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A Note on the Dynamics of Psychiatric Classification

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Abstract

The question of how psychiatric classifications are made up and to what they refer has attracted the attention of philosophers in recent years. In this paper, I review the claims of authors who discuss psychiatric classification in terms referring both to the philosophical tradition of natural kinds and to the sociological tradition of social constructionism—especially those of Ian Hacking and his critics. I examine both the ontological and the social aspects of what it means for something to be a mental disorder, and how the ontological status of these disorders hinges on social causation. Finally, I conclude by suggesting a way in which the biological and the social may be reconciled in an integrative model of variation in psychiatric disorder.

Introduction

A clear definition of mental disorder is of both conceptual and practical importance. On the one hand, precision will better ensure that we are talking about the same thing when we use the concept of mental disorder. On the other hand, a rigorous definition will help diagnosticians, particularly where it is unclear whether a condition is pathological or not.

Consider homosexuality. The first edition of the American Psychiatric Association's *Diagnostic and Statistical Manual of Mental Disorders*, the DSM-I (APA, 1952) classified homosexuality as a sociopathic personality disturbance. Despite controversy, justification for its maintenance in the DSM-II (APA, 1968) was found in a large-scale psychoanalytic study of homosexuality, performed over ten years, which concluded that homosexuality was a pathological fear of the opposite sex caused by traumatic parent–child relationships. This pathologizing of homosexuality endured until a diagnosis of *sexual orientation disturbance* was introduced in the seventh printing of the DSM-II (APA, 1974) to classify the experience of significant discontent with the biological sex one was born with, a condition presently referred to as gender identity disorder. The publication of the DSM-III-R (APA, 1987) saw the disappearance of sexual orientation disturbance, and also of *ego-dystonic sexual orientation*, the latter of which was meant to classify the condition wherein one's sexual orientation is at odds with one's idealized self-image.

The empty space left by the abandonment of ego-dystonic sexual orientation was filled with a residual category, *sexual disorder not otherwise specified*. As Nelson Goodman (1972) once wrote, the wastebasket seems to have been made an integral part of the filing system. If someone presents symptoms that justify a diagnosis of sexual disorder which, in turn, fails to meet the criteria for any of the sexual disorders that have been so far classified, that person is diagnosed as presenting a sexual disorder not otherwise specified. This is understandable, since most psychiatrists working today diagnose disorders looking for symptoms, not causal mechanisms (van Os et al., 2013). Furthermore, psychotherapy that is useful for patients presenting properly classified sexual disorders may be useful for patients presenting those of an unclassified kind. A not otherwise specified (NOS) diagnosis is the next best thing to precision. It is provisional and it is not meant to suggest that a NOS disorder shares the same status of a properly classified disorder. Nevertheless, the very presence of NOS diagnoses may raise suspicions as to the scientific legitimacy of psychiatric nosology (Zachar, 2012).

Examples of prejudice cloaked in pseudoscience are regrettably common in the history of psychiatry. Indeed, they are common enough to intimate skepticism regarding the whole category of mental disorder, a denial embodied in the anti-psychiatry movement that still resonates today (Pickard, 2009). Thomas Szasz, one of its founders, wrote *The Myth of Mental Illness* in 1961, claiming that 'mental disorder' is a mere social construct manufactured by psychiatrists, and that the term can at best be used as a metaphor, given that a proper disorder must be an objectively demonstrable biological pathology. The anti-psychiatry movement's main theoretical tenet is that the definitions and criteria for many, if not most psychiatric diagnoses are vague and arbitrary, failing to meet basic scientific standards.

Since the publication of Szasz's book, however, biological psychiatry has seen many developments. Chemical treatment is widely successful for a large number of disorders such as seasonal affective disorder, clinical depression, bipolar disorder, schizophrenia and obsessive-compulsive disorder. This implies that we have acquired at least some understanding of mental disorders in terms of their chemical effects on the nervous system. Also, and perhaps as important for the scope of this investigation, a definition of mental

disorder was added to the introduction to the DSM-III (APA, 1980) and was modified for the first time since with the publication of the DSM-5:

> A mental disorder is a syndrome characterized by clinically significant disturbance in an individual's cognition, emotion regulation, or behaviour that reflects a dysfunction in the psychological, biological, or developmental processes underlying mental functioning. Mental disorders are *usually* associated with significant distress or disability in social, occupational, or other important activities. (APA, 2013, p. 20, my emphasis)

Unfortunately, there is a manifest vagueness in this definition, and hence in psychiatry's current demarcation of mental disorders, that renders its utility very limited both to theoretical and clinical use. The ambiguity is best detected in the statements that behind the symptoms of a mental disorder there is a 'psychological, biological, or developmental' dysfunction, and that 'mental disorders are usually associated with significant stress', etc. There is no attempt to offer sufficient, let alone necessary, conditions for a condition to be a mental disorder. The vagueness in the DSM's present definition betrays a deeper ambiguity about how psychiatry is conceived and practiced, signalling an old methodological divide between mutually suspicious ways of understanding mental disorder. We will see that the existence of this divide is not gratuitous and, most importantly, that there are reasons to think that it reflects something about the nature of mental disorders themselves, namely, that they are neither exclusively biological, nor exclusively social, phenomena.

Models of Psychiatry

The roots of psychopathology are the subject of the ideological traditions that usually divide psychiatrists into those who favor somatic treatment and those who favor psychotherapy. Each of these orientations stands on a theoretical assumption about the nature of mental disorder. Partisans of what has been called the *biomedical model* (Luhrmann, 2000) look to the brain and its dysfunctions, and employ neuroscience, biochemistry and pharmacology to resolve them. One of its champions, Samuel Guze, defines it as using in psychiatry the concepts and strategies that have evolved in general medicine (1992, p. 129). This approach has achieved scientific success through the increasing understanding of the biological bases of at least some psychiatric conditions and, as a consequence, it has enjoyed institutional

success through widespread prevalence in hospitals and research departments, while also being championed in theory (Murphy, 2009).

The *psychodynamic model*, in turn, treats mental disorder not as the product of a diseased brain but as a malfunction of the *person*. Among its proponents, some deny the pathological status of mental disorder altogether, but most are primarily concerned with how psychopathology is conceived and treated. As critics of the biomedical model, proponents of the psychodynamic model see mental disorders as a response to social and cultural forces, at least to some degree. The successes of dynamic psychiatry lie in the contribution of psychotherapeutic sensibility. Without it, it has been argued, patients 'do less well, are readmitted more quickly, diagnosed more inaccurately, and medicated more randomly' (Luhrmann 2000, p. 262).

While there have been efforts to synthesize these models and consequently achieve an ampler picture of the mind and its problems, it is arguable that the suppression of the psychodynamic by the biomedical model is possibly currently underway (Haslam, 2000). This suppression, however, stands in direct contradiction to the ideas and practices of those who first championed the understanding of mental disorders from an organic basis. The 19th century Swiss psychiatrist Eugen Bleuler provides one of the best illustrations of this point (Ellenberger, 1970). While committed to the organic basis of mental disorders, Bleuler dedicated to establishing personal and social relationships with schizophrenic patients. 'At a certain stage in his career, he lived with them night and day; visitors to the Burghölzli psychiatric hospital were amazed at the ways in which profoundly psychotic patients were able to live in consequence of Bleuler's care. He believed in organic psychiatry, but practiced dynamic psychiatry' (Hacking 1999, p. 118).

The explanatory potential of each of these models can be more easily grasped by looking at specific cases. Schizophrenia, for example, is widely believed to lend itself to a neuroscientific explanation. Overactivity in dopamine systems, a discernible neurological dysfunction, is currently the prevalent hypothesis to explain it (Swerdlow and Koob, 1987). In keeping with this, Shitij Kapur (2003) has recently claimed that an increased release of

dopamine is responsible for contributing to the obsessive focus of subjects on their delusional thoughts. Another competing, although not contradictory, hypothesis based on a set of clinical, neuropathological and genetic findings, points to a hypofunction of glutamatergic signaling as its root cause (Lisman et al., 2008). There is little if any place for social causation in such models. Whatever pressures may affect the schizophrenic patient, they are not taken by most researchers to contribute to its explanation.

In contrast, the psychodynamic or psychosocial approach fares better in the explanation of pathologies such as eating disorders, in the formation of which social forces are undeniably present. As severe as they can be, anorexia nervosa and bulimia are not necessarily accompanied by a single, discernible neurological condition (Hasan and Hasan, 2011). Nevertheless, even if we were to find out they were invariably a result of decreased serotonin activity, this would not necessarily rule out a possible role for social pressures in the causation of such neurological disturbances.

Natural Kinds

One particularly enlightening characterization of the biomedical model's stance towards mental disorder is due to Horacio Fábrega, who observes that it postulates entities that show common features regardless of the person's unique characteristics. These have specific causes, manifestations, and courses and ultimately entail natural objects and natural processes (1997, p. 133). In other words, biomedical psychiatry seems to conceive of mental disorders as *natural kinds* (Luhrmann, 2000, p. 44).

The notion of a natural kind grows out of a long philosophical tradition that accepts that at least some categories are real, in the sense that they 'carve nature at its joints' and are therefore totally independent of human construction. Hence, a natural kind is a grouping or ordering of things that is natural, in opposition to artificial, which implies that these kinds are sharply bounded by the underlying defining properties, or essences, of their objects. Likely candidates for the title of natural kind are chemical elements, elementary physical particles and, arguably, biological species (Ereshefsky 2009).

Diseases are often included among examples of natural kinds. One of Hilary Putnam's favored examples was multiple sclerosis: 'There are objective laws obeyed by multiple sclerosis, by gold, by horses, by electricity; and what it is rational to include in these classes will depend on what those laws turn out to be' (1983, p. 71). Thus his reason for alluding to multiple sclerosis stems from thinking that one of the characteristics that makes something a natural kind is its role in a systematic web of natural laws, and arguably multiple sclerosis fulfills such a role. The law-like regularity of natural kinds and their importance for scientific discourse has also been emphasized by Carl Gustav Hempel:

> Broadly speaking, the vocabulary of science has two basic functions: first, to permit an adequate description of the things and events that are the object of scientific investigation; second, to permit the establishment of general laws or theories by means of which particular events may be explained and predicted and thus scientifically understood; for to understand a phenomenon scientifically is to show that it occurs in accordance with general laws or theoretical principles. (1965/1994, p. 317)

In turn, biomedical psychiatry may be seen as claiming that mental disorders are brain diseases and, therefore, psychiatric conditions (such as schizophrenia) and neurological conditions (such as multiple sclerosis) should be dealt with in the same way. The acceptance that both are natural kinds, in turn, gives rise to the idea that there are *natural kinds of people*, such as the autistic, the schizophrenic, or the clinically depressed. However, in emulating the natural sciences and taking human kinds to be natural kinds, biomedical psychiatry arguably discounts an important aspect in which human classifications differ from 'natural' ones.

While Guze and other proponents of the biomedical model are willing to accept the existence of social forces in the production of mental disorders, they adhere to the view that the brain is 'at the hub of psychiatric thinking' (1992, p. 59). This seems to imply a metaphysical hierarchy in which the organic element has priority over everything else. In so devising such a hierarchy, and in sticking with a natural kind approach to mental disorder, biomedical psychiatry seems at first sight utterly irreconcilable with psychosocial views of disorder. At the opposite side of the spectrum, Peter Sedgwick argues not that mental disorder, but that *disease as whole,* is a social construction.

> All departments of nature below the level of mankind are exempt from both disease and human classifications of disease and treatment—until man intervenes with his own human classifications of disease and treatment. The blight that strikes at corn or at potatoes is a human invention, for if man wished to cultivate parasites (rather than potatoes or corn) there would be no 'blight', but simply the necessary foddering of the parasite crop. Animals do not have diseases either, prior to the presence of man in a meaningful relation with them ... Outside the significances that man voluntarily attaches to certain conditions, *there are no illnesses or diseases in nature* ... The fracture of the septuagenarian's femur has, within the world of nature, no more significance than the snapping of an autumn leaf from its twig: and the invasion of a human organism by cholera-germs carries with it no more the stamp of 'illness' than does the souring of milk by other bacteria. (1973, pp. 30–31, my emphasis)

The moral advanced by Sedgwick is that to be ill is a description or signification we collectively attach to certain conditions and that, as such, it invariably involves value judgments. 'Disorder', 'illness' and 'disease' are, in this view, concepts like 'pest', 'weed' or 'vermin'. They are open-ended and sensitive to human interests, so whether something counts as a disorder or a pest may change as human interests change, and this allows the class to grow or diminish over time (Zachar, 2000). The social process of working out such categories does not make scientific investigation of a species of pest into a normative endeavor (Murphy, 2006, pp. 98–99). Therefore, 'disorder' is strikingly dissimilar to natural kind terms.

As Hacking notes, one of the defects of the social construction talk exemplified by Sedgwick is that it suggests a one-way street. Rather than subscribing to either the natural kinds or the social construction programs, however, when confronted with a mental disorder we want to be able to say that there are at least some real, mind-independent, biological identifiable pathologies and, moreover, that, as a human kind, the labels we attach to those disorders *interact* with its conscious subjects. We don't think of mental disorder as a one-way, but as a two-way street, or rather, 'a labyrinth of interlocking alleys' (Hacking 1999, p. 116).

Human Kinds

Since the publication of his essay 'Making Up People' in 1986, Ian Hacking has been alluding to the 'old and powerful idea that we acquire knowledge of humanity by replacing

human kinds by physiological or mechanical or neuroelectrical or biomechanical ones' (1995, p. 353). He contends, however, that human kinds, unlike natural kinds, exhibit complex interactive relations—between reality, classifications, and things classified—which ultimately frustrate such attempts at replacement.

> 'Interactive' is a new concept that applies not to people but classifications … that can influence what is classified. … We are especially concerned with classifications that, when known by people or those around them, and put to work in institutions, change the ways in which individuals experience themselves—and may even lead people to evolve their feelings and behavior in part because they are so classified. (1999, pp. 103–104)

This classificatory feedback results in what Hacking has called *the looping effect*. Human classifications change people because people can become aware that they are so classified, and people change classifications because people can adapt or adopt ways of living so as to fit or get away from the very classification that may be applied to them. Because human subjects are not stationary, but *moving targets* of classification, what was known about people of a kind may become false because people of that kind have changed in virtue of what they believe about themselves (Hacking, 1999, p. 34). However, Hacking's argument that human kinds are not natural kinds is based not on the fact that this feedback occurs, but that it occurs in a particular way: looping effects are specific to human science classifications because it requires the reaction of conscious subjects. Much of his discussion has been developed in the context of psychiatry. In *Rewriting the Soul*, he pursued his chief example: the invention and development of the category of multiple personality disorder:

> We tend to behave in ways that are expected of us, especially by authority figures—doctors, for example. Some physicians had [individuals with multiple personalities] among their patients in the 1840s, but their picture of the disorder was very different from the one that is common in the 1990s. The doctors' vision was different because … the doctors' expectations were different. … People classified in a certain way tend to conform to or grow into the ways that they are described; but they also evolve in their own ways, so that the classifications and descriptions have to be constantly revised. Multiple personality is an almost too perfect illustration of this feedback effect. (1995b, p. 21)

In response to the objection that a large subset of people to which his theory references are

not fully aware of how they are classified, Hacking (1999, p. 115) points out that looping effects are pervasive: interaction is not limited to the effects on the expectations and behavior of those classified—such as classifying a subject as suffering from mild cognitive impairment, and informing them of the diagnosis—but it also includes effects on the expectations and behavior of those with whom classified individuals interact. One dramatic instance of the effects of psychiatric classifications on people other than those classified was prevalent during the predominance of psychoanalysis in American psychiatry: in the 1960s, the term *refrigerator mother* was coined by Bruno Bettelheim (1967), who advanced a theory, drawing from Leo Kanner (1943), that singled out lack of maternal warmth during child development as a cause of autism and schizophrenia.

Hacking's notion of *interactive kinds* is used in contrast with that of *indifferent kinds*—his name for the equivalent 'natural kinds', a term he resists to avoid the many different senses of this term in the history of philosophy (Hacking, 1991). 'Indifferent' has the advantage of capturing the distinguishing feature of natural kinds he wants to emphasize, namely, that the subjects of these classifications do not interact with them, but are indifferent to how they are classified: 'The classification 'quark' is indifferent in the sense that calling a quark a quark makes no difference to the quark' (1999, p. 105).

In Hacking's terms, then, while 'plutonium' would be an example of an indifferent kind, 'Zulu' would be an example of an interactive kind. There is hardly any space for discussing the fact that the former is natural and the latter artificial. The particular subset of human classifications with which we are engaged, however, seems to blur the boundaries between natural and artificial. What kind of kind would mental disorders be in Hacking's conceptual framework? Hacking's attempt at a reconciliation of the biological and the social dimensions in the explanation of psychiatric classifications results in the composite notion of *interactive and indifferent kinds*. His semantic resolution to our question sets the agenda for the remainder of the present investigation.

Interactive and Indifferent Kinds

How can a kind be both interactive and indifferent, constructed and yet natural? To

understand Hacking's plan of action for bridging the gap between organic dysfunction and social causation, its use of the work of Saul Kripke (1980) and Putnam (1975) must be grasped. Kripke-Putnam semantics envisages the meaning of a term as a *vector*, a finite sequence of descriptive elements, not unlike a dictionary entry. Rachel Cooper (2005, p. 65) notes that these vectors are made up of [1] syntactic markers, [2] semantic markers, [3] a stereotype, and [4] the extension. First there is [1] a part of speech and [2] a general category of the object signified by the word. Together, Cooper observes, the syntactic and semantic markers tell us what kind of a word the word is. For example, 'water' is a [1] mass noun and [2] natural kind term. Then there is [3] a set of typical descriptions of the term, standard examples of its use, and common associations, all of which are subject to change as opinions about the kind vary across time. Thus, the stereotype is that which any competent speaker must know in order to be said to understand the word. For example, in the case of 'water', that it is [3] tasteless, colorless, odorless, used for washing, present in oceans, etc. Finally, there is [4] the class of things to which the term applies, the actual referent of the word, if it indeed has any reference. In the case of 'water' the extension is [4] every single sample of H_2O.

Hacking suggests that we understand the meaning of 'schizophrenia' and 'autism' as Kripke-Putnam vectors. The extension of such psychiatric kinds is the neuropathology that underlies them, such as overactivity in dopamine systems in the case of schizophrenia (if the dopamine hypothesis is correct). The stereotype, however, must include 'the current idea of autism—prototypes, theories, hypotheses, therapies, attitudes, the lot' (1999, 121). This is how social construction finds its way into his account. Hacking claims that, in this way, an author of a hypothetical paper titled 'The Social Construction of Childhood Autism' could maintain that:

> (a) there is probably a definite unknown neuropathology *P* that is the cause of the prototypical and most other examples of what we now call childhood autism; (b) the idea of childhood autism is a social construct that interacts not only with therapists and psychiatrists in their treatments, but also interacts with autistic children themselves, who find the current mode of being autistic a way for themselves to be. (1999, p. 121)

Thus, Hacking believes that the question of whether mental disorders are real or constructed is made dispensable, and that it should be replaced by the technical question 'How to develop a plausible semantics for natural kind terms?' If he is right, then the product of his resolution, the notion of interactive and indifferent kinds, would give us means to successfully be able to talk about interactive kinds that 'pick out genuine causal properties, biological kinds, which, like all indifferent kinds, are unaffected, as kinds, by what we know about them' (1999, p. 123).

Since the publication of *The Social Construction of What?*, however, Hacking's theory has been the subject of a number of objections. In what follows, I will present two of them, which point to conceptual difficulties and question the strength of his arguments, respectively. As Hacking's recent writings indirectly respond to objections, I will also assess how Hacking's recent retractions converge with further criticism concerning the adequacy of his semantic approach to, finally, examine suggestions of how his core insights may be adapted into a productive framework for psychiatric thought.

Criticisms of Hacking's Account

Jonathan Tsou (2007) recently voiced the question of whether Hacking is entitled to maintain that a classification such as 'autism' can be *both* interactive and indifferent. An answer to this question turns on how Hacking presents the interactive-indifferent distinction, which, as we have seen, he does by positing that [1] interactive kinds have looping effects, while indifferent kinds do not (1999, pp. 103–8). Yet we have also seen Hacking contend that [2] there are some human kinds that are both indifferent kinds and interactive kinds (1999, pp. 115–120). Tsou notes that Hacking cannot, without contradicting himself, propose to bring together interaction and indifference, since, in Hacking's own terms, looping exhaustively distinguishes them. Thus, if Hacking's 'interactive and indifferent kinds' exist at all, he is wrong in that looping cannot be the basis of the interactive-indifferent distinction. The moral derived by Tsou is that 'interactive and indifferent kinds cannot be articulated with reference to Hacking's distinction between interactive kinds and indifferent kinds' (2007, p. 334).

According to Tsou, the main problem in Hacking's theory of human kinds is that it suffers

from an equivocation of the term 'indifferent', visible in the above propositions 1 and 2. Whereas in proposition 1 Hacking defines indifference in terms of a lack of looping effects ('calling a quark a quark makes no difference to the quark'), in proposition 2 the meaning of indifference refers to an identifiable biological pathology ('pathology *P*'). Hacking thus unjustifiably conflates the lack of looping effects with the existence of identifiable biological regularities for certain psychiatric conditions.

Such conflation betrays a still deeper confusion. Citing Hacking's claim that 'many believe that specific types of retardation have clear biological causes, to the extent that we can say these disorders simply are biological' (Hacking 1999, p. 116), Tsou notes that Hacking's discussion seems to heedlessly move between two wholly different levels, confusing properties of classifications with properties of kinds of things in nature. Hacking indeed begins with the discussion of human kind terms—classifications—which are or are not subject to social pressures and hence looping. His subsequent account of interactive and indifferent kinds, however, abandons the concern with classifications and takes on kinds in nature. This is not to say, however, that we should forgo Hacking's insight that a biological pathology associated with schizophrenia or autism is not affected simply by the fact that we have found out about it. What it means, Tsou argues, is that Hacking's resolution rests on an inconsistency, namely, that while the distinction in proposition 1 concerns a feature of *classifications*, proposition 2 concerns a feature of *objects of classification*.

Further criticism of Hacking's theory has come from the allegation that his arguments do not establish a real divide between human kinds and natural kinds (Ereshefsky, 2004). For example, Cooper (2004, p. 74) claims that Hacking is not authorized to draw the conclusion that that human kinds are radically unlike natural kinds from the fact that feedback results in human kinds such as 'autistic' have histories unlike those of natural kinds such as 'gold'. She points out that feedback would only be incompatible with human kinds being natural kinds if we adopted an *essentialist* view of natural kinds, that is, if we considered all members of a natural kind to share some essential property (Wilkerson, 1995). She invokes non-essentialist accounts to illustrate the absence of a necessary incompatibility between cultural and conceptual feedback on one side, and the natural kind status of human kinds on the other.

One of the most prominent such accounts is due to John Dupré (1981), whose pluralism, which he formerly labelled *promiscuous realism*, emerged in his paper 'Natural Kinds and Biological Taxa' as a response to the impossibility of accommodating biological species in an essentialist account. He notes that there are reasons for thinking that often no one genetic property or set of properties will be shared by all members of a species (1981, pp. 84–5), especially as gene variation is beneficial as it enable species to adapt to environmental changes. Claiming that there is no reason why the account of species currently offered in classificatory systematics should preclude their being modestly natural kinds, Dupré invites us to imagine a 'quality space' in the form of a multidimensional Cartesian coordinate system. In it, coordinates would be multiplied to as many as there are qualitative dimensions by which living things can be described or picked out. If it were possible to map individual organisms on such a space, Dupré suggests, we would find numerous clusters or bumps. 'In some parts of biology these clusters will be almost entirely discrete' (2002, pp. 33–4). Clusters on the multidimensional quality space would correspond to similar groupings, and some of these would probably correspond to essentialist natural kinds, while others would be the product of our attention being placed on a particular dimension of the map. In this way, Dupré presents an account that is both realist, in that the clusters reflect the real structure of the world, and non-essentialist, in that there will be several incommensurable modes of classification depending on the clusters to which we attend.

Cooper argues that it is really in reference to non-essentialist views that we should ask whether human kinds are fundamentally like or unlike mind-independent, natural kinds. Are Dupré's kinds incompatible with the feedback which, according to Hacking, distinguishes natural kinds from human kinds by way of their indifference? We have seen that Hacking's claim rests on the fact that the people we classify (and those people with whom they interact) are aware of how they are being classified (at least to some degree). While this certainly establishes a significant difference between natural and human kinds, Cooper maintains that 'the fact that only human kinds are affected by the subject's ideas will only be a reason for thinking that human kinds are distinct from natural kinds if an extra premise is added, to the effect that being affected by the ideas is of greater metaphysical significance than being affected by, say, antibiotics' (2004, p. 79).

No indication is given in Hacking's writings to the effect that he would be willing to endorse such a premise. In fact, a telling afore-cited passage hints at the contrary, stating that some human kinds 'pick out genuine causal properties, biological kinds, which, like all indifferent kinds, are unaffected, as kinds, by what we know about them' (1999, p. 123). It is noteworthy that Hacking here contrasts the looping effect with 'genuine causal properties,' as if only biology can be genuinely productive of behavior. As Dominic Murphy (2001, p. 153) points out, that begs the very question it raises, namely, the relation of social construction to biological psychiatry.

Since Cooper's paper, however, Hacking has revised his stance on both natural and human kinds. After passing in review of, and finding flaw in, almost every theory of natural kinds in the history of philosophy since the first uses of the term by William Whewell and John Stuart Mill, Hacking (2007, p. 238) finally concludes that although we may judge some classifications as more natural than others, there is neither a precise nor even a vague class of classifications that may usefully be called the class of natural kinds. Thus, this is how he responds to Cooper's objection:

> A simple deduction: there is no such thing as a natural kind, a *fortiori*, there is no such thing as a human kind. Rachel Cooper in my opinion did not get to the root of the evil in her astute paper, 'Why Hacking is Wrong about Human Kinds' ... She opposes what she calls my 'central claim that human kinds and natural kinds are fundamentally distinct.' In fact, there do not exist two classes (of the sort indicated) that can be defined sufficiently clearly to be either distinct or not distinct. (2007b, p. 291, fn. 17).

Needless to say, this does not mean the end of all our exploring. As Hacking himself declared after his statement of the 'semantic resolution' in *The Social Construction of What?*, 'the dynamics of classification is where the action is' (1999, p. 124). Since they are chiefly methodological, the problems in approaching the issue from a semantic standpoint should by no means hinder us from delving into the pragmatic dimension of human classifications, let alone distract us from the pursuit of a successful reconciliation.

Murphy's Emendation of Hacking's Account

As we have seen, Hacking's 'semantic resolution' states that the referent of a human classification such as 'autism' is the pathology that causes that condition, and that the stereotype associated with the classification will be subject to feedback and, hence, will have looping effects. With Tsou, however, we have seen that 'indifferent kind' does not refer to a classification without looping effects, but to the *causes* of a kind of thing in nature (for our purposes, pathology *P*). Hacking's approach thus fails insofar as it does not show how that psychiatric classifications are indifferent kinds in the sense of being classifications without looping effects.

Still, what can be made by way of a positive reaction to Hacking's core insights, which, despite the failure of their translation into a *semantic* model, strike us as being fundamentally correct? Murphy's emendation of Hacking's reconciliation provides a way, if not an answer. Murphy claims that we are never going to be able to separate out the social and the biological to deal with them individually in the way Hacking seems to have envisaged and that, therefore, we should instead *add* a cognitive dimension to the biomedical model to productively bring together biology and society in the explanation of mental disorders.

A holistic, albeit medical, model of variation in psychiatric disorder would concede that in some cases symptoms are very malleable to modification-by-expectation. This is best exemplified by the existence of so-called *culture-bound syndromes* (Rebhun, 2004). Some of the examples we find in the DSM-IV-TR (APA, 2000) are *running amok* (Malaysia and Indonesia), *brain fag* (West Africa), and *mal de ojo* (Hispanic populations). Hacking proposed an explanation of similar phenomena in *Mad Travellers*, the chief example of which was *dissociative fugue*, a trance-like state characterized by reversible amnesia accompanied by intensive wandering. It was predominantly diagnosed in France in the late 19th century but disappeared by the early 20th century. Analogously to the combination of elements which form an ecosystem, Hacking suggested that various medical, institutional, demographic and cultural vectors serve to facilitate the surfacing of a niche, that is, a particular way of going mad. Hacking's idea is that a disorder, in order to become observable, must provide 'some release for its sufferers that is not available elsewhere in the culture in which it thrives'

(1998, p. 2).

The sudden spreading of anorexia in Hong Kong (Lee, 1991) and clinical depression in Japan (Watters, 2010) following their increasing Westernization suggests that those staples of psychiatric diagnostics may well be some of *our* culture-bound syndromes, which have been 'exported' eastward. A measure of social constructionism might at this point seem healthy. Still, not only social, but more inflexible organic dimensions of disorders might plausibly present variation from one culture to another. What is more, it might present variation from one person to another. In this vein, Murphy points to the importance of considering phenomenological vectors in devising a model of psychopathological variation:

> Under different conditions the same neuropathology might result in different symptoms. Behavior depends not just on brain states but also on the interaction of social, biological, and psychological systems so that similar pathologies may have different prognoses depending on a complicated array of factors. There is good evidence, for instance, that whether or not intense emotional distress becomes pathological anxiety depends not on the nature of the emotional reaction itself but on one's network of support and beliefs about how anxiety should be dealt with [e.g. McHugh and Slavney (1998)]. (2001, p. 154)

This may sound obvious to a practicing psychotherapist. And it sounds familiar to cognitive scientists in virtue of the established fact of neuroplasticity, the changing and reorganization of neurons, their networks and their function through new experiences—an idea first hinted at by William James in chapter IV of *The Principles of Psychology*. Neuroplasticity is not only a good model for the explanation of interpersonal variation, but also illuminates the fact that the form of one brain pathology may vary according to the social forces at play, resulting in different manifestations, as exemplified by culture-bound syndromes. Murphy (2001, p. 154) notes that the brain, as the organ of cognition, changes in response to changes in the social and cultural environment and, thus, concludes that in order that we may understand how behavior might be socially constructed, we need to understand the neuropsychological structures that *mediate* between society and behavior.

Conclusion

The looping effects of human classifications and the social construction of pathological niches occur in virtue of humans having the capacity to form conscious mental representations, which are responsible for the production of behavioral manifestations. The way people think has an impact contiguous with organic dysfunctions—not metaphysically inferior to what may be viewed as 'genuine' causal properties. This is why the biomedical model should not be abandoned, but *amended*. We can understand how the social can be mediated by the biological through the study of how our brains form social representation. And we can understand how different experience of psychological afflictions can arise from similar biology through the study of individual developmental pathways with regard to a variety of inputs (Oyama, 2000). Thus Murphy's view, as Guze's, rightly eschews the common distinction between psychiatry and clinical neuropsychology. But it does so while taking into account the possibility that intrinsic impairment in cognitive architecture might be detectable even in the absence of clear anatomical evidence. Thus, accommodating a theory of psychiatric explanation within the field of cognitive neuroscience with attention to the dynamics of classifications seems like our best shot at finally reconciling biology and society and honoring Hacking's insights.

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