Philosophy of Medicine
Book Review

Review of Jonathan Y. Tsou's *Philosophy of Psychiatry*

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The question "What kinds of things are mental disorders?" has been a central topic in the philosophy of psychiatry for many decades. In his recent book, *Philosophy of Psychiatry,* Jonathan Tsou defends a realist account of mental disorders, which stipulates that "genuine mental disorders are biological kinds with harmful effects" (2021, 4). This counters the proponents of the antipsychiatry movement, such as Thomas Szasz (1960) and R.D. Laing (1967), who argue that mental disorders are oppressive labels applied to socially undesirable behaviors. It also challenges the operationalism of the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association 2013), which defines mental disorders as behavioral syndromes that are assumed to reflect underlying dysfunctional processes.

A key theoretical framework underpinning Tsou's account of biological kindhood is Richard Boyd's (1999) homeostatic property cluster theory. This proposes that members of a given kind are classified together on the grounds that they share enough of a collection of properties that tend to cluster together due to causal mechanisms. While this clustering is not accidental, it is contingent, in that the presence of any given property does not necessitate the presence of any other property. Hence, different members of the kind may possess the properties in different combinations. This framework was initially used to account for biological taxa as natural kinds. For example, due to their shared phylogenetic ancestries and similar ontogenetic environments, vipers tend to be viviparous, have keeled scales, and have hinged fangs, but not all of the members of the kind have all of these features. Notably, *Lachesis muta* is oviparous and *Azemiops feae* has smooth scales. And so, the homeostatic property cluster theory can account for how classification into a kind can support inductive inferences about the kind's members while accommodating variability among the members.

Likewise, Tsou suggests that mental disorders are biological kinds qua homeostatic property clusters. People diagnosed with a given disorder, such as major depressive disorder, share enough of a collection of properties that tend to cluster together due to causal mechanisms. However, Tsou contends that his account "is more restrictive than Boyd's insofar as it demands that at least some of the mechanisms underwriting mental disorders are intrinsic (biological) mechanisms" (2021, 3). This is reflected in how Tsou

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focuses predominantly on biological properties, such as "deficient monoamine activity, hyperactive neuroendocrine response, disrupted neuroplasticity" (2021, 38).

By characterizing mental disorders as homeostatic property clusters, Tsou is able to accommodate the variability exhibited by people diagnosed with a given disorder. Different people diagnosed with major depressive disorder may have different combinations of the aforementioned properties. Moreover, the framework can account for why these properties tend to cluster together in statistically significant ways. Although Tsou acknowledges that homeostatic property clusters are "neither immutable nor constituted by exceptionless laws of nature" (2021, 33), he claims that the causal mechanisms make the clusters sufficiently stable to withstand changes due to social feedback. This is intended to deflect Ian Hacking's (1995) argument that psychiatric kinds are unstable because of the looping effects between the social responses to classification and the behaviors that comprise the kinds. However, as I will later show, I think that Tsou underplays the role of social feedback in the development and progression of mental disorder.

As Tsou recognizes, the application of homeostatic property cluster theory to philosophy of psychiatry is not itself new. Several philosophers have previously proposed that some psychiatric conditions could be analyzed as homeostatic property clusters (Beebee and Sabbarton-Leary 2010; Kendler, Zachar, and Craver 2011; Maung 2016; Zachar 2014). Given the wealth of previous literature on this topic, Tsou's book might be seen as simply revisiting a well-trodden path. However, his original and valuable contribution in this book is the use of homeostatic property cluster theory to develop a general account of the concept of mental disorder that moves away from the field's emphasis on the notion of dysfunction.

The debate over the concept of mental disorder in the philosophy of psychiatry concerns the question of what criteria demarcate genuine mental disorders from healthy states, or from other sorts of problems, such as moral and social problems. Perhaps the most influential philosophical account of mental disorder in recent decades is Jerome Wakefield's (1992) harmful dysfunction analysis, which suggests that a condition is a disorder if (1) it is harmful to the person; and (2) it involves a failure of an internal mechanism to perform its evolutionarily selected function. This is a hybrid account of disorder, as it proposes that a condition's being a disorder is jointly determined by both a value judgment about harm and an empirical fact about dysfunction. Under the harmful dysfunction analysis, then, a dysfunction that does not harm the person, such as the voluntary use of contraception to suppress reproductive function, and a harmful or undesirable state that does not involve a dysfunction, such as infant teething, would not be classed as disorders.

Like Wakefield, Tsou defends a hybrid account of disorder, which acknowledges that a condition's being a disorder is jointly determined by a value judgment and an empirical fact. Moreover, both agree that harm is the appropriate value criterion. As Tsou notes: "The determination of mental disorders (and physical diseases) requires normative judgments (i.e., that a condition is sufficiently harmful to merit intervention)" (2021, 1). However, Tsou and Wakefield disagree about what comprises the factual criterion. According to Tsou, a failure of an internal mechanism to perform its evolutionarily selected function is unnecessary for a condition to be a disorder. Rather, he suggests that a genuine disorder is a biological kind that is harmful to the person, where biological kindhood is defined according to the homeostatic property cluster theory.

The move away from evolutionary dysfunction as a criterion for mental disorder is a merit of Tsou's account. Various scholars have argued that Wakefield's criterion of evolutionary dysfunction is unnecessary for a condition to be considered a disorder (Lilienfeld and Marino 1995; Varga 2011). Some conditions that are rightfully treated as disorders may not be failures of evolutionarily selected functions at all. For example, some may be evolutionary adaptive responses that are now mismatched with the current environment, some may be evolutionarily neutral byproducts, and some may be ontogenetic products of our modern social environment. Moreover, with regard to mental disorders, we may lack the methodological resources to establish whether they are adaptive, dysfunctional, or evolutionarily neutral. As Subrena Smith (2020) notes, psychological traits do not leave unambiguous fossil evidence, the confounding effect of environmental change on psychological development is immense, and the large evolutionary distance between our nearest extant relatives and ourselves renders the comparative method uninformative. Given these methodological limitations, hypotheses about whether some psychological traits are functions or dysfunctions may remain underdetermined indefinitely.

By rejecting the criterion of evolutionary dysfunction, Tsou's account is theoretically capable of bringing a wider range of conditions within the purview of mental healthcare. Furthermore, the homeostatic property cluster theory is a promising framework for a more satisfactory account of mental disorder since it can accommodate complexity, contingency, and variability. However, the potential benefits of the homeostatic property cluster theory are greatly diminished by restricting the properties included in the clusters to biological properties. As I shall argue below, Tsou's privileging of the biological over the social is unwarranted and ultimately problematic.

Underpinning Tsou's analysis is an assumed distinction between biological kinds "underwritten by biological mechanisms" and social kinds "underwritten by social mechanisms" (2021, 3). As noted above, Tsou wants to claim that mental disorders are biological kinds, in contrast to the proponents of the antipsychiatry movement who, according to Tsou, claim that mental disorders are social kinds (Laing 1967; Szasz 1960). It is worth noting here that Tsou's discussion sometimes equivocates between different senses of what it is for a classification to be "underwritten by social mechanisms." The first sense is that social kinds are "socially constructed classes that are invented and reflect the values of classifiers" (2021, 1). That is to say, our social values and interests inform the ways in which we decide to sort things into categories, such as by influencing which properties we consider to be salient for our purposes. The second sense is that social kinds are "determined by social causes rather than biological ones" (2021, 1). In other words, the things that we classify are brought about by social causes, such as defiance being brought about by oppression. Importantly, these different senses are not entirely coextensive. For example, a classification may include only things that have biological causes, but the ways in which they are classified could reflect the values and interests of the people who are doing the classifying.

As Tsou's hybrid account explicitly acknowledges that a condition's being a mental disorder is partly informed by a value judgment about harm, it could be said to be "underwritten by social mechanisms" in the first sense of reflecting "the values of classifiers." This is especially apparent in his claim that the aim of psychiatry is "to help individuals deal with conditions that are harmful insofar as they impede their capacity to

live a normal life" (2021, 44). Here, what is considered by psychiatry to comprise "a normal life" is profoundly informed by social values, although these are not explicated in Tsou's book. Of course, the alleviation of suffering is one such guiding value, but it is not the only one. Given that mental disorders are marked by intentionality and relationality, psychiatric judgments about what is "normal" are also informed by values concerning socially accepted norms of rationality (delusional disorder), affectivity (hypomania), desire (anorexia nervosa), morality (conduct disorder), and interpersonal interaction (borderline personality disorder).

The fact that Tsou's account is "underwritten by social mechanisms" in the first sense is not a weakness of his account per se. Indeed, I would contend that any satisfactory account of mental disorder would have to acknowledge the value-ladenness of the concept, and so Tsou's inclusion of a value criterion is actually a strength. However, it does weaken his objection to the proponents of antipsychiatry, whom Tsou criticizes for characterizing mental disorder as a "deviation from social and ethical norms" (2021, 7), insofar as these norms are also implicit in the evaluative component of his own account. The problem with the antipsychiatry argument is not the premise that the concept of mental disorder is laden with social values. Rather, the problem is the illegitimate move from "the concept of mental disorder is laden with social values" to "mental disorders are not genuine disorders" (Szasz 1960). Indeed, philosophers have long objected to the antipsychiatry argument precisely by contending that the concept of somatic disorder is also value-laden (Fulford 1989; Nordenfelt 2007).

This brings us to the question of whether mental disorder is "underwritten by social mechanisms" in the second sense of being "determined by social causes rather than biological ones". As noted above, Tsou wants to maintain that mental disorders are biological kinds. Hence, he claims that "biological mechanisms determine the general causal features of mental disorders" while "social mechanisms contribute to the specific *expression* of disorders in particular cultural contexts" (2021, 19). However, recent work on ecological, pluralist, and enactive approaches to mental disorder have suggested that such a distinction is untenable (Fuchs 2012; Mitchell 2009; Nielsen and Ward 2018). After all, people do not subsist in isolation, but are always embedded in a social environment, with which they interact in dynamic and recursive ways. Through these interactions, biological mechanisms and social mechanisms continually influence and sustain one another. For example, Thomas Fuchs (2012) notes that major depressive disorder is marked by feedback loops between the individual and the environment, which sustain the symptoms and influence the course of the illness, while Matthew Broome and Lisa Bortolotti (2009) cite evidence showing how a busy urban environment has a profound and immediate impact on the development, intensity, and content of paranoid ideation.

Importantly, the discussion above is not merely the claim that mental disorders can be externally caused by social factors in the way, for example, that lung cancer can be externally caused by tobacco smoking. While lung cancer may be brought about by an external antecedent cause, its projectable feature is an internal biological process whose subsequent progression is independent of its antecedent cause. However, often in the case of mental disorder, the internal and external processes continually influence one another, in such a way that the progression is constitutively dependent on how they interact. Indeed, the effectiveness of psychological therapy is premised on the fact that cognitive, behavioral, and social changes can alter the structure and course of the illness. This is further supported by

evidence that psychological therapy is associated with alterations in neurobiological mechanisms (Karlsson 2011). Given these dynamic and recursive interactions between the neurobiological and the psychosocial, it is doubtful that mental disorders can be said to have underlying "general causal features" that are wholly independent of social mechanisms. Accordingly, Sandra Mitchell (2009) proposes that mental disorder must be understood as a complex behavior of a complex system that is jointly dependent on multiple interacting causes at multiple organizational levels.

Such joint dependence suggests that the empirical data on their own do not justify the privileging of the biological over the social because both sorts of factors have active roles as difference makers in the development and progression of mental disorder. As noted above, Tsou claims that "social mechanisms contribute to the specific *expression* of disorders in particular cultural contexts," while "biological mechanisms determine the general causal features of mental disorders," but it would be just as acceptable in some contexts to say that social mechanisms determine the general causal features of mental disorders while biological mechanisms contribute to how the mental disorders are expressed in different people. For example, an abusive environment is a well-established causal factor that can occasion the development and persistence of mental disorder, while biological, psychological, and other contextual differences could be said to influence whether the affected people present predominantly with internalizing or externalizing symptoms (Keyes et al. 2012).

This raises the question of why Tsou chooses to privilege the biological over the social in his account. I consider two of his reasons here. First, Tsou appeals to fact that schizophrenia appears in all cultures and suggests that a "way to interpret these findings is to regard the uniformity of a condition across cultures as a measure of the extent that a disorder is determined by biological mechanisms" (2021, 40). However, this does not necessarily follow. For example, almost all contemporary cultures use money as a medium of exchange, but it would be wrong to suppose that this uniformity across cultures indicates that money use is determined by biological mechanisms. Such an interpretation potentially underestimates the extent to which social environmental factors are shared across cultures, especially in an increasingly globalized, postindustrial world. Accordingly, the uniformity of the prevalence of schizophrenia could, at least partly, be the result of uniform aspects of the social environment that have been taken for granted. It should also be noted here that Tsou is possibly overestimating the uniformity of the prevalence of schizophrenia, as there is a large amount of evidence which suggests that urban areas, especially those marked by social fragmentation and deprivation, are associated with increased rates of schizophrenia (Heinz, Deserno, and Reininghaus 2013).

Second, Tsou suggests that biological explanations are preferable for psychiatric research because "classifications that individuate biological kinds yield more stable projectable inferences than classifications formulated at more specific (folk) levels that incorporate the effects of social mechanisms" (2021, 43). However, this claim is largely promissory and does not reflect the currently available evidence. As Kathryn Tabb and Maël Lemoine (2021) note, there are currently no mechanistic biomarkers in psychiatry that reveal the causal structures of mental disorders, or yield stable projectable inferences of clinical relevance. On this particular issue, Tsou's presentation of the empirical research sometimes seems uncritical and incomplete. Notably, the evidence regarding his chosen example of "deficient monoamine activity" in major depressive disorder has been suggested

to be less consistent and of less causal significance than Tsou supposes (Pies 2019). By contrast, psychiatric epidemiology has made many successful projectable inferences about mental disorders based on social mechanisms. I have already mentioned the examples of child maltreatment (Keyes et al. 2012) and urban social deprivation (Heinz, Deserno, and Reininghaus 2013) as social mechanisms that occasion and sustain mental disorders. Other examples include the impact of austerity on the incidence of major depressive disorder (Stuckler et al. 2017) and the impact of family rejection on mental health in the transgender community (Klein and Golub 2016). Such evidence would suggest that the most projectable features of mental disorders are unlikely to be found at the biological level but, rather, are likely to be the stable patterns that obtain across a wider system that encompasses the biological, the psychological, and the social.

The claim that biological explanations are preferable for psychiatric research because "classifications that individuate biological kinds yield more stable projectable inferences" is also undermined by recent research on psychological kinds in psychiatry. Notably, the network analysis approach of Denny Borsboom (2008) and Eiko Fried and Angélique Cramer (2017) models mental disorders as clusters of psychological variables that reinforce one another through causal relations. At least implicitly, this is an application of the homeostatic property cluster theory (Kendler, Zachar, and Craver 2011). However, the properties in the clusters are psychological rather than biological. This shows how projectable inferences about mental disorders, including inferences about comorbidities and targets for therapeutic interventions, can be based on psychological mechanisms, without any need to attempt to reduce them to the biological level.

Furthermore, it is doubtful whether successful biological reductions of these psychological processes are even feasible. To be clear, this is not to say that the psychological processes are not implemented by biological processes. Of course, we are embodied in biological systems, and so our cognitive and behavioral activities are implemented by our brains and bodies. Rather, it is to say that the features that are the most projectable are not found at the biological level but at the psychological level. For example, the stable patterns that obtain at the psychological level may be multiply realized, in that the biological mechanisms that implement them are too heterogeneous to support inductive inferences (Maung 2016). Also, given that our behaviors are embedded in a social environment, some of the psychological processes may be constitutively dependent on their interpersonal contexts, so that their projectable features are not the ways in which they are biologically implemented but the social circumstances that occasion them. For example, research has suggested that suicidal behaviors in young people are often shaped by interpersonal contexts of domestic violence, bullying, bereavement, and academic stress (Rodway et al. 2020).

The strength of Tsou's book lies in its negative thesis that mental disorders do not necessarily involve failures of evolutionarily selected mechanisms. Indeed, I would contend that mental healthcare is justified by the presence of suffering that could be alleviated by a mental healthcare intervention, regardless of the evolutionary history of the affected system. However, the positive thesis that mental disorders are biological kinds is less successful. Such privileging of the biological over the social seems somewhat outdated and unwarranted in view of some key features of the contemporary research landscape. These include (1) the relative successes of projectable inferences based on social and psychological mechanisms, as exemplified respectively by the aforementioned research in psychiatric

epidemiology and network analysis; (2) the failure, which is not for a want of trying, to find mechanistic biomarkers that reveal the causal structures of mental disorders; and (3) the increasing knowledge of the dynamic and recursive interactions between biological, psychological, and social processes. In light of the above, philosophers of psychiatry have been turning increasingly to ecological and pluralist approaches to mental disorder (Fuchs 2012; Kendler, Zachar, and Craver 2011; Maung 2016; Mitchell 2009; Nielsen and Ward 2018). To be clear, none of the above critique undermines the importance of biological research in psychiatry. Biological considerations are clearly important to our understandings of the processes involved in mental disorders and the effects of pharmacological treatments. However, given the complexity of psychiatric suffering, our inferences about mental disorders may be profoundly limited if we restrict our characterizations of them exclusively to the biological level. By relaxing our characterizations of mental disorders to include the stable patterns that occur across biological, psychological, and social levels, we may yield more robust and informative homeostatic property clusters.

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