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The Ontology of Posttraumatic Stress Disorder: Natural Kind, Social Construction, or Causal System?

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The ontological status of posttraumatic stress disorder (PTSD) has long been a focus of intense controversy. Is PTSD a natural kind discovered recently by astute clinicians, but present throughout history and across diverse cultures? Or is it a socially constructed artifact arising in the wake of the Vietnam War? In addition to covering issues relevant to this debate, I describe another interpretation of PTSD orthogonal to the natural versus artifactual dichotomy. Inspired by the causal systems approach to mental disorders pioneered by Borsboom and his colleagues, I suggest a causal system interpretation of PTSD is a scientifically more profitable approach than either the social constructionist or natural kind interpretations of this disorder.

Key words: **causal systems, natural kind, network analysis, posttraumatic stress disorder, psychiatric diagnosis, social construction.** ***[Clin Psychol Sci Prac 19: 220–228, 2012]***

Posttraumatic stress disorder (PTSD) has the unusual distinction of being highly studied yet highly controversial (Brewin, 2003; McHugh & Treisman, 2007; McNally, 2003b). One persistent controversy concerns its ontological status. That is, what kind of thing is PTSD anyway? In response to this question, scholars have proposed two characteristic answers. According to the one favored by most traumatologists, PTSD is a timeless natural kind, a universal psychobiological entity emerging in response to extreme stressors (e.g., Osterman & de Jong, 2007). Its presumptive timelessness arises from PTSD's roots in ancient, evolved fear circuitry whose activation pathologically persists long after mortal danger has passed.

Another answer, often favored by historians, anthropologists, and some clinicians, is that PTSD is a social construction, a cultural artifact arising in the wake of the Vietnam War (e.g., Summerfield, 2001). As Young (1995) put it: "The disorder is not timeless, nor does it possess an intrinsic unity. Rather, it is glued together by the practices, technologies, and narratives with which it is diagnosed, studied, treated, and represented and by the various interests, institutions, and moral arguments that mobilized these efforts and resources" (p. 5). He added, "Traumatic memory is a man-made object. It originates in the scientific and clinical discourses of the 19th century; before that time, there is unhappiness, despair, and disturbing recollections, but no traumatic memory, in the sense that we know it today" (p. 141).

In this article, I assess these characteristic answers to the ontological question about PTSD as a prelude to suggesting a third one inspired by the *causal systems* view of mental disorder pioneered by the psychometrician Denny Borsboom and his colleagues at the University of Amsterdam. Their conceptualization of mental disorders is radically different from both the natural kind and social constructionist perspectives. They have applied their conceptual insights and computational analyses mainly to depression, panic disorder, and generalized anxiety disorder, but their approach can deepen our understanding of PTSD as well (Borsboom, 2008; Borsboom, Cramer, Schmittmann, Epskamp, & Waldorp, 2011; Borsboom, Epskamp, Kievit, Cramer, &

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Schmittmann, 2011; Cramer, Waldorp, van der Maas, & Borsboom, 2010; Schmittmann et al., in press).

PTSD AS A SOCIAL CONSTRUCTION

Social constructionist interpretations vary in their emphasis regarding the role of culture in shaping mental disorders (for a review, see McNally, 2011, pp. 128–158). For some syndromes, culture barely scratches the surface, chiefly shaping symptoms in subtle ways. For example, Cambodian (Hinton et al., 2006) and American (McNally, 1994, pp. 108–115) panic disorder patients both respond fearfully to bodily sensations, but cultural differences shape the kinds of catastrophes these patients fear the symptoms signify. Yet other syndromes seem almost entirely a product of culture, best exemplified by dissociative identity disorder (Hacking, 1995; Nathan, 2011). Moreover, shifts in the ambient culture can destroy the niche that sustains these syndromes, causing their disappearance from the landscape of psychopathology (Hacking, 1998).

It is unclear where PTSD may fall on this continuum. However, any formulation that does not assert its status as a timeless, natural kind discovered by astute clinicians will incite the ire of many traumatologists. To question its natural status is to imply its artifactual character, and this, many suspect, amounts to silencing the voices of survivors, delegitimizing their suffering, and aligning skeptics with the perpetrators of trauma. However, to affirm something's artifactual character does not necessarily entail that it is not real. Money, for example, is a social construction having profound consequences, yet no one doubts its reality.

However, these observations are unlikely to satisfy many traumatologists who draw moral implications from ontological controversies. As Yehuda and McFarlane (1997) argued, "Biological findings have provided objective validation that PTSD is more than a politically or socially motivated conceptualization of human suffering" (p. xv). They added that biological research provides "concrete validation of human suffering and a legitimacy that does not depend on arbitrary social and political forces. Establishing that there is a biological basis for psychological trauma is an essential first step in allowing the permanent validation of human suffering" (p. xv).

Such moral considerations seem orthogonal to the ontological debate. Indeed, empathy for trauma victims need not wait for the discoveries of biological psychiatry, and their suffering is no less real in the absence of these discoveries.

A more promising avenue of rebutting the social constructionist thesis is to search for PTSD among trauma victims in diverse cultures around the globe. Cross-cultural researchers have to ensure that respondents understand the meaning and purpose of diagnostic interviews when they interview trauma survivors in non-Western settings. For example, some survivors of the Asian tsunami in Sri Lanka reported PTSD symptoms, mistakenly believing that affirmative responses to questions about PTSD were prerequisites for receiving food, clothing, and other forms of material aid (Watters, 2010, pp. 65–125). Another potential problem is that standard PTSD assessments based on the *Diagnostic and Statistical Manual of Mental Disorders* (4th ed., text rev., *DSM-IV-TR*; American Psychiatric Association, 2000) criteria will miss any posttraumatic psychopathology indigenous to certain cultures. Finally, some data suggest that PTSD in the developing world appears to be partly a function of exposure to Western trauma culture (Yeomans, Herbert, & Forman, 2008). These caveats notwithstanding, cross-cultural traumatologists have reported PTSD around the globe (Osterman & de Jong, 2007).

Another possible avenue for rebutting the social constructionist interpretation of PTSD is to seek evidence for the disorder throughout history. In fact, many scholars have suggested that shell shock in World War I and battle fatigue in World War II constituted PTSD appearing under different names. However, despite some symptomatic overlap among these three syndromes, their differences are equally prominent (Jones & Wessely, 2005). In fact, historical scholarship on war-related psychiatric syndromes within Anglo-American culture provides far less evidence for the cultural timelessness than contemporary cross-cultural scholarship does (McNally, in press).

Jones et al. (2003) studied the medical files of severely traumatized British war veterans who had received pensions for chronic psychiatric illness. They tested whether complaints noted in the files corresponded to today's PTSD symptoms. They were espe-

cially keen to test whether flashbacks—vivid, involuntary sensory memories of trauma—were evident. Strikingly, despite their salience today (Brewin, 2011), flashbacks almost never appeared in the medical files until the Persian Gulf War. Indeed, only three of the 640 patients from World War I reported phenomena even remotely suggestive of flashbacks (0.5%), and only five of 367 patients from World War II did so (1.4%). Among the 428 psychiatric casualties of the Victorian campaigns and the Boer War, not one mentioned flashbacks. However, 36 of the 400 patients of the Persian Gulf War veterans mentioned them (9%). In fact, of the 1,007 patients from World Wars I and II, only eight had flashbacks (broadly defined), and only five of these patients would have qualified for PTSD in the *DSM-IV-TR* (American Psychiatric Association, 2000). Indeed, in the entire data set, hallmark PTSD symptoms, such as intrusive memories and avoidance of reminders, were so uncommon that Jones et al. (2003) concluded that PTSD might be a "contemporary culture-bound syndrome" (p. 162).

Further documenting the symptomatic diversity among survivors of war trauma in the British military medical records, Jones et al. (2002) identified three partly overlapping clusters that have emerged among psychiatric casualties from the Boer War through the Persian Gulf War. The "debility" cluster included complaints of chronic fatigue, anxiety, weakness, and breathlessness. The "somatic" cluster included symptoms such as dizziness, anxiety, rapid heart rate, and breathlessness. The "neuropsychiatric" cluster comprised depression, anxiety, fatigue, sleep difficulties, startle responses, irritability, personality changes, and chronic pain. Strikingly, none of these clusters map directly onto PTSD despite some overlap.

Taken together, we confront the paradox that PTSD appears throughout the world today, yet it does not consistently appear in its current form even within the Anglo-American military historical record. It appears cross-cultural without appearing transhistorical.

PTSD AS A NATURAL KIND

How might natural kind theorists resolve this paradox? Certainly the "operational" approach to conceptualizing mental disorders will not work. In fact, this approach, supposedly embodied in our diagnostic manual ever since 1980, was a nonstarter right from the beginning (McNally, 2011, pp. 203–207). The scientific rhetoric of operationism notwithstanding, few, if any, psychopathologists would endorse the austere implications of this doctrine as set forth by Bridgman (1927). Operational definitions define concepts by their method of measurement, thereby exhausting their meaning. As applied to psychiatric diagnosis, there is nothing more to a disorder than its defining signs and symptoms. Accordingly, any change in symptoms means that we are no longer dealing with the same disorder. The virtue of the *Diagnostic and Statistical Manual of Mental Disorders* (3rd ed., *DSM–III*; American Psychiatric Association, 1980) and its successors was that its authors provided explicit inclusion and exclusion criteria for assigning diagnostic labels; but this is not the same thing as providing an operational definition. Most psychopathologists do not conceptualize the relation between symptoms and diagnoses as one of *formative* measurement as do sociologists who define socioeconomic status (SES) as a composite of income, educational attainment, and occupational status (Schmittmann et al., in press). The three indicators determine—indeed, define—one's value on a measure of SES. Hence, an increase in one's income or educational attainment boosts one's SES rather than vice versa. The causal arrow runs from the indicators to the SES—the construct they form.

In contrast, many, if not most, psychopathologists view the relation between symptoms and diagnoses as one of *reflective* measurement (Schmittmann et al., in press). That is, to explain why symptoms cohere as a syndrome, they assume that symptoms reflect an underlying disease process, and that this process is the common cause of the symptoms. Symptoms are fallible indicators of an inferred entity, whether one conceptualizes this variable as a latent category or latent dimension. This latent variable approach is one version of philosophical realism, and it comports well with a natural kind approach to mental disorders. For one thing, it permits error. Because symptoms are imperfect indicators of an inferred disease entity, one can be mistaken about the true diagnosis. In contrast, the operational approach is stipulative, thereby defining diagnostic errors out of existence. From an operational

perspective, if the symptoms are present, so is the disorder because there is nothing more to the disorder than the symptoms themselves. Yet from a natural kind perspective, the symptoms are imperfect indicators of the inferred entity, permitting the possibility of errors about the presence of the disorder. More importantly, successive revisions of the *DSM* presuppose philosophical realism, the doctrine that postulates a mind-independent reality that scientists endeavor to describe and explain. Changes in diagnostic criteria represent attempts to carve nature at its joints, enabling the elucidation of the underlying processes that give rise to mental illness whether construed as categories or dimensions. Indeed, as Putnam (1984) once remarked, realism is the only doctrine that does make the success of science a miracle.

How might this work for PTSD? If symptoms are natural, but imperfect, indicators reflective of the underlying disorder of PTSD, then we might squeeze shell shock, battle fatigue, and other posttraumatic syndromes into the same PTSD box. On the other hand, when can we tell whether these really are diverse presentations of the "same" disorder, especially when we do not have additional evidence of the underlying latent variable? That is, *something* must occur in common across these diverse presentations to allow us to consider them all reflective of the same underlying entity. *Something* must unify them if we aim to claim that each is a variant form of the same condition.

Perhaps the common factor is the antecedent trauma. In other words, we might conceptualize PTSD as any outcome following exposure to a fixed set of traumatic stressors. This would amount to a one-to-many mapping whereby the common cause of trauma would produce a range of pathological outcomes. Hence, we could justify bracketing the "hysterical" blindness and the motor abnormalities of shell shock with the numbing and flashbacks of PTSD. What unites them is trauma, combat trauma in this case.

However, the term *trauma* has undergone a conceptual bracket creep whereby the range of qualifying events continues to expand across successive editions of the *DSM* (McNally, 2003a, 2009). The original definition of trauma, appearing in *DSM-III*, implied that PTSD could only arise following exposure to a limited set of *traumatic* stressors, such as rape, combat, torture, and natural disasters. Yet in the current manual, one need not even be physically present at the scene of the trauma to qualify as a trauma survivor (McNally & Breslau, 2008). In fact, someone who feels helpless when learning about threats to other people qualifies as a trauma survivor just as much as do the recipients of the threats themselves. For example, 4% of Americans living far from the sites of the terrorist attacks of September 11, 2001, apparently developed PTSD by viewing scenes of the violence on television (Schlenger et al., 2002).

Further complicating matters, some studies show that stressors falling short of the current definition of trauma can produce more PTSD symptoms or higher rates of the disorder than stressors that do meet the current definition of trauma (Gold, Marx, Soler-Baillo, & Sloan, 2005; Long et al., 2008; Mol et al., 2005; Van Hooff, McFarlane, Baur, Abraham, & Barnes, 2009).

How should we interpret these findings? One possibility is that people experiencing subtraumatic stressors misconstrue normal reactions with pathological ones or otherwise misunderstand the meaning of the questions, especially if the assessment procedure involves a questionnaire, not a structured diagnostic interview. Indeed, even among infantry veterans of Iraq, questionnaires tend to overestimate PTSD relative to blind, structured diagnostic interviews (Engelhard et al., 2007).

Another possibility is that people who develop apparent PTSD after exposure to relatively mild stressors possess significant psychobiological vulnerabilities that amplify the effect of these stressors. That is, such cases may exemplify a background/foreground inversion whereby the trauma recedes into the causal background, and the risk factors move into the causal foreground (McNally, 2009). The causal burden for producing the syndrome is borne chiefly by the risk factors, not by the nominal trauma. However, there is very limited evidence relevant to this hypothesis (Breslau, 2010), and only mixed support for it (McNally & Robinaugh, 2011).

Yet another possibility is that stressors that would not have produced PTSD in the past are now capable of doing so. Vicarious trauma provides especially dramatic examples. Witnessing the torture and execution of human beings was long a form of entertainment throughout the world. In ancient Rome, amphitheaters featured Christians, criminals, and others tied to stakes

as hungry lions devoured them alive to the delight of thousands of cheering fans (Auguet, 1972, pp. 93–96). Unsurprisingly, perhaps, philosophers in classical antiquity considered mercy and pity as pathological emotions (Stark, 1997, p. 212). Public hangings were a form of family entertainment in 19th-century America (Domino & Boccaccini, 2000), and photographs of gleeful mobs attending the lynching of African Americans are among the most ghastly of the 20th century. Thankfully, times have changed. The very fact that witnessing such horrors today would constitute trauma, not entertainment, surely must count as moral progress.

In fact, the horrors of World Wars I and II notwithstanding, the world continues to be a far less violent place than in the past. Contrary to what one might infer from today's media, the decline in the rate of violence continues to accelerate (Pinker, 2011). Since World War II, worldwide conflicts have been less frequent, shorter in duration, and less lethal (Goldstein, 2011). One consequence of an increasingly peaceful world is that the range of stressors capable of producing *DSM* PTSD might broaden. Members of the generation that survived the horrors of World War II (Snyder, 2010), especially the Nazi death factories, such as Treblinka (Rajchman, 2011), that far exceeded the horrors of even the Nazi concentration camps (Grossman, 2010), are unlikely to be much affected by some of the relatively mild stressors that seemingly incite PTSD today. That is, if what counts as a traumatic stressor depends on the context of one's environment, then the massive decline in violence today results in a massive broadening in the kind of things capable of producing posttraumatic psychopathology. The relatively greater comfort, safety, health, and well-being of the 21st-century world may have rendered us less resilient to stressors far less psychologically toxic than the ones occurring during World War II.

Regardless of whether the decline in violence and the overall increase in safety, health, and comfort render us vulnerable to develop PTSD to stressors that would have seldom troubled our ancestors, recent PTSD research indicates that the concept of *trauma*, or at least the range of stressors apparently capable of producing PTSD, is far from a stable category of causes. In other words, we seem stuck with a many-to-many mapping, not a one-to-many mapping.

Finally, Borsboom, Cramer, Kievit, Scholten, and Franić (2009) have identified a fatal flaw with the entire latent variable approach to conceptualizing mental disorder. The approach rests on the assumption of local independence of the indicators of the underlying construct, yet this does not seem to work for mental disorders. To see why violation of the axiom of local independence presents a fatal problem, consider three thermometers, each yielding a value for temperature (Schmittmann et al., in press). The reason why the values of the three thermometers are so highly intercorrelated is that they share a common cause: the ambient temperature in the room. There are no causal connections among the thermometers whatsoever; the value of one thermometer is causally independent of the others, thereby meeting the criterion of *local independence* that reflective models (e.g., item response, latent trait) require. If we control for ("conditionalize on") the temperature in the room, the values of the three thermometers would no longer intercorrelate because the only remaining variance would arise from error in the thermometers measuring ambient room temperature.

The latent variable approach to psychological attributes rests on the axiom of local independence (Borsboom, 2008). That is, to explain why a set of symptoms cohere into a syndrome, theorists postulate a latent cause of their covariance. Hence, to explain why the symptoms of major depression tend to occur together, latent variable theorists assert that symptoms are the effect of an underlying cause: depression. The same logic applies to PTSD. An underlying psychobiological entity is the cause of the intrusive thoughts, numbing, and so forth. The underlying disorder is the common cause of its symptomatic, co-occurring manifestations. The symptoms themselves have no correlational, let alone causal, interconnections.

Consider the symptoms of sleep disturbance, fatigue, concentration impairment, and irritability. The latent variable construal of depression presupposes that the inferred entity of depression produces these symptoms, and the symptoms themselves are unrelated to one another among people with depression. That is, conditionalizing on the presence of depression, the symptoms are statistically uncorrelated with one another (i.e., they show local independence). In the general

population, the symptoms do correlate, and their statistical coherence is what counts as the syndrome of depression. The explanation for their hanging together in the general population is that they share the common cause of underlying depression.

PTSD AS A CAUSAL SYSTEM

Borsboom, Epskamp, et al. (2011) regard mental disorders as networks of "mutually reinforcing symptoms" (p. 610). That is, a network, at an abstract level, comprises a set of entities, the relations or paths among them, and information about the pattern of influence that travels down the paths connecting the entities. Yet the instantiation of the network as a psychobiological causal system offers a realist alternative to the problematic realist latent variable approach. In contrast to the latter, the causal system provides answers to how the causal system operates to cause symptoms over time, whereas it remains mysterious how an inferred, unobservable, latent entity possesses pathways to produce symptoms that unfold over time. The causal system approach permits theorists to elucidate the mechanistic pathways by which symptoms emerge at the intraindividual level of the person.

The causal systems model of mental disorder conceptualizes symptoms as autonomous entities interconnected within a network. The relation between symptoms of depression and depression, or the relation between symptoms of PTSD and PTSD, is mereological, not one of measurement. A mereological relation specifies the relation between a whole and its parts. The symptoms of depression or of PTSD are constitutive of their respective disorders, not reflective manifestations of an underlying construct. Likewise, the relation between the USA and the 50 states is mereological. In this view, the diagnostic terms *depression* and *PTSD* each denote a dynamic system of causally interrelated symptoms that unfold over time and can settle into a pathological equilibrium. There is no underlying essence to depression lying beneath the causal system; there is no common cause producing the symptoms. *Depression* denotes a system, not an underlying latent construct, either category or dimension. Its "essence" inheres in the relations among the constitutive elements (symptoms). The lack of local independence presents no problem for a causal system model. Hence, searching for the biological substrate for a latent entity that does not exist poses a problem. However, the causal network analysis encourages the search for the mediational pathways realized psychobiologically at the level of the person (cf. mediational analysis).

Borsboom's group has used software to model the dynamic connections among symptoms within and across *DSM* disorders, illustrating the connections among the symptoms. For example, a core symptom of a disorder receives multiple causal inputs from other symptoms, and more importantly, projects causal outputs to many different symptoms. Clinically targeting an important core symptom, such as intrusive reexperiencing in PTSD or sleep loss in depression, can produce downstream beneficial effects on other symptoms in a kind of therapeutic cascade (Borsboom, 2008).

Other theorists, often in philosophy, have suggested broadly similar approaches to conceptualizing mental disorders, but without the computer software to model the temporal dynamics among the nodes and edges of the network itself. For example, clinicians have suggested that Boyd's (1991) perspective regarding natural kinds as homeostatic property cluster kinds works for conceptualizing mental disorders without committing oneself to the inferred essences presupposed by the problematic latent variable approach (Kendler, Zachar, & Craver, 2011; McNally, 2011, pp. 203–207).

A causal systems view of PTSD is agnostic about the social or natural origins of its constituent symptoms. However, one can picture ways whereby symptoms interconnect. Exposure to trauma establishes a memory of the traumatizing event, resulting in symptomatic expressions of this memory in recurrent intrusive thoughts, nightmares, and sensory flashbacks. These, in turn, motivate avoidance of reminders of the event, perhaps including numbing. Difficulties sleeping result from intrusive thoughts, and the resultant fatigue may diminish interest in formally enjoyed activities and produce fatigue-related irritability and proneness for explosive outbursts. Computational modeling of PTSD symptoms would help clarify these interrelations.

Flying in the face of the axiom of local independence, other scholars have argued that symptoms of PTSD interact. Horowitz (1986) proposed that intrusive reexperiencing symptoms cause symptoms of avoidance and numbing. He viewed the two sets of

symptoms in an oscillating dynamic. Young (2004) noted that an "inner logic" holds among the symptoms of PTSD (p. 128). What Borsboom, Cramer, et al. (2011) add to these observations is the computational power to elucidate the functional interrelations among the symptoms of PTSD (or other disorders) as they unfold over time. Using data from epidemiological surveys, they have illustrated how network analyses easily accommodate the phenomenon of comorbidity much easier than does the latent category or latent dimension approach. The computational power of network analysis can identify central symptoms in a causal system. That is, the symptom bearing the greatest number of connections to other symptoms is clearly more central than one bearing few direct connections to other symptoms. The output from these analyses provides suggestions for symptom targeting by clinicians. Rather than treating an underlying disease entity, clinicians can target specific symptoms, and when these change, improvement will propagate throughout the network, leading to recovery.

CONCLUSION

Most traumatologists have assumed that the social constructionist and natural kind views of PTSD exhaust the conceptual options for understanding the disorder. The causal network approach provides yet another model for understanding PTSD as well as other mental disorders.

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