidote long-sought, and eventually found (in antibiotics), for Syphilis.

3 | EARLY PSYCHIATRY

As an accident of historical timing, the analysis so apt for Syphilis came to shape and direct how early psychiatry and psychiatric classification developed during, and following, the Kraepelinian era (from the 1880s onwards), remaining the predominant influence in psychiatry today. Diagnostic or DSM psychiatry still adheres to these analogies: Research and practice work on the presupposition that the familiar disease categories reflect a successful “carving of nature at its joints”, in the much-used ancient phrase originating with translations of Plato’s Phaedrus. Common causes remain the quest of most medical research and include the goal of finding the targeted interventions of psychopharmacology in “unlocking” inner mechanisms to provide specific antidotes as “magic bullets” for effective cure, to use David Healy’s metaphors.

Medical science has of course been immeasurably enhanced by the bacterial models. Yet, applied to psychiatric medicine, these models have often been challenged and recognized to have had incomplete success as the basis for understanding mental disorders. (For a summary of some of these concerns, see Insel and Cuthbert.13) Despite criticisms from the social sciences, network models of mental disorder, proposed in the last decade and in many ways reminiscent of the earlier, more Galenic ideas, together with the dimensional emphasis of RDoC,14 are—thus far—the exceptions within psychiatry. Yet the existence of psychiatry as we customarily know it may depend on the fate of innovations like these. And they demand its fundamental transformation.

4 | CONTEMPORARY RETHINKING

4.1 | Network models

Mental disorders may be seen as statistical networks sustained by causal interactions between the symptom cluster or syndrome associated with the disorder, it is hypothesized. This analysis rejects the basic terms of DSM-type "common cause" analysis, where—on the model of bacterially caused diseases—each mental disorder comprises a symptom cluster brought about by some particular underlying brain state within the person.14-16 If mental disorders are better viewed not as manifestations of underlying, idiopathic dysfunction but are instead collections of interacting signs and symptoms linked and fostered through feedback loops, then mental disorders themselves are as much, or more, the product of environment and social context than of any inherent causes. As those in the social sciences have long insisted, we must look to the looping interactions between bodily person, experiences, feelings, cultural interpretations, and the world interpreted through symptoms, to find a complete explanation of the disorder.17

Signs and symptoms form a major part of what is available for diagnosis in any models of mental disorder. For better or worse, without independent means of verification, they are in that respect indispensable.18 But in the network analyses described above, symptom descriptions possess an epistemic status with a different significance. Like any other human response, disorder will in some way depend on underlying biology. But the distinction between the causes of disorder and what, in making up its diagnostic identity, constitutes that disorder has been emphasized for certain disorders such as depression. Depressive disorder is attributed on the basis of psychological symptoms alone even in the absence of biomarkers, it is pointed out, whereas it would not be attributed were the biomarkers present in the absence of those psychological symptoms.19 Similarly, the subjective experience of distress and the extent of impairment of the person’s everyday functioning have been proposed as an intrinsic property of depression.20-22 In the present day, at least, the depression, we can say, is constituted by these distresses which form its (present day) diagnostic identity. Analyses characterizing “intrinsic distress” as a property constitutive of some given psychiatric condition (such as depression) seem to suit recent models focused on the causally interconnected statistical networks making up symptom clusters.
Not only the epistemology but the ontology of network models is different, also. The attribution of disorder (or diagnosis) is determined by stable likenesses between cases—not by evidence of any underlying disease process. Rather than syphilis, or herpes, the analogy suggested by network models fitting the disorder of depression might be the common cold. As the unit of focus, not downstream effects, common cold symptoms are constitutive of disorder. Within a network, internal causal relationships between symptoms—and those alone—determine the characteristic profile of the disorder. In this way, the individual symptoms in the network will appear as more or less stable sets of traits that will in some cases become mutually reinforcing. In depression, for example, the person’s repeated, pervasive moods and states are indications of disorder when, because, and to the extent that, the connected links and loops occur often enough to form a dense network of causal interactions.

Viewed as statistical networks, mental disorders can at best achieve predictive validity. Assuming strong metaphysical realism, there is a person-independent world corresponding to accurate judgments about it. A nonrealist sense of validity can presuppose no such world, there is none; the stability and existence of the “world” lies entirely with a concurrence between thinkers and observers, whose weak “realism” accepts only that properties seem to cluster and change in a reliable fashion. Without the presuppositions of realism, there can still be less and more accurate predictions as to future outcomes, but “validity” is this weaker kind, where getting it right, is predicting with accuracy. Thus, as statistical networks, analyses of mental disorder cannot rely on the ontology grounding traditional senses of scientific endeavour. Yet, rather than a deficiency of the network account, construing mental disorder in this way allows it to conform to more recent thinking in science, much of which similarly divorces itself from strong realist presuppositions and the matching metaphysical conception of validity it entails.23

4.2 RDoC

With analyses of mental disorder relying on networks, the taxa of psychiatric classification such as depression are not bounded entities, but dimensional concepts. And this aspect of disease has been quantified and ordered in what the RDoC associated with Thomas Insel introduced with the complaint described in the above discussion: The present system for understanding and diagnosing mental disorders enshrined in the DSMs was still using 19th-century disease models for diagnosis, a categorical system now replaced by dimensional models in other areas of medicine.22 RDoC offers a schema primarily designed to address insufficiencies in psychiatric research. Its design involves a uniform, and elegant, if complicated, matrix. The rows of the matrix refer to psychological capacities (for example, positive and negative valence [or affect]). These capacities are conceptualized dimensionally as spanning across the normal and the abnormal; they are supposed amenable to explanation by relevant mechanisms at multiple levels of analysis that form the rows of the matrix, include behaviour, and self-report, as well as physiology, neural circuits, and genetics. Everything here is dimensional, not only cutting across but dissolving the entities to which conventional DSM diagnostic categories hitherto referred.

The contrasts between DSM systems and these more recent alternatives were noted at the outset. On network analyses, the loosely bounded, fuzzy, and necessarily dimensional entities that are mental disorders will at best be made up of relatively stable clusters contingently identified through empirical observation of patterns among signs and symptoms and distinguished from nonpathological responses through what must be somewhat arbitrary cut-off points (decided by degrees of suffering and their disabling effects). In addition, we saw that symptoms may be explained by many causes (networks), rather than a single, common cause located in the subject’s body (DSM). However, while both networks and RDoC were presented as improvements on traditional categorical models, these innovations rest on different ontological presuppositions. Strong realism apparently still grounds the RDoC goal of finding genetic and clinical neuroscience biosignatures to augment clinical symptoms and signs.13

5 | THE MEDICAL IMAGINARY

The above contrasts were introduced to illustrate how disease models have influenced understanding. Scientific models are by their nature hypothetical constructs, not items in the real world. They guide meaning-making, engender hypotheses, and place constraints on what is observable, what is selected for study, and what is deemed significant in findings and conclusions. In this respect, disease models of the kind described above engage what can be called the medical imaginary—that which is assumed, projected, and expected, in making sense of the world. And this in turn leads to emphases, oversights, lacunae, and distorted interpretations of ambiguous appearances. (As well as arising from the medical imaginary, categorical disease models of the kind described above may reflect an “essentializing” tendency, identified in lay thinking and observed to affect social, biological, and psychiatric categories.)24

In the widely accepted Popperian view of science, observation always occurs in light of theories. But others have pointed out that metaphors guide observation as much as theories. Metaphors cannot be tested, just rejected or abandoned; they lie so deep in the way we think of and experience things that they are apprehended as part of the experience itself. Analogies are not given directly in experience. Rather likenesses such as those guiding the analogies around mental illness occur once the observed phenomena are categorized as illness (and thus problems arising in the person, and not merely her environment, for example).25

Philosophers of medicine describe the bacterial model of disease as so powerful as to sweep away all previous conceptions of disease. Its power lay in its applicability, which went well beyond the simplest bacterial infections where it was first discovered; in its promise of providing targeted effective remedies, and in the appeal of its universality by which diseases could be expected to behave the same regardless of particular circumstances. But its entrenched persistence in psychiatry, I have tried to show, may also have been an accident of historical timing.

If the analogies between simple bacterial diseases and disorders of thought and feeling are mistaken or exaggerated, as the earlier era’s
understanding of Melancholy might lead us to suspect, their practical and clinical consequences seem likely innumerable, and potentially troubling. Arguably, those consequences pervade every aspect of what has been called the “Psychopharmacological Industrial Complex” making up most of psychiatry today, at least in Western and Westernized health systems. They are implicated in remedial responses, for example, where treatments such as “magic bullet” antidepressants are prescribed to cure disordered moods. The expectation that a single common cause unites the symptom clusters associated with particular diagnoses has directed a quest for underlying causes, even in the face of mounting evidence from genetic, neuroscientific, epidemiological, and other social scientific data that mental disorders result from a multiplicity of factors, many external to the body, natural history, or unique psyche, of the person afflicted. Outcomes of thinking categorically also affect some of the most urgent ethical concerns about mental disorder: its best treatment, and ways to reduce the stigma and discrimination that surround it. Reifying and essentialising medical conditions obstruct the goal of healing; it has been suggested: Patients’ sense of reduced being, accentuated by “substantializing” their disease, augurs poor treatment compliance. And, categorical thinking is allied to, and apparently underlies prejudicial attitudes, including the stigma and self-stigma associated with mental disorder. In the final analysis, the evidence over whether to regard mental disorders as categories (taxa) or dimensions seems to be ambiguous. But in the face of such practical and ethical consequences, we may still have reason to favour dimensional accounts.

Innovations like network analyses and RDoC may auger the demise of DSM psychiatry. (Indeed, we may be entering a period of “extraordinary science” indicative of a paradigm shift of Kuhnian proportions.) They reflect welcome, if belated, reforms. Perhaps, with them, we can also expect a reconsideration of renaissance and early modern conceptions of mental disease.

ACKNOWLEDGEMENTS

This paper grew out of a conference “Rethinking Disease: New Theoretical Foundations for Clinical Treatment”, held on November 10, 2017, at the Research Institute of the University of Bucharest. I am grateful to the Institute for sponsoring this event, and also want to acknowledge the valuable and constructive contributions of my fellow presenters, Professors Heinz Katschnig and Ion Copoeru and Drs Valentin-Veron Toma and Alexandra Parvan. In addition, I am grateful for the opportunity to rewrite the essay in response to helpful comments by two anonymous readers.

ORCID

Jennifer Radden @ http://orcid.org/0000-0001-8640-0264

REFERENCES


