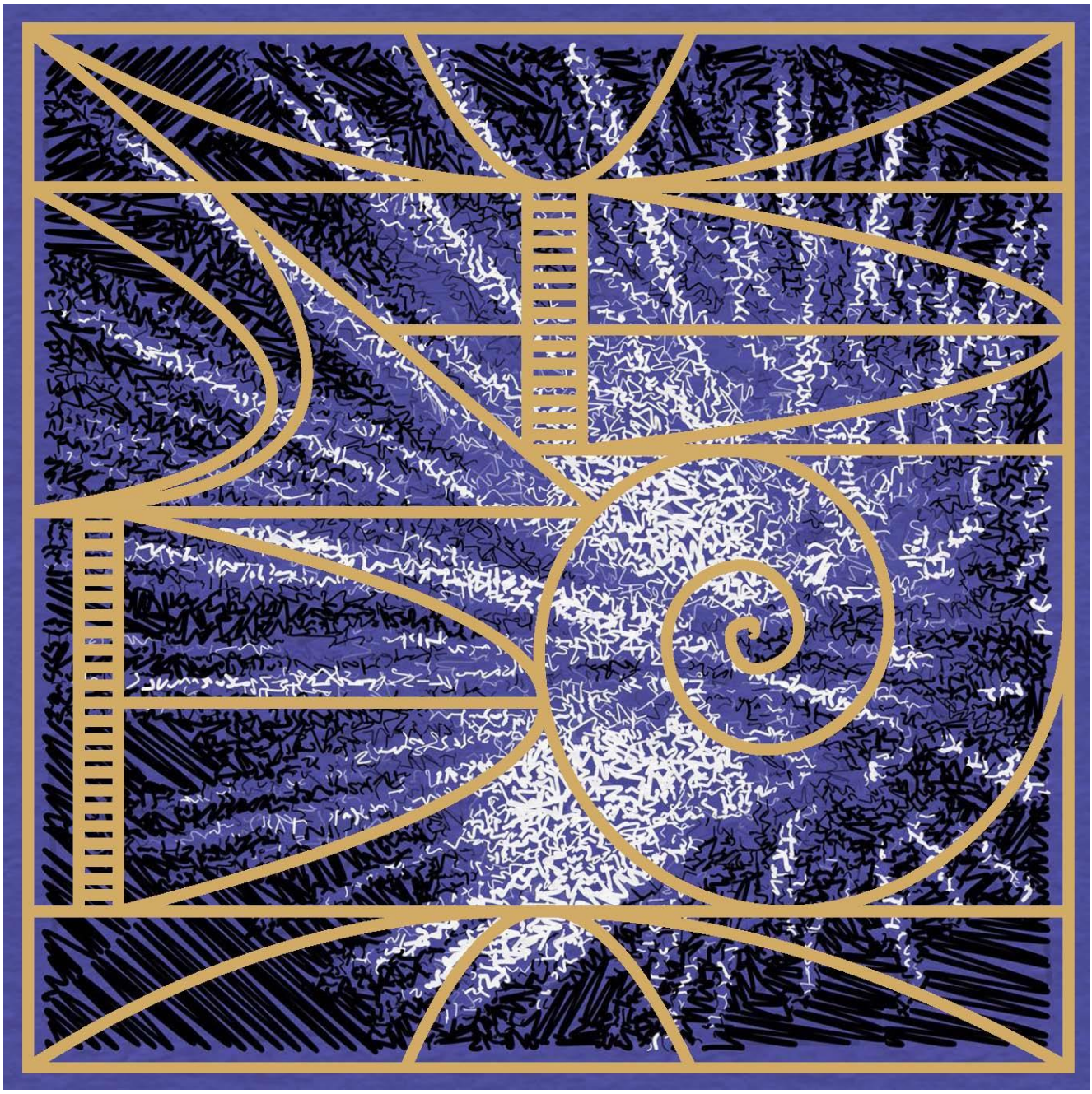

The 28th Annual Meeting of the Association for the Advancement of Philosophy & Psychiatry

Philosophical Issues in Scientific Psychiatry: RDoC, DSM, Mechanisms, & More

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AAPP 2016: ABSTRACTS

RDOC: OUT OF THE FIRE AND INTO THE FRYING PAN?
Robyn Bluhm

For several years now, the National Institutes of Mental Health (NIMH) has been developing a novel framework to guide research on mental disorders. The Research Domain Criteria (RDoC) project is intended to replace the framework provided by current diagnostic categories. According to the former NIMH Director, Thomas Insel, and his colleagues, the current categories are failing both psychiatrists and patients, in large part because "these categories, based upon presenting signs and symptoms, may not capture fundamental underlying mechanisms of dysfunction."

RDoC, by contrast, aims "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." My purpose in this talk is to address some of the challenges that RDoC will face in working toward this goal. In particular, I argue that our "current understandings of brain behavior relationships" raise exactly the same kind of problems as do the diagnostic categories that RDoC hopes eventually to replace. What we know about the brain maps poorly onto both current diagnostic categories and current psychological theories. RDoC may therefore offer only the illusion of an improvement on the current situation.

In making this argument, I will first describe RDoC and show that the presentation of the project as a "matrix" downplays the complexity of its goals. Briefly, RDoC consists of a set of domains of functioning, each of which is subdivided into "constructs" which represent "a specified functional dimension of behavior". These "functional dimensions" are wide-ranging, including various aspects of perception, of social processes, of memory and cognition, and of reward processing. The constructs are listed as the *rows* of a table or matrix. The *columns* of the matrix represent different levels of analysis at which the construct can be studied, including genes, neural circuits, behavior, or self-reports. Currently, the cells are filled with lists of genes, brain regions, etc. that have been associated with the construct. Further research is supposed to provide more detail about the biology or the behavior associated with the construct, as well as "to integrate and synthesize multiple measures to lead to a more comprehensive understanding of the constructs in the matrix, and in turn to the symptoms to which they relate" (NIMH RDoC FAQ page). But in order to achieve the goal of integration, researchers must understand the relationships among the various levels of analysis, including those that focus on biology and those that focus on experiences and behavior (whether assessed experimentally or via self-report).

With this background in place, I will then describe an ongoing discussion among cognitive neuroscientists about the difficulty of linking cognitive and behavioral theories to neuroscientific ones: numerous researchers have argued that progress in neuroscience now depends on the development of a new cognitive "ontology," that is, a new taxonomy of cognitive processes. Even in the case of the best-developed psychological theories, it has not yet been possible to show how the cognitive processes involved are realized in at the neural level. In the remainder of the talk, I will show how these problems will arise using as examples the constructs of "reward valuation" and "understanding mental states". Both of these constructs already have a great deal of information listed for each level, but neither is close to the kind of integration that RDoC both desires and needs.

HANDWAVING AT VALIDITY – CAN WE MEASURE PSYCHOLOGICAL CONSTRUCTS?
Claire Pouncey

The Research Domain Criteria project (RDoC) is meant to make psychiatric research more objective, and psychiatric theory more valid. RDoC departs from traditional psychiatric research in three ways. First, it adds findings from new measurement technologies in molecular biology, neuroimaging, and neurophysiology to the self-reports and psychological assessments traditionally used to identify and measure psychological constructs (theoretical entities). Second, it eliminates DSM/ICD mental disorders as constructs that organize psychiatric research. Third, RDoC includes in its studies the full range of normal to pathological functioning for the constructs of interest. Newer "high-tech" research methods are meant to make psychiatric science more quantitative, and thus more objective, and to provide theoretical validators that were never found for DSM mental disorder constructs. The assumption is that "high-tech" research methods will provide insights into causal mechanisms of mental dysfunction than "low tech" methods allow.

Psychology has developed a number of "validities" – e.g., content-, face-, construct-, and predictive validity – to evaluate how well assessment tests measure what they are intended to measure, and how they may be interpreted. Within psychology, validities fall in and out of favor, their meanings are subject to ongoing debate and revision, and "validity" is not synonymous with "objectivity." Both psychiatry and philosophy of psychiatry tend to overlook this mutability of validity concepts, as well as the conceptual difference between validity and validation. RDoC follows Murphy in assuming that "our knowledge of social, cognitive, and molecular neuroscience is now good enough that we can build a theory of psychopathology that borrows its basic assumptions from neuroscience...". Schaffner calls

this search for causal explanation at lower explanatory levels "etiopathogenic validation," such that psychophysiologic findings "point to the cause of a disorder, or … clarify the pathogenetic process involved in a disorder." This view of validity requires the integration of quantitative measurements at different levels of explanation. Indeed, the means for this cumulative inter-level integration of quantitative data is part of the RDoC project .

The hope that data from research studies at lower levels of explanation can be integrated with the results of studies at higher levels of explanation is understandable, but a hope is all it is. Joel Michell questions whether psychological constructs are quantitative at all, and thus whether there is anything to measure – in a strict technical sense – in either low-tech psychological assessments or high-tech biological research. Michell doubts the axiom (endorsed by Carl Hempel among many others) that as a science develops its theory becomes progressively quantitative. Michell argues that psychological constructs may be ordered without being quantitative, that is, without having consistent ratio intervals between them. He characterizes the "psychometrician's fallacy" of assuming rather than proving that ordinal distinctions identified for psychological constructs are also quantitative. If they are not quantitative, psychological assessment tests do not measure. I apply Michell's work to argue that this concern applies to RDoC's low-level, high-tech biological tests as well: if constructs are not quantitative, then having more precise measurements across a variety of "validators" does not do the work RDoC expects it to do.

RDOC AND THE PROBLEM OF NORMATIVITY
Reinier Schuur

Wakefield argues that RDoC will fail because it doesn't address 'concept validity', the problem of distinguishing pathology from 'normality," i.e., the 'problem of normativity.'

I will argue that, while RDoC can side-step Wakefield's challenge, other problems of normativity still remain that need to be addressed if the RDoC project is to succeed in the future. This talk sets out two arguments for how we should frame this debate; (1) I argue that accounts of pathology in the concept of disease debate are not suited for guiding empirical concerns of normativity, because they are based on conceptual analysis; (2) We should instead turn to the philosophy of biology to frame the problem of normativity facing psychiatric research.

Wakefield argues that the RDoC project will fail because it does not address 'concept validity', the issue of delineating pathological states from normal variation. Others have also argued that RDoC assumes a 'value-free account' of pathology.

I will argue that regarding RDoC as 'value-free' reads too much into it, as it is currently uncommitted on this issue. Indeed, the RDoC project seems to be able to side-step Wakefield's specific challenge, by researching symptom expressions across clinical and non-clinical populations, leaving it to future nosologists to decide on what counts as genuinely pathological.

But there are other ways in which the 'problem of normativity' might come up in research. Even if RDoC remains agnostic about dysfunction, it is still committed to understanding various mental functions. However, understanding and specifying the role of mental functions necessarily involves *some* normative notions of what these mental functions are *for.* If RDoC adopts an overly limited conception of mental functions, it risks impoverishing our empirical understanding of them.

The most obvious place to turn to accounts of pathology-normality to inform RDoC seems to be the concept of disease debate in the philosophy of medicine. However, I will argue that such accounts are ill-suited to guiding empirical research on issues of normativity. This is because such accounts are based on conceptual analysis, which aim to capture how we use the concept of disease and our intuitions about them, not about what the nature of pathology actually *is*. There are no good justifications why empirical research should be constrained by common sense in this way.

I argue that we should instead turn to the literature on functions in the philosophy of biology to frame the problem of normativity facing psychiatric research. We should especially look at new work being done on normative issues of analysing the 'good' of the organism based on its 'lifehistory.' This will give us a good framework to analyse the nature of normative concepts in biomedicine beyond the traditional conceptual analysis that is done in the philosophy of medicine. On this basis we can begin to have a debate about whether or not such an approach can be scaled up to psychiatric research, or if we have to make room for extra-biological and/or cultural conceptions of the norms of mental functions.

LETTING MANY FLOWERS BLOOM: THE IMPORTANCE OF METHODOLOGICAL PLURALISM IN THE STUDY OF MENTAL ILLNESS
Brent Kious

The Research Domain Criteria (RDoC) are intended to rescue the medical model in psychiatry from perceived failings in the DSM. The DSMs themselves were intended to support the development of the medical model by picking out natural kinds—that is, distinct diseases—through symptom clusters. In the main, however, DSM diagnostic categories have not revealed their etiologies, which

suggests to some that they do not correspond to natural kinds. If the skeptics are correct, it may be in part because the symptoms used to make DSM diagnoses are highly subjective. The RDoC aim to rectify this failing: by proposing that investigations in psychiatry should be oriented toward particular domains of functioning, where these include structural, behavioral, and physiological correlates of mental illness, they purport to shift the direction of psychiatric research toward more objective, replicable markers of disease that may correspond more consistently to basic etiologies.

Here I will argue that both the RDoC and DSM-based attempts to structure psychiatric research are misplaced, at least if we assume that the aim of such research is to identify the causes of mental illnesses. This is because both attempts involve an effort to control the direction of psychiatric research in a top-down fashion: effectively, both make *a priori* claims about the level of description of psychiatric phenomena that is most likely to identify causes—for the DSM, symptom clusters, and for RDoC, domains of functioning. But the levels of description that are most likely to be fruitful for causal investigations is actually an exquisitely empirical matter and probably dependent on which disease processes are being investigated. Examples from general medicine demonstrate this: a medical condition like tuberculosis is fairly reliably identified by a symptom cluster: most persons with night sweats, weight loss, hemoptysis, and mild fever exhibit the same underlying physiological process—the formation of granulomas in the lungs—which is likely to be caused by the same condition, namely infection with *Mycobacterium tuberculosis*; and most persons infected with *M. tuberculosis* are likely to have a similar response to the same medication. But for a condition like lung cancer, symptom clusters may be less informative: even if persons with large lung masses all have cancer (and even if those cancers are physiologically or histologically similar), they may exist because of very different underlying genetic changes, which more reliably predict treatment response.

The RDoC suffer from similar failings. Analogizing again to physical illnesses, the RDoC directives are similar to asking physiologists to look for the cause of coughing per se, rather than of the symptom cluster that is characteristic of tuberculosis, which includes coughing. Even if this is likely to be fruitful in some respects—it may help elucidate the mechanisms that produce coughing whatever the disease process—it may not be the most efficient way of identifying particular causes of cough (lung cancer versus TB). To consider one example, fear (one of the RDoC negative valence domains) is obviously present in a wide variety of mental illnesses (and in those who are not ill), so it may not be true that studying fear will quickly lead to insights about the causes of any of those conditions.

Thus, in some cases, DSM-level descriptions of psychiatric phenomena could be quite fruitful as guides for scientific study. In others, RDoC-level descriptions could be. Ultimately, then, the best approach to promoting progress in the science of mental illness—especially for an organization like NIMH, which determines what kinds of research get funded—is to let many flowers bloom, and to support high quality research involving a plurality of methodological and conceptual frames.

NEUROCENTRISM: IMPLICATIONS FOR CONCEPTUALIZATIONS OF MENTAL DISORDER

Scott Lilienfeld

In this talk will examine the recent trend toward *neurocentrism* – the view that the brain is inherently the most important level of analysis for approaching psychological questions – and examine its implications for psychiatric research, psychiatric education, psychotherapy, and other domains. The question of whether mental disorders should be conceptualized as brain diseases will be briefly addressed. In addition, key conceptual challenges to the interpretation of brain imaging and other neuroscience data will be explored. It will conclude with a brief discussion of what such data can add to our understanding of human suffering.

EXTENDED MENTAL DISORDER: WORRIES FOR REDUCTIONISM

Rachel Cooper

In their classic 1998 paper “The Extended Mind”, Andrew Clark and David Chalmers argue that the mind is not contained within the skull but can extend into the world. This paper examines the implications of the thesis of the extended mind for understanding psychopathology. I explore whether and when mental disorders can be extended via considering three sorts of case; difficulties with cognition and memory, impulse control, and affective states.

Problems associated with beliefs, IQ and memory are amongst those that might most easily be understood in terms of the extended mind. In their original paper Clark and Chalmers discuss the case of a person with brain-related memory problems who uses a notebook to record matters to be remembered. In so far as the notebook can fulfill the function of the damaged brain-related memory, they say that the notebook can be considered to be part of that human's mind. In so far as beliefs and memories can be extended this raises the possibilities that when problems arise these may be grounded in events external to the brain. Rather than seeking atrophied brain regions we might need to look for destroyed diaries. I consider whether this way of looking at things can provide a good account of the deterioration that often occurs when patients with dementia are moved from their homes. Can the fact that the individual has been uprooted from the external system that contained their memories and supported their thinking provide an account of their difficulties?

My second case concerns impulse control. Many people manage their anger using techniques that depend on environmental support; they train themselves to walk away from provocation. Walking away both helps the angry person to calm down and prevents them from

reacting violently when provoked. When something goes wrong with such extended systems of anger management, the source of the difficulty may be external to the brain. In certain environments, for example, prison cells and closely controlled school class rooms, people are unable to walk away when angry. Their problems may be rooted in environmental features rather than in their brains.

The final cases I shall consider concern problems with emotion and mood. In so far as such states seem intrinsically linked to their phenomenology, such affective states are amongst those that can least plausibly be considered extended. I use ideas from Colombetti & Roberts to argue that at least certain types of problematic affective state can be considered extended.

Many projects that seek to understand psychopathology implicitly assume that at least the proximate grounds of psychopathology are to be located in the brain. Though it is widely acknowledged that at least certain mental disorders have social causes these are often assumed to act via having effects on brain mechanisms. When mental disorders can be considered extended, this poses problems for the reductionist projects in that play a major role in current psychiatric research. In so far as psychiatric symptoms are grounded in the external world rather than in brains, it will not be possible to understand them by brain scanning studies.

PSYCHOPATHY AND SCIENCE
Emma Satloff-Bedrick & Jeff Bedrick

It is often assumed that understanding normal development and function will help us understand what goes wrong in dysfunction and disease, and, conversely, that understanding what goes wrong in the case of pathology may throw light on what is involved in normal development and function. In this paper I am going to discuss the case of the development of what is known as theory of mind in psychology to show that the relationship between normal development and functioning and pathological development and functioning is not always straightforward, and so we must be extremely cautious in drawing inferences from what we know about one to the other.

Children with callous-unemotional traits present a unique problem for the larger theory of mind literature: their cognitive theory of mind abilities seem to be intact or even heightened compared to typically developing children while their affective theory of mind seems entirely deficient. As I will discuss briefly in this paper, it appears that affective theory of mind (ToM) both precedes and contributes to cognitive ToM among typically developing children; whereas children with callous-unemotional traits, often thought to be a precursor to psychopathy, seem to develop cognitive ToM in the absence of typical affective skills.

What are we to make of the developmental trajectory of children with callous-unemotional (CU) traits? They end up with cognitive ToM skills equal to or better than those of their typically developing peers, yet they clearly arrive there via different mechanisms. Children with autism spectrum disorders have noted difficulties with both cognitive and affective ToM and particularly perspective-taking tasks, and so they do not necessarily challenge the typical model. Children with CU traits, on the other hand, present a clear problem. They lack the foundational understanding upon which cognitive ToM is predicated for typically developing children, yet their cognitive ToM skills do not suffer for it. In this way, children with CU traits present an important challenge to typical developmental models. They suggest that there are multiple, equally successful pathways to the development of cognitive ToM, and that in some children deficits in affective ToM may be separated from any impact on cognitive ToM.

This points to our need for caution in drawing inferences from normal function and development to atypical function and development and vice versa. Our theoretical models must take into account that the underlying development of typical and atypical functioning may proceed along very different lines, and that atypical or even pathological functioning may not represent a simple deviation or problem in the course of typical development and functioning. I have discussed this theoretical issue in the context of one particular difference between typical and atypical development. There is no reason to believe, however, that the development of cognitive ToM represents the only development of a function that would differ significantly between the typical and atypical cohorts. If we are to be sure that we are understanding the typical and the atypical, the normal and the pathological, correctly, we must investigate each independently, and not assume that we can make inferences from one to the other – although we might obviously generate research hypotheses in this way.

ON THE ETHICS OF DESCRIPTION IN PSYCHIATRIC NOSOLOGY FROM DSM TO RDOC
Kathryn Tabb

Psychiatric nosology is going through a profound change, as the hegemony of the Diagnostic and Statistical Manual of Mental Disorders (DSM) is challenged by a new approach to identifying targets for psychiatric research: the National Institute of Mental Health's Research Domain Criteria Project (RDoC). While the metaphysical and epistemological significance of this shift is being explored by philosophers of psychiatry, it is also crucial to assess the ethical implications of the change. After explaining the transformation of psychiatric ontology underway, I explore some consequences of practical import in terms of bioethics' four principles: autonomy, beneficence, non-maleficence, and justice. I conclude by drawing on standpoint theory to argue that there is an ethical obligation to consider the professed care needs of present and future patients before the explanatory needs of scientific investigators.

I begin by discussing work by philosophers on what the metaphysics of psychiatric kinds can tell us about best nosological practices. Philosophers are beginning to offer rewarding philosophical analyses of the sea change that is underway in how psychiatrists conceive of their objects of inquiry. While this work in the epistemology and metaphysics of psychiatric kinds is valuable, I maintain that it is no longer appropriate to continue it without taking its practical implications into account. The metaphysics of kinds has repercussions for an area of psychiatric ethics that does not get much attention, the ethics of classification. But it also has profound repercussions for other themes in psychiatric ethics: the initial act of diagnosis is what makes possible those further acts traditionally of interest to psychiatric ethicists, such as the regulation of patient autonomy, the pursuit of beneficence and the avoidance of maleficence, and the maintenance of justice.

After reviewing the ethical issues that have long been brought to bear around the DSM's conception of disorder, the main focus of my talk will be on new ethical challenges brought on by RDoC's re-envisioning of psychiatry as "clinical neuroscience." In the first place, what sort of extrapolation is at work in the application of conclusions from hypothesis-driven basic science research to psychopathology? My first claim is that the determination of the normal and the pathological is an ethical problem, which cannot be reduced away. Secondly, is the reduction of a sign or symptom to an extreme on a spectrum of variation sufficient, or must psychiatric research do more than provide correlates at lower levels? I argue that the role of the self in psychiatry is crucial, and risks exclusion under RDoC. Finally, what should count as an explanation in psychiatry? I claim that this paradigmatic epistemological question becomes an ethical one when our scientific methods and research priorities determine the distribution of resources between basic science questions – whose payoff is far in the future – and sociological, epidemiological, and clinical studies with more immediate benefits for patients.

Because of the conflicts between the epistemological virtues motivating the turn towards clinical neuroscience and its potential ethical costs, a pragmatic and nuanced response will be necessary for philosophers aiming to offer a prescriptive account of psychiatric kinds. I conclude that the best way to proceed is to take the perspectives of the diverse stake-holders in psychiatry into account, but to privilege first the "prudential values" of psychiatric service-users over the epistemic, financial, or other values of researchers.

THE NORMAL, THE PATHOLOGICAL, AND RDOC: WHAT ABOUT RACE AND GENDER?
Doug Porter

The research domain criteria project (RDoC) is an attempt by the National Institute of Mental Health to develop a new research classificatory system for mental disorders. To some extent the RDoC project can be seen to have emerged from frustration; recent developments in behavioral science and neuroscience have simply not mapped well onto the diagnostic categories currently contained in the DSM. The criteria delineated for a valid RDoC construct, namely that it contain a behavioral component with significance for the study of psychopathology and that the same behavioral component lend itself well to implementation in neurocircuitry, are simple but logically suited to facilitate translational neuroscientific research with significance for the study of psychopathology. Nonetheless, the RDoC project contains many claims and assumptions that are not so simple and straightforward. One assertion that demands scrutiny is that it is the task of RDoC to, "Determine the full range of variation, from normal to abnormal, among the fundamental components to improve understanding what is typical versus pathological." It seems that those involved in the RDoC project believe that differentiating the normal from the pathological is an empirical matter, citing at times concepts of evolutionary science and statistical norms to substantiate those beliefs. Of course there is a legacy of criticism of more carefully formulated thoughts on these matters as they were developed by thinkers such as Boorse and Wakefield. But my concern specifically here is that the RDoC treatment of the normal versus pathological is highly problematic when it comes to categories with social significance such as race and gender.

One caveat that has been pointed out with regard to statistical norms and pathology is the difficulty in determining the presence of meaningful subgroups. Medical research has been criticized in the past for using white males in research and then misapplying those norms to different genders and races. The Negative Valence Systems: Workshop Proceedings for RDoC notes that sex differences may be significant for the study of aggression. Presumably biological differences between the sexes could create meaningful differences in the neurocircuitry of aggression. But, if the RDoC project does succeed in elucidating the neurobiology of aggression, and empirical differences are detected between the sexes, should we attribute these differences merely to biological differences between the sexes? Are social norms with regard to tolerability of aggression in women responsible for different trajectories in neural development? If social factors are responsible for differences between the sexes it appears that RDoC is in a poor position to elucidate them. The explanatory factors contained in the RDoC matrix include genes and physiology, but the matrix does not appear to contain room for socio-cultural explanations of difference. Could we misattribute differences detected between races to differences in genetics: "The Bell Curve" and racial differences in IQ come to mind. The example of aggression in women also points to concerns about using adaptation as a normative barometer of health when we are adapting to contingent social norms that may be patently unjust. What should the norms for fear circuitry be in an era where the phrase "Black Lives Matter" is not a ludicrous overstatement of the obvious? I have no doubt that RDoC as it is currently conceived is in a position to develop evidence that can meaningfully inform the debate about the boundaries of the normal and the psychopathological. But exploration of the meaning of race and gender helps demonstrate the need for humility in determining those boundaries.

ATTACHMENT WITHIN THE RDOC: PROMISING 'BIOMARKERS' CARRYING DECEPTIVELY COMPLEX CONCEPTUAL BAGGAGE
Kevin Keith

The Research Domain Criteria Project (RDoC) has included 'attachment and affiliation' amongst its hierarchically arranged functional constructs under the *Systems for Social Processes* domain. In doing so they argue for a fit between attachment and the project's two main selection criteria: "strong evidence for the validity of the suggested construct" and "strong evidence that the suggested construct maps onto a specific biological system, such as a brain circuit" (Cuthbert & Insel, 2013, p. 6). In this brief presentation, I will argue in favour of this inclusion whilst also advising a healthy dose of conceptual caution.

In addition to the selection evidence cited for inclusion, Attachment Theory has also in places generated just the sort of translational or causal interventionist evidence endorsed by the RDoC. However, included with my endorsement, I also want to engage three important conceptual questions in the study of attachment relevant for consideration by the RDoC project—or similar endeavours. [These questions reflect ideas generated from my current PhD research concluding in 2016: Attachment Theory within a framework of philosophy of psychiatry.] (1) What exactly is an 'attachment' within the field of Attachment Theory? The answer is paradoxically more complex than researchers may acknowledge, especially when considering the seemingly unique *heterotypic lifespan development* of attachment in the human species. (2) What is the current state of the attachment neurobiological evidence that might support inclusion by the RDoC project? Again, although much valuable research exists for attachment at the various neurobiological levels, I also suggest its value may be diminished by a lack of conceptual precision arising from my initial question. (3) And lastly, how might one explain the relationship between the neurobiological and cognitive/affective mechanisms that have been proposed to underpin attachment phenomena? On the one hand, as indicated in responding to question 2, 'attachment' has been shown to encompass multi-level phenomena that clearly include possible neural circuitry and molecular considerations. On the other hand, Bowlby's original recognition of a central role for cognitive/affective mechanisms for understanding attachment phenomena—i.e. Internal Working Models or metal models in cognitive science—has also generated important evidence not reducible to either neurological or molecular levels. In this context, I suggest how an arguably more dynamic or organisational understanding of attachment's multilevel mechanisms may generate additional challenges for the current RDoC matrix approach.

Thomas Insel has previously noted, "Social attachment is a complex process involving changes in sensory, cognitive, and motor functions." By including the attachment construct, the RDoC should expect a productive discernment of valuable biological markers. However, as Insel has intimated, such a task may also prove conceptually challenging when considering the potentially less recognised complexity of human attachment phenomena.

KEEP CALM AND EMBRACE FUTILITY
Alexander Parker

The philosopher of science Thomas Kuhn introduced the idea that scientific research tends to follow a paradigm. He also noted that scientific research periodically reaches a crisis point and that this is typically followed by a paradigm shift. Research in Psychiatry, it seems, has been facing a perennial crisis: failure to achieve results that meet or exceed traditional expectations. Consider, for example, that while the mortality rate due to infectious disease has declined drastically over the past century, deaths due to suicide have remained relatively stable. Such facts have led many academics to question the psychiatric research paradigms of their time. This is consistent with Thomas Kuhn's understanding of scientific progress. Yet, where Thomas Kuhn would expect to see a paradigm shift, what we have witnessed in Psychiatry is paradigm proliferation. Now, a multitude of research paradigms persistently populate the psychiatric research landscape. Not only that, but despite the plethora of research avenues being pursued, our understanding of mental disorder remains relatively incomplete, and the efficacy of psychiatric treatment remains modest.

A possible explanation is that this situation itself is an example of research following a paradigm. The paradigm here is to attribute stagnation in the advancement of our understanding, diagnosis, treatment, and prevention of mental disorder to inadequate investigation. Maybe the solution is to shift away from *this* paradigm and to embrace a more futilitarian mindset. Perhaps we should view the minimal gains that have been made scientifically and clinically in Psychiatry as evidence of a natural limit to what psychiatric research can possibly achieve.

Such a shift would not be without precedent. History has shown that sometimes what appears on the surface to be an extraordinarily challenging version of an established sort of problem turns out to impossible. In physics, for example, the impossibility of knowing the precise momentum and position of subatomic particles is now accepted as a foundational principle, though it took years for such an unlikely premise to gain traction. Now, decades of psychiatric research has inadvertently supplied sufficient empirical data to justify the establishment of its own futility as predetermined by the laws of nature. Apparently, understanding mental disorder as completely as we would like to is impossible. The evidence also suggests that advancement in our endeavor to understand mental disorder will never result in benefits comparable to those seen in other fields of medicine.

As a paradigm for future research in Psychiatry, a futilitarian approach is unlikely to bridge the intractable gap in understanding and clinical efficacy between Psychiatry and the other branches of medicine. History suggests, however, that even shifts towards futilitarian paradigms can lead to unforeseen fortuitous discoveries. In Physics, for example, accepting that measurements of the speed of light would forever return the same result set the stage for the discovery of nuclear power. So even though the conventional aims of psychiatric research may as much be the stuff of science fiction as traveling faster than the speed of light, perhaps if we come to embrace the apparent futility of psychiatric research, rather than dismiss it, fortune may shine down upon us yet, only in some other way.

THE EXPLANATORY IMPORTANCE OF LEVELS AND MECHANISMS
Kelso Cratsley

The identification and explanation of biological mechanisms across integrated levels of analysis is a fundamental commitment of many current psychiatric research programs, including those operating within the framework of the National Institute of Mental Health's *Research Domain Criteria* (RDoC). But recently this approach has been challenged on the grounds that it may not be explanatorily constructive for any number of psychiatric conditions. To begin with, it has been suggested that the increased prominence of multifactorial etiologies that appeal to both neurobiological and social/environmental factors is at odds with the integration of complimentary levels. Briefly put, causal factors that effectively cross levels – for example, social stressors compounding atypical neural development – aren't easily captured by the standard account of levels in cognitive science. This is because the standard account is primarily epistemic, with levels of analysis representing varied perspectives on a single phenomenon, with an interest in implementational relations rather than attending to the *causal* interactions of disparate phenomena required by much recent psychiatric theorizing. What's more, it has been argued that any prior commitment to mechanisms, along with a multiplicity of levels, is a misguided constraint on successful explanation. Here the implication is that causal factors should been taken wherever they are found, regardless of whether they can be characterized in mechanistic terms.

In this presentation I take up these challenges, defending the view that leveled and mechanistic assumptions are indeed essential to psychiatric research (and the study of the mind more generally). This first requires a brief introduction to the basics of mechanistic explanation, with its appeal to functionally specifiable component systems, as well as its application to the study of aberrant mental states. I also draw an initial distinction between more traditional, implementational levels of analysis versus the parts-whole, *organizational* levels of mechanistic explanation. With these conceptual pieces in place, I argue that theory building around many psychiatric conditions will continue to benefit from a commitment to levels and mechanisms. For a start, mechanistic explanation emphasizes the role of contextual factors external to the mechanism in question, rendering it entirely compatible with the recent upsurge of interest in social/environment causal factors in psychiatric research. In addition, the different levels of a mechanism simply refer to the distinct, causally relevant components internal to the system. Levels understood in this way only includes those parts that are doing causal work as part of the larger whole, thus avoiding any implementational – or overly reductive – assumptions. And as regards the search for mechanisms, while there are some serious questions as to whether all mental phenomenon can be mechanistically decomposed, the identification of causal connections leading to the elaboration of mechanisms remains an extremely useful explanatory strategy.

SHOULD PSYCHIATRIC NOSOLOGY BE CONSTRAINED BY UNDERLYING CAUSAL MECHANISMS?
Nicolaus Slouthouber

Recent endeavours to improve the diagnosis and treatment of mental illness have centred around ways in which psychiatric classification can be tied to investigations into the causal structure of mental disorders. The most noteworthy attempt to tie classification to causes has been the development of the Research Domain Criteria (RDoC) project, implemented by the National Institute of Mental Health. In an effort to transform psychiatric classification, proponents of the RDoC project are seeking to identify the causes of mental illness by incorporating evidence from genetics, neuroscience, and cognitive science. The purpose of the present paper is to highlight some of the ways in which philosophical accounts of mechanisms contribute to, or detract from, this approach to psychiatric classification. In particular, I consider Kendler, Zachar, and Craver's "mechanistic property cluster (MPC) kind" model for tying mental disorders to underlying causal mechanisms and etiology. On the MPC view, mental disorders should be seen as sharing "stable similarities" both among their clinical features and among their underlying mechanisms and etiology. The MPC framework, it is argued, is the best current model for thinking about mental disorders, since it is both a plausible account of mental disorders and a useful guide for building psychiatric classifications.

My contention in this paper is that the MPC framework provides limited guidance on how to build classifications of mental disorders. There are two reasons for this. First, within the MPC framework, there is a tension between the dual goals of developing a plausible account of mental disorders (i.e. accurately describing a portion of nature) and developing a useful taxonomy for the diagnosis and treatment of mental disorders. For instance, on the one hand, the MPC framework acknowledges the complex nature of mental disorders—i.e. that mental disorders participate in mechanisms at many levels of analysis (from lower-level biological mechanisms to higher-level socio-cultural mechanisms), as well as in both proximal and distal etiological mechanisms. On the other hand, capturing all of this complexity would, I contend, render the MPC model useless as a practical and implementable guide for classifying, diagnosing, and

treating mental disorders. So, it would seem that advocates of the MPC model must strike a balance between descriptive accuracy and usefulness either by emphasizing some mechanisms over others or by describing mechanisms at a level of detail that is manageable. This, I contend, runs the risk of ignoring mechanisms and details of mechanisms that could prove important for the treatment of mental disorders. Secondly, although advocates of the MPC model do not give us the resources for individuating the mechanisms of mental disorders, I consider whether or not current research on mental disorders meets the norms of mechanistic explanation. In particular, I highlight some of the norms of mechanistic explanation presented by Craver. I argue that research on schizophrenia does not meet the norms of mechanistic explanation. For these two reasons I conclude that, absent reliable methods for individuating the mechanisms of psychiatric disorders, the MPC framework is neither the best current model for building psychiatric classifications nor for tying psychiatric classification to investigations into causal structure of mental disorders.

MISSING THE MIDDLE: PSYCHOSIS AND TEMPORAL LOBE EPILEPSY
Valerie Hardcastle

Shortly after RDoC was officially unveiled, Thomas Insel, the then-Director of the National Institute for Mental Health (NIMH), posted on his blog:

> Unlike our definitions of ischemic heart disease, lymphoma, or AIDS, the *DSM* diagnoses are based on a consensus about clusters of clinical symptoms, not any objective laboratory measure. In the rest of medicine, … symptom-based diagnosis … has been largely replaced in the past half-century as we have understood that symptoms alone rarely indicate the best choice of treatment. Patients with mental disorders deserve better.

He concludes by indicating that, for research purposes, the NIMH will be replacing the *DSM* with "Research Domain Criteria," which define mental disorders based on more specific, underlying, genetic, neural, and cognitive data. He wants to start with our best understanding of brain science and then to link that to clinical behavioral data. In other words, he is advocating for a bottom-up approach to mental disorders: understand first what is going on with the brain without reference to psychological categories, which can potentially mislead.

The case of post-ictal psychosis (PP) stemming from temporal lobe epilepsy (TLE) would seem to be a prime example of the fruitfulness of NIMH's new approach. Based on presenting symptoms, TLE with PP is often misdiagnosed as schizophrenia. However, the psychotic symptoms generally will not recede until the epileptic activity is brought under control. Knowing what the brain is doing during the psychotic episode is crucially important to managing the disorder. In addition, scientists are starting to find common brain pathologies in TLE with PP and schizophrenia.

At the same time, TLE with PP is often first overlooked as the correct diagnosis because treating neurologists can find no evidence of seizure activity, despite extremely sensitive imaging equipment. However, paying careful attention to patients' descriptions of their first-person experiences can provide a differential diagnosis, even absent documented seizures. Perhaps the bottom-up approach is not sufficient after all.

In this presentation, I shall argue that in our rush to reduce "self-report and behavior to underlying neural circuitry and activity" we are overlooking the fundamental importance of the "middle." While both first-person descriptions of experiential disturbances and underlying neuronal or chemical interactions are important for diagnosis and treatment, they often exist in conceptual isolation from one another. RDoC's hope is similar to John Bickle's ruthless reduction: with enough research, we will be able to causally connect cognition, experience, and meaningful behavior to modifications in molecular processes. However, as the case of TLE with PP illustrates, we are also in need of more than mere correlations between the higher and lower levels of description and analysis. For example, we are learning that memory is not just a case of changes in synaptic potentiation, but also of variably affiliated neurons pre-positioned for rapid recruitment. Because causally relevant events occur across multiple levels of organization, attempting to link psychiatric disorders to the lower level mechanisms, or using lower level mechanisms to redefine psychiatric disorders may not be the best research strategies, for both cases can overlook important mid-level events, structures, or states that might be definitive of a disorder or illness. I will provide an example of how the mid-level organization can contribute to diagnoses using recent brain morphometry analyses of TLE patients with PP.

RDOC'S SPECIAL KIND OF REDUCTIONISM AND ITS POSSIBLE IMPACT ON CLINICAL PSYCHIATRY
Simon Goyer and Luc Faucher

RDoC (as well as its European counterpart, ROAMER) is an explicit effort to break free from the constraints of current diagnostic categories and ultimately to rebuild a psychiatric taxonomy on new (biological) grounds. As Insel put it recently, "RDoC's ultimate goal is precision medicine for psychiatry — a diagnostic system based on a deeper understanding of the biological and psychosocial basis of a group of disorders that is unambiguously among the most disabling disorders of medicine" (2014, 396).

At the heart of RDoC is the idea that psychopathologies are the result of "abnormalities in discrete neurobehavioral systems" or "faulty circuits" of the brain. For this reason, the understanding of brain circuits underlying cognitive functions is the focus of RDoC, and neuroscience (both molecular neuroscience and cognitive neuroscience), its basic science.

The focus on faulty circuits has led some to suspect RDoC of being a reductionistic enterprise. And because RDoC will eventually impact clinical psychiatry, some have feared that it will transform clinical psychiatry in a mindless and applied neurobehavioral science. In our presentation, we want to look at RDoC presumed reductionism (section 2). As we will argue, RDoC is officially endorsing a kind of reductionism (which is referred to as "explanatory pluralism" or "patchy reductionism") that does not suffer from the shortcomings of more classical forms of reductionism. Because of that, we will argue that, at least *in principle*, RDoC could enrich rather than impoverish both psychiatric science and clinical psychiatry (section 3). We will end this presentation by trying to show how, despite its revolutionary potential for clinical psychiatry, RDoC could, *in practice* (if it is not true to its professed multi-level form of psychiatry), lead to the transformation of clinical psychiatry into an applied behavioural neuroscience (section 4). We will look at potential pitfalls of this. We will also look at the potential effects of the focus on faulty circuits on patients' self-conception. We will follow Telkin et al. in arguing that certain brain-centered conceptions of mental disorder could constitute an obstacle to recovery. Though we accept their analysis, we want to push it in a different direction by exploring the literature on genetic determinism and essentialism in psychology. This literature leads us to think that the negative impacts of a "biological" conception of mental disorder such as RDoC's is not restricted to the patients, but affect the clinicians and the general population as well. We end our presentation by considering steps to be taken to avoid these effects.

HEMPEL'S ACCOUNT OF PSYCHIATRIC TAXONOMY: ITS HISTORICAL AND CONTEMPORARY INTEREST
Jon Tsou

In the 1950s and 1960s, Carl Hempel advanced a normative account of psychiatric taxonomy in the context of a broader philosophical discussion concerning what constitutes a good scientific taxonomic system. This paper examines Hempel's analysis in order to motivate an argument—in the context of contemporary debates concerning the *Diagnostic and Statistical Manual of Mental Disorders* (DSM)—in favor of a theoretical and causal approach to psychiatric classification. Hempel argues that a good taxonomic system should: (1) reliably describe objects of classification with operational definitions, and (2) provide classificatory concepts that possess *explanatory import*. For this second function to be realized, Hempel argues that the classificatory concepts of taxonomic systems should be organized in terms of more general causal theories that unify the objects of that system. Significantly, Hempel maintains that classificatory concepts with explanatory import provide the methodological basis for explanation, prediction, and scientific understanding. My analysis in this paper emphasizes the importance of theoretically based taxonomic systems for providing projectable and testable classificatory concepts.

Hempel's philosophical analysis is highly relevant for recent debates in philosophy of psychiatry concerning whether the DSM should adopt a purely descriptive or causal approach to psychiatric classification. Since the publication of the third edition of the DSM in 1980 (DSM-III), the DSM has favored a purely descriptive and 'atheoretical' approach to classification that excludes information regarding the causes of mental disorders. From a neo-Hempelian perspective, some of the largest drawbacks of the DSM could be addressed by integrating causal information into the DSM's descriptive categories. In particular, a theoretical and causal approach to psychiatric classification could provide a more effective means for providing valid diagnostic categories ("natural kinds"), formulating testable classificatory concepts, and allowing the DSM to be a reflexive and self-correcting manual. This could alleviate criticisms that the DSM has failed to provide a system of classification that facilitates the treatment of mental disorders, and it would minimize the possibility of reifying artificial kinds (e.g., histrionic personality disorder, narcissistic personality disorder) that do not have a natural basis.

In the final part of my paper, I explore how the Research Domain Criteria (RDoC)—an explicitly theoretical and causal system of psychiatric classification—could be a useful resource for integrating theoretical information regarding the causes of mental disorders into the DSM's diagnostic categories. While the introduction of the RDoC initially placed the DSM and RDoC in an antagonistic relationship, the APA and NIMH have subsequently indicated that the DSM and RDoC are complementary, rather than competing, classification systems. I argue that one way that the RDoC could improve the DSM is by facilitating revisions of the DSM's diagnostic criteria, which are used to operationally define particular mental disorders. More specifically, the RDoC has the potential to identify causal regularities at multiple levels (e.g., at genetic or neurobiological levels), which could help clarify the kinds of psychiatric symptoms (e.g., psychosis, compulsions) that are associated with natural processes. With respect to the DSM, the RDoC could help to revise and refine the DSM's diagnostic criteria by requiring that the operational symptoms that are used to define mental disorders have a clear natural basis. In this way, the RDoC could facilitate the introduction of theoretical information regarding the causes of mental disorders into DSM categories, while retaining the DSM's favored descriptive approach to defining mental disorders.

FACTS AND MYTHS ABOUT RDOC
Uma Vaidyanathan, Dede Greenstein, and Bruce Cuthbert

What exactly is the Research Domain Criteria (RDoC) initiative? In this talk, we will discuss RDoC from two perspectives – one as a set of rules that enable a research framework, and the other as a practical instantiation of those rules (i.e., the RDoC Matrix). In doing so, we will

cover the strategy behind RDoC, the premises it rests upon, and how it attempts to tackle philosophical issues such as the mind-body problem and the definition of impairment when it comes to psychopathology. This discussion is also intended to clear up frequent false myths about RDoC. For example, one criticism that has been lobbied is the notion that RDoC is highly reductionistic and is a system that merely aims to explain mental phenomena through biological means. Others have construed RDoC as a classification system similar to the DSM and ICD. Finally, contrary to frequent misconceptions, NIMH continues to accept research grant applications designed around DSM/ICD disorders. The goal of this talk is thus to clarify what RDoC is and what it isn't, and to outline future steps that can be taken to advance our understanding of mental disorders.

SCIENTISM AND THE ENLIGHTENMENT SPLIT
Nikola Andonovski

In his seminal "Philosophy and the Scientific Image of Man," Wilfrid Sellars diagnosed the 'clash' that plagues contemporary thought: that between the 'manifest' and the 'scientific' image of human beings. According to the manifest, common-sense image, we are *persons* with beliefs, intentions and hopes; we act freely and are ultimately responsible for our actions. In sharp contrast, the emerging scientific image portrays us as complex physical *machines,* composed of simple elements arranged in remarkably intricate ways by the process of natural selection. Nowhere has this clash of images been more apparent than in psychiatry. Since Freud and Kraepelin, psychiatrists have vacillated between the two, often conceptualizing mental disorders as *both* physical illnesses and value-laden failures of persons. In his recent contribution to the topic, John Z. Sadler has convincingly argued that metaphysical 'flip-flopping' between these two worldviews generates many of the problems we see in the theoretical literature and psychiatric practice. These include the prevalence of hybrid and metaphysically suspect "vice-laden" disorders (e.g. Pedophilia is treated as both a disorder and a criminal behavior), the conceptual confusion concerning psychopathologies of morality, as well as the practical problems with the regulation of social deviance. Intriguingly, Sadler argues that *both* wordlviews -the manifest (Judeo-Christian) and the scientific (Enlightenment intellectualist) -are varieties of *folk metaphysics*: collections of unsystematic, naive and variably shared cultural assumptions. Notably, he characterizes 'scientism' -the thesis that "only science can provide us with knowledge about ourselves and the world around us" -as a manifestation of folk-metaphysical views such as naturalism, reductionism and realism. The emergence of scientism -Sadler argues -goes hand in hand with the emergence of instrumentalist thinking, a style of thinking dominated by the values of efficiency, production and outcome. The dominance of instrumentalist thinking in psychiatry has disastrous consequences: marginalization of the patients' narratives, standardization and context-insensitivity of treatments, as well as modification of personal identities in instrumentalist terms (e.g. patients who self-identify as 'schizophrenic').

In this paper, I argue that Sadler's interpretation of the 'clash', although remarkably poignant, is deeply problematic. I make four distinct claims. (1) 'Scientism', properly understood, is not a variety of folk metaphysics *by Sadler's own standards.* Specifically, it lacks some of the distinctive features of folk metaphysics: it is systematic, anti-pragmatic, truth-aspiring, it doesn't structure common-sense intuitions and is open to evidence-based revision. As such, scientism is conceptually independent from the theses of naturalism, reductionism and realism. (2) Indeed, scientism is best understood as a pure *epistemological* thesis motivated by a specific, and remarkably well-developed, view of knowledge and justification. According to this view, science is the *only* source of knowledge because it provides an institutionalized set of (necessary) epistemic filters set up in ways that maximize the avoidance of systematic biases and mistakes in the accumulation of evidence . Importantly, scientism is independent from instrumentalism; one can -and *should* -endorse the former but not the latter. (3) Contemporary science provides *a multi-scale* picture of human beings and their behavior in which 'proper' functioning involves constraints at different scales: genetic, molecular, neural, cognitive, behavioral and social. Understanding this complex multi-scale organization is a first step towards the abolition of psychiatry's troubling dependence on pre-Enlightenment metaphysics. Such understanding can help theoreticians and practitioners in alleviating some of the conceptual and practical problems that Sadler justifiably underlines. It can facilitate the elimination of metaphysically (and politically) suspect hybrid categories, as well as provide explanatory leverage in the treatment of the problematic psychopathologies of morality. (4) This conceptual progress need not be plagued by the disastrous consequences of adopting instrumentalism. (A) Scientism does not entail the abandonment of personal narratives in psychiatric practice. Quite the contrary, personal narratives are treated as *key* sources of evidence, which although not considered sacrosanct or infallible, provide invaluable data for the refinement of complex theories and models. (B) The development of multi-scale models and the rise of precision psychiatry illustrate how understanding the complex, multifaceted picture of human "being in the world" can lead to the development of *personalized* and context-sensitive treatments. (C) This picture, when supported by proper institutional mechanisms, can also lead to formation of comparably complex personal identities, not simple 'label-based' self-identifications.

OUTCOME MEASURES IN SCHIZOPHRENIA RESEARCH
Phoebe Friesen

Woodward's interventionist account of explanation holds that *X* causes *Y* if and only if an intervention that changes the value of *X*, changes *Y* or the probability distribution of *Y* in some regular, stable way[1].

If
I • *X* • *X'*
X' • *Y'*
Then
X causes *Y*

Many concerns have been raised with regards to the boundaries of the *Y*s of psychiatry, which have long been constituted by DSM diagnoses. The shift away from DSM criteria towards RDoC results from these concerns, breaking down *Y* (e.g. schizophrenia) into *Y1* (delusions), *Y2* (disordered thinking), *Y3* (flat affect), etc.

Here, I consider a distinct, but related issue concerning *Y'* in research into interventions for schizophrenia. *Y'* represents the effect that an intervention, *I*, has on a mental disorder, *Y*. Specifically, *Y'* represents how we measure efficacy. In a standard RCT, intervention, *I,* is applied to mental disorder, *Y*, and if *I* leads to *Y', I* is considered efficacious. In research involving schizophrenia, *Y'* usually is represented by a reduction in symptoms[2]. This project explores three reasons to reconsider the use of symptom reduction as an adequate *Y'*, as well as three new models representing treatment in schizophrenia that come into view if we replace *Y'* with an alternate measure, *Y".*

The first reason concerns the gap between efficacy and effectiveness. While several interventions for schizophrenia demonstrate efficacy, expected outcomes for individuals living with diagnoses of schizophrenia are very poor. The median rate of recovery has been close to 13% for decades. This suggests that demonstrating efficacy does not guarantee that an intervention will fare well in clinical practice. The second reason focuses on the most common intervention for schizophrenia, antipsychotics, and their negative effects which are not captured by *Y'*. While antipsychotics are certainly effective in treating (especially positive) symptoms of schizophrenia, debilitating side effects contribute to rates of non-compliance between 40-60%. In recent years, deficits in cortical grey matter and mortality have increasingly been linked to antipsychotics. This evidence indicates that our measure of what a good treatment for schizophrenia is may need to be revised. Finally, a third reason stems from research showing a disconnect between symptom reduction and measures such as psychosocial functioning and overall quality of life (QOL). This implies that the traditional *Y'* may simply be too narrow.

If we take seriously these worries, and consider expanding *Y'* to *Y",* which represents a broader outcome involving overall QOL, as emphasized in the recovery movement, three alternate models of treatment for schizophrenia come into light. The first model shows that multiple interventions are required to reach *Y",* some of which aren't visible when we focus exclusively on symptom reduction. Evidence for this can be found in research that demonstrates links between QOL and various factors like family psychoeducation, hope, and goal attainment. The second model sees symptom reduction as a necessary, but not sufficient, step on the path towards a more complete recovery. Evidence for this can be found in research that finds symptom reduction to be a component of QOL, and theories that suggest that affective or cognitive processes mediate between symptoms and QOL. A third model challenges the necessity of symptom reduction for QOL. Evidence for this model can be found in research that shows QOL is attainable along with regularly occurring symptoms, and within consumer/survivor/ex-patient movements, like the Hearing Voices Network, that champion living with symptoms in a healthy and productive way.

Finally, I consider objections one might raise in response to this expansion, and implications these alternate models have for Woodward's interventionist account of explanation in psychiatry.

WHAT IS A DIAGNOSTIC ERROR IN PSYCHIATRY?
Dany Lamothe and Mona Gupta

In the last two decades, growing concerns about patient safety have led to interest in the problem of medical error. In 1999, the Institute of Medicine released a major report on patient safety estimating that each year in the United States, 44,000 to 98,000 hospitalized patients die from medical errors, including diagnostic error. While morbidity and mortality arising from medication or surgical errors have received a great deal of attention from policymakers, researchers, and clinicians, diagnostic errors figure less prominently. For example, a major Canadian study of adverse events amongst recently discharged medical patients did not even include diagnostic error as an adverse event.

The relative lack of attention to diagnostic error may result from difficulties in defining the concept. Traditionally, the literature concerning diagnostic error has focused on the causes of diagnostic errors simply assuming that an error occurs whenever another diagnosis is made before the correct one. Evolving definitions distinguish between misdiagnosis (the traditional concept) versus diagnoses that are unintentionally delayed or missed entirely (no diagnosis was ever made). More recently, some authors have argued that an error only occurs when there is a missed opportunity to make the correct diagnosis. That is, a diagnosis can be wrong, but it should only be considered a true error if the clinician had all the information necessary to make the right diagnosis at the time of the initial diagnosis. Despite these nuances, both new and old definitions share a commitment to a disease entity assumption, that is, that diagnostic accuracy or error is ultimately assessed in reference to the correct identification of an underlying disease.

Defining diagnostic error poses even greater challenges in psychiatry as there are no objective measures such as physical exam findings or test results which establish the presence of an underlying disease. In the absence of such measures, psychiatrists cannot rely on the correct identification of an underlying disease to serve as the standard by which accuracy or error of psychiatric diagnoses can be assessed. As an alternative, some psychiatrists suggest that an error occurs when a patient receives a diagnosis but does not fulfill the operational criteria associated with that category or vice versa. In this paper, we will address the question of what constitutes a diagnostic error in psychiatry. First, we will review the main models of diagnostic error in medicine. We will then discuss the alternatives offered by authors in psychiatry. We will argue that neither the medical approach nor the psychiatric alternative provides an adequate conceptual basis for psychiatric diagnostic error. The absence of objective measures makes it impossible to assess diagnostic accuracy in reference to an underlying disease while simply fulfilling DSM or ICD criteria may not provide an appropriate characterization of the patient's presenting problem. We will conclude by arguing that diagnostic error may not be the best concept for evaluating the quality of psychiatric diagnostic assessments. Nevertheless, some standard is required in order to prevent the situation in which any diagnosis is acceptable. We propose that the quality of a psychiatric diagnosis should be evaluated on the basis of its ability to represent the patient's problem well from both clinician and patient perspectives.

WHAT DOES IT MEAN TO HAVE A MEANING PROBLEM? MEANING, CONTROL, AND THE MECHANISMS OF CHANGE IN PSYCHOTHERAPY
Garson Leder

Psychotherapy is effective. Meta-analyses, and meta-analyses of meta-analyses, have consistently shown a significant effect size for psychotherapeutic interventions when compared to no treatment or placebo treatments. This effectiveness is taken as a sign of the scientific legitimization of clinical psychotherapy. A significant problem, however, is that psychotherapies with distinct and incommensurate theoretical foundations appear to be equally effective. While individual studies directly comparing therapies, or comparing therapies to placebos, often show the superiority (if often only minor) of one particular therapy over another, meta-analyses of clinical studies consistently show a general lack of statistically significant differences between the outcomes of most forms of standardized psychological interventions. This poses a problem for specific psychotherapies: they may work, but likely not for the reasons that ground their theoretical explanations for their effectiveness.

A prominent explanation for the finding of common efficacy in psychotherapy is to attempt to identify underlying common factors that would explain the efficacy of seemingly disparate therapeutic techniques and theories. The 'common factors' explanation postulates that theoretically non-specific factors (such as an emotionally charged confiding relationship, a healing setting, and a coherent theory/rationale) explain psychological change (as opposed to the specific factors postulated by distinct theories). According to this view, theoretically and functionally distinct therapies such as cognitive behavioral therapy (CBT) and psychodynamic therapies are supposed to be efficacious only because they share particular therapeutic ingredients common to all efficacious therapies. The empirical or theoretical 'truth' of the particular delivery method is taken to be irrelevant; all that matters is thatthe therapy succeeds in delivering the common factors that lead to psychological healing.

The so called 'common factor' theories, while plausible, are also in need of a model of the relation between the common factors of therapeutic change; they need to explain why the common factors are supposed to enable psychological healing. According to influential common factors models proposed by psychologists Jerome Frank, psychotherapies are supposed to work by altering maladaptive meanings and providing patients with new, salubrious, and more adaptive meanings. These models share the assumptions that the alteration of meanings is the primary mechanism of change in psychotherapy and that the problem being addressed in psychological interventions is primarily a problem of maladaptive meanings (or assumptive worlds).

This essay will address three interrelated philosophical and theoretical questions concerning the 'meaning theory of psychological change'. First, what does meaning have to do with psychopathology? Is psychopathology a problem of meaning, or is it merely ameliorated in part by a meaning-based solution? And finally, what is supposed to be maladaptive about the meanings that are altered in psychotherapy (and what is adaptive about the meanings that replace them)? This essay argues that the meaning theory of psychological change is likely mistaken; psychological problems are not normally problems of meaning nor are they directly ameliorated by changes in meaning. Rather, psychotherapeutic change is best explained by the development of the patient's skill of cognitive control of maladaptive cognitive patterns. According to the cognitive control view outlined here, the common factors are effective because they help patients impose coherence on their psychological problems, creating a framework to develop the skill, or ability, of control of their cognitive processes.