What Is Diagnosis For?

Nikolas Rose

Department of Social Science, Health and Medicine
King’s College London
nikolas.rose@kcl.ac.uk

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Introduction

For some, especially British psychiatric clinicians, the debate over the new edition of DSM, the Diagnostic and Statistical Manual of Mental Disorders (American Psychiatric Association 2013), is over-excited. A diagnostic manual in psychiatry, they say, is just a provisional map of territory, a map of the kinds of troubles that psychiatrists work with in their practices. A manual like DSM is a kind of rough guide that will help the practitioner get oriented, maybe to read on the plane but best put to one side when one is actually on the ground, that is to say, in the clinic. And indeed, in the UK, most practising clinicians don’t use DSM classifications in their work, don’t work through DSM checklists in making their differential diagnoses, don’t think of their diagnoses as absolute, let alone believe that they individuate a specific biological substrate in the brain as the cause of the disruption to the lives of the patients. So why all the fuss about DSM 5?

This very British position is attractive. It probably represents the views of most practising clinicians in the UK. It would probably be wise if it were to be widely adopted by everyone involved in mental health–by policymakers, by planners, epidemiologists, the WHO, pharmaceutical companies, the European College of Neuropharmacology, hospital record keepers, insurance companies, employers, researchers, patient groups and patients themselves. But unfortunately, this has not been the case. So let us start by considering what diagnostic manuals do.

Diagnosis as a social phenomenon

Psychiatric diagnoses, like other diagnoses in medicine, play crucial professional, organisational, economic, cultural, and perhaps political, roles. It is easy to think that, alongside aiding professional communication and research, the main purpose of making a diagnosis is to identify the nature of the condition so that, if possible, one can treat its causes or at least mitigate its harmful consequences. We also know that medical diagnoses have long had the function of organizing professional communication, education and research and it is a commonplace to remark on the ambitions for DSMs since 1980 in this respect. But today diagnosis – that is to say, the allocation of a name to a condition - has acquired many other functions. We can start with listing ten: (1) A diagnosis is a condition of eligibility of an individual for treatment – if you have no diagnosis of pathology, there is no case for treating you. (2) In insurance based regimes,
it is a condition of financial coverage of the cost of treatment. (3) For those who are employed, it is a condition of legitimate absence from work. (4) For those who are unemployed, it may be a condition for access to welfare payments. (5) For hospitals and medical establishments it is a central feature of patients records, and these records often shape the allocation of funding from those who commission services for different conditions. (6) For lawyers, it can be a condition for involuntary detention and treatment. (7) In the school system, a diagnosis may be the basis of allocation to special educational provision. (8) For epidemiologists, diagnostic categories are the very basis of their calculations, and of the estimates and predictions that are based on assessments of incidence and prevalence. (9) For planners of services, those estimates and predictions are the essential raw materials for their work. (10) For funders of research, especially charities focused on a particular disorder, it may delineate a problem that is really worthy of investigation.

It is relevant, then, to ask ‘diagnosis for what’ – and what kind of diagnosis is relevant for each purpose. For many of these purposes, an estimate of levels of impairment and capabilities is more relevant than a categorical label. Of course, as in the ‘social model’ of disabilities, levels of impairment and capabilities are inherently dependent on the social norms, expectations and conditions at particular times and places – but that is the reality within which patients and others have to live their lives. For other purposes diagnosis in the clinical sense may also be irrelevant. For example in the criminal justice system, a form of capacity, fitness to plead, is a more relevant than diagnosis in assessing competence to stand trial, and an assessment of responsibility is more relevant than a diagnosis in determining culpability (Szmukler, Daw et al. 2010). Where epidemiological estimates are to be used for assessing the need for certain services or the direct and indirect costs of mental health problems, specific diagnoses are little guide to service use or capacities for employment and are likely to be misleading. It is only since the 1970s that some have dreamed of a single system that could fulfil all these roles, as well as those of clinical judgement, professional communication and research. Perhaps it is time to abandon that dream.

But diagnosis, especially categorical diagnosis – the allocation of a name to an experience of illness – has crucial social and cultural characteristics that make it difficult to abandon altogether.² Diagnosis is deeply embedded in our contemporary notion of medical care. There is an expectation that a visit to the doctor will result in a diagnosis that, among other consequences, legitimates the ailment. And when a diagnosis is given,
it reframes symptoms into a pattern that appears recognizable to both doctor and patient. For the doctor, this enables the patients’ history to be organized into a narrative, which provides a shape for the past and an orientation to the future – it is performative. For the patient, who may be experiencing symptoms that are often diffuse and transient, who may be struggling with behaviours that can seem as merely personal inadequacies, diagnosis reframes these troubles into an illness. The diagnosis enables a story to be created about it – what has led to it, what it is, how it will be treated, what the outcome might be, how it can be spoken about with family, friends, employers and others. Sometimes even the most apparently devastating diagnoses – for instance one of early stage Alzheimer’s disease - can thus provide a certain kind of relief. In non-psychiatric diagnoses, a diagnosis usually – though not always – leads to a reduction in personal responsibility, the mutation of a implied culpability into sympathy and compassion, and a reduction in stigma, although we know that this is not the case in psychiatry. It may open a pathway to effective treatment – although sadly, once again, this is not always with psychiatry.

Of course, sometimes those diagnosed do not find their diagnosis a relief, and do not wish to align their own understanding of their condition with that provided by medical authority. Psychiatry is not alone in generating such contested diagnoses, but the contests in the area of mental disorder are often the fiercest. For some, a psychiatric diagnosis seems a denial of their own account of the causes of their troubles – for instance implying that their physical symptoms are ‘all in the mind’. And psychiatric diagnoses may generate stigma, not just ‘in the community’ but also in wards, clinics, day hospitals, where different diagnoses carry different moral values and elicit different responses from nurses and others. The diagnosis shapes the way in which care staff interpret speech, conduct and distress. Problematic conduct may be a matter for blame where the patient is diagnosed with personality disorder, for sympathy where the diagnosis is bipolar disorder. In short, diagnosis is a transformative moment for the person diagnosed when they embark on a ‘moral career’ as a mental patient, with the changes of self perception and treatment by others that this entails (Goffman 1959). Of course, that moral career need not be all negative: today, diagnoses can sometimes become a basis for social mobilisation, with support groups, pressure groups and charities forming around their diagnostic categories, pressing for better services and research for ‘their’ condition, sharing experiences with other ‘patients like me’ (Rose and Novas 2004).
Thus, even if diagnosis is merely a provisional map for the clinician, it is also a psychological, social, economic, political reality, held in place by many social practices. Doctors, researchers, patients, pressure groups and bureaucrats become attached to these categories, shaped by them, sometimes dependent upon them financially, professionally, politically, morally. Psychiatric categories partake of this reality, despite the disputable definitions and fuzzy borders that frame so many diagnoses. As Charles Rosenberg puts it, an essay entitled 'Contested Boundaries: Psychiatry, Disease, and Diagnosis': "We have never been more aware of the arbitrary and constructed quality of psychiatric diagnoses, yet we have never been more dependent on them than now, in an era characterized by the increasingly bureaucratic management of health care and an increasingly pervasive reductionism in the explanation of normal, as well as pathological behaviour" (Rosenberg 2006). Indeed, as Allan Horwitz and Gerald Grob have suggested, the form of diagnosis embodied in DSMs from 1980 onwards has been so thoroughly institutionalized in the bureaucracy of US psychiatry, that it has proved very difficult to undertake the radical transformations in the move to DSM-5 that some had hoped for (Horwitz 2013). Institutional inertia triumphed despite the well known problems of the system – the multiple comorbidities, the difficulties of taking account of dimensionality, the lack of indices of severity, the absence of reference to development and changes over time. Embedded in psychiatric training, ingrained in the organizational apparatus of hospitals and the procedures of insurance companies, enmeshed in the marketing strategies of the drug companies, DSM-5 would retain the fundamental structure and assumptions of DSM III – categorical diagnosis.

**All in the brain**

Diagnostic manuals have ‘unifying’ consequences – they don’t just ‘sort things out’, they also link things together (Bowker and Star 1999). While several different pathways intertwine in the history of psychiatric classification, the first diagnostic manuals of the modern type were created in the 19th-century asylums (Esquirol 1838; Bucknill and Tuke 1874). These asylums gathered together a heterogeneous group of individuals: voice hearers, those with delusions of grandeur or extreme religious beliefs, alcoholics, unmarried mothers, dementing elderly people, prostitutes, vagrants and many more. (Rothman 1971; Scull 1979; Foucault 2005 [1961]). These people had little in common except for their violation of the web of norms that was coming to pervade conceptions of civility and order in nineteenth century industrializing and urbanizing societies. The early manuals placed descriptions of all these figures within the same covers. In
organising their diverse ailments into a single system, they implied that, despite their many differences, they were all suffering from the same kind of thing – mental disorders. Further, all these troubled individuals came under the care of a single authority figure – the doctor, implying that a medically trained person was the appropriate authority – both in terms of knowledge and in terms of power – to diagnose, to treat and to manage each of them.

Contemporary manuals, like the DSM, classify those on a new ‘territory of psychiatry’ – that of everyday life, or as it is sometimes misleadingly termed ‘the community’. Many have commented on the diagnostic expansionism which we have seen in the subsequent three decades, such that the DSMs went from the 182 diagnoses in DSM II to 265 in DSM 3, to 421 in DSM IV-TR, with diagnoses covering the range A to Z or at least from A to V - Acute Stress Disorder to Voyeurism. Critics suspect that new categories in DSM 5 will exacerbate this expansionism, especially the inclusion of what one might term ‘risk categories’. I think they are right. But my point here is different. It is that, like their predecessors, in placing all the heterogeneous figures that contemporary psychiatrists see – or hope to see – within the same covers, within the same framework, they imply that they are all ail ing from the same kind of thing, and that this is the kind of thing that a particular sort of person – a medically trained psychiatrist – can diagnose, treat and manage.

We can see one consequence of this if we consider estimates of the proportion of the population who could, or should under this way of thinking, be suitable cases for psychiatric treatment. In the United States, Kessler and his colleagues for many years have been estimating "the burden of mental illness" and they come up with a relatively stable figure. It seems that around 25% of adults in any one year or 50% in a lifetime suffer from a DSM diagnosable psychiatric disorder, even though most of them are never diagnosed as such (Kessler, McGonagle et al. 1994; Kessler, Demler et al. 2005). In Europe, Wittchen and his colleagues originally arrived at a similar estimate (Wittchen and Jacobi 2005), but they have recently revised it upwards. They now estimate that around one third of the population of the European Union are so afflicted in any one year (Wittchen, Jacobi et al. 2011). In these later estimates they include substance use disorders like alcohol dependence, psychotic disorders like schizophrenia, mood disorders like depression, anxiety disorders, eating disorders like anorexia nervosa, childhood disorders like ADHD, sleep disorders like insomnia and narcolepsy, and the dementias, PTSD, multiple sclerosis, epilepsy, Parkinson’s, and stroke. They have taken
to describing these as estimates of the size and burden of ‘disorders of the brain’, claiming that mental disorders and neurological disorders share many characteristics in their aetiology and their basis in the brain. Perhaps this phrase – disorders of the brain - was just a wake-up call to politicians to find better treatments for those in distress – for some may think that the term ‘brain disorders’ gives greater objectivity to conditions previously termed ‘mental health problems. And the papers do embody a call to action: they aim to show that ‘brain disorders’ constitute a large pool of unmet need, of sufferers denied access to a diagnosis and hence to treatment – and they also aim to show that there is a strong economic argument for increased investment. But something else is at stake in considering all these are these conditions as disorders of the brain.

Few would doubt that brains are involved, that there are neural correlates of disorders ranging from anxiety to Alzheimer’s disease. But this does not mean that all these conditions have analogous aetiologies, let alone that there should be analogous treatment responses, or analogous research strategies to understand them. As Michael Rutter recently pointed out, infectious diseases and severe deprivation are each associated with pathophysiological changes but the pathophysiology is not causal but consequential (Rutter 2011). This does not, of course, mean that the pathophysiology should not be researched and treated. But it does mean that there should be no assumption that that these conditions have something fundamental in common or even a ‘family resemblance’ (Wittgenstein 1958), let alone that clinical conceptions of aetiology or research into causation should start with neurobiology.

Let me return to DSM and its use of categorical diagnosis. Over the last three decades, categorical diagnosis has had a crucial role in the generation and organization of psychiatric knowledge. The whole apparatus of psychiatric research has become predicated on diagnostic categories – allocation of research funding, methodology of clinical trials, entry of individuals into such trials, choice of treatments, development of hypotheses, interpretation of results, publication in high impact refereed journals and much more. Categorical diagnosis has also been an organizing feature of genetic research, from lineage studies to the more recent investment of enormous amounts of time and energy into genome wide association studies – for how else would one identify ‘cases’ and ‘controls’ if you could not chose the former because they had been ascribed to a particular category by their diagnosis. Attempts to simulate diagnostic categories have also been a driving force behind the creation of animal models, so ubiquitous in psychiatric research that seeks a biological bases for mental disorders – you manipulate genetic and other parameters in order to ‘model’ the key features of schizophrenia or in
your mouse. And, of course, at least since the 1950s, categorical diagnosis has been the basis of pharmaceutical development, and the creation of psychiatric drugs – each targeted and marketed to a particular diagnosis.

As is well known, DSM, since the third edition, has claimed that its mode of categorisation is purely descriptive, eschewing commitment to any particular theory of causation. Spitzer and Wilson, who were largely responsible for that edition, argued that the new classification system was not a “return to a Kraepelinian way of thinking, which views mental disorders as fixed disease entities” but simply an attempt to avoid terms which carry implications about the causes of a disorder, especially where there is controversy over its nature or cause (Spitzer and Wilson 1968: 1621). But this was a little disingenuous. The Feighner criteria that were so influential in the diagnostic style of DSM III and beyond certainly advocated the use of a checklist system for diagnosis, with the stipulation that a certain number of these must be present to warrant a diagnosis. But they stressed the desirability of “laboratory studies” – chemical, physiological, radiological and anatomical, which they considered to be “generally more reliable, precise and reproducible than are clinical descriptions”, although remarking sadly that “Unfortunately, consistent and reliable laboratory findings have not yet been demonstrated in the more common psychiatric disorders” (Feighner, Robins et al. 1972: 57). Nonetheless, the expectation of those who adopted this Feighner diagnostic methodology, and embodied it into DSM III, was that each clinical syndrome “would ultimately be validated by its separation from other disorders, common clinical course, genetic aggregation in families, and further differentiation by future laboratory tests….” (Regier, Narrow et al. 2009).

From that time forward, an ideal image of a psychiatric diagnosis took shape. A diagnosis would identify a specific underlying biological substrate for each unique condition, which, if not the sole cause of that condition, was an ‘obligatory passage point’ for its emergence – that is to say, genetic, developmental, biographical, environmental, psychological or other forces generated that condition by means of their actions upon that biological substrate. Of course, this image is driven by an analogy with the rest of medicine. Charles Rosenberg points out that this idea of an underlying lesion, of reading through the symptoms to their hidden cause, emerges to dominance in general medicine in the late nineteenth century, with the acceptance of the germ theory of infectious disease and the growing significance of postmortem pathology which identified the precise lesion underlying diverse symptoms (Rosenberg 2003). It was strengthened by the growing use of histological, physiological, biochemical and genetic
investigations into pathology - all seemed to confirm that the truth of a disease was to be found in the tissues. Hence it seemed to go without saying that the naming of a disease should, in principle, be a designation of that biological substrate. Psychiatric nosology thought of this as its ideal logic, despite that fact that its searches for those underlying lesions were almost entirely in vain. And it seemed to go without saying, that this biological basis should lie in the brain, and at least to a significant degree in the genes that shaped its development.

By the end of the twentieth century, the truth of this proposition seemed beyond doubt. This, for instance, was the US Surgeon General writing in 1999 (United States. Public Health Service. Office of the Surgeon General, Center for Mental Health Services (U.S.) et al. 1999: 39 - emphases added):

Mental disorders are characterized by abnormalities in cognition, emotion or mood, or the highest integrative aspects of behavior, such as social interactions or planning of future activities. These mental functions are all mediated by the brain. It is, in fact, a core tenet of modern science that behavior and our subjective mental lives reflect the overall workings of the brain. Thus, symptoms related to behavior or our mental lives clearly reflect variations or abnormalities in brain function.

When the planning of the revision of the DSM began, around the time this statement was made, the goal for accuracy in diagnosis and treatment remained that of identifying ideal, though still imaginary, biological substrate, unique for each disease. Steven Hyman, then Director of the US National Institute for Mental Health, writing for a popular audience in Scientific American in 2003 put it thus (Hyman 2003):

By combining neuroimaging with genetic studies, physicians may eventually be able to move psychiatric diagnoses out of the realm of symptom checklists and into the domain of objective medical tests. Genetic testing of patients could reveal who is at high risk for developing a disorder such as schizophrenia or depression. Doctors could then use neuroimaging on the high-risk patients to determine whether the disorder has actually set in. I do not want to sound too optimistic--the task is daunting. But the current pace of technological development augurs well for progress.

In 2009, Darrel Regier and colleagues still expressed their hope that “Mental disorder syndromes will eventually be redefined to reflect more useful diagnostic categories (“to carve nature at its joints”) as well as dimensional discontinuities between disorders and
clear thresholds between pathology and normality.” However, Hyman's optimism had given way to pessimism - the hope of ‘carving nature at its joints' had to be deferred: “our immediate task is to set a framework for an evolution of our diagnostic system that can advance our clinical practice and facilitate ongoing testing of the diagnostic criteria that are intended to be scientific hypotheses, rather than inerrant Biblical scripture.” (Regier, Narrow et al. 2009).

But if DSM categories were intended as scientific hypotheses, they have failed, at least if success is to be found in neurobiology. To put it in the phrase coined by Ian Hacking, DSM has failed to ‘self-vindicate’. As we know, despite a major research effort in which DSM categories were the basis of clinical trials, genetic research using the most advanced techniques, brain imaging and much more, it has proved impossible to identify clear neurobiological bases for the symptom clusters that form the basis of DSM categories. Hence the conclusion expressed by Hyman in 2008, now former Director of NIMH, who had been so influential in the plans for the revision (Hyman 2008: 891):

Efforts to identify risk-conferring alleles … have been largely unrewarding…. The underlying genetics of common neuropsychiatric disorders has proved highly complex … there is much evidence that similar neuropsychiatric symptoms can result from different combinations of genetic risk factors [and] that the same genetic variant may be associated with multiple DSM-IV diagnoses…. The identification of common risk-conferring variants has … proved extremely challenging in most cases, because of their relatively small contribution to the disease phenotype … because of the diverse genetic, environmental and random factors that lead to these common disease

Hyman, concurring with several generations of sociological critics, now concluded that “what we think of as a single ‘disease’ is not in a strict sense a homogeneous entity for which there is a ‘Platonic’ ideal phenotype. Common diseases are more likely to represent families of diseases that share major pathophysiological and symptomatic features, but can differ in important characteristics such as age of onset, severity of symptoms, rate of progression, and response to treatment”. Hence, he believed, we needed to start to think of dimensions without sharp boundaries between distinct disease entities. And, perhaps most damaging for the hopes that were vested in neurobiology to resolve the diagnostic problems of psychiatry, he questioned the view that it might ever be possible to draw clear boundaries between ill and well.
The NIMH, under Hyman’s successor, Thomas Insel, began to set out its alternative approach well before the ink was dry on DSM 5.0. Faced with the reality that DSM does not, and will not, map on to neurobiology, the choice was clear – abandon DSM as a guide to psychiatric research, or abandon the belief that one could discover clinically relevant neurobiological bases for specific psychiatric conditions. In June 2011, when NIMH set out its Research Domain Criteria (RDoC), it acknowledged the failure to ground DSM categories in the brain, but asked “could specificity in fact exist, but not for the currently recognized clinical categories?... If we assume that the clinical syndromes based on subjective symptoms are unique and unitary disorders, we undercut the power of biology to identify illnesses linked to pathophysiology and we limit the development of more specific treatments.” The RDoC, therefore will allow biology, and not the experience of the patient, to delineate diseases – it is, it seems, the body, not the patient, that is sick. This process will be, to say the least “agnostic about current disorder categories. The intent is to generate classifications stemming from basic behavioral neuroscience. Rather than starting with an illness definition and seeking its neurobiological underpinnings, RDoC begins with current understandings of behavior-brain relationships and links them to clinical phenomena.” And at end of April 2013, Thomas Insel announced that: “NIMH will be re-orienting its research away from DSM categories” on the grounds that “Mental disorders are biological disorders involving brain circuits that implicate specific domains of cognition, emotion or behavior’ – and it is in terms of these that diagnosis should proceed and towards these that treatment should be targeted. For now, DSM may be a useful clinical guide and bureaucratic tool. But when it comes to characterising the aetiology of mental disorders, one must trust in the brain, not in DSM.

How we should evaluate Insel’s claim depends, of course, on the evidence - whether it is possible to identify such disordered circuits and link them reliably to an individual ailment. For at present, the hypothesis that there is neurobiological specificity to psychiatric disorders remains an hypothesis. Further, to evaluate this claim requires thinking more about how we understand the words like ‘are’, ‘involving’ and ‘implicate’. This may sound pedantic, but it is not, for the answer will shape the direction of research. Are these disordered brain circuits the reality underpinning the ailment? Or, as I would argue, are these neurobiological disturbances elements in complex biopsychosocial pathways, some if not all of which may be specific to individual patients.
Where is a disease?

In 2006, Charles Rosenberg, perhaps the foremost historian of contemporary medicine, published an essay entitled ‘Contested Boundaries: Psychiatry, Disease, and Diagnosis. His conclusion was challenging: “We have never been more aware of the arbitrary and constructed quality of psychiatric diagnoses, yet we have never been more dependent on them than now, in an era characterized by the increasingly bureaucratic management of health care and an increasingly pervasive reductionism in the explanation of normal, as well as pathological behaviour.” (Rosenberg 2006). Some hoped that the revision of DSM would at least move towards a resolution of the first part of this dilemma, not merely basing diagnoses on less arbitrary categories, but also recognizing the dimensional nature of many disorders, their ‘fuzzy’ borders, their non-categorical realities, and going some way towards a system that diagnosed in terms of degrees of impairment of functioning rather than allocation to static categories. While we have yet to fully digest the revised version and to observe it in action, at first sight it fails these tests. As I have said, diagnostic biomarkers are notable for their complete absence. The symptom based checklists for the allocation of categorical diagnostic codes are, if anything more complex than before. Comorbidities are acknowledged, but nothing more. Dimensions are absent, merely gestured to by the fact that some diagnoses allow for a classification of severity – mild, moderate or severe. In all these respects, despite the involvement of dozens of experts, the investment of hundreds of person hours, the politest verdict one can make is that while DSM, at least in the United States, will remain embedded in health care bureaucracy, DSM-5 makes few if any clinical, scientific or intellectual advances over its predecessors.

This, then, is a good moment to consider the fundamental links between the DSM conception of diagnosis and the prevailing belief that mental disorders take the form of specific diseases. Another of Rosenberg’s challenging essays is helpful here: it is entitled “The Tyranny of Diagnosis: Specific Entities and Individual Sickness” (Rosenberg 2002). Rosenberg points to the fact that, as we entered the 21st Century, specific disease categories were omnipresent: it seems as if we could be objective about disease only when we could pin down the specific character of an underlying pathology – he refers to this as ‘the specificity revolution’. As the phrase suggests, it was not always so. Indeed he is drawing here upon an earlier great historian of medicine, Owsei Temkin. Temkin distinguished two ways of understanding disease – ontological and physiological (Temkin 1977; Rosenberg 2003). Ontological: individual diseases exist independently of
their manifestations in particular men and women. Physiological: disease is a fundamentally individual phenomenon, the consequence of uniquely configured factors in particular men and women interacting with their environmental circumstances. Physiological, here is a strange term: this is the approach that Rosenberg calls ‘individual sickness’.

Temkin wisely warns against celebrating either the ontological or the individual conception. But today, medical research has veered strongly to the former – the ontological notion of disease. An ontological notion of disease does not have to be linked to a categorical idea of diseases as specific entities. And despite their turn away from DSM, NIMH’s RDoC are ontological, based on the hypothesis, or the hope, that mental health problems are diseases like any other, and that different diseases have distinct brain based pathophysologies. However they are based on another assumption, which is that the underlying pathophysiology is multi-dimensional, involving anomalies on a number of distinct neurobiological circuits which together underpin, produce, account for and explain the ailment experienced by the patient.

For now, the RDoC approach is intended to guide research rather than practice, supplementing, not undermining, DSM’s use in practical settings. But the dream remains that, in the future, these will move from research to the clinic, for the RDoC is based on the belief that mental disorders must, in the end, be understood as in some sense disorders of the brain. One wonders how they envisage their brain circuit based diagnoses operating in clinical settings. Could one imagine a clinical diagnosis operating in these terms, for example: “the patient scored in the fourth quartile on the negative valence system, in the second quartile on the positive valence system, was slightly below average in cognitive functioning, had severe disruption in systems for social processes and was in the top centile for sensitivity in the arousal regulatory system.” Evidence in other areas, for example in risk profiles, suggests that the lure of categories is powerful. Both patients and clinicians seek simpler ways of making sense of such dimensions and communicating them, thus it is likely that these algorithms would be regrouped into diagnostic categories, with contestable cut off points, types, subtypes, and so forth, replicating many of the problems encountered with DSM and its search for dimensionality. But we are getting ahead of ourselves. For the fundamental question is whether an appeal to the brain, or to the genetic sequences that shape the structure and function of the neuronal architecture, or to the new visibility apparently conferred by neuroimaging, finally enable clinicians to delineate the boundaries of normality, and to
differentiate within disorder – to decide who is, and who is not, a suitable case for which kind of treatment? Could neuroscience ever provide psychiatry with a meaningful kind of objectivity that it seeks?

Conclusion

It perhaps seemed pernickety when I said that an evaluation of Insel’s claim that “Mental disorders are biological disorders involving brain circuits that implicate specific domains of cognition, emotion or behavior” depended on how we understand the words like ‘are’, ‘involving’ and ‘implicate’ – as fundamental causes, or as elements in a complex and highly individual biopsychosocial pathway. Few, I think, would dispute that, in principle, neurobiological processes are involved in variations in cognition, emotion, volition and other human faculties. But the question is in what way – where is the disease? The RDoC criteria are “organized around basic neural circuits, their genetic and molecular/cellular building blocks, and the dimensions of functioning that they implement”. But RDoC also, so it is claimed, aims to foster two important dimensions that are not ‘in the brain’ – developmental aspects and interactions with the environment and their relation to specific circuits and functions. If those developmental and environmental factors are relevant, then, they are so via their influence on these neural circuits. But I suggested, we need to be even more radical in our approach, and to move towards the conception of disease that Temkin understood as ‘individual sickness’ – and that others who thought like him such as Kurt Goldstein tried to capture in their recognition of disease as a matter of the whole living organism in its milieu. As Goldstein put it in another context “only such methods will really carry us further which consider the single phenomenon in its significance for the whole organism. Only investigations with that emphasis will give us an understanding of the actual meaning of a phenomenon in respect to its functional significance … for the organism in question” (Goldstein 1995 [1939]: 215).

It is not merely in the clinic that we need to question the belief that a disease is specific entity defined by a unique pathophysiology. Psychiatric research should also begin with the person in his or her situation, and seek to trace the constellation of factors across the life-course that have shaped the ailment as a reality that is both biological and social – indeed that requires us to question that misleading distinction. Undoubtedly patterns will arise at this level, indeed the social sciences have been tracing these for over a century. These patterns within the milieu – a form of life, a location in city or
countryside, a rhythm of work, a pattern of interactions - are not merely external to the
disorder, influencing its mode of expression – they are constitutive of the disorder and
its inscription in body and brain. And it is in the milieu, in a form of living with its
possibilities and its limits, that the disease inheres. And only by understanding that will
research translate into to clinical practice.

For both research and treatment then, we should start in the experience of the ailing
individual, the extent of their troubles, the pattern of impairment of some of their
capacities in particular familial, social, cultural, and indeed economic situations, their
compensatory strengths and resiliencies. What would a diagnostic system look like if it
focused, neither on disease entities nor on neurobiological markers, but on patterns of
capabilities, impairment and resilience and the kinds of support that might alleviate or
mitigate them? That, it seems to me, is a challenge both to DSM and to RDoC. It is also,
of course, a challenge to social scientists who wish to resume their truncated dialogue
with biological psychiatry, and to address the challenge of analysing how experience
gets under the skin.

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have not addressed in this draft.
FOOTNOTES

1 This, for example, was roughly the position taken by Professor Simon Wessley of the Institute of Psychiatry at King’s College London in an article that he wrote to coincide with the launch of DSM 5: http://www.guardian.co.uk/science/2013/may/12/DSM-5-conspiracy-laughable

2 There is a growing interest in the sociology of diagnosis: see for example (Jutel 2009; Jutel and Nettleton 2011; McGann 2011)

3 http://www.patientslikeme.com/

4 My argument here, of course, is that what we have in these manuals are institutional epistemologies.


6 http://www.nimh.nih.gov/about/director/index.shtml

7 Of course, this may be true of many other kinds of disease.

8 http://www.nimh.nih.gov/about/director/index.shtml

9 Of course, this does not imply that interventions on the brain and body, for example with pharmacology or by brain stimulation, cannot play an important role in alleviating particular conditions, and perhaps even allowing a return to normal functioning.

10 George Szmukler has pointed out to me that “In the good old days, we used to write a ‘formulation’ of the patient’s problems. This included a summary of the problems; a diagnostic assessment; an account of possible aetiological factors; treatment options; and prognosis.” For further details of this, lost and lamented approach, see (Greenberg, Szmukler et al. 1986)
References


