

Early Career Scholars Conference in Philosophy of Psychiatry: Overcoming Mind-Brain Dualism in 21st Century Medicine

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Keynotes

Jennifer H. Radden

"Melancholy as Disease: Learning About Depression from Burton's *Anatomy*"

The Anatomy of Melancholy (1621) by Robert Burton is a famous and beloved book. But its psychology, normal and abnormal, has always been derided as a humoral muddle. To decide whether he has anything to offer us today, I want to explore Burton's use of the concept of disease in light of present day philosophy of science and medicine. Discussions within those disciplines employ four separate, or at least separable, sets of contrasts. Each is pertinent to whether Burton has anything to offer beyond a muddle, and all are applicable to present day conceptions of the depression that is widely accepted to be the descendant disorder from what was once called melancholy. They are: causes and symptoms (and signs); symptom-based (or descriptive) classifications and etiological ones; categorical and dimensional conceptions of disease and types of classification; reductive and non-reductive etiological accounts. Burton is not always right, I want to show, but his embodied mind, his non-Cartesian interactionism so hospitable to cognitivist analyses, the role he accords the imagination, and his emphasis on pragmatics, particularly self help, combine to provide us with new ways to approach the woeful array of crises confronting psychiatry, and particularly the study and treatment of depression, today.

John Z. Sadler

"Folk Metaphysics as Cultural Confound and Constraint in Mental Health Discourse"

Philosophers of mind, psychology, and psychiatry have used, fruitfully, the concept of 'folk psychology' to do all kinds of intellectual work in the analysis of free will, mind/brain dualism, and problems in moral psychology like compatibilism/incompatibilism. However, the ideas and concepts of 'folk metaphysics' are comparatively neglected in philosophy in these specialized fields. In this lecture, I first frame my own concept of folk metaphysics and how it has been used in my Vice & Psychiatric Diagnosis project, which analyzes the significance of vice (immoral or criminal conduct) in DSM diagnostic categories. Briefly, folk metaphysics are partially shared, philosophically unsystematic and naïve, cultural assumptions held by ordinary Westerners about the nature of reality, human nature, and the sources of morality. I shall then provide a brief historical analysis of the development of two prevailing strands of folk metaphysics in Western culture, based upon what I call the "Enlightenment Split". These strands, I will show, generated two culturally prevailing folk-metaphysical paths for us

Westerners. One, inherited from the Roman Catholic Church, framed the metaphysical assumptions of Western common and criminal law, based upon folk-metaphysical core beliefs in free will, individual (not collective) moral responsibility, and desert (one gets what one deserves). The other strand of folk-metaphysical beliefs was inherited from the Enlightenment European intellectuals who formulated modern science, with its emphasis on complex multifactor causation, wrongful conduct as disease, and the challenge of simple and complex causal determinisms of various kinds. I'll argue from examples that both strands of folk metaphysical beliefs are operative and obfuscating in contemporary mental health, psychiatric, criminal law, and philosophical discourse. Metaphysical flip-flopping between both strands generates many of the practical problems we see in the literature, psychiatric practice, and scientific debate, from compatibilism, to retributive justice, to the insanity defense.

Speakers:

Wesley Buckwalter, University of Waterloo

“The Folk Psychological Basis of Dualistic Psychiatric Approaches”

One approach to psychiatric practice, often referred to as the “biological” or “brain-based” approach, views the mind as a purely physical entity that is completely dependent on brain function. According to the brain-based approach, all mental disorders are biological pathologies of the brain or nervous system. A different approach, often referred to as the “mind-based” or “dualistic” approach, views the mind as a disembodied entity that is somehow distinct from the brain and other physical systems. According to the dualistic approach, mental disorders are not brain or neurological disorders and thus should be treated very differently than other biological pathologies.

Researchers across a diverse range of fields in philosophy, cognitive science, and medicine have rejected this form of dualism as a false theory of the mind. In recent years, psychiatrists have also come to reject dualistic theories as incorrect and potentially harmful accounts of mental disorders (Kendler 2005). But despite calls for biological and brain-based approaches from within the field (Andreasen 1997; Kandel 1998), dualistic approaches still implicitly inform various aspects of clinical psychiatric training, practice and treatment (Kirmayer 1988; Luhrmann 2000; Miresco & Kirmayer 2006). One goal of contemporary psychiatry is to better understand why these dualistic aspects of clinical approaches persist among mental health professionals, and develop strategies for overcoming them.

One recent attempt to track their persistence demonstrates that professional psychiatrists and psychologists reason dualistically about individual clinical scenarios (Miresco & Kirmayer 2006). These researchers have shown that professionals evaluate protagonists of clinical vignettes differently when their symptoms were judged to be psychological rather than biological in origin. Specifically, protagonists displaying symptoms seen as psychologically determined (e.g. personality disorders) were regarded as more blameworthy, in greater control of, and

responsible for their behaviors than when symptoms were seen as biologically determined (e.g. chemical dependence or mood disorders brought about by SSRIs).

The mind-brain dichotomies drawn by mental health professionals in Miresco & Kirmayer's study are also highly consistent with the results of several studies of lay-judgments of mental disorders made by ordinary adults (Weiner et al. 1988; Buckwalter & Turri, manuscript). In one recent study for instance, researchers found that participants were consistently more likely to say that agents "literally" do have the ability to fulfill their obligations, and were judged much more blameworthy for failing to fulfill obligations, when the reasons for failure were described psychologically (e.g. due to serious clinical depression) rather than physiologically (e.g. due to a serious car accident, Buckwalter & Turri, manuscript).

The fact that similar mind-brain dichotomies persist among both lay-people and highly trained health professionals has led some researchers to hypothesize that dualistic approaches to mental disorders "reflects a basic cognitive schema", one that is used to "intuitively to understand human behavior" (Miresco & Kirmayer 2006: 913). But what is this basic cognitive schema? How does it account for differential appraisals, for instance, of the "controllability" of physically and psychologically induced behaviors? And lastly, can understanding this schema help overcome dualistic responses in favor of more scientific treatments of behavioral disorders?

This paper attempts to answer these three questions. Expanding on prior work, I suggest that the tendency to evaluate mental disorders dualistically is deeply rooted in folk psychology, and particularly, the folk psychology of belief. Belief is a central plank of folk psychology, the capacity to predict and explain the mental states and behaviors of others. Detecting when others have beliefs is a central aspect of social cognition, cooperation and communication (e.g., Baldwin and Tomasello 1998; Baron-Cohen 1996). As a pervasive aspect of ordinary life, it is reasonable to suppose that the folk psychological system is also active when professionals and lay people interact with patients with behavioral disorders. In particular, two recently uncovered aspects of the folk psychological system may play an important role in these circumstances.

The first aspect involves the doxastic conception of mental disorders—and especially, delusional disorders. One debate in philosophy of psychiatry is whether the contents of clinical delusion, such as Capgras or Fregoli delusion, should be classified as beliefs. A popular view in philosophy and cognitive science is that such delusions aren't beliefs because they don't always guide behavior and affect in the way that beliefs do (Bayne 2010: 330). Contrary to these views however, recent empirical work demonstrates that folk psychology unambiguously views such delusions as stereotypical beliefs (Rose, Buckwalter & Turri forthcoming). Moreover, this research shows that the assertions patients make about their own conditions are recruited as powerful cues for belief-ascription, suggesting belief ascription may extend to many other forms of mental disorder.

The second aspect involves the question of doxastic voluntarism—the view that we have the same willful control over our beliefs that we have over our actions. Most contemporary philosophers reject voluntarism for involuntarism, the view that we cannot choose or refuse to believe at will (Williams 1973; Alston 1988). Contrary to these positions however, recent empirical work demonstrates that folk psychology clearly views belief as voluntary and perhaps as the most voluntary propositional attitude (Turri, Rose & Buckwalter, manuscript). These researchers have found that both an agent's professed decision to choose or refuse to believe, and the strength of an agent's willpower makes an enormous impact on whether people agree that the person holds a certain belief or can choose to believe voluntarily.

Putting these aspects together, the result is a basic folk psychological schema that (1) classifies the contents of various mental disorders as stereotypical beliefs, and (2) fully embraces voluntarism about those beliefs. I argue that these principles of folk psychology implicitly encourage mind-brain dichotomies in our interpretations and evaluations of mental disorders. On this view, the degree to which a patient is seen as holding a belief consistent with the content of a mental illness contributes to the false perception that the disorder is voluntary, within the control of the agent to choose, and therefore, blameworthy. I discuss the implications of this view and the ways that implicit dichotomies driven by these folk psychological mechanisms might be mitigated.

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“RDoC: Thinking Outside the DSM Box without Falling into a Reductionist Trap”

Just as the DSM-5 was about to be finalized, the National Institute for Mental Health (NIMH) launched its “Research Domain Criteria” (RDoC) initiative. As stated on the website of the NIMH, the RDoC project is an initiative that aims to “define basic dimensions of functioning (such as fear circuitry or working memory) to be studied across multiple units of analysis, from genes to neural circuits to behaviors, cutting across disorders as traditionally defined. The intent is to translate rapid progress in basic neurobiological and behavioral research to an improved integrative understanding of psychopathology and the development of new and/or optimally matched treatments for mental disorders” (NIMH, “Research Domain Criteria” Web. April 5th, 2014 <<http://www.nimh.nih.gov/research-priorities/rdoc/index.shtml>>). That project has been seen by many as a disavowal of the type of nosological enterprise incarnated by the DSM itself (from DSM-III to DSM-5)¹. The latter interpretation of the initiative has been fuelled by statements from individuals either involved in the RDoC initiative or working close to it. For instance, Thomas Insel on his blog on the NIMH website celebrated the future arrival of the DSM-5 by saying that “[p]atients with mental disorders deserve better” (April 2013). In the same spirit, Hyman (a former NIMH Director) has been writing that “it now appears that the accreting failures of the current diagnostic system cannot be addressed simply by revising individual criterion sets and certainly not by adding more disorders to DSM-5 [...] DSM-III was a brilliant advance; *it is now time to move on*” (2011, 3 and 14; our emphasis).

Indeed, RDoC calls for a paradigm shift in research that would align psychiatry with the rest of medicine. Current research in medicine has unearthed etiological mechanisms, discovered biomarkers, identified risk factors -- all of which, separately or in combination -- allows for the development of new treatments. While the rest of medicine is making incredible, progressive strides in the treatment of cancer or Alzheimer’s disease, research for the treatment of major mental disorders has stalled almost completely (Akil et al. 2010; Hyman & Fenton 2003). Insel & Sahakian (2012) describe the situation thusly: “the world is experiencing a crisis in drug development for mental illness; drug companies are withdrawing from the field or redirecting the investments” (269). It is believed that the discovery of new treatments necessitates a more sophisticated understanding of mental disorders. RDoC advocates² argue that this understanding

¹ This is at least how it has been interpreted in the popular press; see for instance, Belluck and Carey 2013; Campbell, 2013; Horgan, 2013; Koven, 2013.

² The NIMH lists a number of publications on its website which describes the RDoC project (<http://www.nimh.nih.gov/research-priorities/rdoc/nimh-rdoc-publications.shtml>). Among the authors of

will come only with the condition that we abandon the DSM's current constructs for new constructs developed in a bottom-up fashion to better mirror "reality". Since RDoC conceptualizes mental illness as brain disorders (Insel et al. 2010, 749), it looks for "reality" at the level of neural circuits. In this respect, RDoC's initiative is brain-centered and might appear reductionist in spirit.

In our presentation, we first want to describe the context in which RDoC appeared and demonstrate that, if it is not a disavowal of the DSM-5's work, it certainly signals the abandonment of a method of trying to establish a valid nosology; a paradigm shift in nosology so to speak (Section 1). We will then question if RDoC is a reductionist enterprise (Section 2). We will explain why RDoC is not reductionist in a strong and naïve sense, but why it could be understood as reductionist in a weaker sense (Section 3). Indeed, RDoC advocates seems to show some philosophical sophistication in labelling their reductionism "patchy" and the type of typical explanation in psychiatry as "interlevel". We'll explain how these two conceptions of the relations between different levels of explanation are to be understood in the context of psychiatry. We'll argue, following Kendler recent proposal (2012), that with this understanding of the relations between different levels of explanation, the choice of a preferred level is grounded in pragmatic considerations. Finally (section 4), while this weaker form of reductionism does not possess the problems the stronger forms of reductionism do, it might nonetheless generate problems of its own that researchers should be aware of. We will try to delineate some of these problems.

Anthony Fernandez, University of South Florida

"Psychiatry and the Poverty of Subjectivity: The Power and the Promise of Phenomenological Psychopathology"

Since the 1980s, psychiatric classification has been dominated by the American Psychiatric Association's *Diagnostic Manual of Mental Disorders* (DSM). However, the DSM-5, released in May of 2013, was the target of searing criticism from patient advocacy groups, psychotherapists, and even psychiatrists (including Robert Spitzer, chair of the DSM-III taskforce, and Allen Frances, chair of the DSM-IV taskforce). However, the criticism with the greatest visibility and most significant ramifications

these papers, we find Thomas Insel (actual director of the NIMH), Bruce Cuthbert (director of NIMH Division of Translation Research), Sarah E. Morris (chief of project of the NIMH Schizophrenia Spectrum Disorders Research Program), Charles A. Sanislow (who now works at Yale Psychiatric Institute, but used to be the program chief of NIMH Extramural Research in the Mood and Sleep Disorders Research), Daniel S. Pine (chief of the NIMH Section of Development and Affective Neuroscience). These researchers, as well a few other co-authors, are responsible for presenting, articulating and defending (if necessary) the RDoC project. It is to them that we are referring when we are talking about the "RDoC advocates". We are aware that other members of the project — for instance, participants to the workshops that were convened to define the constructs that figure in the RDoC framework -- might not share the views expressed by the leading advocates of the project.

came from the National Institute of Mental Health (NIMH). Just weeks prior to the publication of the DSM-5, Tom Insel, head of the NIMH, declared in a public announcement that NIMH funding will be largely reserved for studies that *do not use* the DSM-5 categories of mental disorders. Instead, most funding will be awarded for studies that support the new Research Domain Criteria (RDoC) project in its attempt to develop scientifically (i.e. neurologically and behaviorally) validated categories of disorder.

The major concern held by Insel and the NIMH is that psychiatric research has failed to correlate the diagnostic categories of the DSM with neurobiological mechanisms. In other words, the symptomatically delineated categories of the DSM, drawing primarily on references to patients' lived experience (e.g. cognitive distortions, emotional disturbances, delusions, or hallucinations) and observable behavior (e.g. insomnia/hypersomnia, anti-social activities, or hyperactivity), have not been adequately correlated with relevant changes in the brain. In order to remedy this issue, the RDoC project seeks to delineate preliminary research categories of disorder using only third-person observable data (specifically, neurobiological and behavioral data). As currently formulated, studies of the lived world of subjects with psychiatric disorders will play no role in the delineation of the preliminary research categories that will be drawn up by the RDoC project.

While I share Insel's concerns over the disutility of the DSM categories, especially in regard to their failure to map onto neurobiological mechanisms, I believe he was too quick to dispense with descriptive accounts of lived experience. I argue not only that references to lived experience are conducive to the delineation of preliminary research categories for neurobiological and psychopharmacological research, but also that phenomenological psychopathology (with its roots in the tradition of 20th century continental philosophy) is an invaluable tool for obtaining just such data.

Drawing on work by Gordon Parker, I show that preliminary categories differentiated by exclusive reference to changes in lived experience can (if developed with respect to the correct changes in lived experience) be correlated with neurobiological mechanisms as well as marked differences in the efficacy of targeted psychiatric interventions. However, I also argue that Parker's methods for delineating such categories are fairly superficial, and could be enhanced by the methodological tools available in phenomenological psychopathology.

A similar point has been argued by Parnas, Sass, and Zahavi. However, their proposals for a phenomenologically informed psychiatric classification have failed to adequately distinguish among the various levels of phenomenological investigation. As a result, they have been unable to differentiate between those phenomenological studies that are relevant for neurobiological research, and those that are not.

In order to remedy this issue in the phenomenological and psychiatric literature, I introduce three levels of phenomenological investigation. These levels are (1) structure, (2) mode, and (3) situatedness. (1) Phenomenological studies of structures seek to uncover the existential or ontological constitution of human existence. Structures are generally taken to be general categories of necessary characteristics of human existence. They include what phenomenologists term disposedness, understanding, care, and temporality, among others. (2) Phenomenological studies of modes, in contrast, typically take for granted the framework of existential structures as outlined in previous phenomenological investigations. Rather than seeking to uncover necessary characteristics of human existence, modal investigations seek to understand the variety of ways a particular

structure can manifest. For example, while it is a necessary characteristic of human existence that one is always disposed towards and attuned to the world, one is always disposed and attuned in some particular way. This way, or mode, is a mood, or affective disposition. We can find ourselves in the world through a variety of moods and affective dispositions, many of which phenomenology is capable of revealing through careful examinations of human existence. (3) Phenomenological studies of situatedness are typically understood as the least fundamental level of phenomenological investigation. These studies investigate the actual events that play a role in shaping human life (both social- historically, and personal-biographically) as well as the narrative that we construct in order to make sense of these events and incorporate them into our lives.

All three levels of investigation shed light on the shape and form of the lived world and human existence. However, when it comes to using these investigations for the sake of psychiatric classification and research, not all levels are equal. I argue that the level of situatedness, while perhaps the most important level for effective talk therapy, is unlikely to help us cluster subjects into categories that are relevant for neurobiological and psychopharmacological research. Investigations of changes in mode (e.g. profound changes in moods or modes of temporality) on the other hand, may be relevant for such research in some instances. Investigations of changes in existential structure, consisting in alterations of those aspects of human existence that are typically understood as necessary and constitutive, are likely to be relevant for this kind of research in nearly all instances.

By carefully delineating the levels of phenomenological investigation, research in phenomenological psychopathology puts us in a position from which we can begin legitimate research into correlations between subjective and objective phenomena. The attaining of such a position is a necessary first step towards answering both philosophical and scientific questions about the relationship between the mind and the brain.

Aaron Kostko, University of Minnesota Rochester

“Humanistic and Personalized Psychiatry Without Dualism”

Despite evidence suggesting that Cartesian dualism is still pervasive in psychiatric practice and research (Miresco and Kirmayer, 2006; Demertzi et al, 2009), the view has become a common and easy target of criticism within psychiatry. Physicalist-minded philosophers and psychiatrists (Andreasen, 1984; Martin, 2002; Yudofsky & Robert, 2002; Insel & Quirion, 2005; Kendler, 2005; Murphy, 2006; Reynolds et al, 2009; Andersch, 2012; Zeman, 2014), dualist-minded philosophers and psychiatrists (Switankowsky, 2000; McClaren, 2006), and those more narrowly focused on establishing a more humanistic and individualized psychiatry/medicine (Dubos, 1965; Cassell, 1976; Hastings, Fademan, & Gordon, 1980; Sullivan, 1986; Toombs, 1988; Mehta, 2011) all seem to agree that psychiatry should abandon any commitment to Cartesian dualism and that many of the problems facing contemporary psychiatry stem from failing to recognize this point sooner. Despite this widespread agreement, there is little agreement as to what should take its place. In this paper I consider recent arguments by Switankowsky (2000) and McClaren (2006, 2010) that purport to show the necessity of adopting

an interactive form of property dualism in order to establish a more humanistic and personalized psychiatry that can successfully treat the whole patient. After outlining each of these proposals in detail, I argue that they fail to provide an adequate account of the causal efficacy of irreducibly mental properties and that this failure undermines the alleged necessity of adopting an interactive form of property dualism to establish a more humanistic and personalized psychiatry. Moreover, even if Switankowsky and McClaren could provide an adequate account of the causal efficacy of irreducibly mental properties, I argue that a general physicalist framework can equally realize the goal of establishing a more humanistic and personalized psychiatry that treats the whole patient. I conclude by briefly considering how recent research in environmental epigenetics (Weaver et al, 2004) and pharmacogenomics (Mrazek, 2010) may help to realize this goal.

McClaren's (2006) proposal relies upon the application of Chalmer's (1996) naturalistic dualism to psychiatry. He argues that adopting such a view "restores the essence of humanity, our mentalism, to rightful primacy (1173)." Following Chalmers, McClaren divides mental phenomena into the experiential realm and the cognitive realm. The experiential realm is immediate, private, ineffable, irreducible, and carries no informational content. On McClaren's model, the experiential realm includes exteroceptive sensations such as sight, sound, smell, touch, pain, sexual sensations, etc., interoceptive sensations such as hunger, thirst, nausea, etc., emotions such as anxiety, anger, joy, humor, sadness, etc., and compound emotions such as despair, suspicion, guilt, etc. Cognitive functions, by contrast, are fast, unconscious, communicable, reducible, and have no experiential component. Examples of cognitive functions include deciding, judging, planning, calculating, hoping, recalling, being aware, intending, realizing, meaning, implying, deceiving, etc. The most important difference between the experiential and cognitive realms is that the latter are causally efficacious whereas the former are epiphenomenal. Although this would seem to be problematic since many of the mental phenomena that McClaren lists in the experiential realm seem to be constitutive of and causally relevant to the onset and maintenance of many psychiatric conditions, he contends that there is no need to worry. He argues that although "disordered conscious experiences...comprise the core of mental disorder as we define it," they are "secondary to disturbances in the cognitive realm" and that "we don't need a theory of conscious experience to be able to explain the causation of mental disorder (1172)."

McClaren's application of Chalmer's naturalistic dualism to psychiatry either fails to accommodate the symptomatology associated with many psychiatric conditions or is internally inconsistent. McClaren construes the experiential realm as epiphenomenal and "secondary to disturbances in the cognitive realm." However, it is not clear in what sense the experiential realm is secondary to disturbances in the cognitive realm. On one interpretation, being "secondary to" might mean that conscious experience occurs later in a causal chain that leads to behavior. If this is the case, then conscious experience would be causally efficacious and McClaren's view would no longer constitute a form of dualism. On the other hand, being "secondary to" could entail that conscious experience is simply a causally inert by-product of cognitive functioning. While this is a possibility, many of the mental phenomena that McClaren lists as belonging to the experiential realm, e.g., sexual sensations, anxiety, sadness, and despair, are typically considered to be both constitutive of and causally relevant to the onset and maintenance of many psychiatric conditions. To deny this would be to seemingly ignore the constitutive and causative roles that these phenomena play in psychiatric conditions. Thus, it is

not clear that McClaren can maintain that one does not need a theory of conscious experience to be able to explain the causation of mental disorder.

Switankowsky's proposal relies on the application of Nagel's (1986) dual-aspect theory. She argues that adopting such a view is necessary to successfully treat the whole patient. Although her argument is intended to apply to medicine more broadly, it generalizes to psychiatry as well. Following Nagel, Switankowsky conceives of the individual as a duality of physical properties and emergent, irreducibly psychological properties. However, unlike McClaren, her proposal construes all psychological properties as causally efficacious. She appeals to evolutionary theory as support for this claim, arguing that irreducibly psychological properties must be causally efficacious if one accepts that organisms with conscious experience were better able to survive than those without it. Switankowsky then outlines several practical benefits that follow from adhering to this form of interactive property dualism. First, she contends that such a view enables a physician to treat the whole patient, particularly the self or the "lived body" of the patient, rather than just the body of the patient. Second, she argues that an interactive property dualist framework enables the physician to treat the "lived experience" of an illness, by which she means the psychological disturbances that affect the patient's whole life. Third, she claims that such a view is necessary for a physician to tend to the subjective features of an illness, e.g., the inner, qualitative experiences associated with the illness and the personal meaning that the patient attaches to these experiences. Finally, she contends that an interactive property dualist framework better enables a physician to take seriously the patient's clinical narrative.

Switankowsky's application of Nagel's dual-aspect theory to psychiatry suffers from two major shortcomings. First, her appeal to evolutionary theory as a defense of the causal efficacy of irreducibly psychological properties fails to establish its intended conclusion. Asserting that organisms with conscious experience were better able to survive than those without it does not explain how an irreducible, non-physical property can generate behavior(s) that would confer an adaptive advantage on the organism. At best, Switankowsky's argument establishes that conscious experiences could be causally inert by-products of cognitive functions that do confer and adaptive advantage on the organism. However, this would lead to the same concerns raised for McClaren's view. One would have to deny the causal relevance of mental phenomena that are typically considered to be causally relevant to the onset and maintenance of many psychiatric conditions. Moreover, this conclusion would seem to undermine the practical benefits that Switankowsky claims to follow from adopting an interactive property dualist view, at least to the extent that these benefits depend on the causal efficacy of psychological properties. Second, even if Switankowsky's appeal to evolutionary theory could establish the causal relevance of psychological properties, it is not clear why it would be necessary to adopt an interactive property dualist view to realize the practical benefits that she mentions. One need not conceive of psychological properties as irreducible in order to pay attention to a patient's clinical narrative, to listen to a patient's subjective experience associated with an illness, to tend to the personal meaning that he/she attaches to these experiences, or to recognize that an illness impacts a patient's whole life. A physicalist-minded psychiatrist can just as easily tend to these features as a psychiatrist who adopts an interactive form of property dualism. He/she simply contends that the subjective features of the illness on which the patient reports are reports of disturbances in the patient's body that may impact multiple aspects of the patient's life. Thus,

one need not adopt an interactive form of property dualism to establish a more humanistic and personalized psychiatry that treats the whole patient; one only need be a good psychiatrist.

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Mael Lemoine, University of Tours

“Neurobiological redefinition of a psychiatric symptom: elimination, reduction, or what?”

‘Reduction’ has become a very widespread term in philosophy of neuroscience to account for the kind of relation that holds between the neurobiological and the mental or the behavioral. Yet a search in *Molecular Psychiatry*, *Nature Neuroscience*, or in standard textbooks like Eric Kandel's (2011), shows how rarely the term is used in this sense by scientists themselves. Recently philosophers of neuroscience (and psychiatry), like Schaffner (1998, 2000), Craver (2007) and Murphy (2006), have been using the term with more caution than was the case, say, in the 1980s (P. S. Churchland 1989).

Here I would like to focus on the ‘reduction’ of psychiatric symptoms to neurobiological processes by considering the case of anhedonia. Carnap proposed a useful distinction between ‘reduction’ and ‘derivation’, the former concerning the translation of a term into another vocabulary, and the latter corresponding to the classic view of logical deduction of nomological

statements (Oppenheim and Putnam 1958; Nagel 1961; Hempel 1965). The reduction of a symptom is of the first kind: it is not concerned with explaining regularities (empirical laws), which would be the case if reduction were about a syndrome or about the course of a mental disorder. The goal is to identify a neurobiological process that bears a specific relation to one symptom. The usual question is whether it is 'reducible', 'irreducible' or 'eliminable' (P. M. Churchland 1981).

My contention is that this kind of 'reduction' should be thought of as a 'redefinition' of the term referring to a set of phenomena of the same kind – in the case at hand, a redefinition of 'anhedonia'. It is similar to a linguistic translation in that, ideally, the extension of the term should mostly remain the same (except maybe for marginal cases). In this case, however, the intension of the term will change dramatically in the process, using neurobiological descriptors instead of clinical ones. In short, a redefinition seems to be a shift towards different, neurobiological predicates, to describe the same set of phenomena previously referred to and described thanks to clinical predicates. In the case of anhedonia, "the decreased ability to experience pleasure from positive stimuli or a degradation in the recollection of pleasure previously experienced" (dsm 5, p. 88) becomes something like a chronically under-activated dopaminergic mesolimbic circuit of reward (Berridge 2006). Reduction can then be defined as a redefinition where the new description successfully *applies to the same set of patients*. If it cannot successfully be so applied, then it is a case of either *irreducibility* (if the new, neurobiological description does not discard the old, clinical one) or *elimination* (if it makes the old one useless or inappropriate).

What I would like to show is that reduction, irreducibility and elimination are only ideal cases that are never actually encountered in neurobiological psychiatry. Most of the time, the problem is that sets of phenomena referred to by neurobiological and clinical descriptions only *partially* overlap: they do not identify enough for a claim of reduction to be made, nor do they differ enough for a claim of irreducibility or elimination to be made. For instance, in a review of the role of the reward system in depression, Nestler and Carlezon never clearly say what proportion of anhedonic patients have an impaired reward circuit: they merely show that most depressed patients are anhedonic and most impairments in this circuit in animal models lead to depression-like behaviors (Nestler and Carlezon 2006). This is far from identifying or distinguishing biologically-defined anhedonia and clinically-defined anhedonia.

Thus, the relationship between the old description of the phenomena, the set of patients, and the new description of the phenomena should be considered differently. I propose that it should be thought of as *triangulation*. What researchers are trying to do is localize something they believe is real, and tentatively call 'anhedonia'. They are provided with three definitions of it: 1) an ostensive definition - a set of patients suffering of the 'same condition' -, 2) a descriptive, clinical definition, and 3) a new explanatory, model-based, neurobiological description. The 'reduction or elimination' problem holds that all three definitions claim to *define* the thing as it itself is, and not only to determine how to detect it. In Peter Achinstein's terms, they would be composed of terms referring to "semantically relevant properties," like 'atomic number' is for gold, rather than "non-semantically relevant terms", like 'shiny yellowish' is for gold (Achinstein 1968).

Yet the important, though often forgotten, fact is that these descriptions are not definitions, but simple detection tools. Each tries to detect a different type of signal coming from the same direction. Thanks to all three, we try to localize more precisely the area of investigation. It is often the case that in so doing we have to tweak one, two or all three of our

definitions in order to improve the quality of the image. This is actually what is currently happening in research on anhedonia: a triangulation process comparing the results of epidemiological, clinical and neurobiological studies. I will show how new concepts such as Klein's clinical distinction between liking and wanting and between anticipatory and consummatory pleasure (Klein 1987), or Berridge's model of a dopaminergic circuit of 'incentive salience' (Berridge 2007), have been attempts at manipulating these three sorts of definition until a sharper image is provided. It has to be done carefully, with minimal changes, so that one perspective does not completely exclude one of the others, which would result in a completely new object. This conservative method could explain resistance to reduction: it is caused by a well-justified reluctance to abandon a way of describing the phenomenon at hand that worked so far, albeit imperfectly. And the impatience of a scientist of one kind, say, a neurobiologist, to make his points and renounce the contributions of others might explain eliminativism. It is sometimes our best move when we face inconsistent or shallow constructs such as 'phtisis', a term that used to refer to many different lung conditions.

Rather than a quest for reduction, this process of triangulation is probably better described as the search for robustness of a construct (Wimsatt 2007). It is an essential aspect of the 'naturalization' of psychiatry through neuroscience.

Lauren Ross, University of Pittsburgh

“Psychiatric Genetics: Advances in Explaining and Understanding Psychiatric Disorders”

Attempts to clarify the etiology of many clinically accepted psychiatric disorders seem to continually meet with unexpected challenges. Perhaps the most recent example of such a challenge has been the inability of genome-wide association studies (GWAS) to identify the specific genomic sequences that were thought to be associated with, and perhaps causally relevant to, particular psychiatric disorders. While the expectation of such findings may have been unrealistic, the failure of such studies to deliver these results had been viewed by some as supporting the claim that genetic and molecular features of neural systems are not relevant to understanding and explaining psychiatric disorders. Such claims resonate with a form of mind-brain dualism that has significantly influenced the development of modern clinical psychiatry, viz. the view that processes of the mind have very little to do with the processes of the brain (Kandel 1998; Kendler 2005). The acceptance of these dualistic claims in psychiatry and the field's general “drift away from biology” has been attributed to slow advances in the brain sciences and challenges in using them to better understand psychiatric disorders (Kandel 1998, 458).

In this presentation I indicate how current advances in psychiatric genetics contrast with these strong dualist claims. I argue that recent progress in identifying causal genes and their relationships to specific psychiatric disorders has successfully addressed significant challenges to better understanding and explaining them. The main challenge I consider is the unexpected and increasing evidence indicating that many psychiatric disorders are caused by different “rare” mutations in different individuals, e.g. disorders like schizophrenia and autism spectrum disorder (Sullivan, Daly, and O'Donovan 2012). These findings contradict the common disease-common variant (CDCV) hypothesis, which suggested that these common disorders were likely caused by common genetic mutations (Visscher, Brown, McCarthy, and Yang 2012). Furthermore, these

findings indicate a more complex etiology for such disorders and complicate our attempts to understand and explain them. How do we explain the development of schizophrenia or autism spectrum disorder in humans, or at the type-level, if the same disorder has different causes in different individuals? Furthermore, how can different genetic causes produce the same psychiatric disorder?

Recently advancements in gene expression profiling and pathway analysis have begun to answer these questions and provide causal explanations for these complex disorders. I describe these techniques and how they have revealed information about the many interrelated causal factors that lead to specific psychiatric disorders. I then examine a novel type of explanation that has emerged from their results: explanations that invoke the concept of a causal pathway that leads to a particular psychiatric disorder. In psychiatric genetics the concept of a causal pathway is often discussed in regard to pathways from genes to behavior. Pathways represent causal connections between upstream genes and the downstream behaviors they produce (Plomin, DeFries, Knopick, and Neiderhiser 2012). Such a concept provides a straightforward way of beginning to explain how different gene mutations can produce the same effect of interest: different upstream genes can have distinct pathways that all lead to (or converge on) the same single disorder of interest. In situations where many genes can cause the same phenotype an important distinction is often made among these any pathways from gene to behavior. This distinction is that the many pathways may be causally distinct or causally related to one another. Such a distinction is mentioned by Kendler, who states that “[a]t one extreme, there may be dozens of biologically distinct pathways to illness with little or no sharing between them. At the other extreme—etiologic homogeneity—just one pathway to illness awaits discovery” (Kendler 2013, 1060). When many distinct genes produce the same phenotype via pathways that are causally related, these causal relations can sometimes be integrated into the representation of a single shared or common pathway. These common pathways represent a type of “etiologic homogeneity,” because they indicate how different causal inputs can alter a single shared pathway to produce the same outcome. In these cases the many individual causal pathways from gene to phenotype can be collapsed into a single common pathway, which abstracts from differences among the many pathways to represent a single causal route that all the causal genes share. Thus, referencing the shared pathway can allow for an explanation of complex psychiatric disorders that have different genetic causes in different individuals. This is because all of the distinct causes alter the same common pathway that leads to the single psychiatric disorder of interest.

Explanations that invoke causal pathways have become increasingly more common in psychiatric genetics and take a variety of forms. I analyze examples of these explanations to further clarify their structure and content. I discuss two related explanations that such explanations are intended to address, viz. explaining (1) the cause of the disorder at the type-level and (2) why the disorder is genetically heterogeneous (i.e., why it has many distinct genetic causes). I indicate how explanations that invoke causal pathways are well equipped to answer these questions and I examine how current philosophical accounts of explanation fare in accommodating and elucidating their explanatory structure. I argue that Woodward’s (2003) interventionist account is well-equipped to elucidate the structure of such explanations and the causal information they contain. Finally, I highlight certain challenges that an interventionist interpretation of these explanations is likely to encounter and how such challenges might be addressed.

Explanations that invoke causal pathways appear to represent a significant advance in our understanding of many psychiatric disorders. They have led many researchers to view schizophrenia and other psychiatric disorders as “pathway diseases,” which are best understood as involving perturbations of causal pathways that lead to these conditions (Sullivan 2012). Viewing these disorders as caused by the alteration of a common causal pathway has been considered a “novel explanation for the observed genetic heterogeneity” that is increasingly identified in these disorders (Luo, Huang, Jia, Li, Su, Zhao, and Gan 2014). Better understanding these novel explanations and related techniques reveals how neurobiological information contributes to our understanding and explanation of psychiatric disorders.

Katrina Winzeler, University of California-Berkeley

“The ineliminability of psychotherapy as a treatment for depression: path-dependency and resilience”

In the treatment of clinical depression, two empirical claims are supported by multiple studies: 1) Antidepressant medication (ADM) and psychotherapy are equally effective in treating depression, and 2) of those treated with either ADM or psychotherapy, those treated with psychotherapy tend to stay well for longer and to suffer fewer relapses after treatment is terminated. In conjunction, these claims appear to support a conclusion that not only is psychotherapy an effective treatment for depression, but also that it provides benefits that ADM does not. Any effort to reduce psychiatry to a physical science needs to be able to explain such results, and the view that I offer in this paper can do that. As such, my view supports the ineliminability of psychotherapy from a completed science for the treatment of depression. By using the term “completed science,” I indicate that this ineliminability is not merely pragmatic; that is, it is not just that psychotherapy does certain things that we cannot yet accomplish through medical methods. Rather, my point is the stronger claim that it is not possible for psychotherapy ever to be eliminated by solely physical methods as a treatment for clinical depression, because no merely physical method could ever duplicate the results of psychotherapy.

I make this argument within a physicalist framework. It is easy to defend the ineliminability of an intervention typically couched in mental terms – psychotherapy – via an assumption of substance dualism about mind and body; fundamentally different substances will always require different types of interventions. But my argument is that dualism is not necessary for defending the ineliminability of psychotherapy. My view explains how both primary types of treatments for depression – ADM and psychotherapy – are efficacious, in part via an appeal to neurobiology. It also explains why some outcomes of psychotherapy cannot be replicated by ADM.

In this paper, I introduce an interpretation of the empirical facts, on which two general categories of both symptomology and interventions can be mapped onto distinct brain areas, brain areas that I call “lower” and “higher.” Respectively, these are the limbic system and the prefrontal cortex, and I argue that this distinction applies to interventions as well. In other words, ADM acts primarily on the lower brain areas and lower symptoms, while psychotherapy acts

primarily on the higher brain areas and higher symptoms. The efficacy of all interventions, therefore, can be accounted for by neurobiological explanations.

Of course, these categories of “higher” and “lower” are not meant to be wholly discrete, and there is high explanatory power in considering the interactions between the two levels. A third empirical claim that bears on my work is: 3) Patients who undergo effective monotherapy (either ADM or psychotherapy) typically will show many of the same long-term brain changes as one another, though there are systematic differences both with regard to a) final results and b) timeline of changes. I argue that interactions between the lower and higher levels, in the form of both bottom-up and top-down causal processes, can account for this evidence.

This emphasis on the brain might make it seem mysterious why I am looking to defend psychotherapy from elimination. In fact, my opponent is not just the dualist, but also the physicalist who supports the collapse of psychiatry into another branch of physical medicine. This opponent might pose the question: if psychotherapy changes the brain via the indirect route of discussing one’s problematic emotions and cognitions, and bottom-up ADM would eventually make the same brain changes, then wouldn’t it make more sense to find a way to bypass this psychotherapeutic medium, thereby changing the brain directly? I argue that neurobiological interventions alone could never replace what psychotherapy does, even at the level of the brain. My reasons for this claim ultimately have to do with the manner in which psychotherapy is performed. I claim that psychotherapy exhibits what I call interventional autonomy. Two features comprise this interventional autonomy: 1) the first feature has to do with the holism of the substrates of psychotherapeutic interventions. However, this supposed feature of interventional autonomy is vulnerable to the charge that it is important only in the context of present scientific limitations. It looks like we can imagine “brain tweaking surgeries” that are capable of replicating the neurobiological results of psychotherapy. Some mental health professionals might even claim that treatments like rTMS already do this, and that I have been remiss in not comparing psychotherapy to these higher-level techniques, instead of just comparing it to lower-level techniques.

I argue against such an objection based on 2) the second feature of interventional autonomy, which I call path-dependency. Such path-dependency entails that psychotherapy is necessary to recovery, in the non-pragmatic sense that no other intervention – not even a physical intervention at the “higher level” - could lead to all of its same results. I claim that the future neural interventions suggested by my objector would fail to reproduce the accompanying changes in neural states that are dependent on the process of psychotherapy. Furthermore, I maintain that these accompanying changes – for example, memories of self-efficacy - are not just idle side-effects of therapy, but rather, that they are precisely those that are responsible for psychotherapy’s increased resilience against relapse when compared with ADM.

The obvious response from my opponent is to push the original objection so that the brain surgery produces these accompanying neural changes as well. Thus, we imagine the patient to have implanted memories of positive experiences and self-efficacy, which underlie a state of mental health and resilience. What would be wrong with this result? I claim that the veridicality of these memories matters, not just because deception is not a good thing, but rather, because the patient’s non-veridical memories will fail to line up with the memories of important people in his life. These clashes will lead to hardships in navigating the social world. Thus, my paper ends

with a discussion of why this objection still fails to undermine the path-dependency that makes psychotherapy ineliminable as a treatment for clinical depression.

Serife Tekin, Daemen College

“Operationalizing the Self in Scientific Psychiatry: Perils and Promises”

A core feature of the last three editions of the Diagnostic and Statistical Manual of Mental Disorders (i.e., DSM-III, DSM-IV, and the DSM-5; henceforth DSM-III+) is the operational approach, according to which mental disorders are individuated through an operational criteria that consists of a sufficient number of symptoms (experienced by the patient) and signs (observed by the observer). What is also called the descriptive or atheoretical approach had developed as a reaction to earlier etiological approaches grounded in psychoanalytic theory, which had relied on empirically undefended theoretical assumptions, and involved a narrative prototype-description and a process of matching the individual patient to such prototypes. Operationalism was considered as a necessary step for making psychiatry more scientific, by way of identifying and investigating its readily measurable targets, i.e., the clusters of symptoms and signs that would serve as outwardly observable correlates of disease and bases for genetic and neural research into illness etiology. Operational criteria were also deemed useful in the clinical contexts where the DSMs are used by psychiatrists, medical doctors of different specializations, nurses, counsellors, etc., easing the process of diagnosis and treatment. DSM-III+'s operationalism has been a target of significant criticism for its efficacy in meeting psychiatry's scientific and clinical goals (Sadler 2005; Fulford, Thornton, Graham 2006; Tekin 2014; Pearce 2014; Parnas and Bovet in press; Schaffner and Tabb in press).

In this paper, I use historical and philosophical methods to appraise one particular criticism of operationalism, i.e., the neglect of the complexity of the self in the operational descriptions of mental disorders such as depression and schizophrenia (Sadler 2005; Dean 2012, Parnas and Bovet in press; Schaffner and Tabb in press; Parnas and Sass 2003; Tekin in press). It has been argued that the DSM categories' abstraction (or bracketing) of the self-related aspects of the encounter with mental disorders, its silence on how the illness experience is integrated into the patient's life as a whole, and on how symptoms are experienced phenomenally, make the categories simply a “repertoire of behaviour” (Cohen 2010) and the manual merely a “drug cartography” (Radden 2009). I trace the emergence of the problem of the missing self in psychiatric taxonomy to the historical evolution of operationalism in the DSM categories. In this historical review, I discuss several –not mutually exclusive– reasons, including the increasing resistance against psychodynamic psychiatry, Carl Hempel's influence on the image of science in psychiatry, pragmatic goals of finding reliable universal criteria for diagnosis for epidemiological measurements, etc. In this review, I also engage with some of my own findings in the Hempel archives at the University of Pittsburgh.

What I dub the problem of the missing self in psychiatric taxonomy is manifest in two ways, both the result of this history. First, although crucial to the understanding of its nature, the self-related aspects of the encounter with mental illness are not part of the DSM descriptions.

The excluded self-related aspects include the individual's particular life history; interpersonal relationships; biological and environmental risk factors; gender, race, class, and status; the developmental trajectory of mental disorder in the individual from childhood to adulthood; and the meaning the individual ascribes to these elements of life in her socio-cultural context. This feature of the DSM is called "hyponarrativity," or the abstraction of the illness category from the particular experiences and contingencies of the individual patient (Sadler 2005). Second, the first-person-specific dimension of the encounter with mental illness are not part of the DSM descriptions, such as the content of what the individual hears when she hears voices; or the distortions in the sense of self, prior to the full development of psychotic episodes, even though these very experiences are integral to understanding the onset of illness. DSM's deficiencies in engaging with the first person aspects of illness encounter as an obstacle for the development of effective treatment methods, phenomenologically oriented philosophers and psychiatrists have even developed a scale to measure disruptions in the sense of self to help in clinical assessment and treatment of the patients (Parnas and Sass 2003).

The often-cited reason for the problem of the missing self in psychiatric taxonomy is the conviction that the use of the concept of the self will hinder psychiatry's commitment to be scientific because it is not readily observable and measurable. A scientific classificatory system has to rely on observables, and thus it has no room for not readily observable or measurable targets. Clusters of symptoms and signs, on the other hand, are readily observable and measurable, and facilitate objective scientific research and clinical diagnosis, because these "consensus-based lists" afford clinicians a sense of certainty in an area of medicine where no physiological tests are plausible, increasing the reliability of diagnostic categories. I conclude by arguing that the worry that the concept of the self will hinder objectivity in psychiatric research is ungrounded and that employing a concept of the self in mental disorder descriptions/categories is not an obstacle for objectivity and reliability that psychiatry strives towards. In this discussion, I also review what I consider as unsuccessful attempts to operationalize the concept of self in scientific psychiatry, by referring to some of the recent work done in neuroscience on brain's default network (Qin and Northof 2010). In addressing the shortcomings of such operationalization, I lay the groundwork for successful uses of the concept of the self in psychiatry.

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Kathryn Tabb, University of Pittsburgh

“Philosophy of Psychiatry after Diagnostic Kinds”

A significant portion of the scholarship in analytic philosophy of psychiatry has been devoted to the problem of whether or not psychiatric disorders are natural kinds, and if they are not, what kind of thing they are (Zachar 2000; Zachar 2014; Kendler et al. 2010; Haslam 2003; Cooper 2005; Kincaid and Sullivan 2014). My contention is that this problem is fast growing less relevant to the concerns of practitioners and service-users of psychiatric medicine. Dissatisfaction with what I call the “diagnostic kind model” of psychiatric objects is currently appearing on a variety of fronts. Among clinicians of diverse orientations, it manifests as a dislike of, and in some cases open rebellion against, the hegemonic authority of the American Psychiatric Association (APA)'s *Diagnostic and Statistical Manual of Mental Disorders (DSM)*. Among clinically-oriented researchers, it presents as a frustration with the demarcation of nosological boundaries that has so long occupied psychiatry as a science (Poland and Eckardt 2013). Among scientifically oriented researchers, it is most obvious in the introduction by the National Institute of Mental Health (NIMH) of an alternative tool for classifying psychiatric research, the Research Domain Criteria project. The basic charge of the NIMH is that the *DSM* does not allow for the construction of patient samples that are homogenous in the relevant way to facilitate scientific progress (Tabb, forthcoming). The NIMH has determined that psychiatry does not in fact need a taxonomy to progress, though they acknowledge that the *DSM* (or some other manual) may be requisite for many pragmatic aims such as treatment, epidemiology, and communication with patients.

I argue that by focusing on the problem of kindhood, philosophers have neglected the development of a conceptual account that could explain the sort of scientific progress that the NIMH is envisioning, which could also accommodate the sorts of challenges raised by practitioners about the insufficiency of the *DSM* to describe their patients or promote their best care. I take as my examples the employment of Richard Boyd's homeostatic property cluster (HPC) kind account by various philosophers (Kendler et al. 2010), and the “exemplar” account promoted by Dominic Murphy. I argue that Murphy's account is more admirable insofar as it addresses, head on, the idea of an underlying mechanism and thus can accommodate the sort of etiological projects pursued by psychiatric researchers. I demonstrate, however, that Murphy's account still relies on what I call the “received authority” of diagnostic kinds, the very assumption that the NIMH is criticizing. Murphy's notion of the psychiatric “exemplar,” while it avoids the metaphysical pitfalls of earlier talk of natural and HPC kinds, still obfuscates, rather

than highlighting, the problem with this sort of thinking. And insofar as Murphy's account still relies on a categorical model, it will become obsolete if the NIMH's vision for psychiatric research into domains of functioning rather than diagnostic categories is realized.

To show how philosophers of psychiatry might better describe psychiatric research, I take the case of auditory-verbal hallucinations (AVH). The relation of this symptom to various diagnostic categories, and how it should best be conceptualized (on the level of the person, the neuron, etc.), are, I argue, appropriate and exciting topics for the philosopher. I sketch the contours of a philosophical treatment of contemporary debates over the relationship between AVH and thought insertion, and rather than taking it for granted that the aim of these debates is to improve our conception of schizophrenia and related diagnostic kinds, I show how the symptom challenges the utility of these categories. I argue for a pluralistic treatment of the taxonomical issues raised by presence of AVH in some patients and not others, suggesting that patient populations could be divided in a variety of ways on the basis of current research into the symptom, each of which may be useful for different projects. Once diagnostic kinds are not the target of research, it may well be that our ability to recognize clinically-relevant subtypes of patients will be enriched, though these subtypes may cross-cut the population of service-users, rather than neatly demarcating them into categories.

In other words, while there may be no good answer to the question of what kind of thing diagnostic kinds are, I think there are a lot of good answers to the question of what kind of things psychiatry can study and intervene upon. In fact, I will conclude my talk by suggesting that this latter question is an importantly *ethical* one. Once it is no longer taken for granted that the proper objects of psychiatric research are diagnostic kinds, it must be asked where psychiatry should be devoting its resources. Here again I advocate a form of pluralism. While the circuit-centric approach of the NIMH is already paying dividends for psychopharmacological intervention (and, I suggest, there are reasons to be optimistic about this despite past failures), it neglects other objects of research to which psychiatry, as a practice as well as a science, has obligations. It may be that thinking in terms of diagnostic kinds is required to think about some of the ethical aspects of psychiatric practice, such as the looping effects of diagnostic acts. But it may be that other experiential aspects of psychiatric patienthood cannot be accommodated by analysis at the level of the circuit, the gene, or the diagnosis, but demand an engagement with currently neglected research targets, such as the afflicted self (Tekin, forthcoming).

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Poster Presenters:

Phoebe Friesen, CUNY Graduate Center, "Let A Thousand Ontologies Bloom"

"Let A Thousand Ontologies Bloom"

To heal and to explain are two goals that stand at the forefront of psychiatric research. Both complementary and competitive, these goals have led to a dualistic picture of psychiatric phenomena in scientific practice. As investigations take shape beginning with a focal point of either the experience of the patient or the investigation of a pathology, two ontological frameworks have developed that roughly originated from the dual goals of healing and explaining. We might call these two frameworks the Psychological Framework and the Biological Framework; they can be seen in the contrasts between the mind and the brain, between psychotherapeutic and pharmacological treatments, and between social constructionist explanations and explanations that align with the medical model. This paper seeks to explore the way in which these two ontological frameworks pose a barrier to the goals of psychiatry and consider solutions that have been offered in response to this dualism. I will demonstrate how two proposals that have been offered fail to overcome the primary worry that arises from psychiatric dualism. I propose that these failures are a result of their focus on integration, which is problematic because of the complexity of the phenomena under investigation in psychiatric research. I offer reasons to reject an integrative approach in favour of a pluralistic one, and illustrate how this alternative ontological picture might be better suited to the goals of psychiatry.

In section one, I explore the myriad number of levels or perspectives that can be considered when one is researching a particular disorder, ranging from biochemical to genetic to psychological to anthropological, with many more in between. I argue that while theories developed at different levels of research are often compatible, the real worry within psychiatric research is based on conceptions of ontological priority. I define ontological priority as an attribution of a certain kind of causal reality to one or more levels, at the expense of others. Drawing on the insights of N.R. Hanson as well as those of Helen Longino and Ruth Doell, I seek to demonstrate the way in which these attributions of ontological priority constrain what hypotheses will be tested, what is seen as relevant data, how that data is interpreted, and eventually what explanations and treatments will be developed (Hanson, 1958; Longino and

Doell, 1987). I further illustrate this point by way of examples paradigmatic to both the Psychological Framework and the Biological Framework (Warner and de Girolamo, 1995; Wilson, 2004). This analysis suggests that the contemporary dualism in psychiatric research may be leading us to miss out on potential explanans and treatments. I argue that this is the primary worry that falls out of psychiatric dualism, as it directly threatens the two goals of healing and explaining.

In section two, I briefly outline two solutions that have been proposed in order to overcome this dualistic picture, both of which take an integrative approach. The first is that of Ian Hacking, who offers the fruitful distinction between interactive and indifferent kinds, suggesting that we might see the underlying neuropathology of a disorder as an indifferent kind, but those experiencing the disorder as interactive kinds, in order to integrate both the psychological and biological into one account (Hacking, 1999). Along with Dominic Murphy, I reject Hacking's account because it assumes that there is a stable underlying neuropathology behind each experience of mental disorder. Murphy himself aptly extends Hacking's discussion of interactive kinds to apply to neuropathologies themselves, and suggests that we can overcome psychiatric dualism if we recognize the way in which the same disorder could arise by way of either a biological cause (a defect in the mechanism) or a psychological one (the mechanism received the wrong input) (Murphy, 2001). I reject Murphy's account as well, arguing that it still encompasses the dualism he seeks to overcome.

Regardless of the fact that both of these accounts fall short, I argue in the third section that integrative solutions to the problem of psychiatric dualism are a step in the wrong direction. The reason for this stems from the primary issue of dualism that is identified in the first section, concerning the likelihood of missing out on potential explanans and potential treatments when working from a narrowly focused ontology. In attempts to integrate the biological and psychological pictures of psychiatric phenomena, this issue becomes even more pronounced. The aim of combining the two dominant ontological frameworks into one amalgam becomes either too restrictive, embracing some levels of investigation and leaving out the rest, or too ambitious, seeking to examine all levels of investigation at once, a goal which is both impractical and impossible. This brings me to the (perhaps) surprising conclusion that an incomplete ontological framework is inevitable in psychiatric research. Furthermore, this implies that the worry regarding missing explanans and treatments will arise in any particular investigation. For this reason, I argue that the widespread adoption of one ontological framework for all psychiatric research would only exacerbate this concern.

In the fourth section, I lay out what I think to be the most promising solution to psychiatric dualism. The strategy is one of ontological pluralism, which encourages an exploratory and open-ended approach to ontology within psychiatric research. Such an approach aims to lessen the impact of restrictive ontological frameworks on our potential for discovering relevant explanans and effective treatments that might not otherwise come into view. Drawing on the work of Paul Feyerabend, I outline the way in which his 'theoretical anarchism' might be adapted into an 'ontological anarchism' that is in line the pursuits of psychiatry (Feyerabend, 1975). In particular, each level of investigation in psychiatry may be seen as holding ontological primacy within different domains of research, as opposed to there being a widely held hierarchical picture of ontological status. I argue that ontological pluralism is well-suited to the

field of psychiatry because of the sizable number of levels of investigation, the lack of consensus on many research questions, and the regular involvement of external interest groups. Along with this proposal, I consider an important objection to this pluralistic approach, which asks how we might be able to choose between competing scientific explanations that rely on different ontologies. The answer I think is a simple and pragmatic one. I suggest that scientific explanations in psychiatry should embrace ontological frameworks that are suited to the explanatory goals of each particular investigation.

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Eric Hochstein, Washington University in St Louis

“Methodological Dualism as a Virtue in Psychiatry”

There is often taken to be a conflict within psychiatry between two different models of the patient. One model interprets patients as agents, with beliefs, desires, pains, hopes, fears, and other psychological states. This model is often contrasted with a more mechanistic interpretation of patients in terms of the neurophysiological mechanisms that transform sensory inputs into behavioral outputs. The tension between these two models stems from the fact that they seem, at least prima facie, to be irreconcilable. They posit different sorts of objects and relations in their

characterization of the same behaviors (one posits a set of neurophysiological mechanisms, while the other posits a set of psychological states). The agent-based, or goal-directed, model of the patient is often associated with the medical aspect of psychiatry, while the more causal mechanical model is often associated with the more scientific aspect of psychiatry. How then should psychiatry proceed given this tension between these two fundamental perspectives?

In this paper, I argue that there is a misconception at the heart of this tension. The assumption that the agent-based model is medical and not scientific, while the mechanistic model is scientific but leaves out something essential in the understanding of patients from a medical perspective, is often motivated by metaphysical worries. Namely, it assumes that science is only concerned with the description of physical structures and their causal interactions, and not unobservable entities such as psychological states and experiences. Meanwhile, the medical perspective suggests that a strictly mechanical description often leaves out something important in understanding the distress of patients. Is there more to understanding psychological agents than what can be conveyed through mechanistic models? If so, does this imply Cartesian Dualism? If we wish to avoid such a dualism and still be in accord with what our best science tells us, must we abandon agent-based models altogether in favor of the more scientifically respectable mechanistic model?

I will argue that the dualism implied by this tension between the agent-based model, and the mechanistic model, is not metaphysical, but merely methodological. In other words, it is true that the different ways of modeling the patient capture things that the other model cannot. This, however, does not imply anything like metaphysical dualism. Nor does it suggest that an agent-based model is unscientific in virtue of not being mechanistic. On the contrary, scientific practice often requires the kind of methodological dualism we see within psychiatry. The necessity of using non-mechanistic interpretations of complex systems in order to understand their behavior, while using mechanistic models to understand features of their underlying implementation, is something that is actually quite common throughout the sciences. Once we understand this, we can see that psychiatry need not choose between medical and scientific interpretations, since scientific practice allows for both interpretations. It simply means we cannot integrate the two interpretations into one and the same model.

To demonstrate, consider fluid mechanics (a branch of physics). Fluid mechanics is concerned with scientifically modeling behavioral features of various fluids in a variety of different contexts and environments. Interestingly, what we find in fluid mechanics is that in order to model many common behavioral features of fluids, we must deliberately ignore the mechanistic details of the system (i.e. model it in non-mechanistic ways), or say deliberately false things about its causal mechanisms. Accurate mechanistic models are simply unable to characterize the sort of behavioral features we wish to understand. Consider the way in which water moves as it flows through pipes, or the way in which waves propagate when we throw a rock into a pool of water. Mechanistic models (i.e. models which characterize the behavior of water in terms of the causal interactions of its molecular components) are unable to adequately model phenomena such as water-flow and wave propagation. Instead, if we wish to model such behavior, we must adopt models which interpret water as a continuous fluid, and not a collection of molecular mechanisms (for details, see: Morrison 1999; Teller 2001; Thomson-Jones 2005; Giere 2006). Likewise, if we wish to model other behavioral features of water, like phase

transitions, we must say deliberately false things about the mechanisms of the system, assuming things like infinite volumes, or infinite molecular components (Granger 1995, p.17; Batterman 2002, 2011).

We can find similar examples in domains like evolutionary biology. If we wish to model the evolution of phenotypic traits within a population, we often can only do so if we assume that population sizes are infinite, and if we likewise ignore many of the causal processes we know to be involved in the evolutionary process (see: Beatty 1980, 1981; Orzack & Sober 1994; Potochnik 2007, 2010; Rice 2012). Meanwhile, the inclusion of these correct details actually detracts from our understanding of the phenomenon we are trying to model.

The reason for this is because when we deal with extremely complex systems, we lack the resources and ability to model everything about the system within a single representational format. A single type of model simply can't usefully capture everything we need to know about the target system. As such, we must develop different sorts of models which are useful for characterizing the system in different ways depending on what we wish to learn about it. Thus, the duality in psychiatry between the agent-based model, and the mechanistic model, reflects this pragmatic feature of scientific practice. There are aspects of human behavior that cannot be captured using a strictly mechanistic model. This is not for metaphysical reasons, however. This is not because humans must be something more than a vast collection of physical mechanisms. It is because complex mechanistic systems often act in different ways depending on the context in which they are placed, and mechanistic models often cannot tell us this information given their focus only on the internal features of systems. Meanwhile, agent-based models often ignore the mechanistic details of systems, and instead characterize regularities and patterns in the way in which people interact with others, and deal with situations arising in their environment (Dennett 1987, p.257; Bechtel 2007, p.10). As such, we use the two models to understand different facets of a patient's behaviors and inner life. Both have an essential role in psychiatry, but this shouldn't be surprising given that we find a similar duality between models throughout the different sciences.

Jelena Krgovic, University of Buffalo

“Mental Disorder - Between the Medical Model and Anti-Psychiatry”

The problem of mind-dualism in psychiatry concerns the divide between the focus on the scientific and, consequently, third-person perspective and the insistence on the subjective and first-person aspects of psychopathology. One position that challenges the existence of mental disorders is a metaphysical one. This view challenges the claim that mental disorders are mental; that is, it claims that “mental” in mental disorders should be replaced by a brain-based or physicalist understanding. It rests on the body/mind problem in philosophy of mind. Underlying the view against mental disorders being mental is the claim that all mental states are physical states, and thus, all mental disorders are physical disorders. As George Graham remarks, the DSM –IV states the following: “Although the book is titled the *Diagnostic and Statistical*

Manual of Mental Disorders, the term *mental disorder* unfortunately implies a distinction between “mental” and “physical” disorders that is (an) ... anachronism of mind/body dualism.” (APA, 1994, xxi)³ Moreover, Thomas Szasz’s critiques of mental disorders has also, in addition to the ethical criticism he makes to the concept of mental illness, subscribed to this understanding of mental illness. Namely, Szasz would accept that schizophrenia is an illness provided brain lesions were found that account for schizophrenia. However, schizophrenia would no longer be, on his understanding, a mental illness but rather, a physical one.

I shall argue that identification of mental states with neurological ones is unwarranted. The critique of identity-theory will be based on Sartre’s philosophy of mind which I will elaborate on. Sartre’s rejection of the reducibility of mental to physical rests on the notion of intentionality which cannot be so reduced, as I will argue. I will here rely on Phyllis-Sutton Morris’ elucidation of Sartre’s philosophy⁴. On a positive side, I aim to elaborate on a Sartrean understanding of mental illness which, I argue, can incorporate both the scientific as well as the subjective aspects of psychopathology. Namely, Sartre’s position on illness is one of weak constructivism such that it involves both a value and an objective part, rather than only a value part. Additionally, Sartre’s position is that mental illness is not socially constructed, but individually constituted, such that mental illnesses are constructed by a person’s linking of painful experiences into a unified psychic project. The advantage of his view is that it acknowledges the suffering component involved in mental disorders. Moreover, it also avoids Boorse’s criticism of weak constructivism when it comes to illnesses. Namely, in his criticism of weak constructivism, Boorse argues that the problem with such views is that they make disease *prima facie* undesirable. He claims that this is not so since the flat feet of a draftee may in fact be desirable, or similarly a mild infection produced by inoculation. The view criticized here does not represent Sartre’s view, however, since for him the evaluative component concerns the individual experiencing the illness and not the concept of disease. While “illness” refers to the weakly constructed set of symptoms for the individual, “disease” is its objectification from the point of view of others, above all on the part of the medical-psychiatric profession. The objectification here involves knowledge of illness. Sartre claims that illness is only constructed, it is not known, but is instead suffered. Now, since we do not know our illness as an objective fact, but only “give it its matter” and evaluate it, knowledge of our illness comes through others. Sartre writes of disease that it is different from illness in that it is “objectively discernible for Others. Others have informed me of it, Others can diagnose it; it is present for Others even though I am not conscious of it.”⁵ This means that disease escapes the person suffering it because it is the physician who diagnoses it and knows its cause.

For Sartre, it can be the case that something is a disease and that it be desirable depending on the individual’s situation and what meaning the disease has as an illness in that situation. The distinction between illness and disease allows Sartre to avoid the pitfalls of weak normativism because he distinguishes between the objective state of one’s body—diagnosed essentially by others—and the way a person lives that state for herself. Moreover, Sartre’s view of mental illness involves a normative part, which is certainly important when it comes to physical illness as well, but as Fulford observes, our values when it comes to physical illness are in agreement,

³ Graham, *The Disordered Mind*, p.75

⁴ Phyllis Sutton Morris, *Sartre’s Concept of a Person: An Analytic Approach*, University of Massachusetts, 1976

⁵Sartre, *Being and Nothingness.*, p. 467

which is not the case regarding mental illness. Given that disease component is such that involves causes and knowledge of illness, Sartre's understanding of illness allows for scientific research of etiology of illness > however, given that disease is reductive and cannot incorporate the subjective experience nor meaning the ill person gives to her illness, it is necessary to account for the subjective phenomenological understanding of illness as well.

I will thus argue that what needs to be done is to incorporate the study of the phenomenological understanding of the person with a mental illness together with an objective understanding of disease. I will also argue that existential psychoanalysis can provide a method for such phenomenological understanding superior to the one given by Freudian psychoanalysis.

Valentina Petrolini, University of Cincinnati

“NEITHER BRAINLESS NOR MINDLESS”: TOWARDS AN INTERACTIONIST VIEW IN PHILOSOPHY OF PSYCHIATRY

Most recent debates in philosophy of psychiatry stem from opposing views about the status of the discipline and advocate different approaches towards the understanding of mental disorders. The competing accounts can be roughly grouped in two categories:

1) *Reductionist* accounts. Psychiatric disorders should be regarded as biological kinds and the nature of mental illness is better explained by reference to the bottom level (e.g. neural correlates) rather than to high-order cognition (e.g. introspection).

2) *Anti-reductionist* accounts. Psychiatry cannot be successfully - or meaningfully - reduced to neurobiology and the nature of mental illness is better explained by reference to the higher levels (e.g. disruption of the sense of agency) rather than to the bottom ones (e.g. chemical imbalances).

The goal of this paper is to outline an *interactionist* view in philosophy of psychiatry and to show that such an account fares better than the reductionist and anti-reductionist alternatives. The paper is divided in three sections: in the first I raise some issues with 1) and 2), articulating the need for a view able to integrate reductionist and anti-reductionist elements. In the second I individuate three desiderata for an *interactionist* account: a) special attention to the connection between the patient's *phenomenology* and neural correlates; b) special attention to the relationship between *biological* and *environmental* factors; c) endorsement of a *dimensional* approach to mental illness. In the third I briefly discuss the implications that an interactionist view may have on *psychiatric practice*, focusing on the phases of prognosis, diagnosis and treatment.

§1. The *reductionist* accounts described in 1) present three problems. First, they seem to undercut psychiatry's main goal as a discipline, which is to promote the well-being of *entire persons*. In this sense, reductionist approaches can be regarded as "mindless" because their exclusive focus on neurobiology runs the risk of ignoring or downplaying other crucial factors (e.g. behavioral, emotional, psychological, social). Second, overly reductionist accounts do not advance our understanding of the nature of mental illness and of what makes psychiatric

disorders distinctively *pathological*. Delusions are an interesting case in point here: even if significant neurobiological abnormalities can be detected (e.g. dopamine dysregulation, anomalous MPFC activity), we tend to regard delusional subjects as pathological in virtue of their behavioral, social and emotional manifestations. Third, a fully reductive approach runs the risk of neglecting the *intersubjective* character of mental disorders and in particular the connection with shared epistemic norms (e.g. what counts as rational, what constitutes a good reason or explanation). However, the anti-reductionist accounts described in 2) also exhibit certain problems. In particular, the focus on higher-order cognition and the characterization of mental disorders as failures of inference or self-monitoring run the risk of neglecting the aspects of mental illness directly connected to neural activity. Therefore, a "brainless" approach in philosophy of psychiatry seems to equally misrepresent an important target of the discipline.

§2. The first desideratum for an interactionist view would be a special attention to the *phenomenology* of the patient's experience and its connection with *neural correlates*. Focusing on the phenomenology of the patients means exploring the importance of personal life *narratives* - e.g. how the patients mentally place themselves in the environment, how they think of themselves in relation to others, etc. Once the patient's narratives have been adequately mapped out, the next step would be to ask what makes them disordered or pathological.¹ At the *top* level, we see a lack of intersubjective recognition and violations of norms of rationality: we - as "others" - are unable to make sense of the other person's narrative and frame it in a way that would render her thoughts intelligible. At the *bottom* level, there are several connections that can be drawn between phenomenological manifestations and cognitive or neural dysfunctions. For example, if the patient's narrative focuses on experiences of aberrant salience like the ones described by Kapur (2003) - e.g. "My senses seemed alive, things seemed clear cut, I noticed things that I had never noticed before" (p. 15) - we may want to look for alterations in dopamine release.

The second desideratum would be a special attention to the relationship between *biological* and *environmental* factors in the development and manifestation of mental illness. These factors are often separated in diagnostic classification systems, as suggested by the divide between *neurosis* and *psychosis*.² Yet, a number of recent empirical studies challenge this sharp distinction and highlight a closer connection between neurobiological and socio-cultural factors. A seminal work on this topic is the meta-analysis offered by Cantor Graae & Selten (2005), providing an overview on the studies conducted in different European countries to explore the high incidence rate of schizophrenia among first and second generation migrants. The data collected contributed to the formulation of the so-called "Social Defeat Hypothesis" (SDH), according to which discrimination and perception of social inequality would have a significant impact on the development of schizophrenia. The SDH represents an interesting case of interactionist explanation in which environmental circumstances are actively affecting brain changes causing psychiatric disorders to emerge.

The third desideratum would be the endorsement of a *dimensional* approach to mental illness, where psychosis is regarded as lying on a continuum with non-psychotic traits. Rather than circumscribing mental disorders in clear-cut categories grouped via symptoms or syndromes, a dimensional approach proposes to consider psychiatric traits as appearing in degrees among the general population.³ The embracement of a dimensional perspective sits comfortably with an interactionist solution. On the neurobiological side, we explore the neural correlates of widespread psychological phenomena (e.g. social anxiety) rather than investigating

dysfunctions on a group of patients that we have already identified categorically. On the environmental side, we focus on the occurrence of psychotic traits in the general population with particular attention to the role of emotions, inferential processes and coping mechanisms.

§3. Here I propose an application of the interactionist view to psychiatric *practice*. In the prognostic phase, the endorsement of an interactionist approach would be reflected in a heightened attention to *vulnerability markers* and *risk factors*. A more precise articulation of these factors may be helpful at different levels, such as prevention, policy making and fund allocation (e.g. mental health centers in large urban areas, governmental programs targeted to migrants). In the diagnostic phase, the adoption of an interactionist view seems particularly important in order to refine our understanding and classification of mental illness. The focus on phenomenology suggests an approach based on *structured interviews* to assess the patient's first-order experience, with special attention to the construction (and disruption) of self-narratives, while the endorsement of a dimensional approach suggests revising the diagnostic categories by rendering them closer to *prototypes* than to checklists. In the therapeutic phase, the interplay between levels seems crucial in order to address different aspects of mental illness. At the top level, *cognitive* and *emotional-cognitive behavioral therapy* (CBT and CEBT) may be interesting ways to act upon the emotional and affective imbalances connected to factors like social defeat or isolation. At the bottom level, psychiatric practice should intervene to adjust chemical imbalance through *pharmacological* intervention while observing the interaction with higher-order manifestations.

¹See Gallagher 2003

² See Freeman & Garety 2003.

³ See Johns and van Os 2001

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Sarah Robins, University of Kansas

"Confabulation and Constructive Memory"

Participants have been led to believe that they had been hospitalized overnight, or that they had an accident at a family wedding...they have fallen sway to the suggestion that they were once victims of a vicious animal attack (Loftus, 2003: 869).

The participant reported that in response to hearing 'piano' he created 'image of trying to get a grand piano through the front door at home.' But he was never presented with 'piano' (described in Dewhurst and Farrand, 2004).

These are not cases from the annals of psychiatry: they are reports from non-pathological adults, volunteers for psychology experiments. They reflect a significant, if startling, discovery from the last few decades of memory science: namely, that memory errors are a persistent and pervasive feature of everyday life. Philosophers and scientists who theorize about memory argue that these errors compel a *Constructive View of Memory* (e.g., de Brigard, 2013; Michaelian, 2012; Sutton and Windhorst, 2009). Memory should no longer be considered a capacity for preserving and retrieving representations of the past. Instead, memory is the act of *constructing* representations at the moment of recall, using any and all available resources—general knowledge, contextual cues, current emotional state, etc.

While the *Constructive View* offers an intriguing and promising approach to memory, its development has proceeded in a way that is insensitive to the role that memory errors—specifically, confabulations—play in psychiatric theory and practice. This neglect has significant consequences, both for our understanding of memory and of mental illness. Or so I shall argue. In this paper, I first identify the conflict between the philosophical and psychiatric approaches to confabulation. Next, I suggest a rapprochement, encouraging a more elaborate taxonomy of memory errors, beginning with a distinction between *misremembering* and *confabulation*.

The Constructive View of Memory collapses the distinction between memory errors and successful remembering—on this view, “veridical memories...are no less constructed than false memories” (Sutton and Windhorst, 2009: 87). Doing so makes it possible to accommodate and recast memory errors. Both are results of the same cognitive process, by which one builds representations of what might have happened during a past event. On this view, memory errors are no longer seen as mistakes or malfunctions; rather, they are harmless side effects of a system that is functioning as it should. Some go so far as to propose that the tendency to produce such errors is adaptive (Schacter and Addis, 2007). They note, for example, that susceptibility to memory errors is well associated with measures of creativity (Dewhurst, Thorley, Hammond, Ornerod, 2011).

This approach to memory errors has implications for psychiatry. By treating all attempts at remembering—the errors *and* the successes—as (harmless) confabulations, the Constructive View of Memory lacks the resources required for distinguishing between pathological and non-pathological forms of memory error, as psychiatric treatment requires.

In psychiatric theory and practice, confabulation is an important diagnostic symptom of disorders such as Korsakoff’s syndrome, Alzheimer’s disease, and schizophrenia. Korsakoff’s patients, for example, are often willing to tell stories of events from their past, but these stories vary widely over time and are often wildly implausible or in conflict with established facts (Kopelman, 1987). Similarly, it is thought that nearly 50 percent of those with schizophrenia experience *anosognosia*, an inability to remember that they have a disorder (Gilleen, Greenwood, and David, 2010; NAMI, 2005).

Within psychiatry, there are ongoing debates over how to best define confabulation (Bortolotti and Cox, 2009), with some favoring a narrow focus on memory (e.g., Moscovitch, 1995) while others prefer to encompass all delusional or unjustified beliefs (Berrios, 2000). There are also disagreements over etiology. Some view confabulation as retrieval failure (Berlyne, 1972), whereas others consider it the result damage to the executive system (Hirstein, 2005) or a lack of source memory (Johnson, Hashtroudi, and Lindsay, 1993). Regardless of how—or whether—these issues are settled, confabulation remains a critical concept for diagnosing, treating, and theorizing about mental illness.

If the Constructive View of Memory is right, however, confabulation fails to pick out a distinctive form of memory error, or even an error at all. All memory productions are the result of the same, well-functioning, process of constructing representations that strike the rememberer as plausible accounts of past events. Some memory theorists accept this, casting everyday memory errors as “a kind of confabulation in non-clinical subjects” (Garry, French, and Loftus, 2009). I believe that this conclusion should be resisted, for two reasons.

First, to treat all memory errors—everyday mistakes and florid confabulations—as on a par is to ignore the disorder and suffering these errors reflect in clinical populations. As Hirstein explains, “confabulation in the clinic can be severely debilitating” (2009: 1). We owe it to those whose lives are disrupted by confabulation to continue the pursuit of ways to identify, treat, and prevent these memory disorders.

Second, the Constructive View's inclination to treat all memories as confabulations is not compelled by, nor even the best interpretation of, the data on memory errors in everyday and clinical populations. While the terms "false memory," "confabulation," and "misremembering" are often treated as synonyms, used interchangeably in current theorizing, closer attention to subtle differences between these errors suggests there are multiple, distinctive ways in which attempts to remember can go awry.

The route toward rapprochement begins, I suggest, with an improved taxonomy of memory errors. As an important step in this direction, I advocate for a distinction between the errors of *misremembering* and *confabulation*. When a person misremembers a past event, what she reports about that event is inaccurate and yet the error is explicable only on the assumption that she retains information of the mischaracterized event. Confabulations, in contrast, are wholly inaccurate, reflecting no influence of information retained from the past event. It parallels the distinction between illusion and hallucination in perception, and further, offers the best way to characterize many familiar paradigms for eliciting memory errors in experimental contexts. As most non-pathological cases of memory error are best interpreted as misrememberings, this distinction paves the way for the demarcation between pathological and non-pathological errors that is urged by psychiatric theory and practice.

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"Normative Standards for Scientific Psychiatry"

I am interested in a particular conception of the discipline of psychiatry known in the philosophical literature as *scientific psychiatry*. Briefly, scientific psychiatry is concerned with the study and treatment of mental disorders, where mental disorders are real entities with discoverable causal etiologies. While ideally consilient with the sciences of the mind/brain, psychiatric diagnosis also, crucially, involves normative and evaluative concepts such as health and well-being. *How do we decide who is better or worse off? What differentiates mental illness from mental health?* In this paper I will examine the distinction and what it means to apply these terms to individuals in clinical contexts.

As I will argue, mental health is a notion that has much in common with notions of well-being, flourishing, and value—a cluster of normative issues in positive psychology which have generated a lot of recent excitement among philosophers. Part of my job here, therefore, will be to orient the philosophical and empirical debates surrounding well-being with those in the philosophy of psychiatry. In a sense I will use the toolkit of the positive psychology movement in thinking about *positive psychiatry*. The main goal of the paper will be to use this discussion to articulate standards for the normative theory of scientific clinical psychiatry's second stage. With these standards in place, I will offer a view which I think adequately addresses the relevant concerns.

To get a feel for the kinds of standards I have in mind, consider: mental health, like well-being, should have some motivational or reason-giving content. It should be good *for* an individual. For this reason, norms that have authority in virtue of something external to the

subject are suspect, which suggests that mental health should be located in the subjective point of view. But being mentally healthy cannot be as simple as believing so. A good theory of mental health will countenance the possibility that someone who thinks they are well is actually disordered.⁶ Motivating and justifying reasons are crucial for a theory of mental health if we want it to be something we have reason to promote in ourselves and others. These competing concerns parallel the standards of normative and empirical adequacy which Valerie Tiberius and Alexandra Plakias identify in their 2010 paper “Well-Being.” Normative and empirical adequacy give a theory its “normative authority”, “the feature in virtue of which people have a reason to follow the imperatives of a normative theory” (Tiberius & Plakias, 2010).

With regard to a theory of mental health, then, if we have no reason to care about the theory or if it gives us no reason to follow its imperatives, then it fails to be normatively adequate. In other words, the first thing that psychiatric practice do is reliably distinguish between cases of mental illness and mental health, and thus refrain from being paternalistic. Historically, the discipline of psychiatry has been less than successful at this task. Because diagnosing an individual as having a mental disorder can be a way of saying that they have a condition that is bad and ought to be corrected, and that their pattern of behavior is somehow deviant or harmful, psychiatric diagnosis can be, and has been, used as a tool of social control. There are clear historical cases of diagnostic categories which functioned as nothing more than a means of institutionalized oppression. Surely runaway slaves diagnosed with ‘*drapetomania*’ did not think of themselves as unwell or needing some sort of treatment, nor should they. Whatever else a good theory should do, it must avoid this threat—it should not characterize individuals as unwell who aren’t. Who gets to decide who is mentally ill, and when, is of grave importance when the downstream effects of receiving a psychiatric diagnosis can include limiting the autonomy of the diagnosed.⁷

Moreover, the insight that culturally relative norms about what behaviors are and aren’t desirable can compromise the objectivity of a taxonomy of mental disorders, highlights the need for empirical adequacy. The second thing a good nosology must do is be objective. Mental disorders should be grounded in objective facts about the agents they characterize. If a theory implies that it cannot be investigated, measured, and achieved, then it fails to be empirically adequate. Our mental health should be every bit as scientifically investigable as the cognitive mechanisms that make it up. Happily, we can again look to positive psychology as a model for how to proceed, and a wealth of data on *happiness* and *life satisfaction*, two commonly endorsed empirical measures of flourishing.

Ultimately, the view I favor, like Tiberius and Plakias’ account of well-being, grounds mental health in flourishing with respect to ones values. As they argue, “As long as we make room for the possibility of defeaters when the context requires, a psychologically realistic conception of a person’s values can do the necessary work in a normative account,” (Tiberius & Plakias, 2010). Thus, suppose that I have both the symptoms and the distinctive underlying

⁶ The question of whether someone is mentally ill is slightly different from the question of whether they are disordered, but the two notions are intimately linked. To diagnose someone with a mental disorder is to say that they have a condition which inhibits their mental health. This leaves the possibility open that someone may not have a disorder, per say, but still fail to be mentally healthy.

⁷ Also, while diagnostic categories themselves already carry evaluative content—we tend to treat individuals diagnosed as mentally ill prejudicially in comparison to those we consider healthy (Banaji, 2013)—it has often been the case that those on the receiving end of psychiatric diagnosis come from stigmatized, disadvantaged, or disenfranchised groups (Satcher, 2001).

causal etiology of clinical depression, and am seeking help from a therapist. I might describe how my low energy and affect are impacting my relationships and my ability to work—both of which I report to her as valuing. Since the cause of my inhibited flourishing is this particular mental condition, I can be said to be have a mental disorder. The goal of psychiatric treatment is to intervene on my mental functioning in order to allow me to flourish.

This view can also make sense of more difficult cases, like the “successful psychopath” or high-functioning autistic. These individuals may be atypical in both behavior and cognitive makeup, but we cannot say they are disordered unless they are failing to achieve a good life *as they define it*. It can also say something about an artist in the grips of a manic episode, who claims that she has no disorder because alcohol and a functioning word processor are the only things she values. She may just be wrong.

Georg Repnikov, University of Sydney

“Psychiatric Nosology and Our Changing Understanding of Mental Disorders”

Psychiatric classification systems both reflect and influence the understanding of mental disorders prevalent at a given time. In this paper, I will explicate some of the conceptual assumptions our current classifications are based upon, show how they contribute to what critics believe is a crisis in psychiatric nosology, and determine what kind of shift in our understanding of mental disorders is presupposed by, or likely to follow from, a new research program meant to ameliorate this crisis - the Research Domain Criteria Project (RDoC). The following provides a summary of the points to be discussed in the final version of this paper.

1. The Crisis in Psychiatry - The DSM and its Problems The DSM Approach to Nosology

In the first part of this paper, I will introduce the currently dominant DSM approach to classifying mental disorders and explain the reasons for its "theoretical neutrality" with respect to questions of etiology and pathophysiology as well as its reliance on "phenomenology" - the clinical observation of symptoms, signs and the course of illness. I will show that the neo-Kraepelinian approach to research and clinical practice embodied by the DSM is committed to the medical model of mental disorder, viewing psychiatry as a branch of medicine and people diagnosed with a mental disorder as sick individuals in need of medical attention. Mental disorders are conceived of as problems arising and residing within the individual, to be classified, studied and treated along the lines of diseases in biomedicine. Although the DSM officially holds its diagnostic categories to be mere constructs, *in practice*, in the clinic as well as

in the laboratory, these categories usually become reified and get treated as representing natural kinds - discrete disease entities with boundaries dividing them from other disorders as well as from normality (Kendell & Jablensky 2003; Hyman 2010).

Problems with the DSM Approach

Psychiatry is in crisis. Compared to other areas of medicine, and despite advances in basic biomedical research, knowledge about the etiology or underlying dysfunctions and pathophysiology of DSM disorders is still limited and progress in the development of effective individualized treatments has been slow as a result (Cuthbert & Insel 2013).

In this part of the paper, I will explain why many critics attribute this dismal state to the DSM with its categorical structure and operationalized polythetic diagnostic criteria. I will show in what way these features of the DSM have led to extremely high rates of comorbidity, heterogeneous patient groups and an excessive use of Not-Otherwise-Specified diagnoses.

Besides pointing out some of the negative consequences these problems have had on research and treatment practice, in the final version of this paper, I will explore the DSM's struggle to establish both reliable and valid diagnoses from a psychometric perspective. In particular, I want to demonstrate how the DSM's failed attempt to simultaneously improve internal consistency and interrater reliability as well as content and external validity could readily be explained by a mismatch between the assumed categorical nature of mental disorders and the latent dimensional structure of certain parts of psychopathology - which would also sit well with recent research on the contributions of a variety of causal difference-makers in psychiatry (Mitchell 2008; Kendler 2012a,b; Berenbaum 2013), given an interventionist understanding of causation (Woodward 2003).

2. The Making of a New Paradigm - The RDoC Project

The Basic Structure of the RDoC

Confronted with this crisis in psychiatric classification, the leadership of the National Institute of Mental Health (NIMH) decided to initiate a new funding program meant to generate "a research literature that can inform future versions of psychiatric nosologies based upon neuroscience and behavioral science rather than descriptive phenomenology" (Cuthbert 2014, p. 28). The ultimate goal of the NIMH is to build a new classification system, based on breakthroughs in scientific research and independent of the traditional DSM categories, that will facilitate future studies and improve treatment outcomes.

While the RDoC itself is *not* a classification of mental disorders, it does provide a conceptual research framework - the RDoC matrix. In the final version of this paper, I will introduce the different elements of this matrix - the constructs and sub-constructs making up the "Domains of Functioning" to be studied (e.g., cognitive systems, social processes) as well as

the various classes of measurement assessing the "Units of Analysis" (e.g., genes, brain circuits, behavior). I will emphasize the dimensionality of these constructs, discuss the reasons for their inclusion and explain how the RDoC is supposed to produce a classification that will not suffer from the same problems confronting the DSM now. In this part, I will also address some of the initial criticisms leveled against the RDoC that are based on a failure to acknowledge some of its central features.

The Philosophy of RDoC

The final part of my paper is devoted to a discussion of some of the conceptual issues arising in the context of the RDoC project. My point of departure is the observation that while the DSM approach embodied a "soft" or "minimal" version of the medical model (Kendler 2012a, Murphy 2013), the RDoC is committed to a stronger view in that it "conceptualizes mental illness as brain disorders. In contrast to neurological disorders with identifiable lesions, mental disorders can be addressed as disorders of brain circuits" (Insel et al. 2010).

First, I will show how this assumption constrains the research funded by RDoC. Second, I will argue that the RDoC research will likely result in a "creeping reductionism" - the explanation of parts of psychopathological phenomena in terms of multilevel mechanisms or "pathways" (Schaffner 2013) - which is at odds with the conception of mental disorders as *mere* brain circuit dysfunction. Third, I will demonstrate that while some advocates of the RDoC think they are "neutral" with respect to fundamental definitions of mental disorder (Cuthbert & Kozak 2013), they do, in fact, adopt a *practical kind* view of mental disorder (Haslam 2002). Fourth, given this view of mental disorders, what is to count as "pathological" becomes a matter of clinical utility, not natural necessity, and I want to explore what kind of psychological effect this might have on how we conceive of psychopathology. Fifth, and lastly, I want to discuss the integrative power of the RDoC project to cross the explanatory divide enshrined in contemporary forms of mind-brain dualism, and whether there might be something essential left out of the RDoC picture of psychopathology.

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Alexander Jeuk, University of Cincinnati

LINGUISTIC DUALISM AND THE EMBODIED APPROACH TO MENTAL DISORDER

An influential source of dualistic intuitions in psychiatric research derives from an implicit, paradigmatic conception of cognition. This conception assumes an explanatory gap between conscious experience, semantic-linguistic mental representations and the neural mechanisms that realize them.

The main assumptions behind this conception are (i) that experience is non-linguistic; (ii) that cognition is realized by mental representations that are linguistic-(symbolic); (iii) that embodied and neural mechanisms are not representable in a way so that they are mechanistically continuous with experience and cognition.

What underlies this assumption is the conviction that cognition, in particular conceptual cognition, is not only expressed linguistically-symbolically, but that it is linguistic-symbolic in kind.⁸ According to this conviction, the models that explain neural mechanisms are described in (a) a linguistic format and (b) in a linguistic format that implements a language that is markedly distinct from the language with which experience or mental representation processing are described.

Though we might be able to conceive of certain neural networks and their working principles in terms of higher-order functions like sensorimotor processing, prediction or simulation, we are not able to map linguistic-symbolic mental representations in an intelligible way on the neural level. This assumption entails a dualism between cognition and the neural mechanisms that underlie it. Accordingly, what seems to be at the core of the dualism⁹ between experience, cognition and neural mechanisms is the assumption that cognition is linguistic-symbolic - i.e. a Linguistic Dualism.

Indeed, Linguistic Dualism has conceptual consequences for psychiatric research in terms of (a) how researchers and practitioners conceptualize symptoms or anomalies and (b) with regard to the observational expectations they form about the co-occurrence of symptoms in a particular mental disorder. The concrete consequence is that symptoms of mental disorders seem unconnected, disparate and the question arises whether mental disorders are well-circumscribed kinds.

For instance, concerning Autism Spectrum Disorder it seems difficult to connect sensorimotor deficits,¹⁰ that are describable partly by means of breakdowns in neural mechanisms, with social deficits, linguistic deficits and cognitive deficits. Clumsiness,¹¹ the inability to track eye movements,¹² problems in understanding metaphor¹³ and the inability to process abstract concepts¹⁴ seem neither experientially nor mechanistically connected and utterly disparate¹⁵ according to theoretical frameworks adhering to Linguistic Dualism. Similar problems apply to Schizophrenia. How can we connect patient reports about inserted thoughts,¹⁶ co-occurring breakdowns in motor control that cause delusions of alien control¹⁷ and imaging data that suggest the activation of the auditory cortex in the case of thought insertion?¹⁸ Thought insertion seemingly pertains to inserted thoughts, i.e. linguistic-symbolic entities, and

⁸ This means that we do not only take recourse to language as a means of communication to express our thoughts, but that cognition or thought is housed in a linguistic-symbolic representational format.

⁹ We might even speak of a 'trialism'.

¹⁰ cf. Fournier 2010.

¹¹ cf. Fournier 2010.

¹² cf. Klin et al. 2003.

¹³ cf. Glezerman 2013.

¹⁴ cf. Groen et al. 2009.

¹⁵ cf. Happé et al. 2006.

¹⁶ cf. Waters & Badcock 2010.

¹⁷ cf. Frith et al. 2000.

¹⁸ cf. Moritz & Larøi 2008.

not to auditory verbal hallucinations or auditory phenomena in general. And delusions of alien control are seemingly constrained to the explanation of effector movements.

I argue that this seeming symptom disparity vanishes if we reject the main assumption behind Linguistic Dualism: that cognition is linguistic-symbolic. I propose an alternative framework that is based on a synthesis of Enactivism, Grounded Cognition and Mechanistic Explanation; a synthetic approach that I call 'The Embodied Approach to Mental Disorders' in the context of psychiatry.

Enactivism locates meaning in lived experience and sense making practices. Enactivism explicitly rejects the separation of experience and meaning by grounding meaning in experience, in particular in the experience of action and perception.¹⁹

Grounded Cognition explains how experiential meaning is realized by embodied and neural mechanisms by means of modal-sensorimotor representations, simulation and motor control mechanisms, i.e. mechanisms that realize experiential meaning that is grounded in action and perception. Grounded mechanistic models can explain how sensorimotor mechanisms can realize not only basic sensorimotor functions, but also how they can explain social,²⁰ linguistic²¹ and cognitive conceptual functions.²² They can explain this through models of offline re-enactment (simulation) of sensorimotor processes in motor control mechanism that are ordered in nested hierarchies,²³ mostly for the purpose of prediction and error reduction.²⁴

Mechanistic Explanation allows us to frame how models from Grounded Cognition and phenomenological approaches from Enactivism can be brought into accord. Mechanistic Explanation models themselves can be represented by means of sensorimotor representations and provide a theoretical justification for the connection of sensorimotor mechanisms, social cognition, language processing, conceptual cognition and experience.

Bechtel²⁵ explicitly identifies Barsalou's Perceptual Symbol Systems²⁶ model as the model to bridge this gap. The content of a representation is individuated by experience in action and perception, the mechanisms spelled out are higher order sensorimotor representations and simulation mechanisms that are realized in a network model that is projectable on neural networks. Machamer et al.²⁷ and Machamer²⁸ mention the importance of embodied sense making mechanisms underlying the meaning of the representational machinery of mechanistic explanations.

If this synthesis holds, Linguistic Dualism is rejected and the problem of disparate symptom disunification that is entailed by it vanishes. I spell this out using the example of

¹⁹ cf. Fuchs 2009; Klin et al. 2003; Silberstein & Chemero 2012; Thompson 2007.

²⁰ cf. Gallese 2003.

²¹ cf. Pickering & Garrod 2013.

²² cf. Barsalou 1999, Gallese & Lakoff 2005, Grush 2004.

²³ cf. Simmons & Barsalou 2003.

²⁴ cf. Clark 2013; cf. Hohwy 2013.

²⁵ cf. Bechtel 2008.

²⁶ cf. Barsalou 1999.

²⁷ cf. Machamer et al. 2000.

²⁸ cf. Machamer 2004.

thought insertion. If we do not conceive of thought anymore in terms of a linguistic-symbolic process, we will not conceptualize patient reports literally as 'thought insertion', but likely as misguided inner speech or an auditory verbal hallucination.²⁹ If we do not conceive of inner speech anymore as conscious thought or the expression of conscious thought, but as a sensorimotor event that is the prediction of a motor intention to act linguistically,³⁰ then we can apply the standard comparator model of delusions of alien control³¹ to thought insertion. This entails not only a non-mysterious explanation of a symptom of Schizophrenia, but it also unifies symptoms by subsuming 'thought insertion' under delusions of alien control. It needs to be shown that this approach applies to other mental disorders as well, but work already done by some researchers³² is indicative that the source of an important dualism in psychiatric research can be located in Linguistic Dualism.

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²⁹ cf. Langland-Hassan 2008.

³⁰ If we 'de-lingualize' cognition, we also 'de-cognize' language. Language is only a means of communication that is not reflective of cognition itself. Rather it is a sensorimotor phenomenon that is comprised of a motor command to utter a sentence and phenomenologically of an auditory episode that resembles attenuated outward directed speech.

³¹ cf. Frith et al. 2000.

³² cf. Klin et al. 2003; Oberman & Ramachandran 2007.

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Jasmin Ozel, University of Pittsburgh

“Early Intervention in Schizophrenia: Costs and Benefits of Including a "Psychosis Risk Syndrome" in the DSM”

Despite significant progress in the psychopharmacological treatment since the 1950s, schizophrenia is still one of the main causes for permanent disability and approximately one third of all patients still have a poor prognosis. Hence, new routes to the treatment or even prevention of schizophrenia are increasingly being explored. One of them, the early intervention at prodromal stages of schizophrenia, seems to be a particularly promising approach.

In my talk, I will argue for the following three points: First, shifting the focus of schizophrenia research to prodromal stages will shed more light on the actual nature of the disorder. It has been suggested that it is in fact a "disorder of the self", which lies at the center of schizophrenia (Nelson, Parnas, & Sass, 2014). Second, focus on the prodromal stages of the illness will considerably impact treatment options and outcome: Recent research in psychiatry suggests that treatment at prodromal stages might lead to complete remission of symptoms in some cases, and could hence be an actual cure for schizophrenia. Third, the eventual decision not to include an at-risk diagnosis into the DSM-5 needs to be carefully reconsidered for the next version of the DSM. Research that understands schizophrenia in terms of its effects on the self instead of in terms of what potentially are mere surface symptoms, such as delusion and hallucinations, will give us a much more adequate picture of the illness. Moreover, such a disorder of the self would already show up much earlier in the patient's development and could hence be a more reliable indicator of prodromal stages of schizophrenia.

The most important benefit of early diagnosis and treatment is that it vastly improves the prognosis. The earlier schizophrenia is being treated, the less severe its course tends to be. There are various reasons why such an early treatment can lead to a so much better outcome. On the one hand, there is evidence suggesting that psychosis itself may be toxic to the brain (Wyatt 1991), and that early intervention would hence prevent the patient's brain from being harmed. On the other hand, early treatment would also diminish the impact on the psychological, social and socioeconomic state of the patient—which often constitutes a different kind of toxicity, namely "psychosocial toxicity of the impact of the subthreshold prodromal symptoms" (Schaffner & McGorry, 2001, p.5).

Yet, there are also significant possible costs connected with attempts to diagnose and treat patients at prodromal stages. First of all, given the age of the patients, there are issues with informed consent that go beyond the ones we usually encounter in adult patients. The stigma that is associated with the diagnosis of schizophrenia is a known issue. And such a stigma will most likely also apply to cases in which schizophrenia is diagnosed at a prodromal stage. Patients might be discriminated against, and the diagnosis will most certainly have an impact on the image they have of themselves. Furthermore,

antipsychotic medications, which in a low-dose would be the treatment of choice, have side effects that impact the patient's life in various ways. Given that the risk of false positives is significant, one might argue here that this doesn't justify such serious medical intervention on the mere basis of a predicted psychosis risk. Yet, I will argue in my talk that ultimately, the benefits of early intervention outweigh these costs.

Research in the prodromal stages of schizophrenia is of particular importance for another reason: More and more research on schizophrenia suggests that psychotic symptoms, which are usually taken to be characteristic of schizophrenia, are merely "surface-level" phenomena. Underlying, at the core of the disorder, is a disturbance of the patients' "self-experience or selfhood". By disturbances of the self, the following features are usually referred to: depersonalization, distortions of the first-person perspective, a "diminished sense of coherence and consistency in fundamental features of self" (such as identity confusion), and unclear boundaries between self and world / others (Nelson et al., 2014). This disorder of the self has often been characterized in terms of a disturbance of the "minimal self", which refers to the phenomenal character that the first personal experience comes with. In schizophrenic subjects, it doesn't hold anymore that "I am always already aware of 'I-me-myself,' with no need for introspection or reflection to assure myself of being myself" (Nelson, Parnas, & Sass, 2014).

The main advantage of using disturbances of the self as diagnostic criteria in the diagnosis of prodromal stages of schizophrenia is that even a "minimal self-disturbance" already strongly predicts a later onset of schizophrenia. Moreover, self-disturbance seems to be highly correlated with a later onset of schizophrenia, but much less so with psychotic disorders outside of the schizophrenia spectrum.

Another reason why disturbances of the self are a promising new route to progress in the research on and treatment of schizophrenia, is that they provide us with a "richer understanding of psychopathology in research and clinical training, thereby mitigating over-reliance on 'symptom checklists'" (Nelson et al., 2014). If schizophrenia's "checklist symptoms", namely delusions, hallucinations, disorganized speech, catatonic behavior and negative symptoms (American Psychiatric Association, American Psychiatric Association, & DSM-5 Task Force, 2013; 295.90 / F20.9) turn out to be mere surface phenomena, then the actual nature of the illness can most likely not be found here. I will argue that we can achieve a much deeper understanding of the illness if we focus on its actual core, namely disorders of the self. The symptoms that individuals display when they haven't entered openly psychotic stages yet are much less dramatic than the ones we find in psychotic patients. And yet, they might be the only way we can make progress in understanding the nature of schizophrenia.

Rik Hine, Texas Christian University

“(Mis)representing and intervening: Diagnosing the crisis in contemporary psychiatry”

“Science is said to have two aims: theory and experiment. Theories try to say how the world is. Experiment and subsequent technology change the world. We represent and we intervene. We represent in order to intervene, and we intervene in the light of representations.”

(Hacking,

p.31)

How we represent the world to be has important implications for how we try to change it. Nowhere is this more apparent, and pressing, than Psychiatry. Internal disagreements about the direction of the field reflect fundamental differences about the issues that fall under its aegis. Often to the detriment of those it seeks to treat. On the one hand, are those who think that Psychiatry should focus on subjective, first person aspects of mental illness.³³ On the other, advocates of objective, third person investigation. Never the twain shall meet³⁴, it seems. And this is no surprise given that the crisis is merely a modern scientific strain of an old metaphysical malady: dualism. The underlying motivation for this schism is a division of opinion about the possibility (and desirability) of an in-principle reduction of mental illness to brain malfunction³⁵. At stake, both sides argue, independently, is therapeutic progress. And for some, associated concerns about Psychiatry’s claims to scientific status.

In what follows, I will argue that Psychiatry can have its cake and it eat, too. That is, the field can keep subjective experiences at its explanatory core and still be a branch of science, in good standing. Resolution of this debate, though, involves rejecting the underlying reductionist assumptions that influence the way in which it is currently conducted. To be clear, this is not about taking sides on the debate *within* Psychiatry. It is instead, a rejection of the very terms under which it takes place.

Indeed, it is ironic that some factions within Psychiatry are trying to secure its scientific *bona fides* on an out-dated model of ‘science.’ This is one which assumes both that inter-theoretic reductions are rife in the natural sciences, and that reducibility is a normative constraint on scientific legitimacy (Horst, 2007). But these suppositions are simply not supported by contemporary scientific practice. There are no genuine cases of inter-theoretic reduction. And reductions, of any kind³⁶, are rare *within* individual sciences, too. Moreover, the “aprioristic normative agenda of the Positivists has been abandoned in favor of approaches that study the various methods and models of individual sciences, and the prevailing view is that the special sciences are autonomous and not in need of vindication by proving their reducibility to physics.” (Horst, p.47)

Relatedly, this position also labours under an outmoded notion of laws. Central to common-sense psychological explanation is the conception of causal relations between beliefs and desires. But it is generally agreed that the covering causal laws at work in this domain are radically

³³ I’ll leave aside, here, ontological questions about the existence of ‘mental illness,’ rather than individual ‘mental illnesses’ (see, for instance, Bentall, 2004).

³⁴ See Thornton’s take on the ‘interface problem.’ (2009)

³⁵ This is not the place to investigate what *the* ‘fundamental’ level of explanation amounts to.

³⁶ I will say more about varieties of ‘reduction’ in the longer paper.

different to those in the physical sciences. That is, the former are merely “*ceteris paribus*,” compared to the strict and exceptionless laws of the natural sciences (Fodor, 1974)³⁷. If, as is common, one assumes that the latter laws are a hallmark of scientific legitimacy, then the scientific aspirations of Psychiatry are called into question. The anti-reductionist, of course, fares no better, finding herself with a ‘counterfeit’ notion of causation at the very heart of her explanatory scheme.

But, as Cartwright has argued:

“The laws that describe this world are a patchwork, not a pyramid. They do not take after the simple, elegant and abstract structure of a system of axioms and theorems. Rather they look like...science as we know it...the cover of law just loosely attached to the jumbled world of material things.” (p.1)

The point, then, is that these beliefs cast a pox on both houses. Those seeking Psychiatric intervention are held hostage to representations rooted in an obsolete scientific worldview. The field fails to reflect the methodologies of the very sciences it aims to be positively associated with.

So what is the way forward? Well, whilst provision of a detailed picture is beyond the scope of this paper, one issue is abundantly clear: the discipline needs to prioritize developing the *proprietary* methods and models it requires, not concern itself over its scientific standing. Ironically, though, the way to accomplish this is by recognizing, and embracing, contemporary scientific pluralism. For instance, biologists employ a wide variety of classificatory schemes, which usually cross-classify. Nevertheless, this is not seen as an impediment to proper explanation, nor is it immediately assumed that it calls into question the legitimacy of the kinds invoked. Rather than being treated as rival positions, it is argued that they merely reflect the multiplicity of interests within the domain (Kitcher, 2003).

Undoubtedly, this approach carries concerns about an attendant *ontological* promiscuity in its wake. But for now, at least:

“The worry is not so much that we will adopt wrong images with which to represent the world, but rather that we will choose the wrong tools with which to change it. We yearn for a better, cleaner, more orderly world than the one that, to all appearances, we inhabit. But it will not do to base our methods on our wishes. We had better choose the most probable options and wherever possible hedge our bets.” (Cartwright, pp. 12-13.)

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³⁷ See Davidson (1970) for a similar set of concerns.

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