



Re-socializing the Vulnerable Brain: Building an Ethically Sustainable Brain Disease Model of Addiction

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According to the brain disease model of addiction (BDMA), substance addiction is a chronic, relapsing brain disease. The BDMA is currently influential in informing addiction policy and the development of new treatments, but remains highly controversial across the addiction research community. We draw on resources from philosophy of science and applied ethics to re-examine the methodological and ethical implications of the BDMA and offer a new forward-looking and constructive conceptualization of the BDMA as a heuristic reductionist research hypothesis. We argue that this not only allows a sharper delineation of the empirical shortcomings of the BDMA, but also helps skeptical social scientists appreciate and incorporate the empirical successes of the BDMA to a broader, social understanding of addictions. We apply this view to the ethical implications of the BDMA, especially to the key concept of vulnerability. The BDMA states that it is the brain that has been hijacked by the drug and the brain thus compels the individual to act in ways that are often disastrous for the individual. The proponents of the BDMA ascribe “vulnerability” to multiple levels of organization, such as genes, specific neural systems, the brain, and the person, thus resulting in confusion and highly problematic ethical, social and even legal implications. The BDMA locates the vulnerability firmly within the individual and treats it as a matter of susceptibility to changes in brain chemistry. This may well be a part of the phenomenon, but the kind of vulnerability relevant for understanding and treating addiction is, however, embedded in normativity, as it concerns the agency of individuals with addiction. Agency is not simplistically reducible to the competencies of the individual, but rather it is also constituted in the interactions with their environment.

Keywords: brain disease model of addiction, reductionist heuristics, vulnerability, localization, agency

INTRODUCTION

The human brain is susceptible to psychoactive substances. This fact has been widely acknowledged and utilized in medicine, recreational activities, spiritual experiences, as well as in the enhancement of physiological and psychological performance (see e.g., Goodman et al., 2007). As the human brain’s susceptibility to such substances is shared by all human kind, societies have felt the need to control the substances in various ways (e.g., establishing agencies, such as European Monitoring Centre for Drugs and Drug Addictions). With psychoactive substances, there is a risk of abuse and misuse. The pharmacological effects of psychoactive substances often lead to using more than

initially intended or using a prescribed substance to purposes not covered by the original purpose of the prescription. As such misuse can result in substantial harm, control, and governance is often warranted. In fact, one of the motivations for control stems from the fact that individuals are in danger of losing control in drug use, i.e., becoming addicted (e.g., Manchikanti et al., 2010). While we acknowledge that the concept of addiction is contested, historically contingent and that addictive behaviors display significant cross-cultural variation, in the following we adopt a suitably loose and broad understanding of addiction as a persistent problem in self-control related to substance use possibly resulting in harm to the agent.

Addiction has been viewed as one of the undesirable consequences of psychoactive substances, no matter what the original purpose of the consumption has been. However, the nature of addiction is contested; there are various competing and complementary views on the condition, ranging from a non-pathological condition grounded on subjective preferences to an agency-deleting disease. Whilst each view has its own challenges and strengths, one account has received a great deal of attention (and funding). The brain disease model of addiction (BDMA) states that addiction is a chronic relapsing brain disease. This immediately suggests that the right response to addiction is the development of pharmaceutical and other medical interventions for the destructive condition that is portrayed in the model. Although substance use is initially voluntary, the pharmacological effects in the brain reduce the individual's ability to control her own behavior (e.g., Leshner, 1997). Whether or not this is factually so, the view has nonetheless ethical, legal and social implications that need scrutiny. In what follows we will focus on the brain disease model of addiction, as it has been argued to be the view that is shared by *scientists* (Animal Farm, 2014). Although neuroscientific (especially dopaminergic) perspectives have also gained prominence in the scientific and public understanding of behavioral addictions, most notably of pathological gambling, we focus here on substance addictions, as these are the phenomena to which the full BDMA has been most explicitly formulated (for a neuroscientific model of pathological gambling, see, e.g., Potenza, 2008).

Nevertheless, the full scientific picture is not that simple. BDMA is a deeply contested model (Hall et al., 2015; Heather et al., 2018)—despite its proponents' insistence on its prominence in the US or even worldwide (Animal Farm, 2014)—and it faces many empirical and normative challenges. Despite its original rationale of reducing the moralistic stigma associated with addiction (e.g., Leshner, 1997), the BDMA is seen as empirically and ethically problematic in downplaying the role of social and cultural context in creating and sustaining addiction. Existing surveys suggest that among scientists and clinicians, the model is usually endorsed only with a string of caveats about ignored contextual factors attached (Bell et al., 2014; Barnett et al., 2018). The empirical and normative problems of the model arise out of the fundamental difficulties in integrating the ever-increasing empirical findings about altered brain functionality to the broader social scientific bodies of knowledge about the social

reality and the lived experience of people using psychoactive substances.

This tug-of-war between the neurobiological and sociological perspectives on addiction is but one front in the broader re-negotiation of boundaries of expertise and authority on normatively contested behavior brought about by advances in neuroscience (e.g., Pickersgill, 2013; Rose and Abi-Rachid, 2013). However, in few other areas of application has the neuroscientific picture gained such prominence in the public as well as in the governing sphere (in the United States) as in addiction research. In what follows, we consider these fundamental challenges from the perspectives of philosophy of science and applied ethics, in that order.

DIAGNOSING THE BDMA

The brain disease model of addiction was “launched” as a specifically scientific way of understanding addiction by the then director of the National Institute of Drug Abuse (NIDA) Alan Leshner (1997). BDMA was positioned explicitly as an alternative to the stigmatizing view of addiction as a personal moral failure and as a political tool for securing better access and insurance cover for treatment, as well as for influencing public opinion and legislation (Dackis and O'Brien, 2005). Underneath the two main components of “disease” and “the brain,” the model is in fact more like an umbrella term that covers various alternative and often competing neuroscientific models that focus on habits, learning, pleasure, and incentive salience. As a poignant symbol of the prominence of the BDMA within the institutions responsible for control and treatment, the directors of the NIDA and the National Institute on Alcohol Abuse and Alcoholism have provided an integrative neurobiological account that attempts to combine all of these mechanisms into a coherent whole (Koob and Volkow, 2016).

The model is based on diverse experimental and physiological evidence. The results of animal models on self-administration of drugs and of neuroimaging studies on individuals with drug addiction support the idea that dopamine activation plays a key role in the “addicted brain,” especially in the mesocortical, mesolimbic and mesostriatal pathways (Volkow et al., 2011). The model is also supported by genetic research on addiction in twin-studies, not only demonstrating the (population-level) heredity of the increased risk of addiction, but also suggesting corresponding biological pathways from the genome to behavioral patterns (Kendler et al., 2012).

The opponents of the BDMA nevertheless deride the model as being a dogma upheld more by appeals to authority rather than by solid empirical evidence (Satel and Lillenfeld, 2014). They, for instance, refer to epidemiological evidence that most people “mature out” of problematic patterns of substance use on their own (Heyman, 2009; e.g., Granfield and Cloud, 2001), and suggest that addiction as such does not amount to a (medical) problem, but that its problems are mostly due to comorbidities (e.g., Pickard and Pearce, 2013). The agency-deleting disease model is also hard to reconcile with the fact that most people with problems related to substance use do nevertheless respond to

even small changes in incentives or personal situations (Heyman, 2009; Satel and Lillenfield, 2014), and that only a subset develop these problems in the first place. More generally, and what is most important for our present purposes, BDMA is accused of leading to an impoverished and reductionist understanding of addiction, devoid of important social and cultural contextual variables (Fraser et al., 2014; Reinerman and Granfield, 2015). Although we agree with most of the opponents' criticism, the problem with this confrontational attitude is that it also renews and maintains the barriers between the vast bodies of empirical understanding about the social and societal factors relevant for understanding addiction and recovery, and the neuroscientific insights about the sub-personal mechanisms relevant for understanding the self-regulation of our behavior. We take our intervention to be timely as the newly launched European Addiction Theory–network positions itself explicitly in opposition to the BDMA (Heather et al., 2018).

Fenton and Wiers (2017) correctly identify the BDMA as actually consisting of a cluster of empirical hypotheses and that, given the evidence at hand, the jury should still be out on all of them. Their take is that, at the moment, evidence seems to point that the full BDMA does apply to a small segment of people with substance use problems, but that most within this population fall short on what the BDMA seems to entail. Although in broad agreement with the assessment of Fenton and Wiers, we now present an alternative way of conceptualizing the BDMA. We take the most constructive way of understanding the BDMA not simply as (a set of) empirical hypotheses, but as a *strategic* simplification—a *heuristic reductionist* hypothesis. We will next show how the BDMA can be seen as a natural consequence of applying a particular set of research heuristics aimed at understanding complexity: first try to understand the phenomena by treating it as a sum of the properties of its parts and then learn about the role of context, organization and interaction by looking at the specific ways in which this initial simplifying hypothesis *fails*. The question of whether the BDMA is “true” or “false” is therefore transformed into whether and when it is false in an informative way.

We do not claim that this is what the proponents of the BDMA have in mind (on the contrary, we take the proponents to fully intend that the BDMA is the final scientific verdict on the matter). Instead, we propose this as a forward-looking way of repositioning the debate—one that also helps the opponents of the model to appreciate more what has been learnt about the constitution of agency in addiction by looking inside the heads of individuals with addiction. Whereas, much of the earlier criticism of the BDMA has concentrated on the agency-deleting aspect (the disease component), our conceptualization additionally explicates the empirical and normative limitations of the reductionist localization aspect of the model (the brain component).

The concept of reductionist research heuristic is taken from the work of philosopher of science William Wimsatt, who has studied the epistemic strategies used for untangling and understanding (especially biological) complexity (2007). Although much of his work has been about explicating the fallacies arising from reductionist hypotheses, he nevertheless

insists that such false hypotheses are useful, even necessary. The reason is that it is epistemically useless to approach complexity holistically, by simply acknowledging it or by amassing observations without any roadmap for putting these observations into a coherent whole. Reductionist hypotheses, although literally false, are useful in that by looking at *the way* in which they fail to empirically account for the full richness of the phenomenon studied provides further hypotheses about *the way in which* the whole is more than simply a sum of its parts. The necessity of such simplifying working hypotheses is deeply ingrained, although often implicit, in the training of natural scientists, but may be a more alien mindset to many social scientists. We hypothesize that the failure to recognize this insight is a major factor in the skepticism toward neuro-centered perspectives on addiction, especially among researchers with a more social scientific or anthropological orientation.

Adapting this key lesson to our context, we hold that pointing out that a social-behavioral phenomenon, such as addiction is complex, context sensitive, historically contingent and socially embedded (e.g., Reinerman and Granfield, 2015), is not, as such, yet a deep insight about the phenomenon. It is more of an admission that the phenomenon is not yet well-understood. As Derek Heim (2014, 40), along with 94 signatories, puts it in his response to the prominent BDMA editorial in *Nature* (2014), “Addiction is too complex to be fought on a medical-research front alone. A variety of approaches based on diverse levels of analysis is required.” Again, few would disagree with this statement. The real challenge is in figuring out the form of complexity, the dynamics of the different aspects, ways in which context matters, and when and how the extrinsic social and environmental factors influence the phenomenon. The challenge is all the more daunting in a case, such as addiction, in which the biological, and the social are so obviously intertwined in the treatment and regulation of the phenomenon. Answering these challenges, and moving beyond the *mere acknowledgment* of the interconnectedness of the brain, the body and the social, requires epistemically strategic simplifications¹—a point often misunderstood and overlooked by the critics of the BDMA. Our modest contribution to answering these challenges is the suggestion that we should treat the empirical failures of the BDMA as opportunities to learn more about the *form* of the interaction between the neurobiological and the social. We will next delineate the empirical failings of the BDMA as consequences of reductionist heuristics, and point to ways in which these failings could serve as empirical entry points for understanding the interaction between the biological and the social.

The BDMA is a reductionist hypothesis on two levels. First, it conceptualizes the problematic behavior of the individual as

¹Behavioral analytical perspectives on addiction utilizing economic reasoning (e.g., Rachlin, 2007; Ainslie, 2013) are a good example of a successful integration of the neuroscientific, behavioral, and to a modest extent also social dimensions in a tractable manner. Nevertheless, even these models and associated experimental designs are subject to the reductionist fallacies delineated below. Furthermore, in practice the piceconomic models emphasizing the dopaminergic system end up having much of the same problematic ethical implications as “standard” BDMA, outlined in the latter sections.

being exclusively caused by “abnormalities” or “dysfunctions” in specific brain systems, caused in turn by continuous substance use (Vrecko, 2010). The main locus of addiction-within-the-brain has long been thought to be the mid-brain dopaminergic system realizing a reward-based reinforcement learning mechanism (for criticism, see Nutt et al., 2015), but the current picture now includes other systems and functionalities as key components of addictive behavior (for a review see Volkow and Morales, 2015, Koob and Volkow, 2016). Functional and physiological correlates for compromised top-down executive control, skewed cue salience, and reduced ability to handle stress and anxiety, now figure prominently in the neuroscientific understanding of addiction (Koob and Volkow, 2016). These empirical advances show that the reductionist strategies have proven powerful in disentangling the complex organization within the inner mechanisms regulating our behavior; the empirically exposed limitations of the original “reward-system” model of addiction, localizing addiction to the mid-brain dopaminergic system, have provided opportunities to learn piecemeal lessons about the role of other systems, such as the consequences of the deterioration in the handling of internal and external stressors. Nevertheless, even the current model is clearly “incomplete” in not yet including neural mechanisms responsible for, for example, false beliefs induced by motivated reasoning or representations of long-term goals. Note that this is not intended as a criticism of the current model, since simply pointing out that such things are important is not very constructive until we have learned in *what way* they should be incorporated into the full picture of the neurology of addiction.

Second, and much more problematically, BDMA conceptualizes addiction as a stable property of the individual. The locus of the problems in the alignment of short-term desires and long-term goals, commitments to others, and the normative and legal expectations of the society, is taken to be a specific deficit in individual agency, caused by the use of a foreign psychoactive substance or excessive engagement in an addictive behavioral pattern, such as gambling. This second reductionist localization hypothesis probably seems so obviously correct to the proponents of the brain disease model that its nature as a drastic simplification is anything but transparent.

Nevertheless, we hold that also this second localization step is also literally speaking false. Addiction is a phenomenon of self-control and agency, and agency, as such, is also constituted by the social relations we have with each other (e.g., Mackenzie and Stoljar, 2000; see also Jennings, 2016; Enfield and Kockelman, 2017). Coherent and meaningful agency or *autonomy*, i.e., acting according to *our* long term goals, reflectively endorsed beliefs and self-identity, is possible and meaningful only with the help of social scaffolding and socially distributed strategies of self-control (Enfield, 2017, Stoljar, 2018), as well as of the socially provided symbolic resources for envisioning possible alternative courses of action and for constructing a narrative self, capable of holding time-consistent plans and projects (McConnell, 2016).

The much criticized *empirical* shortcomings of the BDMA are direct consequences of the predictable and, in a sense, unavoidable biases of these reductionist simplifications. First, BDMA suggests that explanations for puzzling social behavior

should first be looked for inside the individual agent and any such inner difference-makers found should be regarded as “the essence” of the behavior (Wimsatt, 2007, 348). Within the individual, this suggests that addiction “boils down” into a dysfunction of a specific subsystem, such as the hysteresis of the dopamine system with its associated biomarkers. This one true cause of the behavior is also taken to be particularly “real” in being robustly physical. The immediate, and mistaken, implication is that the most effective strategy for influencing the phenomenon is to intervene on this inner essence. As Nora Volkow has stated in an interview, “we will be treating addiction as a disease, and that means with medicine.”²

The idea of an inner essence of addiction leads to default biases in the explanation of related social problems and comorbidities: if and when individuals with addictions exhibit a clustering of different social and health problems, the default explanation is that the inner essence is the common cause of these problems. For example, the concurrence of heroin and amphetamine use in many US cities is by default taken to result from the common neurological vulnerability, rather than from the fact that draconian anti-vagrancy laws force homeless individuals with heroin-dependence to use amphetamines to stay awake and avoid confrontation with the law—a structural cause (Langegger and Koester, 2017).

A more subtle bias resulting from these localization steps is the default assumption that all the relevant interactions respect the agent-environment boundary (“interface determinism”) (Wimsatt, 2007, 348). For example, the current integrated neuroscientific model seems on the outset to take into account many context sensitive aspects of addictive behavior by emphasizing, e.g., the neurological basis of skewed incentive salience and cue reactivity (Jasinska et al., 2014) and the importance of environmental stressors in compromising top-down executive control in terms of physiologically measurable changes in cortical connectivity and the amygdala (Kwako and Koob, 2017). What falls beyond this localization is that agency itself is distributed into the wider social context and that agency-undermining vulnerabilities can arise directly from changes in these social relations (see e.g., Anderson, 2017). For example the draconian anti-vagrancy laws that ban loitering in public and thus deprive the individual basic human rights of homeless people to be able to rest (Langegger and Koester, 2017). This turns into a vicious circle for such vulnerable individuals, as their attempts to reduce the need to rest with meth they further dispose themselves to other agency-undermining vulnerabilities, e.g., sleep deprivation.

Localizing the problem to be within the agent leads one to ignore or downplay observations of historical and interactional patterns. If addiction is a chronic brain disease, then all information about the etiology of the condition is screened-off by the resultant changes in brain functioning, i.e., knowledge of the history of the addictive behavior is relevant only to the extent that it helps in diagnosing what is wrong with the agent (or her brain) here and now. This rules out the possibility that the

²Nora Volkow, interview by Jeneen Interlandi, “What Addicts Need” *Newsweek* February 23, 2008.

problematic behavior is, at least partly, due to the development of behavioral patterns detrimental to agency (within a particular social context), but which do not necessarily correspond to any “pathological” changes in the brain. Heavy substance use is often accompanied with ways of organizing one’s life and friends facilitating the use, and it is often as hard to quit these as it is to quit the consumption of the substance itself. In contrast, many frameworks for understanding addiction not based on the BDMA emphasize the systematic differences in the histories of different kinds of problems of self-control, differences which are very much relevant to whether treatment should be considered, and what form this treatment should take. A prominent example in the context of behavioral addictions is the “pathways model”—typology of problem gamblers, which takes the history of the problematic gaming behavior into account—with implications to effective treatment (Blaszczynski and Nower, 2002).

The two localization steps also have pronounced effects on the experimental designs used to study addictions. The reductionist hypotheses imply a preference for experimental designs with constant environmental variables and manipulation of the intrinsic states of the subject, over designs in which environmental variables are manipulated (Wimsatt, 2007, 349–350). This makes the BDMA partly self-vindicating. The classic paradigm of drug self-administration in rats is a good example of this bias: controlling for the experimental environment produced strongly biased results concerning the behavioral consequences of self-administration, whereas as soon as the animals had access to meaningful social interaction with their fellows, they stopped “choosing” self-administration of even highly “addictive” substances, such as cocaine or heroin (For instance Solinas et al., 2008, see also Alexander et al., 1978, Alexander et al., 1981). When talking about neuroscientific experiments on human subjects, it is, of course, prohibitively difficult to devise meaningful designs which would not impose drastic controls on the environment. It is simply not feasible to, for instance, scan the subject’s brain across different social contexts of substance use. Nor are we aware of studies correlating physiological changes in the brain to substance use across different social contexts.³

Agency is also a *folk-psychological* concept: we are agents in virtue of our behavior being conceptualized in terms of *beliefs* and *desires*. By folk-psychology (or common-sense psychology) we refer to the set of more-or-less automatic capacities and practices used to interpret and predict the behavior of others and ourselves in terms of internal “mental states” like beliefs and desires (Stich and Ravenscroft, 1994). One of the main reasons for the endless controversies in theorizing addiction is that addictive behavior does not fit neatly into this folk-psychological template for understanding human behavior. One of the key insights into addiction, based partly on the neuroscientific picture of the reward system, has been that addictions really do decouple wanting from liking: individuals with addictions can be, at least momentarily, motivated to pursue goals that they do not really

value or enjoy. Folk-psychological concepts are also ill-fitted to handle “loss of control” that comes in degrees: we are either in or not in control—our behavior is caused by *our* beliefs and desires—or it is caused by something else entirely. Hence the empirically suspect dichotomy between the “rational” or volitional view of addiction as choice behavior and the agency-deleting view of the BDMA.

The tension between addiction and our folk-psychological understanding of action is one of the key hurdles in constructing an integrated and socially enriched empirical picture of addiction. Our understanding of social interaction is, almost by necessity, based on folk-psychological concepts, whereas the neuroscientific picture of the drivers of addictive behavior does not map neatly unto these concepts. We suggest that *both the empirical failures of the reductionist BDMA hypotheses as well as the mismatches of folk-psychological accounts of action and addictive behavior flag contact points between the biological and the social in need of a new conceptualization*. This should be taken as the heuristic for building a sociologically and neuroscientifically informed model of addiction. The empirical failings also point to potentially informative foci for future empirical research on the interaction between the biological and the social: the possibly self-enforcing feedback between changes in, say, cue salience or affective self-regulation, and changes in the social relations or self-identity; comparison of comorbidities across social contexts; comparison of life-histories and current behavioral patterns within groups of subjects with similar biomarkers associated with heavy use, to name a few. We return to these tentative suggestions in the concluding remarks.

VULNERABILITY AND AGENCY

We will now move from the purely empirical to more normatively laden implications of the two reductionist hypotheses by looking at how they mold the way in which individuals’ *vulnerability* in addiction is conceived as one of the important reasons for prevention and treatment. Vulnerabilities in addiction are typically connected to various aspects of drug use and its induced pathologies of the brain (see e.g., NIDA., 2014), but what exactly does this vulnerability amount to? In the psychological literature on psychopathology, there seems to be no general agreement (see e.g., Ingram and Price, 2010) on how to define vulnerability. Yet, a significant corpus of knowledge exists, as researchers are taken to “already know vulnerability when they see it,” and to also be able to identify groups of vulnerable people using mainly pre-theoretic criteria (Ingram and Price, 2010, 5, for discussion on the concept of vulnerability in bioethics, see Rogers et al., 2012; ten Have, 2016). We now question the view that vulnerabilities in addiction can always be traced down to brain pathology, as the BDMA seems to suggest. We argue that the reductionist fallacies localizing agency inside individuals and unto particular neuronal populations have pernicious effects on the identification and treatment of substance-related vulnerabilities.

To give some idea of how the BDMA shapes the conception of vulnerability, let us review the ways in which two leading

³For example, Siegel (2001) reviews experiments studying contextual effects (the presence of cues previously paired with the substance) on the physiological effects of a substance on rats. We acknowledge the difficulties in translating such designs to humans.

figures in the BDMA camp—Koob and Volkow (2016)—use the concept in several points in their review of the neurobiology of addiction. We take this authoritative review to exemplify ways in which vulnerability *can* be unreflectively localized to different levels of organization—unless due care is taken to keep in mind that such localizations are not really valid outside their heuristic use. Koob and Volkow discuss “initial vulnerability” of transduction and transcription factors in addiction-relevant neurocircuitry (Koob and Volkow, 2016, 760) and “molecular basis of vulnerability to relapse” (Koob and Volkow, 2016, 767) not only on neurobiological level, but also on that of the individual, where these vulnerabilities are directly translated into individuals subjected to “greater vulnerability” to addiction and “vulnerability to relapse” (Koob and Volkow, 2016, 761). In using the concept of vulnerability, the article bundles up many levels of explanation: it discusses “genetic and environmental vulnerability to addiction” (Koob and Volkow, 2016, 766), and “[genetic] vulnerability in the human population” (Koob and Volkow, 2016, 768), but also “vulnerability for drug and alcohol use disorder” (768), and more generally “vulnerability to drug use and relapse” (Koob and Volkow, 2016, 768). The authors do not limit their interest to strictly endogenous features and are also interested in environmental factors, but only insofar as they contribute to “resilience against vulnerability” (Koob and Volkow, 2016, 761) and to ways to “influence vulnerability to the development and perpetuation of addiction” (Koob and Volkow, 2016, 764). They claim to do so by identifying features that contribute “to an individual’s vulnerability to addiction” (Koob and Volkow, 2016, 766). Vulnerability is therefore attributed to everything from genes and brain-chemistry to persons and populations. Is it really the same notion or does the meaning vary from use on one level to use in another? Purely descriptive notions relying on statistics and biochemical mechanisms differ in an important sense from notions that refer to socially meaningful action. From a normative perspective, it arguably matters whether we are discussing vulnerability on the level of the population, the individual or that of genes and molecules.

Vulnerability as susceptibility on a genetic and molecular levels is constituted and modified by a range of different predispositions and modifiers, which are often multifactorial. Although detailed analyses of the molecular and genetic susceptibilities are beyond the scope of our paper, what needs to be emphasized is that the relation of these descriptive sub-personal attributes to the normative sphere of individual action regarding substance use is anything but straightforward. According to Ingram and Price, 2010, the core features of psychopathological vulnerability include that it is a stable trait, endogenous and latent in nature, and that stress has a role in “actualizing vulnerability” (Ingram and Price, 2010, 6). What is noteworthy about this is that it, first, firmly locates the vulnerability within the individual. It is a property of the agent in question. Second, the “actualization” of vulnerability implies a deviation from the agent’s normal functioning. Third, vulnerability is a matter of degree: one can be more or less vulnerable due to changes in brain chemistry. This third point can be traced back to a point much discussed in the bioethical literature on vulnerability (see discussion e.g., Mackenzie et al.,

2014; ten Have, 2016; Straehle, 2017): all living organisms are vulnerable in the sense that they are finite and fragile to many forces. Psychoactive substances, by definition, are substances that bring about reactions in the brain and the human brain is therefore inherently susceptible to these kinds of substances. Of course, the intoxication that, say, a suitable dose of opiates brings about in humans may vary from individual to individual, but all human beings become intoxicated after consuming enough opiates. In the same vein, long-term use results in changes in the brain and again there is individual variation. These susceptibilities and changes nevertheless, as such, do not necessarily result in individual harm with the same inevitability as neuronal degeneration in Parkinson’s disease, for instance.

Human brain’s susceptibility to psychoactive substances is therefore a fact that need not be normatively loaded, as it does not in and of itself entail positive or negative outcomes of the use of these substances. In this light, susceptibility of the brain to psychoactive substances does not, as such, constitute a threat to human agency: Our brains react to psychoactives in different ways, depending, partly, on our genetic make-up, the constitution of the substance, dose, and the circumstances in which the anticipated reaction takes place. By consuming psychoactive substances, we are able to intervene and modify the (dys)functioning brain. Opioids take away pain, sedatives relieve anxiety, and stimulants help us to stay focused and alert, and the context partly determines whether those pharmacological effects on human agency result in such desired outcomes. Of course, medical use of psychoactive substances is not and has not been the only purpose of these substances. Be it for a shaman or a raver, psychoactive drugs have also been used as enhancing some desired traits, sources of feeling pleasure and for reaching altered states of consciousness (Goodman et al., 2007) These purposes typically involve different people in different social and cultural circumstances.

If all consumption of psychoactive substances leaves a mark on the brain, research on mere use, and the marks that this use leaves on the brain, does not suffice for something to be identified as an addiction, as not all people lose control over their drug use, even when the use is frequent and heavy. For instance, many chronic pain patients could qualify for this group. They consume opioids for their pain, and this, as such, does not make them individuals who suffer from addiction—even if they are physically (and even psychologically) dependent on the substance that they are using. Their prescription is typically such that it does not (further) undermine the agent’s ability to act in ways in which they see best in the light of their identity and long-term goals. Yet Rehm et al. (2013) have argued that the very fact that current neuroscientific studies cannot reliably differentiate between truly addicted individuals and heavy users (because all the subjects used in the studies have had a long history of substance affecting the brain) as reason to doubt the scientific or even clinical meaningfulness of the distinction. Nevertheless, there seem to be populations of heavy user that do not have the kinds of problems true addiction is likely to bring about (see e.g., Van der Pol et al., 2013). Epidemiological studies show that vulnerabilities are dependent on many demographic, social and historical variables (e.g., Van der Pol et al., 2015). Insofar as addiction is

not automatically developed simply after a certain amount of drug use, it could be the case that long-term effects differ in non-addicted heavy drug users and addicted drug users. Some animal models seem to suggest interesting differences between individuals subjected to heavy use in terms of “compulsive drug consumption that occurs in spite of adverse consequences” (Volkow and Morales, 2015, 715). However, care should be taken in extrapolating these model findings to human agency in that the “adverse consequences” in the form of immediately administered electric shocks are something altogether different than long term problems in the (lack of) control of one’s life (Ahmed, 2018). Willingness to undergo pain for an expected reward hardly tells us inevitably about loss of control.

Nonetheless, most of the neuroscientific science evidence rests on changes in the brain processes of chronic drug users. But one can be physically and psychologically dependent on things that are beneficial for one’s identity and long term goals, like chronic pain patients. The concept of vulnerability should also be seen in this broader normative context, not just in terms of physical susceptibility: it implies that in certain circumstances, the population or the individual are in an increased, or even likely, danger of being harmed and wronged (see Hurst, 2008; Martin et al., 2014). Insofar as the susceptibility and dependency to psychoactive substances actualizes the descriptive vulnerability of individuals and their constitution, it as such does not suffice for automatically amounting to problematic behavior and suffering.

There are no neuroscientific studies (that we know of) comparing the physiological or functional differences between people with heavy substance use patterns and people with addictions. However, we do not take this to be an argument against the distinction. In contrast, we surmise that the very absence of such studies is itself a consequence of a reductionist bias, localization of a systemic function (self-control required for full-blown autonomous agency) to a single neuronal subsystem directly affected by the substance. Yet it is arguably the individual’s self-endorsed agency and the vulnerabilities related to it that matter normatively, rather than the ever-present sub-agentive biochemical susceptibility to substances *per se*. We would therefore, contrary to Rehm et al. (2013), like to see future neuroscience of addiction take the distinction between heavy use and addiction more seriously. This may present quite a challenge to research. Insofar as the research is conducted with animal models, the distinction between heavy use and addiction on the basis of self-control may understandably be difficult to operationalize (these are the same challenges that are discussed in research on decision-making and problem gambling with animal studies, see Ahmed, 2018).

We definitely do not intend to downplay the causal role of the psychoactive substance, however. One could, for example, bring up the increasing problematic opioid use in the US, the opioid epidemic (Global Commission on Drug Policy, 2017b; US Department of Health Humanities Services (HHS), 2018). The mere fact that the number of people who develop problematic opioid use in the US is increasing does not, of course, by itself imply that the only cause of this increase is the drug and its effects in the brain and that no other factor plays a role. Although

the prevailing prescription practices and the heavy promotion of drugs and the active suppression of concerns about over-prescription by the pharmaceutical industry certainly played a key role (Manchikanti et al., 2010; Fischer et al., 2014; Global Commission on Drug Policy, 2017a, 3) has released a position paper in which they state that most addictions in the US opioid crisis start with “diverted supplies rather than among pain patients.” Most prescription opioid users fail to develop addiction (furthermore, heroin, not prescription opioids, has now become the most common *first* opioid). These facts suggest that an exclusive focus on the direct pharmacological effects of the drugs falls short of explaining the crisis (Dasgupta et al., 2018 and Rigg et al., 2018 review institutional and socioeconomic factors explaining geographical heterogeneity in the opioid epidemic). Nevertheless, when weighing in on the crisis in a recent comment in *The Lancet*, Volkow emphasized the importance of the access to treatment and that “medications are the gold standard of treatment for opioid use disorder” (Volkow, 2018, 285). For her, the answer to the epidemic seems to lie not in figuring out why so many people suddenly engage in patterns of opioid use deleterious to their health and autonomy, but in therapy making use of medication “allowing the patient’s brain to heal” (cf. Volkow, 2018, 285).⁴

In sum, the BDMA seems to flirt with an unfair description of vulnerabilities in its conflation of the levels of explanation and in lumping together the different kinds of vulnerabilities that are in play in addiction. The kind of vulnerability we should be interested in here is embedded in normativity, as it concerns the *agency* of individuals with addiction. The reason we are concerned with certain behavioral patterns partially resulting from the use of psychoactive substances is the associated loss of control, impaired agency. But agency is not situated within the brain, or even within the individual. Agency is more than merely the competencies and psychological states of the individual: it is also constituted by the individuals’ interactions with their social and material environment (e.g., Enfield and Kockelman, 2017). Agency, especially pathological agency, is a normative concept and, as such, is socially constituted. We are in control of our own behavior not *in virtue* of having the right sort of psychological processes causing the behavior, but in virtue of our behavior being *appropriately* responsive to reasons and incentives in our social environment. We form intentions about future actions and hold on to them unless a right kind of reason requires us to adjust the intentions (see Holton, 2009). When we are considering vulnerabilities in addiction, we should be considering the susceptibility of this socially upheld responsiveness to reasons to all the consequences of *the practices of using* psychoactive substances, not merely the susceptibility of the brain to the substances themselves.

⁴Very much in line with this statement, NIDA is also part of the NIH HEAL Initiative seeking answers to the opioid crisis on two fronts: improving medication-assisted treatment and researching alternative resources for pain management (<https://www.nih.gov/research-training/medical-research-initiatives/heal-initiative>). Again, this reflects a very individualistic understanding of the epidemic; a brain disease caused by opioids originally taken as pain killers.

REFLECTIONS ON THE ETHICAL IMPLICATIONS FOR PRACTICE

Assumptions within the BDMA have ethical implications on the society and stakeholders, especially for individuals with addictions. When the reductionist hypotheses are bundled together, the individual is deprived of full agency. As Pickard (2013) points out, it is challenging to form effective intentions if one does not believe one is capable of executing those intentions to begin with. Like the rest of us, people with problems in controlling their substance use have a folk-psychological understanding of their own agency and they may well experience frustration and confusion about their own actions. It may be difficult to understand why they fail to act in ways they regard as better and explain why they continue to act in ways in which they know to have grave consequences. In this light, it is reasonable that they may come to question their agential competence, their efficacy for desired action. On the first blush, BDMA may sound like a plausible solution, as it offers a compelling explanation for this behavior: the drug has “hijacked” the brain. This solution, however, seems to come with a high price as motivation has been identified as a key factor in recovery in addiction (see e.g., Sayegh et al., 2017) and the BDMA implies that the agent’s efficacy is lacking. Moreover, the BDMA implies that agency is impaired in a stable manner, grounded in uncontested physiological reality. Consequently, an individual with addiction seeking help may find it impossible to be motivated and form intentions of acting contra-addiction due to BDMA-informed beliefs about his or her condition. Their self-efficacy is likely to be low to begin with and this kind of further “evidence” may run the risk that their beliefs are further confirmed. Depriving agency will also reduce “positive stigma,” i.e., the normative expectations of others, crucial for the social constitution of long-term agency. Some studies suggest that a biological disease-conceptualization of addiction actually increases the social distance kept by other people to those with addiction (Satel and Lillenfield, 2014). If we are not treated as responsive to reasons and incentives by our fellows, we lose a key resource for actually doing so. We can therefore argue that the picture portrayed by the BDMA’s reductionist view questions the agency of individuals with addiction on a very fundamental level and thus amounts to a violation of one of the main ethical principles in biomedicine, namely the principle of respect for autonomy (e.g., Beauchamp and Childress, 2001).

It seems clear that if we took the vocal proponents of the BDMA seriously and applied the model to all cases of problematic substance use, we would end up misdescribing a great number of substance abusers and misusers. To be clear, this is not what happens in the practice of prevention and treatment. Nevertheless, it could still be argued that BDMA in its severe form applies to a sub-population and that these individuals are, in fact, beyond non-medical help. These individuals may find it comforting to understand why they persistently fail to act in ways they would like to and keep on acting in ways in which they accept to have detrimental consequences for them and others. Narrowing down the population regarded as “addicted” may generate yet another problem for prevention and treatment, however. As the severity of the condition is highlighted, it runs

the risk of more and more individuals failing to identify with the symptoms. Consequently, the chances of early prevention and treatment may decrease due to this non-identification with the BDMA. It may even raise the threshold for seeking help, as individuals do not consider their state severe enough to seek help. In the worst scenario they may choose not to seek help at all, as they reject the idea that they are suffering from an agency-deleting disease of the brain, the treatment of which takes place in psychiatric wards. These issues matter on individual as well as on public health scale. People’s willingness to seek help may also depend on the kind of place in which the service is offered (see Lagisetty et al., 2017). The same kind of advice and help may be easier to accept for instance in a safe injection site than in medical facility, even if the personnel have the same training. Vulnerability that one may experience in losing control over one’s drug consumption involves feelings of shame (see e.g., Wiechelt, 2007). These kinds of states are likely to make the individual with addiction even more vulnerable and further complicate addressing the problem and seeking help.

A question can be raised whether BDMA, in its current formulation, creates unfair circumstances to individuals with addiction: Not only does it highlight the automatic aspects of human behavior and thus runs the risk of eating away the agency of individuals with addictions, but also seems to complicate access to help by raising the threshold of what constitutes addiction and by labeling it in a way that may result in individuals not identifying with the condition.

CONCLUDING REMARKS: TOWARD A MORE SUSTAINABLE NEUROSCIENTIFICALLY INFORMED PICTURE OF ADDICTION

Nobody denies that addiction involves changes in the brain—any significant behavioral change probably involves alterations in the brain—and that, in the case of substance addictions, substance use is one important cause of these changes. The challenge is to articulate how these changes in the brain affect, and are also partially caused by, changes in the socially constituted guidance of our behavior in the light of our long-term projects and goals, i.e., agency. According to our proposal, we should look for the empirical shortcomings of the reductionist hypotheses implied by the BDMA for lessons about the persistent problems in self-control related to the use of psychoactive substances—something over and above the truism that these problems are complex, contextual and socially embedded.

A socially embedded neuroscientific picture of addiction should recognize that the contrast between choice and disease is a false dichotomy. Individuals with addictions do respond to incentives and, in fact, the current integrated neuroscientific model (e.g., Koob and Volkow, 2016) already points to the ways in which environmental and inner factors skew decisions toward substance use even when other considerations speak against it. The ways in which addictive substances link environmental cues to motivational states (skewed incentive salience), reduce the

efficacy of representations of long-term goals (executive control), compromise affective self-regulation and increase the role of stress function all point ways in which the folk-psychological template of action should be enriched and modified in the context of use of psychoactive substances.

But most importantly, a neuroscientifically informed model of addiction should not localize agency itself to be a property of the brain. Agency is not a homunculus inside our skulls, waiting to be hijacked by devious substances. We control our own conduct not only by “sheer willpower,” but, perhaps more importantly, by also making commitments to others, by modifying our lived environment so as to make certain courses of actions more “costly” than others, and by re-writing the narratives that define who we are.

Consequently, whereas the BDMA conceptualizes the inner essence of addiction as the primary target of pharmacological and other medical interventions, such as Deep Brain Stimulation (for a review on neuromodulation in treating addictions see Bari et al., 2018), a socially integrated brain perspective would conceptualize medication as a possible additional resource in reorienting and reconstituting socially distributed agency. There is a fundamental difference in conceptualizing medication as restoring “a healthy brain,” and using psychoactive substances to manage oneself and one’s life, so as to facilitate the creation and pursuit of meaningful long-term goals. This broader understanding of “medication” could therefore also remove some of the moralistic skepticism regarding the therapeutic use of the addicting substance itself in helping to regain control of one’s life (such as heroin-assisted treatment, see Ferri et al., 2012).

In the end, even when one regards the advances in the neuroscience of addiction as greatly contributing to our understanding of not only addiction, but also to our understanding of the motivational architecture and self-control in general, the empirical shortcomings and the practical and

ethical costs of the brain disease model of addiction should force us to reconsider the usefulness of the notion. Addiction is certainly not a disease of the brain in the same sense as neurodegenerative diseases are. As addiction as a social phenomenon is affected by the self-understanding of the people involved (i.e., via the “looping effect”), the concept of a brain disease, no matter how well-informed and nuanced, may be too liable for harmful public misunderstandings as to warrant scientific or administrative usage. An empirically and ethically viable neuroscientific picture of addiction should not conceptualize addiction neither as a biological disease nor as localized in the brain. An ethically sustainable neuroscientific picture should focus on tractable (piecemeal) models of the *interaction* (feedback) between the cognitive, affective, and motivational changes brought about by substance use and the lived social environment, and treat cognitive and affective processes within the individual only as one resource among many in the constitution of personal autonomy.

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All authors listed have made a substantial, direct and intellectual contribution to the work, and approved it for publication.

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REFERENCES

- Ahmed, S. H. (2018). Individual decision-making in the causal pathway to addiction: contributions and limitations of rodent models. *Pharmacol. Biochem. Behav.* 164, 22–31. doi: 10.1016/j.pbb.2017.07.005
- Ainslie, G. (2013). Intertemporal bargaining in addiction. *Front. Psychiatry* 4:63. doi: 10.3389/fpsy.2013.00063
- Alexander, B. K., Beyerstein, B. L., Hadaway, P. F., and Coombs, R. B. (1981). Effect of early and later colony housing on oral ingestion of morphine in rats. *Pharmacol. Biochem. Behav.* 15, 571–576. doi: 10.1016/0091-3057(81)90211-2
- Alexander, B. K., Coombs, R. B., and Hadaway, P. F. (1978). The effect of housing and gender on morphine self-administration in rats. *Psychopharmacology*, 58, 175–179. doi: 10.1007/BF00426903
- Anderson, J. (2017). “Vulnerability, autonomy gaps and social exclusion,” in *Vulnerability, Autonomy, and Applied Ethics*, ed C. Straehle (New York, NY: London: Routledge), 49–68.
- Animal Farm (2014). Editorial. *Nature* 506:5. doi: 10.1038/506005a
- Bari, A., DiCesare, J., Babayan, D., Runcie, M., Sparks, H., and Wilson, B. (2018). Neuromodulation for substance addiction in human subjects: a review. *Neurosci. Biobehav. Rev.* 95, 33–43. doi: 10.1016/j.neubiorev.2018.09.013
- Barnett, A. I., Hall, W., Fry, C. L., Dilkes-Frayne, E., and Carter, A. (2018). Drug and alcohol treatment providers’ views about the disease model of addiction and its impact on clinical practice: a systematic review. *Drug Alcohol Rev.* 37, 697–720. doi: 10.1111/dar.12632
- Beauchamp, T. L., and Childress, J. F. (2001). *Principles of Biomedical Ethics*. New York, NY: Oxford: Oxford University Press, p. 102.
- Bell, S., Carter, A., Mathews, R., Gartner, C., Lucke, J. and Hall, W. (2014). Views of addiction neuroscientists and clinicians on the clinical impact of a ‘Brain Disease Model of Addiction’. *Neuroethics* 7, 19–27. doi: 10.1007/s12152-013-9177-9
- Blaszczynski, A., and Nower, L. (2002). A pathways model of problem and pathological gambling. *Addiction* 97, 487–499. doi: 10.1046/j.1360-0443.2002.00015.x
- Dackis, C., and O’Brien, C. (2005). Neurobiology of addiction: treatment and public policy ramifications. *Nat. Rev. Neurosci.* 8, 1431–1436. doi: 10.1038/nn1105-1431
- Dasgupta, N., Beletsky, L., and Ciccarone, D. (2018). Opioid crisis: no easy fix to its social and economic determinants. *Am. J. Public Health* 108, 182–186. doi: 10.2105/AJPH.2017.304187
- Enfield, N. (2017). “Distribution of agency,” in *Distributed Agency*, eds N. Enfield and P. Kockelman (New York, NY: Oxford University Press), 9–14.
- Enfield, N. J., and Kockelman, P. (eds.) (2017). *Distributed Agency*. New York, NY: Oxford University Press.
- Fenton, T., and Wiers, R. W. (2017). Free will, black swans and addiction. *Neuroethics* 10, 157–165. doi: 10.1007/s12152-016-9290-7
- Ferri, M., Davoli, M., and Perucci, C. A. (2012). Heroin maintenance for chronic heroin-dependent individuals. *Cochrane Database Syst. Rev.* 2011:CD003410. doi: 10.1002/14651858.CD003410.pub4

- Fischer, B., Keates, A., Bühringer, G., Reimer, J., and Rehm, J. (2014). Non-medical use of prescription opioids and prescription opioid-related harms: why so markedly higher in North America compared to the rest of the world? *Addiction* 109, 177–181. doi: 10.1111/add.12224
- Fraser, S., Moore, D., and Keane, H. (2014). *Habits: Remaking Addiction*. Basingstoke: Palgrave.
- Global Commission on Drug Policy (2017a). *The World Drug Perception Problem. Countering Prejudices About People Who Use Drugs*. Report. Available online at: http://www.globalcommissionondrugs.org/wp-content/uploads/2018/01/GCDP-Report-2017_Perceptions-ENGLISH.pdf
- Global Commission on Drug Policy (2017b). *The Opioid Crisis in North America*. Position paper. Available online at: <http://www.globalcommissionondrugs.org/position-papers/opioid-crisis-north-america-position-paper/>
- Goodman, J., Lovejoy, P., and Sherratt, A. (2007). *Consuming Habits: Global and Historical Perspectives on How Cultures Define Drugs. 2nd Edn*. London; New York, NY: Routledge.
- Granfield, R., and Cloud, W. (2001). Social context and “natural recovery”: the role of social capital in the resolution of drug-associated problems. *Substain. Use Misuse* 36, 1543–1570. doi: 10.1081/JA-100106963
- Hall, W., Carter, A., and Forlini, C. (2015). The brain disease model of addiction: is it supported by the evidence and has it delivered its promises. *Lancet Psychiatry* 2, 105–110. doi: 10.1016/S2215-0366(14)00126-6
- Heather, N., Best, D., Kawalek, A., Field, M., Lewis, M., Rotgers, F., et al. (2018). Challenging the brain disease model of addiction: European launch of the addiction theory- network. *Addict. Res. Theory* 26, 249–255. doi: 10.1080/16066359.2017.1399659
- Heim, D. (2014). Addiction: not just a brain malfunction. *Nature* 507:40. doi: 10.1038/507040e
- Heyman, G. M. (2009). *Addiction: A Disorder of Choice*. Cambridge, MA: Harvard University Press.
- Holton, R. (2009). *Willing, Wanting, Waiting*. Oxford: Oxford University Press.
- Hurst, S. (2008). Vulnerability in research and health care; describing the elephant in the room? *Bioethics* 22, 191–202. doi: 10.1111/j.1467-8519.2008.00631.x
- Ingram, R. E., and Price, J. M. (Eds.) (2010). *Vulnerability to Psychopathology: Risk Across the Lifespan. 2nd Edn*. New York, NY: The Guilford Press.
- Jasinska, A. J., Stein, E. A., Kaiser, J., Naumer, M. J., and Yalachkov, Y. (2014). Factors modulating neural reactivity to drug cues in addiction: a survey of human neuroimaging studies. *Neurosci. Biobehav. Rev.* 38, 1–16. doi: 10.1016/j.neubiorev.2013.10.013
- Jennings, B. (2016). Reconceptualizing autonomy: a relational turn in bioethics. *Hastings Cent. Rep.* 46, 11–16. doi: 10.1002/hast.544
- Kendler, K., Chen, X., Dick, D., Maes, H., Gillespie, N., Neale, M. C., et al. (2012). Recent advances in the genetic epidemiology and molecular genetics of substance use disorders. *Nat. Neurosci.* 26, 181–189. doi: 10.1038/nn.3018
- Koob, G. F., and Volkow, N. D. (2016). Neurobiology of addiction: a neurocircuitry analysis. *Lancet Psychiatry* 3, 760–773. doi: 10.1016/S2215-0366(16)00104-8
- Kwako, L. E., and Koob, G. F. (2017). Neuroclinical framework for the role of stress in addiction. *Chron. Stress* 1, 1–14. doi: 10.1177/2470547017698140
- Lagisetty, P., Klasa, K., Bush, C., Heisler, M., Chopra, V., and Bohnert, A. (2017). Primary care models for treating opioid use disorders: what actually works? A systematic review. *PLoS ONE* 17:e0186315. doi: 10.1371/journal.pone.0186315
- Langegger, S., and Koester, S. (2017). Moving on finding shelter: the spatiotemporal camp. *Int. Sociol.* 32, 454–473. doi: 10.1177/0268580917701584
- Leshner, A. I. (1997). Addiction is a brain disease, and it matters. *Science* 278, 45–47. doi: 10.1126/science.278.5335.45
- Mackenzie, C., Rogers, W., and Dodds, S. (2014). “What is vulnerability and why does it matter to moral theory,” in *Vulnerability. New Essays in Ethics and Feminist Philosophy*, eds C. Mackenzie, W. Rogers, and S. Dodds (Oxford: Oxford University Press), 1–29.
- Mackenzie, C., and Stoljar, N. (Eds.) (2000). *Relational Autonomy – Feminist perspectives on Autonomy, Agency, and the Social Self*. New York, NY; London: Oxford University Press.
- Manchikanti, L., Fellows, B., Ailani, H., and Pampati, V. (2010). Therapeutic use, abuse, and nonmedical use of opioids: a ten-year perspective. *Pain Physician* 13, 401–435.
- Martin, A. K., Tavaglione, N., and Hurst, S. (2014). Resolving the conflict: clarifying ‘vulnerability’ in health care ethics. *Kennedy Inst. Ethics J.* 24, 51–72. doi: 10.1353/ken.2014.0005
- McConnell, D. (2016). Narrative self-constitution and recovery from addiction. *Am. Philos. Q.* 53, 307–322.
- NIDA. (2014). *Drugs, Brains, and Behavior: The Science of Addiction*. Available online at: <https://www.drugabuse.gov/publications/drugs-brains-behavior-science-addiction/drug-abuse-addiction>
- Nutt, D. J., Lingford-Hughes, A., Erritzoe, D., and Stokes, P. R. (2015). The dopamine theory of addiction: 40 years of highs and lows. *Nat. Rev. Neurosci.* 16, 305–312. doi: 10.1038/nrn3939
- Pickard, H. (2013). Psychopathology and the ability to do otherwise. *Philos. Phenomenol. Res.* 90, 1–29. doi: 10.1111/phpr.12025
- Pickard, H., and Pearce, S. (2013). “Addiction in a context. Philosophical lessons from a personality disorder clinic,” in *Addiction and Self-Control: Perspectives From Philosophy, Psychology and Neuroscience*, ed N. Levy (Oxford; New York, NY: Oxford University Press), 165–189.
- Pickersgill, M. (2013). The social life of the brain: neuroscience in society. *Curr. Sociol.* 61, 322–340. doi: 10.1177/0011392113476464
- Potenza, M. N. (2008). The neurobiology of pathological gambling and drug addiction: an overview and new findings. *Philos. Trans. R. Soc. Lond. B Biol. Sci.* 363, 3181–3189. doi: 10.1098/rstb.2008.0100
- Rachlin, H. (2007). In what sense are addicts irrational? *Drug Alcohol Depend.* 90, S92–S99. doi: 10.1016/j.drugalcdep.2006.07.011
- Rehm, J., Marmet, S., Anderson, P., Gual, A., Kraus, L., Nutt, D. J., et al. (2013). Defining substance use disorders: do we really need more than heavy use? *Alcohol Alcoholism* 48, 633–640. doi: 10.1093/alcal/agt127
- Reinarman, C., and Granfield, R. (2015). “Addiction is not just a brain disease: critical studies of addiction,” in *Expanding Addictions: Critical Essays*, eds C. Reinarman, and R. Granfield (New York, NY: Routledge), 1–24.
- Rigg, K. K., Monnat, S. R., and Chavez, M. N. (2018). Opioid-related mortality in rural America: geographic heterogeneity and intervention strategies. *Int. J. Drug Pol.* 57, 119–129. doi: 10.1016/j.drugpo.2018.04.011
- Rogers, W., Mackenzie, C., and Dodds, S. (2012). Why bioethics needs a concept of vulnerability. *Int. J. Femin. Approach. Bioethics* 5, 11–38. doi: 10.3138/ijfab.5.2.11
- Rose, N., and Abi-Rachid, J. M. (Eds.) (2013). *Neuro: The New Brain Sciences and the Management of the Mind*. Princeton, NJ; Woodstock, GA: Princeton University Press.
- Satel, S., and Lillenfield, S. O. (2014). Addiction and the brain disease fallacy. *Front. Psychiatry* 4:141. doi: 10.3389/fpsy.2013.00141
- Sayegh, C. S., Huey, S. J., Zara, E. J., and Jhaveri, K. (2017). Follow-up treatment effects of contingency management and motivational interviewing on substance use: a meta-analysis. *Psychol. Addict. Behav.* 31, 403–414. doi: 10.1037/adb0000277
- Siegel, S. (2001). Pavlovian conditioning and drug overdose: when tolerance fails. *Addict. Res. Theory* 9, 503–513. doi: 10.3109/16066350109141767
- Solinas, M., Chauvet, C., Thiriet, N., El Rawas, E., and Jaber, M. (2008). Reversal of cocaine addiction by environmental enrichment. *Proc. Natl. Acad. Sci. U.S.A.* 105, 17145–17150. doi: 10.1073/pnas.0806889105
- Stich, S., and Ravenscroft, I. (1994). What is folk psychology? *Cognition* 50, 447–468. doi: 10.1016/0010-0277(94)90040-X
- Stoljar, N. (2018). “Answerability: a condition of autonomy or moral responsibility (or both)?” in *Social Dimensions of Moral Responsibility*, eds K. Hutchison, C. Mackenzie, and M. Oshana (New York, NY; London: Oxford University Press), 231–252.
- Straehle, C. (Ed.) (2017). *Vulnerability, Autonomy, and Applied Ethics*. New York, NY; London: Routledge.
- ten Have, H. (2016). *Vulnerability – Challenging Bioethics*. New York, NY: Routledge.
- US Department of Health and Humanities Services (HHS) (2018). *What is the U.S. Opioid Epidemic?*. Available online at: <https://www.hhs.gov/opioids/about-the-epidemic/index.html> (accessed June 10, 2018).
- Van der Pol, P., Liebrechts, N., De Graaf, R., Korf, D. J., Van den Brink, W., and Van Laar, M. (2013). Predicting the transition from frequent cannabis use to cannabis dependence: a three-year prospective study. *Drug Alcohol Depend.* 133, 352–359. doi: 10.1016/j.drugalcdep.2013.06.009
- Van der Pol, P., Liebrechts, N., De Graaf, R., Korf, D. J., Van den Brink, W., and Van Laar, M. (2015). Three-year course of cannabis dependence and prediction of persistence. *Eur. Addict. Res.* 21, 279–290. doi: 10.1159/000377625

- Volkow, N. D. (2018). Medications for opioid use disorder: bridging the gap in care. *Lancet* 391, 285–287. doi: 10.1016/S0140-6736(17)32893-3
- Volkow, N. D., and Morales, M. (2015). The brain on drugs: from reward to addiction. *Cell* 162, 712–725. doi: 10.1016/j.cell.2015.07.046
- Volkow, N. D., Wang, G.-J., Fowler, J. S., Tomasi, D., and Telang, F. (2011). Addiction: beyond dopamine reward circuitry. *Proc. Natl. Acad. Sci. U.S.A.* 108, 15037–15042. doi: 10.1073/pnas.1010654108
- Vrecco, S. (2010). Birth of a brain disease: science, the state and addiction neopolitics. *Hist. Hum. Sci.* 23, 52–67. doi: 10.1177/0952695110371598
- Wiechelt, S. A. (2007). The specter of shame in substance misuse. *Subst. Use Misuse* 42, 399–409. doi: 10.1080/10826080601142196
- Wimsatt, W. (2007). *Re-Engineering Philosophy for Limited Beings: Piecewise Approximations to Reality*. Cambridge, MA: Harvard University Press.

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