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<Commentary on Borsboom et al., BBS Vol. 41, 2018 – revised by CCE>

<COMP: BRITISH SPELLING AND USAGE OK>

<CT>Indeed, not really a brain disorder: Implications for reductionist accounts of addiction

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<C-AB>**Abstract:** Borsboom et al.'s formulation provides an opportunity for a fundamental rethink about the "brain disease model" of addiction that dominates research, treatment, policy, and lay understanding of addiction. We also demonstrate how the American opioid crisis provides a contemporary example of how "brain disease" is not moderated by the environmental context but is instead crucially dependent upon it.

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The dominant explanation of addiction (substance-use disorder) is that it is an acquired brain disease (Leshner 1997; Volkow et. al 2016). In recent years, many academic researchers,

clinicians, and philosophers have objected to this characterization (e.g., Davies 2018; Heather 2018; Heather et al. 2018; Levy 2013; Lewis 2017; Satel & Lilienfeld 2014). We commend Borsboom and colleagues for outlining a convincing alternative to biological reductionism as an explanation for mental and behavioral disorders. In this commentary, we outline how their approach provides the foundation for a fundamental rethink about the role of the brain in addiction, one that is able to retain many of the important contributions of neurobiological research to our understanding of the disorder without the requirement to accept the "greedy reductionism" (Dennett 1995) inherent in the "brain disease model of addiction" (Volkow et al. 2016).

First, consideration of Borsboom et al.'s notions of *rational relations* and *intentionality* highlights the lack of explanatory power of the brain disease model of addiction. Current brain disease model of addiction accounts are able to characterise the molecular, structural, and functional adaptations in distinct brain regions that are correlated with distinct symptoms or "stages" of addiction, that is, multiple overlapping "brain diseases." For example, Volkow et al. (2016) distinguish three recurring stages of addiction, each of which has a distinct neural substrate: (1) binge and intoxication, characterised by rapid learning about the incentive-motivational properties of the drug and associated cues; (2) withdrawal and negative affect, characterised by hyposensitivity of the brain reward system and an exaggerated stress response; and (3) preoccupation and anticipation, characterised by impaired decision-making and inability to resist strong urges.

Borsboom et al.'s notions of *rational relations* and *intentionality* can be applied to make sense of the addict's behaviour and how it is related to, but not fundamentally determined by, the underlying neurobiological changes. For example, regarding *intentionality*, within the "withdrawal and negative affect" stage, one must invoke intentionality in order to understand why the addict uses the drug to manage negative mood (because the drug has provided short-term relief in the past), and why medications that can alleviate withdrawal symptoms, such as nicotine replacement therapy or methadone, can reduce tobacco smoking and opiate use, respectively (Mattick et al. 2009; Stead et al. 2012). Regarding *rational relations*, to give one example, the observed "impaired control" over substance use seen in the "preoccupation and anticipation" stage (stage 3) can be understood as a direct consequence of increased valuation of the drug coupled with reduced valuation of alternatives (to drug use) that characterise stages 1 and 2, respectively (Berkman et al. 2017; Heyman 1996). Thus, there is no requirement to interpret the observed structural and functional changes in prefrontal brain regions as indicative of "impaired ability to resist strong urges" (Volkow et al. 2016). Our point is that attempts to use neurobiological changes to explain behaviour can lead to very misleading explanations that are contradicted by behavioural data.

Second, consideration of intentionality can account for an important observation about the long-term course of addiction: Most addicts eventually recover from addiction, and most of those that recover do so without any treatment (Heyman 2013). If addiction is an acquired chronic brain disease, how can this be so? Demonstrations that addicts are less likely to recover if they believe that they suffer from a chronic disease (rather than, for example, an unhealthy habit that could be overcome; see Eiser & Van der Pligt 1986; Eiser et. al 1985; Miller et al.

1996) make sense when viewed through Borsboom et al.'s framework: Addicts can change their behaviour and give up drugs, but only if their attributions for their addiction permit them to do so.

Finally, we suggest that the current "opioid crisis" in the United States provides a pertinent demonstration that addiction can be *primarily* determined by the broader social, environmental, cultural, and historical context (cf. Hart 2013). The origins of this crisis coincided with the de-industrialization, economic decline, and urban decay in the "Rust Belt" and Appalachian regions (Quinones 2016). Together with alcohol poisonings, suicide, and chronic liver disease, increasing death rates from opioid overdose occurred among middle-aged, white, non-Hispanic men and women of low educational levels – the so-called "deaths of despair" (Case & Deaton 2015). There is also a strong inverse correlation between levels of "social capital" in United States counties and age-adjusted drug overdose mortality (Zoroob & Salemi 2017). More generally, there is evidence that deaths and emergency department visits related to opioid use vary with macroeconomic conditions (Hollingsworth et al. 2017).

It could perhaps be argued that these variables exert their effects on rates of addiction merely by increasing the prevalence of drug use, so that more people are susceptible to the brain changes that then lead to the development of addiction. But in our view it is far more likely that the variables in question are significant elements in the kind of broad causal network that Borsboom et al. describe. For example, "people discover that opioids are an excellent short-term balm for existential maladies like self-loathing, emptiness, erosion of purpose, and isolation. Years of heavy use condition people to desire drugs at the first stab of distress" (Satel &

Lilienfeld 2017). So, too, the easy availability of opioids, whether by prescription from local medical practitioners or through the skillful marketing of illicit suppliers (Quinones 2016), make attempts at behavioral change less likely, and relapse (if change is attempted) more likely to occur. The overarching point is that these broad contextual determinants should be regarded as part of the casual nexus of the disorder of addiction, not merely as "social factors" that might moderate the expression of an underlying brain disease.

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[COMP: DO NOT SET REFERENCES FROM HERE; USE CONSOLIDATED REFERENCES REVISED FILE]

<RFT>**References** [Matt Field, Nick Heather, and Reinout W. Wiers] [MF]

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