Re-Visioning Psychiatry

Cultural Phenomenology, Critical Neuroscience, and Global Mental Health

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The historian of medicine Charles Rosenberg wrote a few years ago that "[w]e have never been more aware of the arbitrary and constructed qualities of psychiatric diagnoses, yet we have never been more dependent on them than now" (2007, p. 50). This argument could be easily extrapolated from diagnoses to cover psychiatry’s categories and concepts more generally, and it would aptly describe the way in which the notion of addiction as a "brain disease" is simultaneously ubiquitous and highly contested today. In North America and elsewhere, the idea of addiction as a "chronic, relapsing brain disease" serves as a guideline for prioritizing funding for basic research on addiction and substance use and has been adopted as an official definition by influential professional organizations such as the American Society of Addiction Medicine (ASAM, 2011). Closely linked to this concept are claims that addiction is not limited to "substance abuse" or "substance use disorders," but encompasses pathological, harmful, or distressing patterns of gambling, eating, sex, technology use, and other behaviors.

Yet one doesn’t have to look far to find criticisms of these brain-centered accounts or expansive definitions as reductionist, biomedicalizing, or focused on the wrong factors. As mainstream, a publication as the New York Times could in 2014 ask six experts the basic question, "What is Addiction?" and receive six substantially different answers – each emphasizing factors as wide-ranging as poverty, personal choice, genetics, and spirituality ("What is Addiction," 2014). The specifics of distinct definitions, and their attendant critiques of the brain disease model, vary widely. Psychiatrists, psychologists, behavioral economists, and philosophers argue that the brain disease model obscures the role of choice, agency, and social environment in producing the destructive behavioral patterns we associate with addiction (Foddy & Savulescu, 2010; Heyman, 2009; Levy, 2012; Satel & Lilienfeld, 2013). These critics point to the often forgotten finding that many people who use opiates and other addictive drugs habitually for a period of time either "mature out" of heavy use or are able to stop without any therapeutic
intervention (Robins, 1993). Other observers argue that designating addiction as a "disease" or "disorder" medicalizes conditions that are more properly understood as "problems of living," social deviance, or simply the contingent (and sometimes harmful) outcomes of people interacting with particular environments (Keane, 2002, p. 568; Peele, 1985; Reinaman, 2005, p. 308). For many critics, this biomedicalization of addiction obscures patterns of structural violence, particularly in light of the overwhelming associations between addiction-related harm and social inequalities, poverty, and incarceration (Bourgois, 2003; Singer, 2007). Anthropologists, historians, and sociologists have played an important role in this critical discourse, drawing attention to the ways in which framing addiction as a brain disease elides psychological, familial, social, economic, institutional, and global systemic factors.

Until recently, most social scientists working in these traditions have treated the brain disease model of addiction and its forerunners, along with other biological knowledge, either as a "black-box" to leave untouched or as rhetoric, ideology, or social construction to be critiqued. However, over the past several years, a growing number of social scientists studying addiction have begun paying attention to materiality, embodiment, and biology in potentially novel ways. We have seen a growing number of calls for a robust and thoughtful engagement between the social and biological sciences of addiction, or at least for an approach that, as Scott Vreco has put it, "maintains critical ambivalence toward the reality of addiction as a disease, but nevertheless commits to thinking seriously about the physiological" (Campbell, 2010, 2011; Courtwright, 2005; Fraser, Moore, & Keane, 2014; Hansen & Skinner, 2012; Kaye, 2012; Kushner, 2010; Lende, 2005, 2012; Saris, 2013; Singer, 2001; Weinberg, 2011; Vreco, 2010a, p. 38). These calls are part of a broader shift among many social scientists toward various modes of engagement with the biosciences, many of which aim to contribute to biosocial or ecological accounts of health, illness, and disease (Downey & Lende, 2012; Fitzgerald & Callard, 2014; Lock & Nguyen, 2010; Rose, 2013; Slaby & Choudhury, 2012). This reorientation is often linked to an understanding of biological

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1 Throughout the chapter, I draw on the work of social scientists in order to illustrate broader arguments, not in an effort to comprehensively review current research. For useful mappings of the social science literatures on addiction see: Weinberg (2011) on sociology; Kushner (2010) on the history of addiction; and Marshall, Ames, and Bennett (2001) and Singer (2012) on the anthropology of alcohol and drugs. Nicher and colleagues (2004) examine a somewhat wider set of qualitative literatures, with a particular focus on methods and applied or engaged social science, as do Page and Singer (2010).
processes as “porous to social and even cultural signals to an unprecedented extent” (Meloni, 2014, p. 2). And indeed, when we look beyond the headlines highlighting addiction as first and foremost a brain disease, we find many researchers in neuroscience and psychiatry seeking to move beyond potentially reductionist interpretations to investigate the multiple links between biological mechanisms, environmental effects, and developmental histories (Volkow, Wang, Fowler, & Tomasi, 2012).

But what does it mean, concretely, for social scientists to think seriously about materiality and biology while maintaining a “critical ambivalence” toward prevailing notions of addiction as brain disease? Or, to put the question in another way, how might the social sciences of addiction advance a critically productive engagement with neuroscience and psychiatry without either treating biology as only a discursive product or making it the foundation of an ontological hierarchy? Moreover, how might we do so in a way that builds upon the decades of important ethnographic and historical research on addiction?

In thinking about these questions, I have found it particularly useful to return to Gregory Bateson’s theory of alcoholism as a problem of epistemology (1972). In his influential article “The Cybernetics of ‘Self,’” Bateson argues that alcoholics are often unable to maintain sobriety because they identify the “will” or the “self” with conscious mental processes, a misrecognition that is ultimately just an “unusually disastrous variant of Cartesian dualism, the division between Mind and Matter” (1972, p. 313). In conceptualizing their “will” as distinct from and opposed to the “unconscious processes,” which they experience as “urges” and “forces,” alcoholics commit themselves to a struggle with a reified disease entity that can only escalate as their social relationships deteriorate. Indeed, the reason Alcoholics Anonymous (AA) worked, according to Bateson, was that in declaring their “powerless[ness] over alcohol,” its participants were effecting “a change in epistemology, a change in how to know about personality-in-the-world” – turning away from a fatal dualism to a way of knowing and acting with a distinctly different set of assumptions about “mind,” “self,” and “volition” (1972, p. 313).

Some forty years later, Bateson’s argument remains radically suggestive. As I read him (perhaps somewhat idiosyncratically), Bateson urges us to think of alcoholism not only in ecological terms (as a system that encompasses the person and her milieu), but also in a way that recognizes that “ontology and epistemology cannot be separated” in the study of human beings (1972, p. 314). In other words, the ways in which we act in the world (including those experienced as distressful or deemed pathological) cannot be meaningfully disentangled from our knowledge of the
world – or more specifically, our categories and their attendant logics. This basic premise has, of course, been echoed by much of the literature in science studies and the social sciences of knowledge, particularly in the work of Ian Hacking – who, like Bateson, has often turned to ecological metaphors and emphasized the centrality of epistemology in his efforts to reconceptualize relationships between processes traditionally demarcated as “biological” and “social” (cf. Hacking, 2002).

Working from the idea that a biosocial or ecological account of addiction should start with examining “addiction” as a problem of knowledge, I devote the first part of this chapter to a discussion of the brain disease model of addiction as an epistemic object, tracing its emergence from a particular scientific style of reasoning and examining some of its key social effects. In the remainder of the chapter, I briefly review the social science of addiction literature associated with four conceptual frameworks, which I suggest highlight domains and mechanisms of biosocial entanglement – and are thus particularly fruitful as potential sites of conversation between social scientists and bioscientists. These frameworks are: (1) embodied sensations, (2) will and habit, (3) social and material milieu, and (4) trajectories.

A final caveat regarding the conceptual framing of this chapter: while I use the term “addiction” throughout, I do not restrict myself to its “core” behavioral symptoms, as defined by the current psychiatric literature (e.g., compulsive drug-seeking despite severe consequences); rather, I examine the broader terrain within which substances and practices may or may not be understood or experienced as distressful. Although this approach makes for a somewhat more unwieldy examination of the literature, it is necessary in order to understand distressful or harmful patterns and experiences involving psychoactive drugs, if one accepts that any definition of pathology inherently requires some normative claim or stance.

**Addiction as Brain Disease: The NIDA Model**

As historians of science and medicine have shown, today’s “chronic, relapsing brain disease” model is just the most recent of a long series of attempts to conceptualize addiction to alcohol, opiates, or other drugs as a disease (Acker, 2002; Campbell, 2007; Valverde, 1998). Although these models share certain characteristics, they invoke distinct loci and mechanisms of addictiveness and privilege different forms of intervention and lines of scientific research; in addition, all have been shaped both by their contemporary political and social milieu and by the styles of thought prevailing in contemporaneous scientific communities (Berridge, 2013;
Courtwright, 2005; Gusfield, 1996; Vrecko, 2010b). For example, one of the earliest articulations of the disease concept is often attributed to an eighteenth-century Philadelphia physician, Benjamin Rush (Levine, 1978). Rush understood the habitual drunkard’s desire to consume alcohol as a chronic, progressive compulsion that eventually and inevitably led to a loss of control. As with later disease or medical models of addiction, the key point often emphasized about Rush’s framing of alcoholism as disease was its distinction from understanding drunkenness either in moral terms (as “bad behavior,” laziness, etc.) or in terms of individual choice. The historical moment at which this concept emerged was, of course, no accident. During the early industrial period – and increasingly during the nineteenth century (when disease concepts of addiction became much more widespread) – drinking practices were problematized for their perceived incompatibility with the behavioral strictures then valorized, particularly those of self-reliance, independence, and productivity (Levine, 1978; Room, 2003).

Yet, despite echoes of these predecessors, recent claims that addiction is a brain disease are the product of a distinct style of thought, one that emerged from the expansion of basic research on neurochemistry under the auspices of Richard Nixon’s War on Drugs during the early 1970s. Key to this stream of research was the isolation of opiate receptors in nervous tissue in 1973, which helped to consolidate a biomolecular model of addiction by demonstrating a specific mechanism through which drugs could have biological effects (Vrecko, 2010b). During the 1980s and 1990s such basic research on the neurochemical underpinnings of craving and pleasure gained strength from the widespread availability of new imaging technologies that permitted noninvasive study of brain structure and function (Campbell, 2007; Fraser et al., 2014; Vrecko, 2010b).

All of these conceptual, technical, and political developments made possible the consolidation and public articulation during the 1990s of the idea that addiction is a “chronic, relapsing brain disease” linked to the neurobiology of reward, attention, motivation, and decision making (Kalivas & Volkow, 2005; Leshner, 1997). Because the U.S. National Institute on Drug Abuse (NIDA) has been central to research behind its development and recent NIDA directors Alan Leshner and Nora Volkow have been instrumental in its articulation both within professional circles and among the lay public, the brain disease approach has also been referred to as “the NIDA model” (Courtwright, 2010).^2

^2 In this chapter I use the terms “NIDA model” and “brain disease model” interchangeably.
Perhaps most significantly, this model argues that chronic use of addictive substances results in certain neuroadaptations of brain systems involved in reward, attention, and motivation, with enduring effects.

There is an important distinction to make here. The brain disease (or NIDA) model of addiction represents a particular interpretation of experimental findings in neuroscience, and many researchers have used neuroscience to critique the model and propose alternatives (e.g., Levy, 2014). Indeed, as sociologists Suzanne Fraser, David Moore, and Helen Keane have argued, it may be more useful to think of this “model” as a relatively simple narrative, “distilled from the complex neuroscience of drugs and reward” and “strengthened by the appeal of evolutionary logic” (2014, p. 52). Here is a version of the narrative drawn from a review article by Charles Dackis and Charles O’Brien, two leading neuroscientists of addiction:

Addiction is best conceptualized as a disease of brain reward centers that ensure the survival of organisms and species... Given their function, reward centers have evolved the ability to grip attention, dominate motivation and compel behavior directed toward survival goals, even in the presence of danger and despite our belief that we are generally rational beings. By activating and dysregulating endogenous reward centers, addictive drugs essentially hijack brain circuits that exert considerable dominance over rational thought, leading to progressive loss of control over drug intake in the face of medical, interpersonal, occupational, and legal hazards. (2005, p. 1431)

At the center of most versions of this narrative is the neurotransmitter dopamine, understood to modulate circuits in the midbrain associated with desire, pleasure, and reward. However, there is considerable debate among neuroscientists about the specific causal role played by dopamine in reward — and particularly, whether it mediates the pleasurable effects of reward (glossed in the literature as “liking”); the prediction of future reward (“learning”); or “incentive salience,” that is, “the ascription of attractiveness to ‘intrinsically neutral’ stimuli” (Berridge & Valenstein, 1991, p. 9), also described as “wanting” or “desiring” (Berridge, 2007; Berridge, Robinson, & Aldridge, 2009). As this third “incentive salience” theory argues, organisms come to experience desire (“wanting”) in response to stimuli that were initially associated with pleasurable experiences (“liking”) through processes of conditioning (Berridge, 2007). As certain drugs “plug ... directly into the neurobiological mechanism that ordinarily adjusts learned incentive salience in accordance with physiological states” (Berridge, 2007, p. 413), “wanting” becomes “craving,” a response that can be triggered and amplified by the environmental stimuli or cues that have become associated with particular drugs, as well as by stress (Dackis & O’Brien, 2005, p. 1432). Such
“cue-induced craving” is widely understood in this literature as a central cause of relapse after long periods of abstinence, both because the neuroadaptations resulting from chronic drug use are considered long-standing and because the entire process may take place outside the conscious awareness of potential users (Campbell, 2013; Childress et al., 2008). A key aspect of the “incentive salience” hypothesis is that it frames addiction as less a problem of pleasure than one of anticipation or desire. For Robinson and Berridge, “incentive salience” or “wanting” is distinct from the experience of pleasure itself – “liking” – which represents a separate process associated with a different set of brain circuits. Daniel Lende suggests that “incentive salience” may be semantically closer to “passion” than “wanting” (Berridge et al., 2009, as cited in Lende, 2012), adding that this view of addiction has implications for the way addiction fits into a broader interpretation of contemporary consumer capitalism (Lende, 2012, p. 350). That is, a theory of addiction as a pathology of anticipation and desire (rather than pleasure) suggests that addictive experiences may have particularly strong affinities with the central logics and affective textures of consumer capitalism (cf. Saris, 2013; Schüll, 2006, 2012).

One of the most significant effects of the NIDA model has been to fuel the expansion of “addiction” as a cultural idiom for understanding multiple pathologies and forms of distress. For many addiction researchers (as well as laypeople), the model’s emphasis on the interaction between drugs and basic neurobiological pathways has validated the idea that “various drug dependencies should be conceptualized as a single disorder” (Dackis & O’Brien, 2005, p. 1433). Not only has the model fostered the grouping of alcohol, tobacco, opiates, and many other intoxicants under a single conceptual rubric, but it has brought them together with pleasurable activities such as gambling, video gaming, sex, and eating (Petry, 2006; Volkow & O’Brien, 2007). This idea is based on the finding that such activities activate the same brain circuits as drugs of addiction and that they may do so intensely enough to be subject to the same processes of dysregulation, leading to “compulsive drug-seeking” (Dackis & O’Brien, 2005). Although some scholars have interpreted the

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3 There is, of course, much more to this model. Drug reward is understood as associated with other neurotransmitters, in addition to dopamine (Volkow et al., 2012). Many neurobiologists also argue that addiction is associated with reductions in metabolism in the prefrontal cortex and, in the case of certain drugs, “reductions in frontal gray matter density” leading to disruptions in the decision-making and risk assessment capacities often glossed as “executive function” (Dackis & O’Brien, 2005, p. 1432; Volkow et al., 2012).
expansion of addiction-logics as another instance of biomedicalization, others have argued that in broader cultural terms, it may not only normalize pathology (by emphasizing its foundation in basic biological processes rather than ascribing it to characteristics of particular individuals or kinds of people) but also render everyone potentially pathological (Rose, 2003; Schüll, 2006). Despite the continued debates and marked skepticism of many researchers, the idea of behavioral addictions received institutional legitimation with the recategorization of compulsive gambling as an addiction in DSM-5 (APA, 2013).

However, the relationship between the NIDA model and addiction diagnostics and therapeutics remains profoundly ambivalent. The description and categorization of addictions in the DSM-5 reflect both the ambition of its developers to bring nosology closer to neurobiology (through the use of diagnostic biomarkers) and its ultimate failure to achieve this goal. On the one hand, the newly formed category of “Substance-Related and Addictive Disorders” consists of brain disorders; this overall framing echoes the idea of addiction as a disorder independent of substances (Fraser et al., 2014). On the other hand, the most significant change between DSM-IV (APA, 1994) and DSM-5 – the erasure of the distinction between substance abuse and dependence, and the replacement of both categories by the heterogeneous category, “substance use disorder” – was justified as much on the basis of the categories’ social effects as it was on their neurobiological validity (Fraser et al., 2014, pp. 37–45).

The brain disease model’s relationship to addiction therapeutics is similarly equivocal. The rise of the brain disease model been accompanied by a growing enthusiasm for new pharmaceutical treatments for addiction, as well as for interventions involving neurological modulation (such as deep brain stimulation and transcranial magnetic stimulation). Unlike older pharmacological treatments such as disulfiram, which was used as a tool in an essentially behavioral intervention for alcoholism, newer drugs such as naltrexone and acamprosate are understood to directly modulate the neurochemical effects of substances or to reduce sensations of craving (O’Brien, 2005). Similarly, buprenorphine, used in the treatment of opiate dependence since the mid-1990s in a number of countries, was specifically developed as a opiate-substitution therapy

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4 Such debates over the validity of behavioral addictions point to another important effect of the “chronic, relapsing brain disease” model of addiction: whereas earlier models emphasized a distinction between “physiological” and “psychological” addiction, or attempted to encompass the two, the NIDA model arguably blurs any clear distinction between these categories.
because its biochemical properties were understood to reduce its potential for dependency, abuse, and "diversion" – problems that had become evident with methadone maintenance therapy since the beginning of its widespread use in the 1970s (Campbell & Lovell, 2012; Meyers, 2013). Simultaneously, many therapeutic and recovery fields throughout the world that are dedicated to addiction employ a dizzying range of psychosocial, talk, and behavioral therapies, which may encode models of personhood and enactments of agency that differ radically from those associated with pharmaceutical interventions. For example, although treatment modalities in North America are legion, none have had the cultural impact nor attained the prevalence of the Twelve Step program and numerous related modes of talk-based therapy, which, as Summers' Carr has argued, share a conception of addiction as a "disease of denial" (Carr, 2013).

In focusing on the effects, consequences, and receptions of the brain disease model, I have perhaps refuted it unfairly, paying less attention to instances of profound disagreement, internal critique, and acknowledgment of uncertainty by neuroscientists of addiction. And yet, as Nancy Campbell has pointed out, while most researchers are quite frank about the enormous gaps that remain in explaining mechanisms linking neurobiology to social behavior in compulsive drug use, "there is a persistent gap between what scientists humbly admit to one another and what the public understands them to be saying" (2011, p. 207). Arguably, this gap is at least partly the product of prominent neuroscientists' assumptions that public acceptance of a neurobiological model of addiction will promote rehabilitative and therapeutic (rather than penal and coercive) approaches to intervention and will lead to reduced stigma and, ultimately, to fewer harmful health outcomes for patients (Campbell, 2013). Of course, these aspirational claims sit quite uneasily next to empirical findings of deeply ambivalent social effects of framing addiction in neurobiological terms (Buchman, Illes, & Reiner, 2011; Pescosolido et. al., 2010; Satel & Lilienfeld, 2013), not to mention the prevailing modes of regulating narcotic drugs in many countries, where criminalization and mass incarceration seem to go hand-in-hand with the acceptance of addiction disease models by criminal justice professionals (Courtwright, 2010; Garriott, 2011; Kaye, 2012).

**Biosocial Entanglement and Ecologies of Addiction**

Even if we set aside the usefulness of such "strategic reductionism," the more vexing question remains of how to link knowledge about neural processes to our explanations of the actions and experiences associated
with addiction, and how (if at all) these explanations should guide our interventions. It is sometimes pointed out that even Alan Leshner, while famously arguing that “addiction is a brain disease, and it matters,” explained that, in fact, it is “a brain disease for which the social contexts in which it has both developed and is expressed are critically important” (1997). Indeed, researchers building on this model increasingly take into account other internal and external factors shaping addictive behavior, including genes, development, stress, early trauma, social and physical environmental cues, and comorbidity with other psychiatric disorders (Volkow et al., 2012).

However, there is a significant difference between (1) conceptualizing a set of “core” biological processes or “single biological essences” (Kendler, 2012, p. 11) that “develop” and are “expressed” in particular social contexts and (2) thinking about the social and biological as fundamentally inextricable. Even the NIMH’s Research Domain Criteria (RDoC) system of classification, which begins not with a category of “addiction” or “substance abuse,” but at the level of “positive and negative valence systems,” such as reward learning and habit, has been criticized for its inadequate attention to social processes in their own right (as opposed to neural correlates or preconditions for social processes) (Kirmayer & Crafa, 2014). If we understand “addiction” to be a name we give to a set of affords, experiences, and patterned actions that are “emergent” in interactions taking place across different levels of complexity between brain circuits, substances, self-systems, social networks, and markets (to name a few), then we need to pay closer attention to the specific mechanisms through which these interactions take place (Kirmayer & Gold, 2012; Lock & Nguyen, 2010, p. 90). To rephrase it as a question: If we think of addiction not as a brain disease, but as a phenomenon that emerges under a particular set of biosocial and institutional conditions, what do we need to pay attention to?

In the remainder of this chapter, I briefly review the social science of addiction literature associated with four conceptual frameworks or problem spaces, all of which highlight the domains and mechanisms of biosocial entanglement: (1) embodied sensations, (2) will and habit, (3) social and material milieu, and (4) trajectories. These may seem to unlikely categories. Where are the substances? Where are individual psychologies? Where are structural inequalities? As will become clear, I have chosen these categories precisely because they cut across some of the other frameworks, hopefully putting them into conversation with one another. Rather than a systematic review of social science research or an overarching model of addiction, these categories represent conceptual and discursive spaces where social scientists and bioscientists
(and particularly neuroscientists) working on addiction might productively communicate and engage.

**Embodied Sensations**

The embodied sensations and states of consciousness associated with both the habitual use of psychoactive substances and what some researchers understand to be the “core” behavioral components of addiction (such as “cue-induced craving”) are an important domain where the social sciences can both speak directly to and potentially develop arguments emerging from the neuroscience of addiction. Anthropologists and sociologists have emphasized that the phenomenology of drug use and addiction is not determined solely by the psychochemistry of ingested substances or (in the case of gambling and gaming) by the characteristics of immersive technologies. Rather, a range of perspectives suggests that embodied sensations and states of consciousness are coproduced or mediated by the interaction of these material properties and expectancies, conceptual models of the body, somatic modes of attention, and bodily memories, which are themselves the instantiations of social values, cultural scripts, and patterns of experience (Csordas, 1993; Hinton, Howes, & Kirmayer, 2008). In part, this complexity occurs because many psychoactive substances simultaneously affect multiple circuits in the nervous system, so users must learn to interpret their drug-related sensations and experiences in particular ways. Howard Becker made such a claim years ago when he argued that one becomes a marijuana user by learning to pay attention to certain sensations and subjective experiences (1953).

In an important early study along these lines, Craig MacAndrew and Robert Edgerton (1969) drew on ethnographic evidence of societies in which heavy drinking did not result in disinhibition or negatively valorized behavior to make a strong claim for the cultural specificity of such effects and more generally to argue that “drunken comportment” is determined culturally, rather than pharmacologically. Much of the subsequent discussion in anthropology and sociology focused on the question of whether this argument romanticized indigenous drinking, or at least failed to account for the potential transformation of valorized and socially constructive practices of heavy drinking into more disruptive and painful patterns with the advent of markets and wage labor (Heath, 2004; Marshall, 1982; Room, 1984). However, other work—primarily in experimental psychology—focused on the effects of expectancies related to intoxicated behavior and aggression, with somewhat mixed results (Marlatt & Rohsenow, 1980; Room, 2001; Testa et al., 2006). In
general, however, experimental, qualitative, and ethnographic work asking such questions remains wanting.

Close attention to the phenomenology of gambling, gaming, and other activities and practices that are increasingly framed as potentially addictive is especially important, given that these claims are justified primarily on the basis of neurobiological correlates with certain kinds of experience. In her ethnography of video machine gambling in Las Vegas, anthropologist Natasha Dow Schüll pays close attention to the accounts of players – particularly their descriptions of being “in the zone,” the “world-dissolving state of subjective suspension and affective calm” that some machine gamblers seek to attain again and again, despite experiencing extremely painful consequences in many domains of their lives (2012, p. 19). Similarly, Jeffrey Snodgrass and his colleagues have argued for an interpretation of World of Warcraft and similar online games as “technologies of absorption,” which facilitate states of dissociation and serve to relieve stress, resulting in positive experiences of achievement, competition, or sociality among some players and distressful or problematic patterns among others (Snodgrass, Lacy, Dengah II, Fagan, & Most, 2011).

Setting and meaning deeply shape both the embodied sensations associated with psychoactive drug use or the experience of immersive technologies and the experiences of “cue-induced craving” associated with the brain disease model of addiction. For example, Daniel Lende has argued that the experiences of “wanting more and more” reported by the young drug users in Colombia with whom he worked resonate with the theory of addiction as “incentive sensitization,” but in a way that depends on particular social and experiential settings – in this case, the concrete settings of these adolescents’ everyday lives (Lende, 2005, 2012; Robinson & Berridge, 1993). Other studies have not only found significant differences in kinds of craving, but also made clear that processes involved in amplifying feelings of wanting, or their resurgence after periods of abstinence, can only take place within specific social and material environments and are mediated by individually or culturally specific meanings, sensations, and expectancies, where multiple kinds of cues may work synergistically to initiate sensations of craving (Bruehl, Lende, Schwartz, Sterk, & Elifson, 2006). Rather than occasioning drug use in some direct or unambiguous way, these experiences of craving may lead potential users to employ various strategies to control drug use – with differing degrees of success and with expectancies or beliefs about craving itself potentially playing an important role (Bruehl et al., 2006; Lee, Pohlman, Baker, Ferris, & Kay-Lambkin, 2010). Finally, as Allison Schlosser and Lee Hoffer have shown in their research with
heroin users with co-occurring mental illness, the effects of various psychoactive substances (heroin “highs”, the “side” effects of psychiatric medications) and their absence (withdrawal, craving) have complex interactions, which are often managed in relation to one another (2012).

**Will and Habit**

Much of the conceptual difficulty surrounding ideas of addiction has to do with the way in which the affects, behaviors, and experiences associated with it run counter to widely held Euro-American assumptions about volition, self-control, choice, agency, and autonomy – assumptions that are often reflected in scientific thinking (in both social sciences and biosciences), as much as they are in lay ideas. Thus, during the nineteenth century, alcoholism and other addictions were often framed as “diseases of the will,” and it has been argued that this concept arose as a kind of shadow to the normative ideal of the freely choosing subject, in much the same way as Michel Foucault and others have argued that the concept of madness emerged in a mutually constitutive relationship to reason (Foucault, 1965; Valverde, 1998). To put it in very rough terms, the addict was seen as one who was unable to align his actions with his intentions because of a weakness or failure of the will, which was conceptualized as a human capacity alongside reason and emotion. Recent definitions of addiction translate these problems of the will into a language of “self-control” – the loss of which is understood as a core diagnostic criterion (Baler & Volkow, 2006; Weinberg, 2013). Parallel to ideas about the will, a long-running tension has persisted between theories emphasizing the addictiveness of psychoactive substances (or technologies or practices) and those focusing on the vulnerabilities (whether understood in genetic, psychosocial, or cultural terms) of particular individuals or populations (Campbell, 2007; Valverde, 1998).

Significantly, scholars of addiction in the social sciences increasingly discuss it in ways that avoid the binaries of free will and determinism, conceptualizing volition, control, or agency as partial, fragmented, emergent, or distributed across individuals, social networks, or assemblages comprising humans and nonhuman actors (Duff, 2011; Gomart, 2004; Weinberg, 2013). Such work is particularly suggestive because its conceptualizations of volition seem to dovetail with those arising from the neuroscience of addiction. As Jamie Sarris has argued, “theorizing in both psychopharmacology and [the neuroscience of] addiction increasingly has given us a sense of the will as an uncertain achievement, less of an essence and more an epiphenomenon of discrete processes that are subject to both degradation and enhancement” (2013, p. 273).
The idea of habit has emerged as particularly useful in this regard. Mined by both Eve Kosofsky Sedgwick and Mariana Valverde in respective writings on addiction, as a concept that sidesteps the "metaphysical absolutes" of free will and determinism, the notion of habit has recently reemerged as an object of more general interest among social theorists (Bennett, Dodsworth, Noble, Poovey, & Watkins, 2013; Fraser et al., 2014, pp. 22–23; Sedgwick, 1993, p. 137; Valverde, 1998, pp. 36–41). In his neuroanthropological account of addiction, Lende also refers to habit to explain a mode of learning that begins to dominate if and when drug use becomes repetitive, patterned, and compulsive. For Lende, the key point is that from both a neurobiological and a cognitive perspective, such habit learning seems to take place through the same processes associated with the internalization of "cultural models" or practices "through training, skill acquisition, and other forms of patterned practice" (2012, p. 258). Although these uses of the term "habit" are clearly distinct in many ways, each use can be seen as an attempt to displace tensions between free will and determinacy with accounts of human action as embedded in or emerging from individual particularity.

**Social and Material Milieu**

Social scientists have long emphasized the significance to addiction and substance use of what is variously referred to as milieu, environment, or setting, and more recent work has expanded these arguments, suggesting the literal interpenetration of bodies and environments, both material and social, complicated by the articulation between milieu and the global circuits through which substances and technologies move. For many researchers in the health sciences, the key question regarding environments or settings concerns the availability and accessibility of substances (or technologies). Thus, epidemiological studies have shown that risk of alcohol dependence is substantially affected by the spatial and temporal availability and pricing of alcohol in a given setting (Wagenaar, Salois, & Komro, 2009). Research from neuroscience and psychiatry has transformed this perspective on environments, emphasizing that it is not simply the availability of substances or devices that defines the "riskiness" of an environment in this sense, but the degree to which it is saturated with cues that have become associated with use for the addict (Childress et al., 2008; this idea was, of course, recognized and institutionalized somewhat earlier in AA as an injunction to avoid the "people, places, and things" associated with drug use).

However, for other researchers, the notion of milieu suggests an even more fundamental starting point: the idea that drug use (or any other
activities associated with addiction) always takes place in a social setting, which, at least in part, provides actors with a meaningful frame or reference point and shapes motivations. Clearly, the consumption of psychoactive substances is often constructive in that such practices are a means of producing and maintaining social relationships, even when it occurs through disagreement and negotiation, rather than ritual and consensus (Douglas, 1987; Colson & Scudder, 1988; Moore, 2004). The social settings of use are equally important in shaping the ways in which the uses or potential benefits of psychotropic effects are understood (McKenna, 2013).

Moreover, milieu can be conceptualized in relation to the broader circuits of production, exchange, and consumption — through which psychoactive substances flow as commodities. Anthropologists and historians have documented the key roles played by the trade and use of various psychoactive substances (particularly alcohol, tobacco, and opium) and certain foods in extending the reach of capitalism and colonialism over the past three hundred years (Mintz, 1985; Mills & Barton 2007). David Courtwright (2005) calls this “limbic capitalism,” arguing that the prevalence of such substances is directly linked to what neurobiology tells us about their inherent addictiveness for humans. Although such a claim perhaps begs for closer attention to the particularities of substance use and meaning, it is clear that embodied desires are often linked to the circuits of consumer capitalism, and that this linkage is facilitated and amplified by numerous entities seeking to profit from it (ranging from narcotrafficking operations to the alcohol, tobacco, gaming, and — as Kalman Applbaum (Chapter 21, this volume) reminds us — pharmaceutical industries).

Moreover, social scientists have emphasized that material and social environments not only shape patterns of substance use and behavior but that environments — as well as the capacity of individuals to move between them — are in turn deeply structured by broader political, economic, and social factors in ways that concentrate the incidence of drug-related harm and infectious diseases (particularly HIV) among particular populations (Bourgois, 2003; Page, 1997; Rhodes, 2002; Singer, 2007). State and social policy is extremely significant in this regard, as it often sets the overall framework for understanding particular substances, behaviors, or environments as problems of a specific kind, often with far-reaching unintended consequences. Thus, criminalizing certain drugs has often had the effect of shaping social and material milieu in ways that arguably increase the risk of harm to those with less capacity (in the form of economic or social capital) to move outside of those often marginalized environments (cf. Lende, 2012, p. 349).
The importance of accounting for milieu extends to our understanding of addiction interventions and therapeutics as well. My own ethnographic work has examined the largely behavioral and suggestion-based techniques for managing sobriety that have been popular in Russia for the past thirty years. While these methods are often criticized as paternalistic and for fostering the dependence of patients on physicians, I argue that particular arrangements of authority in the clinical relationship have less to do with particular therapeutics than they do with the broader institutional and social settings in which these therapies are enacted (Raikhel, 2010). The work of anthropologists Anne Lovell and Todd Meyers on the configurations of pharmaceutical therapy for opiate addiction in Marseille, France, and Baltimore, Maryland, respectively, similarly highlights the mutual shaping of therapeutics and social and institutional milieu (Lovell, 2013; Meyers, 2013).

*Trajectories*

A range of perspectives in the social sciences have emphasized a temporal perspective on illness and health, on individuals undergoing changes, and the relationship between experiences, life-course events, and environmental processes, which William Garriott and I have referred to collectively as “trajectories” (2013). Such a focus on trajectories draws our attention to the key temporal aspect of addiction, framed by philosopher Gilles Deleuze in the question: “Why and how is this experience [of drug use], even when self-destructive, but still vital, transformed into a deadly enterprise of generalized, unilinear dependence? Is it inevitable? If there is a precise point, that is where therapy should intervene” (Deleuze, 2007, p. 254). Almost all attempts to conceptualize addiction address the temporal aspect in some way, whether framed as a shift from use to abuse, from incentive salience to habit, or any other number of distinctions. Significantly, a focus on trajectories also allows for potential conversations between the increasingly neurodevelopmental approaches of neurobiologists and psychiatrists to mental illness, on the one hand, and the emphasis given by social scientists to structural inequities in material resources and power, on the other (with such inequities often intertwined with class and ethnic distinctions). Highlighting the effects of stress and trauma on selves and bodies, as well as the ways in which these effects can create path-dependent outcomes, a trajectories framework permits us to

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5 See Raikhel and Garriott (2013) for a more detailed discussion of several social science traditions using the concept of trajectories.

6 Thanks to Jeffrey Snodgrass for emphasizing this point.
think closely about epidemiological findings such as the association between risk for alcohol dependence and the experience of early childhood adversity (Kessler et al., 2010).

However, work in the social sciences also emphasizes contingency, differentiation, and singularity in the study of addiction trajectories. Anthropologically informed literature on illness and drug-use trajectories emphasizes that norms, role expectations, and understandings of pathology are culturally determined, and that they are differentiated across local conceptualizations of the life course (Nichter, Quintero, Nichter, Mock, & Shakib, 2004). Other research suggests the ways in which institutionalized, enacted, and materialized ideas about specific life trajectories may work to perpetuate themselves, creating a kind of looping effect. For example, in her study of heroin addiction in New Mexico’s Española Valley (the area of the United States with the highest per capita rates of heroin overdose), Angela Garcia argues that for some users, the clinical concept of chronicity they encounter in recovery programs dovetails with local Latino tropes of loss and endlessness (themselves shaped by many decades of land loss and expropriation) and reinforces a sense of hopelessness and “no exit” from addiction (2010). Finally, a very important aspect of this idea is that it draws our attention not to the trajectories of disease entities, but of particular people, highlighting the contingency and irreducibility of individual human lives.7

Conclusion: Steps to an Ecology of Addiction

In 1997, the same year that then NIMH director Alan Leshner urged his readers to attend to addiction as a brain disease, Howard Shaffer, a leading researcher on compulsive gambling, described the field of addiction research as beset by “conceptual chaos,” (Shaffer, 1997; Leshner, 1997). The subsequent years have seen many researchers, clinicians, policy makers, and patients turn to the brain disease model, at least partly, as a solution to the profound uncertainty and disagreement which has historically characterized knowledge about addiction. Yet, perhaps instead of seeing “chaos” in the multiple conceptual frameworks which inhabit addiction studies, we might see at least the potential for a vibrant “epistemic pluralism,” encompassing not only different research styles in neurobiology and the biosciences but also distinct approaches to psychoactive substances and addiction in the social sciences. In other words,

7 Indeed, it is precisely an emphasis on the potential of open-endedness and contingency that distinguishes the notion of trajectory from that of the “career” – an idea that has been widely used in the ethnography of drug and alcohol use (e.g., Waldorf, 1973).
maybe the first “step to an ecology” of addiction is not a unified “model,” but a greater engagement between research agendas that approach the question in fundamentally distinct ways. In this chapter, I have described four problem areas which are especially promising for such vital engagement, but there are certainly others as well.

What relevance does this “epistemic pluralism” have for the psychiatry of addiction? Setting aside the potential of more longitudinal approaches in clinical psychiatry, as well as the important arguments for more attention to social, cultural, and political-economic factors in the clinic, such an epistemologically engaged approach draws our attention to whether and how our conceptual categories shape the very behaviors they seek to describe. Depending on which definitions we adopt, our sense of the problem’s scale changes dramatically, as do any implications concerning the ontological status of addiction and which interventions are best suited to address the problems. Rather than asking which interpretive framework more closely “carves nature at its joints,” perhaps the place to start is to ask which is best suited for the particular purpose at hand, both for psychiatrists and for the patients they care for and serve.

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