2013

Neo-Kraepelinian Divergences from Kraepelin : What are They and Why They Matter

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by

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An undergraduate honors thesis submitted in partial fulfillment of the

requirements for the degree of

Bachelor of Science

in

University Honors

and

Philosophy

Thesis Adviser

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2013

Introduction

Throughout its development, psychiatry has struggled to legitimate itself as a scientific and medical discipline. Much of this struggle has been attributed to a lack of consensus regarding the nature of mental illness as well as a standard methodology for making diagnoses. In an attempt to eliminate this impediment to psychiatry's scientific advancement, the third edition of the Diagnostic and Statistical Manual of Mental Disorders (DSM) was published in 1980 with significant methodological changes to the nature of classification and approach to clinical diagnoses. Similar to the characterization of modern psychiatry as being amidst a Kraepelinian revival, this highly influential text is often associated with and regarded as an adaptation of the psychiatric nosology of Emil Kraepelin (1856-1926). This paper begins by asking, what lead to this Kraepelinian revival? Secondly and as the primary focus will be an investigation of how "Kraepelinian" this "neo-Kraepelinian" manifestation in the DSM III actually was. Addressing the latter will lead to an examination of (1) the technical and contextual differences between Kraepelin's and the DSM's classification of the major psychoses and (2) the ontological differences between and consequences of the respective nosologies. Ultimately, it is argued that the DSM's neo-Kraepelinian translation of Kreapelin's work has deviated from it in consequentially problematic ways. But first, it is necessary to define the technical terms which will be used throughout this paper.

Defining the technical terms

The term *reliability* refers to the degree to which a measuring procedure of a particular phenomenon yields consistent results in repeated trials. In psychiatric diagnoses, *inter-rater reliability* refers to how often clinicians arrive at the same diagnosis given an identical set of data and a given patient. A central goal of psychiatric nosology is diagnostic reliability, and a central goal of psychiatric practice is to demonstrate a high degree of inter-rater reliability.

The term *validity* refers to the degree to which a measuring procedure measures what it purports to measure. As it applies to psychiatric nosology, it refers to whether or not a diagnostic category maps onto a true disorder. Kendell and Jablensky propose the following condition for determining validity:

(1) If the defining characteristic of a category is a syndrome, then the syndrome must be separated from other syndromes and normality by a "zone of rarity" criterion, i.e., *"natural boundary"* (clear-cut boundary at the level of defining characteristic) (5).

In addition, there are two other forms of validity which are pertinent to the discussion of DSM categories (Carmines and Zeller, 17, 22-23);

(1) *Predictive validity* concerns the degree to which a measuring instrument accurately predicts some form of behavior which is external to the measuring

instrument itself. If a diagnostic category is meant to predict how a person with that diagnosis will behave, it can be said to have predictive validity if it accurately predicts how a person with that diagnosis will behave.

(2) *Construct validity* is the extent to which there is a correlation between a particular measurement and other measures consistent with theoretically derived hypotheses concerning the constructs that are being measured. As an example, to measure the construct validity of a particular measure of narcissism, say DSM-defined Narcissistic Personality Disorder (NPD), one would consider the relation between the construct and a theoretical hypothesis derived *from* the construct of DSM-defined NPD. If there is a high correlation between these two measurements then it would constitute a piece of evidence in support of the construct validity of DSM-defined NPD.

The term *pathognomonic* refers to a sign or symptom which is definitively indicative of a particular condition. While it is not necessary for the associated diagnosis, it is sufficient in that it is specific to a particular condition and not any others.

In what follows is a consideration of a brief history of psychiatry which will be helpful in understanding the factors that contributed to the "Kraepelinian revival" characterizing modern psychiatry.

Part I A Brief History: The Development of Psychiatry and Resurgence of Kraepelin

Ideas about the nature of mental illness are as old as our history allows. The ancient Greeks viewed madness as a result of elemental and humoral imbalances. While this perspective persisted in part through the Middle Ages in Europe, religious influences at the time effectually placed the mentally ill within the domain of morality. Throughout most of the eighteenth century, individuals were held in asylums and subjected to cruel environments as a means of separation from the rest of society rather than therapeutic intervention. It was not until approximately the middle of the nineteenth century that psychiatry emerged as a medical specialty. The German psychiatrist Emil Kraepelin (1856-1926) was a prominent figure in this development.

Before Kraepelin, there was little to no consensus on the nosology of mental illness. A proponent of biological psychiatry, he paved the way for a more scientifically based study of and classificatory system for mental illness. After categorizing as many mental illnesses in accordance with biological etiology, he was left with a large group of patients with psychotic conditions that lacked any apparent pathophysiologic explanation (Klerman, 100). His differentiation of these psychoses into dementia praecox and manic-depressive insanity was criticized for precisely this reason. Despite this, today Kraepelin's legacy endures in virtue of this distinction. While most psychiatrists in Europe continued to follow Kraepelin through the 1900s, American psychiatry shifted away from biological psychiatry toward environmental and psychoanalytical psychiatry (Decker, 341).

During World War II, soldiers presenting cases of combat neuroses provided a platform for practicing psychoanalysts. Many psychiatrists witnessed the successes of these therapeutic interventions and consequently, post-World War II saw a marked increase in the amount of candidates seeking psychoanalytic education (Decker, 342). The general belief was that psychoanalysis could alleviate most mental illness. As opposed to the biological view of mental disorder as brain disease and thus clearly distinguishable from normal mental functioning, the Freudian view of continuous mental life became predominant. In addition, the American Psychiatric Association and psychiatry in general began attending to the social bases of mental disorders. Accordingly, DSM I and II reflected a psychodynamic slant (Mayes and Horwitz, 249). The shift away from biological psychiatry to psychodynamic psychiatry after the war represented a time of optimism for the field at large. This, however, was short lived.

During the 1960s and 1970s, psychiatry was met with growing opposition and challenges. For one, the chronic state of most patients in state hospitals led to a depressing and discouraging atmosphere (Decker, 343) and with an inadequate amount of professional staff, therapeutic treatments were severely lacking. The prolonged institutionalization of psychiatric patients in combination with unsuccessful treatment brought about greater scrutiny directed toward the broader practice of institutionalization and cast doubt on the once optimistic approach of psychoanalysts and social activists. The image of American psychiatry was growing increasingly tarnished. The 'anti-psychiatry' movement began to form during the 60s, and its ideas were even supported by the psychiatrist Thomas Szasz who authored *The Myth of Mental Illness* (1961). Szasz argued that psychiatry equated non-conformist behavior with mental illness and therefore mental illness is a myth. In the year 1973, an article by the Stanford psychologist and lawyer D. L. Rosenhan was published in the journal *Science*, which provided experimental evidence in support of the ideas of Szasz shared by much of the general public. By demonstrating that individuals merely posing as real patients could gain admission to psychiatric hospitals based on a single vague description of an auditory hallucination, the study showed that psychiatrists were incapable of distinguishing between real and faked symptoms. These findings provided a tangible source with which to attack the scientific validity of psychiatric diagnoses.

At the end of that same year, the APA made a decision that seemed to confirm the implications of the Rosenhan experiment; having been pressured and protested by the Gay Liberation movement for years regarding the diagnosis of 'homosexuality' as a mental illness, the APA voted to have it removed from the DSM II (APA, 1968). This decision fueled public criticism regarding the scientific basis for DSM diagnoses; the simple fact that a 'decision' about the legitimacy or illegitimacy of a psychiatric diagnosis could be made as a result of social pressures reiterated the inadequately scientific basis for the DSM's method of diagnosis and differentiation between the mentally well and the mentally ill. American psychiatry was no longer rightfully afforded a place among other medical specialties and as an autonomous discipline it was in a state of crisis. To make practical matters worse, third-party payers began refusing to

pay psychiatrists for the treatment of illnesses not considered to be 'real diseases' (Decker, 345). Successful outcomes of treatment needed to be secured, and in the eyes of third-party payers psychotherapy was incapable of fulfilling that expectation.

In order to salvage its status as a legitimate discipline and alleviate its critical state, there was a clear historical necessity to return psychiatry to a medically-modeled, scientific psychiatry. It was around this time that a group of psychiatrists at Washington University in St. Louis (later considered the 'neo-Kraepelinians') banded together with a common goal of restoring biological psychiatry in place of psychoanalytic and environmental psychiatry (Decker, 345). In addition to the necessity at hand, new findings in psychopharmacology at the time encouraged this Kraepelinian revival. Specifically, between the 1950s and 1970s, the drugs lithium, chlorpromazine and imipramine were thought to be successful treatments for mania, schizophrenia and depression, respectively (Ghaemi). This finding was significant in that it provided a type of treatment validation for Kraepelin's nosology; a specific correlation between the administrations of these drugs to the respective disorders suggested that Kraepelin's classificatory system carried greater therapeutical relevance than Freud's neurosis-psychosis continuum. In addition, these psychopharmacological discoveries provided a convenient solution to the practical problems concerning third-party reimbursement for treatment, i.e., treatments should be for 'real *diseases.*' It was within this context that the "Kraepelinian revival" emerged and biological psychiatry became the dominant approach. With several self-identified neo-Kraepelinians on the task force for the drafting of DSM III (APA, 1980), it too was a reflection of so-called "neo-Kraepelinian" principles.

The resurgence of Kraepelinian psychiatry thus appears to have been the product of, (1) a historical "necessity" to establish a valid and reliable basis for psychiatric diagnoses and practices in the face of overwhelming public and professional criticism, as well as (2) apparent treatment validation of Kraepelin's classifications. It should be asked whether these reasons were sufficient enough to validate the revival *itself*. Though it is not the aim of this paper to offer an in-depth analysis of and answer to that question, a few remarks on the issue will suggest that perhaps they were not and that this insufficiency, in part, portends the nature of the problematic deviations of the 'neo-Kraepelinian' DSM from Kraepelin's work.

First, it is important to keep in mind the distinction between (1) the perceived *necessity* of some entity to have particular qualities in order to *achieve* some end result, and (2) the reality concerning that entity's ability to actually demonstrate or *have* those qualities. In other words, the *necessity* of 'X' having the properties of 'A' and 'B' *in order to achieve* 'C', does not validate the assertion that 'X' indeed has the properties of 'A' and 'B' and therefore achieves 'C'- no matter how dire that necessity is, no matter how important it is (for whatever reason) that 'X' achieve 'C' and no matter how evident it may appear that 'X' already achieves 'C'. This is an obvious logical distinction, but still pertinent in considering whether, when faced with an overwhelming necessity fueled by

public pressure and scrutiny, the neo-Kraepelinians properly validated their assertions that, for example, mental illness is brain disease, that it is categorical by nature or that, as suggested by the DSM III, definitive diagnoses can be made on the basis of signs and symptoms alone. If true, these properties would allow the practice of psychiatry and its use of the DSM the status of a legitimate and scientific discipline. Though this would be clearly beneficial for stakeholders and provide solutions to the problems of psychiatry, that does not *make* them true. Asserting the existence of these properties without sufficient empirical support would lead to their indemonstrability in practice.

Secondly, that the alleged specificity of the above mentioned pharmaceutics to treat mania, schizophrenia and depression was later found to be empirically weak (Healy, 849), suggests an unscientific assumption made more so on the basis of convenience to meet third-party payer's expectation of paying for the treatment of 'real diseases', rather than rigorous empirical verification. The neo-Kraepelinians relied on the assumption of agent-specificity in order to establish empirical support for their nosology. However, the agent specificity itself was not sufficiently empirically supported. Thus, it appears that neo-Kraepelinian enthusiasm regarding this apparent correlation and its ability to solve one of psychiatry's central problems took precedence over the empirical bases they initially tried to establish.

Having illustrated the context of this Kraepelinian revival, as well as some problems with the foundation of the revival itself, I move on to answering the primary question of how Kraepelinian the highly revised 'neo-Kraepelin' DSM III actually was. Specifically, this will be done by (1) an examination of the DSMs translation of Kraepelin's most well-known work in differentiating the major psychoses, followed by (2) the ontological differences between and consequences of the respective nosologies in part III.

Part II Kraepelin Classification vs. DSM III Classification: The Case of the Major Psychoses

The most borrowed aspect of Kraepelin's work by the DSM III and IV is his differentiation of the major psychoses into dementia praecox and manic-depressive insanity (APA, 1980/1994). Kraepelin described manic-depressive insanity as being characterized by mood excesses and described dementia praecox by two general 'maladies'; dissociative pathology, i.e., disorganization of thought and behavior, and avolition. But more specifically, it was dissociative pathology closely linked with avolitional pathology that distinguished dementia praecox from manic-depressive insanity (Fischer and Carpenter, 2081). The DSM III and IV maintain this nosological classification of manic-depressive insanity and dementia praecox with bipolar disorder and schizophrenia, respectively. But a comparison of the DSMs diagnostic criteria for each with Kraepelin's original construct reveals fundamental differences between the two.

First, the DSM III adopted the concept of "nuclear schizophrenia" which was

based on two hypotheses; Schneider's First Rank Symptoms (1959) which were proposed to be highly discriminating of schizophrenia, and Langfeldt's proposed distinction between true and pseudoschizophrenia (1969). As a result of incorporating this concept in the DSM III, the idea that there existed pathognomonic symptoms for schizophrenia was embraced. In particular, it held that only hallucinations and delusions or just "bizarre delusions" would satisfy criteria A for schizophrenia (APA, 1980). Kraepelin's dissociative and avolitional pathologies and the close linkage between the two was not allotted significance at all for the purposes of diagnosis. In fact, avolition was not even included as a criterion in DSM III. The intention of incorporating Schneider's hypothesis was to increase inter-rater reliability of diagnoses (Fischer and Carpenter, 2082), and to be fair, a simple deviation from Kraepelin's diagnostic criteria for these categories does little more than dampen the connection and oft referred similarity between the two. But a closer examination shows that the APA's decision to incorporate it into the DSM was based on presumed construct validity rather than empirically supported construct validity.

During the late 60s and 70s, Schneider's hypothesis which was central to the concept of nuclear schizophrenia was tested. It was found that his First Rank Symptoms were found in psychosis unrelated to schizophrenia, falsifying his hypothesis that these First Rank Symptoms were pathognomonic to schizophrenic psychosis (Fischer and Carpenter, 2083). Furthermore, none of the definitions of nuclear schizophrenia which relied on pathognomonic reality distortion predicted course and outcome, thus demonstrating poor predictive validity of the diagnostic criteria. Instead, it was found that the greatest distinction between schizophrenic and non-schizophrenic psychosis were symptoms of restricted affect, poor rapport and poor insight. Despite this empirical data that had been available before the publication of the DSM III in 1980, the validity of nuclear schizophrenia was simply presumed and incorporated into the diagnostic scheme.

This specific alteration of Kraepelin's original diagnostic criteria directly contradicts Kraepelin, who was particularly careful to avoid any claims about pathognomonic symptoms. Specifically, he held that utilizing the idea of pathognomonic symptoms in diagnostic practice is empirically untenable. (Jablensky, 384). The falsification of the hypothesis central to nuclear schizophrenia during the 60s and 70s was at once a confirmation of Kraepelin's position and a testament to the empirically inadequate methods of determining diagnostic criteria for DSM diagnoses.

The DSM alteration of Kraepelin's original criterion effectively shifted the diagnostic categories between bipolar disorder and schizophrenia much closer together (Fischer and Carpenter, 2083). By emphasizing common features and de-emphasizing the pathological attributes that Kraepelin used to distinguish the conditions, it also increased the likelihood of false positives in diagnosis. The phenomenon of psychosis occurs in myriad conditions, many of which are distinguishable at the level of etiology, i.e., sensory isolation, temporal lobe epilepsy,

Huntington's disease, and can also occur in reported instances of religious ecstasy (Fischer and Carpenter, 2081-2). It was determining the nature of the patient's distress (if any) accompanied by these episodes of psychosis that was important in distinguishing between psychotic schizophrenia and bipolar disorder. This is what Kraepelin had done by examining the pattern of pathology associated with each, and it is precisely what the DSM discarded and replaced with the concept of nuclear schizophrenia, thereby relying on the idea of pathognomonic signs.

This discrepancy between Kraepelin and the neo-Kraepelinian diagnostic formulations brings to light another significant difference, and that is Kreapelin's insistence on viewing the whole of the clinical picture in diagnostic efforts (Decker, 339). Kraepelin stated in the fifth edition of his textbook that his work is a:

> Decisive step from a symptomatic to a clinical view of insanity...The importance of external clinical signs has...been subordinated to consideration of the *conditions of origin*, the *course*, and the *terminus* which result from individual disorders. Thus, all purely symptomatic categories have disappeared from the nosology (Engstrom, 1995: 294; Kraepelin's italics).

This quote shows that Kraepelin doubted the ability of external clinical signs to sufficiently define a nosologic category. Kraepelin never even issued a *definitive* list of diagnostic criteria for dementia praecox or manic-depressive insanity but rather, advocated for the consideration of the comprehensive case at hand, including even the characteristics of the personality being invaded by the illness (Kraepelin, 2002).

In contrast, one of the defining features of the so called 'neo-Kraepelinian' DSM III and IV (APA, 1980/1994) is a purely symptomatic nosology whereby a detailed checklist of symptoms can be used as a method toward securing a diagnosis. Thus, emphasis was placed on the importance of external clinical signs, thereby failing to consider other factors such as conditions of origin, course of illness, terminus resulting from disorders, or characteristics of the individual personality being invaded by the illness. These details are important in developing 'the whole of the clinical picture' which was the rule Kraepelin taught his students (Kraepelin, 2002). Contrary to this rule, it has been argued that since the publication of DSM III in 1980, American psychiatric education has placed increasingly less emphasis on the importance of comprehensively attending to individual cases (Andreasen, 111). Instead, students are taught to memorize the symptomatic categories of the DSM nosology, thus neglecting important aspects of the condition as well as clinical signs potentially detectable through considering the characteristics of a patient's personality.

Through an evaluation of Kraepelin's enduring contribution to modern psychiatric nosology, namely the differentiation of [now] bipolar disorder and schizophrenia, it has been shown that significant alterations were made which contradict many of Kraepelin's principles, such as the empirical tenability of

pathognomonic symptoms, definitive diagnostic criteria in general, and the necessity of considering the entire clinical picture rather than a reliance on pure symptomology. The consequences of such changes have not only effected patients receiving differential diagnoses of bipolar disorder and schizophrenia by shifting the two groups much closer together, but has also lead to a narrowed focus on external clinical signs in diagnostic efforts. Neither consequence serves the best interest of patients or, arguably, aspiring clinicians. It has also been shown here that neither alteration in the DSM III diagnostic scheme would have likely been supported by Kraepelin. Despite this, these alterations are representative of the practice that the DSM has wholly embraced in its neo-Kraepelinian reformulation of the DSM. The following section addresses this expansion in more detail and considers the ontological commitments of both Kraepelin and the DSM III.

Part III From Kraepelin to the DSM III: From Disease-entities to Disorders and Syndromes

Emil Kraepelin began by classifying as many cases of mental disorder as possible based on biological etiology, for example those which were due to infection or endocrine disorders (Klerman, 100). Such a classification can thus be regarded as consisting of disease-entities. However, the differentiation for which he is most well-known- dementia praecox and manic-depressive insanity- was made in the absence of any discovered pathophysiological explanation. It was based on a *presumed* biological etiology instead. Thus, Kraepelin was unable to demonstrate that Dementia Praecox and manic depressive insanity were actual disease-entities. Consequently, the division was often criticized (Kraepelin, 1919).

As opposed to Kraepelin, the neo-Kraepelinian revolution of the DSM forfeited *any* ontological commitment from the start but, nonetheless, expanded Kraepelin's nosology beyond the major psychoses to eventually include over 400 diagnoses including affective disorders (non-psychotic unipolar major depressive disorder being frequently diagnosed), anxiety disorders (GAD, panic, social anxiety, OCD), personality disorders, and other conditions such as ADHD and PTSD (APA, 1980/1994). These changes characterize the shift from Kraepelinian disease-entities to the DSM's syndromes and disorders. A legitimate assertion that certain mental illnesses are disease-entities would require a demonstration that such entities have neuropathological or other biologically-based causal mechanisms or factors. As a result, valid categorizations could be made if these demonstrations showed that they are discrete entities with natural biological boundaries that separate them from other disorders (Kendell and Jablensky, 7). The DSM decision to remain ontologically neutral resulted in the shift from disease-entities to disorders and syndromes. The latter categorize mental illness solely on the basis of signs and symptoms, ones that are frequently observed to cluster or intercorrelate. This "atheoretical" character of the DSM III and IV means that the diagnostic categories do not (explicitly) involve or

presuppose any particular theory of etiology or pathology (APA, 1994). Given the normally assumed connection between theories and ontology, in virtue of atheoreticity, clinically defined and theoretical DSM categories are applicable to a variety of ontological positions held by clinicians and researchers in the field of psychiatry.

One problem with the DSM's neo-Kraepelinian divergence described above occurs in practice, when syndromes and disorders are treated as proxies for diseases. It has been argued that this assumption is the result of a reification fallacy that occurs merely as a result of giving diagnostic concepts an official nomenclature and precise operational definitions (Kendell and Jablensky, 5). Reification in this sense occurs when DSM-defined diagnoses come into general use and begin to be perceived and utilized as if they are actual entities that can be unquestionably invoked to *explain* the patient's symptoms. However, since most diagnostic concepts defined by their syndromes have not been shown to have natural boundaries separating them from other disorders, they should not be regarded as valid (Kendell and Jablensky, 5). Furthermore, it is argued by Ghaemi that symptoms 'hypertrophied' into diagnoses appear to provide justification for medication, since clinicians often make biological assumptions about treatment. Thus, he argues that the neo-Kraepelinian attempt to stay neutral ontologically is, in practice, trumped by biological assumptions regarding treatment. Assuming Ghaemi's ideas to be correct, elaboration on his claims would be helpful in understanding more specifically *why* DSM diagnoses appear to provide justification for treatment with medication and *why* clinicians tend to make biological assumptions about treatment.

If reification of DSM diagnostic concepts can occur without any resultant specificity in regards to the *treatment* of conditions falling under such concepts, and if Ghaemi's argument is correct, then there must be a secondary assumption contributing to the truth of his claim. I side with Ghaemi, and thus posit that there is a secondary assumption at work, namely, the assumption that mental illness is brain disease. This assumption can easily be derived from the denial of dualism stated in the DSM itself;

> "the term mental disorder unfortunately implies a distinction between 'mental' disorders and 'physical' disorders that is a reductionist anachronism of mind/body dualism...There is much 'physical' in 'mental' disorders and much 'mental' in 'physical' disorders...unfortunately the term [mental] persists in the title of DSM-IV because we have not found an appropriate substitute" (APA, 1994).

What this statement amounts to is the conclusion that there is no significant distinction between the mind and the brain for the purposes of psychiatry. If there is no significant distinction here, then anything regarded as 'mental' is conceptually reducible to the physical and vice versa. Therefore it can be said that 'mental' illness is really not different in any significant way from 'physical' illness, i.e., brain illness or brain disease.

It is generally regarded as a metaphysically extravagant notion to assert a fundamental distinction between the mind and the brain, and as such it is commonly rejected in scientific communities. Psychiatry, although dealing explicitly with 'mental' activity, e.g., consciousness, subjectivity and intentionality, nonetheless reject dualism. A common misconception which may contribute to this assumption is that psychiatry has proven dualism to be false (Cooper, 104-105). To elaborate, it is not incompatible with a dualist position to accept that neuroscientific research has shown correlations between properties of the brain and subjective experiences. For example, a dualist can accept that a brain scan gives good reasons to think someone is in pain by offering a reliable marker for pain, rather than offering evidence of the pain itself. Similarly, it is compatible with a dualist position that in certain cases medications reliably bring about changes in mood. Therefore these reasons are not reasons to reject dualism.

It is not my aim here to offer a defense dualism, but rather to draw attention to some significant implications that accompany a clear denial of it. For one, it seems to suggest a false dichotomy; if the mind is not the brain, then the mind is a mystical, inexplicable entity that does not belong to the realm of science. This is a false dichotomy because the idea of the mind as something fundamentally distinct from the brain is not incompatible with neuroscientific findings. Additionally, this false dichotomy excludes from the outset any alternative theories of mind, such that it is an information processor or a process itself rather than a 'thing' reducible to other physical 'things.' A priori exclusions such as these are not only based on misconceptions of dualism but also stymy potential developments in psychiatry that could not only be consistent with a scientific model but potentially provide a revolutionary way of understanding mental activity.

The considerations discussed above help to elucidate Ghaemi's claim that the neo-Kraepelinian neutral ontology contributes to diagnostic confusion since clinicians often make biological assumptions concerning treatment. In other words, the difficulty in the neo-Kraepelinian attempt to remain ontologically neutral becomes apparent when considering the context in which that supposed neutrality is immersed, i.e., one in which psychiatry is viewed as a definitively scientific discipline, misconceptions of dualism lead to the exclusion of potentially viable theories of mind and thus the acceptance of mental illness as nothing other than brain disease prevails. Thus it seems to be a reification phenomenon occurring *within this context* that appears to justify treatment *with medication* and that explains, at least in part, *why* clinicians tend to make biological assumptions regarding treatment for DSM diagnoses. Consequently, there appears to be at least some degree of incompatibility between the supposed ontological neutrality of the DSM and an unquestioned denial of dualism, because the latter implies a biological theoreticity in the form of the dictum 'mental illness is brain disease', rather than theoretical neutrality. One cannot logically claim neutrality with respect to the ontology of mental illness while also asserting (directly or indirectly) that mental illness is nothing other than brain disease.

The result, as Ghaemi argues, is the justification of medication for the treatment of DSM-defined disorders. For, if mental illness is brain disease, and if brain diseases are treated with medication, then it should be sufficient to treat mental illnesses with medication. Ghaemi provides an example of this phenomenon using the rather new diagnosis of Adult ADHD. This condition, not even recognized in the psychiatric literature until the 1990s, carried little evidence of nosological validity. While the National Comorbidity Survey analysis found that 3% of the adult population met criteria for the condition, 84.1% of this population were also diagnosable with mood disorders. While these statistics suggest a lack of syndromal specificity and diagnostic invalidity, there was a significant increase in the diagnosis of adult ADHD in 2002 after a new drug had been marketed in the U.S. to treat its symptoms.

This example, in addition to showing that DSM diagnoses appear to provide justification for treatment with medication, also shows that the apparent specificity of a drug to treat the symptoms of a condition outweighs other evidence that suggests the nosologic invalidity of the condition itself. If there were strong empirical demonstrability that specific drugs worked for specific conditions, then this would provide some empirical justification for diagnostic categories. However this is not the case, as seen with problems of comorbidity and the lack of specificity of most current psychopharmacological treatments. Aragona argues that in cases such as these, the problem is rooted in the heterogeneity of DSM diagnostic categories, and not the drugs themselves (5). To elaborate, the testing of psychopharmacological drugs becomes problematic in that it relies on the formal DSM-diagnoses given to a group of test subjects. Thus, the empirical performance of the drugs is reliant on the rules which contributed to the conceptual construction of those diagnostic categories. Problems with the nature of these constructions (lack of specificity of symptoms, lack of qualitative determination and use of a polythetic rule coupled with a quantitative diagnostic threshold) allow for patients who have received the same formal diagnosis to retain many significant differences. These differences become uncontrolled variables in experiments that aim to test the specificity of certain drugs to treat certain DSM diagnoses. From an understanding of this problem, it follows that positive correlations between drug treatments and alleviation of symptoms provide only weak empirical validation, i.e., treatment validation of DSM diagnostic categories. In this sense, treatment validation of diagnostic categories effectually masks problems of heterogeneity and encourages the assumption that DSM-defined disorders and syndromes are valid, as seen with the increased diagnoses of Adult ADHD concurrent with a new drug marketed for it.

This interplay of DSM diagnoses and the medications purported to treat them has a second and perhaps more obvious consequence; treatment that is designed to eliminate only symptoms. Since psychiatric conditions are defined by their signs and symptoms, there are no objective tests (standard methods objectively applicable to any

patient which exclude the subjectivity of relying on verbal expression and interpretation) with which to validate suspected diagnoses. This is perhaps the primary and most consequentially detrimental way in which psychiatry deviates from other medical disciplines.

Consider, for example, an Endocrinologist seeing a patient who is suspected of having diabetes mellitus. The physician might suspect this diagnosis based on patient-reported symptoms of fatigue, excess thirst and blurry vision. The physician may also find high levels of sugar in a urine analysis. These symptoms, though commonly seen in patients with diabetes, could manifest in an array of medical conditions and do not represent the variation of symptoms that can be present. In no way do they, by themselves, indicate the presence of a particular medical condition. It is the physician's job to determine the possibilities and the most probable conditions that would be the cause of the signs and symptoms. Part of this process involves considering the *individual* case at hand. For example, the physician might find that although the patient is a normal weight (while obesity is a common associated condition), they practice risk behaviors for developing diabetes such as excess alcohol consumption. All such details still do not confirm a medical diagnosis of diabetes. Instead, signs and symptoms as well as a consideration of the individual patient, direct the physician to order particular objective tests, e.g., specific blood tests, in order to confirm or rule out the suspected diagnoses. Once this is done, medication is justifiably prescribed to correct the underlying biological process. In this case it would most likely be insulin to correct the insulin deficiency which caused the condition and the resulting symptoms to begin with. Therefore symptoms are alleviated by virtue of the medications that target the underlying pathophysiological processes that caused them in the first place.

In contrast, modern diagnostic practices based on the DSM not only bypass the individual and environmental factors that could indicate a particular condition (like obesity or alcoholism in the case of diabetes) but also makes definitive diagnoses on the symptoms alone. As discussed, such diagnoses are often followed by unjustified prescriptions of medications. They are unjustified because their employment alone presumes a biological cause correctable with a specific type of medication without there being a known biological cause. The consequence is that only outward symptoms are treated, masking and allowing any underlying mechanism to continue. The idea of any other medical discipline practicing this way is nearly unimaginable. In the absence of objective tests which validate suspected diagnoses by detecting underlying biological causes, psychiatric diagnoses are subjectively made on the basis of interpreted signs and symptoms alone.

Part IV What should be done?

In 1887 Kraepelin lectured that "Unlike other branches of medicine, psychiatry

has to do with two fundamentally different categories of phenomena...The impossibility of a satisfactory solution to the fundamental psycho-physical problem inherent to psychiatry has had two consequences...numerous attempts to bridge the gap separating events of the body and mind by means of airy constructions of speculative fantasy...[and]...a strict and resigned determination to focus only on establishing what is real" (Kraepelin, 1887/2005: 351).
In an attempt to avoid the former, the neo-Kraepelinians aimed to establish the latter, but failed due to an inherently flawed approach that focused solely on symptoms. Though Kraepelin relied in part on symptoms for his differentiation of dementia praecox and manic-depressive insanity, he acknowledged the limitations of doing so, and toward the end of his career even admitted that "we must, then, accustom ourselves to the idea that the phenomena of illness which he have hitherto used are not sufficient to enable us to distinguish reliably between manic-depressive illness and schizophrenia in all cases" (1920/1974: 29). Had sufficient evidence supported Kraepelin's belief that the ultimate validation of this differentiation would come from neuropathology, physiology and biological chemistry of the brain (Jablensky, 383), then it would serve as support for his nosology, providing a biological explanation that went beyond symptoms. Similarly, many of the issues discussed in this paper concerning the neo-Kraepelinian DSM would not be existent or relevant if sufficient empirical evidence supported the delineation of the myriad DSM-defined conditions. However as of today, only 3% of psychiatric conditions have been causally established (Stevenson). Therefore it is likely that for the other 97%, Kraepelin would have denied reliability of differentiating them for the purposes of diagnosis, just as he did regarding his own work.

Kraepelin held the belief that scientific knowledge comes only through empirical research. Similarly, the neo-Kraepelinians believed that only empirical psychiatric research with a focus on biology held any hope for the improvement of psychiatric practice (Decker, 339). Kraepelin attempted to establish the applicability of a medical model to psychiatry, and the neo-Kraepelinians assert that psychiatry *is* a medical discipline adhering to a medical model (Klerman, 104). If the goal is to ground the practice of psychiatry on valid and reliable bases comparable to other medical disciplines, then the data acquired and the diagnoses made must be validated by objective tests.

In response to the fifth and latest edition of the DSM published in May of 2013, the National Institute of Mental Health has proposed a new project, Research Domain Criteria (RDoC), which rejects the use of DSM categories from the outset, since doing so simply presumes DSM classifications to be accurate reflections of the reality of mental illness, thereby excluding any objective findings which are not consistent with DSM categories (Insel). The RDoC project will begin by "collecting the genetic, imaging, physiologic, and cognitive data to see how it all clusters – not just the symptoms – and how these clusters relate to treatment response..." (Insel).

A significant shift away from the current DSM categories is necessary in order to make claims about reliability and validity. There is no doubt that the above approach would provide an objective source of information geared toward identifying biological causal processes of mental illnesses. However, it would be a mistake to assert that such a model would have the ability to address the *whole of clinical psychiatry,* since doing so would presume that *all* psychiatric symptoms, conditions and cases are merely biological. This presumption would dismiss the unique feature of psychiatry that even Kraepelin acknowledged in his statement "...Psychiatry has to do with two fundamentally different categories of phenomena..." (1887/2005: 351) by assuming the reducibility of all mental activity to physical activity. This is why caution regarding extrapolations of the project's findings to the entire realm of mental life and illness would be important in order to avoid prematurely assuming a fundamentally biological basis to every individual psychiatric case from the simple fact that *some* conditions have been found to have biological bases. This is similar to the misconception occurring in the rejection of dualism; since neuroscience has identified correlations between brain activity and subjective states, all subjective states must be correlated with or caused by brain activity. Instead, a system like this should be used as an objective *guide* for clinicians, giving them a better idea of what diagnoses are likely biological disease-entities and which are not, and therefore better equipping them to make decisions regarding which type of treatment is prima facie appropriate for individual cases.

Conclusion

The Kraepelinian revival characterizing modern psychiatry began on a shaky, unscientific foundation. With social pressures mounting and threatening the discipline of psychiatry as whole, there was a historical necessity to establish its scientific validity and reliability. Overzealously made correlations between discoveries of drugs and their ability to specifically treat certain disorders provided only a weak empirical basis for using and expanding a Kraepelinian nosology. The latter of these factors which contributed to its revival foreshadowed the practice that has consumed modern psychiatry, namely the use of prescription medications for treatment of symptoms and empirically weak treatment validations of DSM diagnostic concepts.

The actual implementation of Kraepelin's work in the neo-Kraepelinian DSM III and IV differed from Kraepelin in crucial ways which exacerbated the reasons for the criticism of psychiatry to begin with, i.e., low reliability and low validity, or otherwise high reliability at the expense of validity and thus widespread and consistently inaccurate diagnoses. The reification of DSM diagnostic concepts in conjunction with the ontological shift from disease-entities to syndromes and disorders lead to the justification and unquestioned treatment of these reified concepts with medication. The fact that diagnoses and respective treatments address only superficial symptoms contradicts the neo-Kraepelinian claim that psychiatry practices as a legitimate

medical discipline, and persists in the name of Kraepelin despite his acknowledgment of the inadequacy of such approaches.

As a result, once again the discipline of psychiatry has found itself in a state of continued crisis. The recent research proposal by the National Institute of Mental Health moves psychiatric research in a more promising direction by liberating itself from the constraints of DSM categories. By endeavoring to gather through neuroscience and genetics a collection of objective data, the RDoC more accurately follows Kraepelinian goals than have the neo-Kraepelinians in their reliance on symptoms. Nonetheless, the nature of psychiatry demands that a method which gathers objective, biological data comprises only a partial picture of it, not to be viewed as sufficiently holistic. Doing so would unjustifiably exclude other potentially viable perspectives. While science provides an avenue for the search of empirically tenable, reliable and valid *aspects* of psychiatry, there is no justification in assuming that these aspects comprise a complete understanding of the complexities of the human mind.

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