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Toward a cultural biology of addiction

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Abstract Addressing the issues examined in the introduction, this article suggests an integrative 'cultural biology of addiction', which aims at encouraging a continuing dialogue between neuroscientists and those engaged in social studies of addiction. This cultural biology of addiction provides an alternative framework that brings together seemingly contradictory social-constructionist and biologically reductionist claims about addictions.
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Introduction

In the last two decades addiction scientists have come to view addiction as a brain disease. This claim was popularized by Alan Leshner, the director of the National Institute for Drug Abuse (NIDA) from 1994 to 2001, who asserted that prolonged substance use turned on 'a switch in the brain' that permanently transformed brain mechanisms, making interventions aimed at reversing addictions extremely difficult (Leshner, 2001). This view has been refined in subsequent years by Leshner's successor at NIDA, Nora Volkow and her collaborators (Kalivas and Volkow, 2005; Volkow, 2005). Addictive substances, write neuroscientists Eric Neslter and Robert Malenka, hijack the brain's reward circuit and the malfunctioning limbic system overrides the regulatory functions of frontal lobes, hijacking the brain's checks and balances. Thus, 'chronic drug use induces changes in the structure and function of the system's neurons that last for weeks, months, or years after the last fix' (Nestler and Malenka, 2004). Moreover, argues neuroscientist Michael Kuhar, 'studies of effects of drugs on receptors have shown us how drugs can change gene expression and how drugs can change the biochemical makeup of the brain' (Kuhar, 2010).

In contrast to the brain disease model, historians, sociologists and anthropologists of addiction have suggested that the classification of certain substances as illicit or licit tells us more about social norms and power relationships than about the psychopharmacological properties of the substances themselves. Social studies of addiction have examined past and current definitions of addiction and concluded that alcohol prohibition and the criminalization of narcotics and stimulants reflected dominant cultural values rather than robust scientific findings (Levine, 1978; Kushner, 2006, 2010). Thus they have insisted that

addiction should be situated in the context of wider cultural and environmental frames (Kushner, 2009).

These studies pose an *intellectual* challenge to the research, treatment and control of addiction. But, so far, addiction scientists have paid scant attention to them. There are many reasons for this lack of engagement. In part it is due to the fact that neuroscientists and social scientists do different work. Neuroscientists attempt to identify the processes and mechanisms of addictions; historians, sociologists and anthropologists of addiction see their role as contextualizing these claims through explorations of the political, social and cultural meanings of addiction. As important, as Courtwright suggests, the lack of engagement also reflects the fact that 'both social scientists and neuroscientists still live in their own gated academic communities, that they engage in vigorous boundary maintenance, and that they champion their own disciplinary and subdisciplinary master variables' (Courtwright, 2010, p. 144). This article suggests some ways to open these gates or at least provide more access to diversity inside these communities.

Academic History

Academic historians are not simply engaged in telling a chronological story; nor, since the late nineteenth century have they assumed that they can uncover 'facts' that recreate the past as it was. Rather, academic historians insist that historical sources do not speak for themselves, but are subjects of contested interpretations framed by current and past cultural and political contexts. From this perspective, there can never be a final 'factual' reading of the past; today's landmark interpretation is regularly subjected to tomorrow's reinterpretation because, odd as it may sound to the non-academic historian, the past is always subject to change as historians redefine the contexts in which events occur.

For historians, sociologists and anthropologists of addiction the claim that addiction is a brain disease requires contextualization. For social theorists the bottom line is that whatever its biological substrates, the identification and meaning of addiction are framed by changing social, cultural and political values. The rhetoric of addiction, according to William White, reflects the needs of professionals more than it does the population of the afflicted (White, 2004).

Taking White's view further, sociologist Helen Keane's *What's Wrong With Addiction?* focuses on how addiction rhetoric is constituted in current discourses (Keane, 2002). Although addiction has been portrayed as restricting freedom and individual autonomy, Keane argues that discourses of addiction have tended to limit freedom as they have authorized the prohibitive power of the family, the state and the corporation.

A second reservation about the brain disease paradigm is that it is not new, but rather is a restatement of long-held views repackaged in the current vocabulary of neuroscience. Often, writes historian Nancy Campbell, what has been learned in addiction science has been ignored in succeeding paradigms. More than a half-century ago, Campbell finds, addiction researchers Maurice S. Seever and Abraham Wikler had independently concluded that addiction was a chronic relapsing/remitting condition. Campbell also points to a rhetorical resilience of a traditional 'moral lexicon' of addiction. Citing the work of current NIDA director, Nora Volkow and her colleagues as exemplars, Campbell finds that their

notion of 'disrupted volition' parallels nineteenth century constructions of addiction 'as a "disease of the will" subject to voluntary control'. Thus, writes Campbell, with 'amnesiac gesture toward its own repressed past, the addiction enterprise comes full circle into the present' (Campbell, 2007, pp. 221, 237).

The claims that addiction is a brain disease would sound familiar to nineteenth century neurologists. In many respects, current views resemble degeneration theory as expounded by the French physician Théodule Ribot in his 1883 study *Les Maladies de la Volonté*, which was reissued in 32 subsequent editions in French and English (Ribot, 1900). Degeneration theory offered a hereditarian explanation for a variety of disorders including retardation, depression, depravity and sterility. Behaviors that today would include addictions to alcohol, eating and sex were alleged to have a cumulative destructive impact on the nervous system that was inherited by succeeding generations (Dowbiggin, 1991). Practitioners took extensive family histories and prepared elaborate pedigrees that sought to explain a current disorder by uncovering patterns of disease and behavior in a patient's family. Adherents sought to portray degeneration as organic, but much like addiction therapies today, treatment revolved around an array of psychological and moral interventions under the rationale that alterations in habits had a direct physiological influence on the nervous system (Nye, 1984; Pick, 1989; Dowbiggin, 1991; Micale, 1995).

Degeneration theory meshed with the views of the influential neurologist, James Hughlings Jackson, whose 'dissolution theory' was based on his claim that lesions in the neo-cortex reversed the evolutionary process in which the 'higher' cortical structures restrained the 'lower' emotive, limbic functions. Jackson's hydraulic theory reinforced the assumptions that addictions reflected a hijacking by these more primitive structures, often referred to as the 'reptilian brain'. Thus, sounding very much like the mechanisms described by current addiction neuroscientists, addiction was a brain disease resulting from damage to the cortical censors (Harrington, 1987). Because these behaviors appeared to run in families, it was a small step to connect Jackson's dissolution with degeneration.

Both degeneration and dissolution were translated into early twentieth century popular scientific explanations of the physical effects of alcohol and other drugs. For instance, historian Susan Speaker writes of Richmond P. Hobson, a retired naval officer and three-term congressman from Alabama, whose 1919 *Alcohol & the Human Race* was portrayed as based on the best 'evolutionary science' of the time (Hobson, 1919). Hobson, who founded the American Alcohol Education Association in 1921, wrote that alcohol was a toxin that paralyzed white blood cells making them unable to 'catch the disease germ' that was 'devouring' the drinker. This led to the destruction of the 'centers of the brain upon whose activities rest the moral sense', resulting in what Hobson labeled 'retrograde evolution' (Speaker, 2004, p. 214).

Hobson's 'science' both influenced and was influenced by early twentieth century prohibitionist sentiments. With the end of Prohibition, a new science of alcoholism emerged. Americans, according to Speaker, ceased 'demonizing alcohol after Prohibition, and chose to deal with its risks largely through regulation, education, and harm-reduction strategies'. However, she writes, 'they have resisted' treating most other psychoactive drugs in a similar manner (Speaker, 2004). What emerged were distinct attitudes, policies and sciences that separated alcohol from other addictive substances. But, Speaker implies, these distinctions

were based less on objective evidence than on the cultural, social and economic attitudes toward alcohol and other mind-altering substances.

Alcohol and Other Drugs

American addiction scientists generally study alcohol separately from other substances. Indeed, the histories of alcohol addiction have much in common with those of other drugs, but unlike illicit and (still) legal ones such as nicotine, alcohol putatively poses a danger only to predisposed alcoholics. The prevailing scientific view in America is that moderate consumption of alcohol by those without a predisposition is safe and not addictive. In contrast, the dominant media and scientific view today holds that, although some people are more prone to addictive behaviors than others, no predisposition is necessary for addiction to illicit substances and nicotine; any exposure potentially places any user at risk (Nestler and Malenka, 2004; Dalgarno and Shewan, 2005).

The construction of alcoholism as a disease, according to sociologist Ron Roizen, meshed with the values of both the 'spiritual orientation' of Alcoholics Anonymous and the 'disinterestedness, objectivity, and empiricism', of contemporary science. Ironically, the notion that alcoholism was a disease 'also offered destigmatization to the alcoholic and a measure of new symbolic legitimacy for [the] beverage alcohol itself'. From the disease perspective, alcohol 'harbored little more responsibility for alcoholism or alcohol related troubles than did sugar for the disease of diabetes' (Roizen, 2004, p. 64).

The current concept defining alcoholism as a disease – that moderate drinking is safe for all but the potentially and actually alcoholic – comes from the work of the director of the Yale Center for Alcohol Studies, E. [Elvin] M. Jellinek, in the 1940s (Jellinek, 1960). However, Roizen's examination of the science of alcoholism reveals 'its strongly social-constructionist character and flimsy science base' and 'invites our attention to the relationship between alcohol science and the wider society' (Roizen, 2004, p. 74).

One of the lynchpins for the diseasing of alcoholism is the widespread popular belief that Native Americans are genetically vulnerable to alcoholism. This view has been challenged by a number of recent studies. John W. Frank and his colleagues emphasize that beyond the obvious 'risk factors in contemporary life', there is the need to consider the historical sources of Native American drinking problems. For instance, one must acknowledge 'the extraordinary barrage of inducements to drink heavily in the early years after European contact' (Frank *et al*, 2000, pp. 349–350).

Historian Peter C. Mancall agrees that some individuals 'seem to possess an inherited predisposition toward alcohol abuse', but he insists that 'there is no convincing evidence suggesting that Indians as a group are more inclined to possess these traits than the general American population' (Mancall, 2004, pp. 99–100). Historical research, according to Mancall, reveals that 'there has been no single Native American response to liquor. Consumption patterns have differed over time by region and even in specific communities'. They also have varied by age and gender. Europeans, Mancall reminds us, who had been exposed to alcohol for centuries, 'had developed rules for its consumption'. Nevertheless, they too experienced 'periods of wide-spread alcohol-related problems', including the so-called gin craze in the mid-eighteenth century that 'occurred in part because of wider

availability of more potent alcohol during the early phases of the industrial revolution when the English and other Europeans drank more alcohol' in an attempt to 'escape from the disorienting social changes of their everyday lives' (p. 100).

Mancall's thesis is built on a number of studies (Kunitz and Levy, 1994) including Craig MacAndrew and Robert B. Edgerton's 1970 cultural anthropology classic, *Drunken Comportment: A Social Exploration*, which explored variations in behaviors observed in different populations when they are drunk (MacAndrew and Edgerton, 1969). In relatively simple societies people learn how they are supposed to behave when intoxicated; in more complex societies the cultural expectations may vary, but the same principle holds. Edwards supports the conclusions of MacAndrew and Edgerton's ethnography. Although he acknowledges that 'alcohol is a drug which has the inherent capacity to interfere with brain function and produce a state of intoxication', Griffith Edwards, former chairman of the United Kingdom's National Addiction Center, nevertheless, argues that 'intoxication is not, however, a fixed and monolithic state' (Edwards, 2002, p. 52). A similar point is made by psychologist Michael Windle who argues that multiple factors influence alcohol use and abuse, including 'genetic, biochemical, physiological, cognitive, social, neighborhood, and societal' (Windle, 2009).

Although today alcoholism is widely assumed to be organic, mid-twentieth century psychiatry focused on gender role confusion. Alcoholic males, writes Michelle McClellan, were characterized as effeminate with homosexual tendencies manifested by employment difficulties. In contrast, psychiatrists portrayed female alcoholics as displaying 'masculine traits such as aggressiveness', and they 'were often promiscuous or frigid' and inadequate mothers (McClellan, 2004, p. 274). Gendered assumptions, according to historian Lori E. Rotskoff, also informed psychiatric views about the role that sober wives played in their husbands' alcoholism (Rotskoff, 2002, 2004). Underlying many of these observations was the tension of the post-war readjustment of gender role expectations, with returning males displacing working women. The task, seen by many psychiatrists and social workers in the 1940s and 1950s, was to re-establish traditional gender roles within the American family. Thus by the 1950s, psychiatrists and social workers advocated group therapy for alcoholics' wives. 'Given the nation's deep psychological investment in marriage', Rotskoff concludes, 'it is apt that alcoholism's deleterious effects would increasingly be measured in marital terms' (Rotskoff, 2004, p. 321).

What these and other historians have argued is that the theories informed the diseasing of alcohol-reflected dominant social values in the guise of science. One might insist that current scientific claims about alcoholism as a disease rely on a completely different science, informed by neurobiology, biochemistry and genetics (Braun, 1996; Oscar-Berman and Marinkovic, 2003). But having shown the culture-bound nature of earlier scientific theories supporting the diseasing of drunkenness, historians are skeptical of current scientific assertions that alcoholism is a disease.

Opiates and Other Illicit Drugs

The same science and psychiatry that have consistently viewed host predisposition as the trigger for alcohol addiction have as consistently viewed opiates as posing an addictive risk

for all who use them. According to Edwards, this is because alcohol intoxication 'is remarkably susceptible to cultural prescriptions and proscriptions' and alcohol is 'a widely accepted recreational drug'. But in contrast, 'intoxication with crack cocaine, or injected amphetamines, or with a heavy dose of LSD, is not so easily shaped, and these are not drugs which society is ever likely to accord a licit recreational status' (Edwards, 2002, p. 57).

In *Creating the American Junkie* (2002) and her subsequent publications, Caroline Acker traces this history of opiate prohibition through an examination of the experience of users as they negotiated a world in which opiate use increasingly became criminalized (Acker, 2002). Acker's work reinforces David Courtwright's study, *Dark Paradise* (2001), which, using similar narratives, demonstrates that 'what we think about addiction very much depends on who is addicted' (Courtwright, 2001a, p. 4). In the early twentieth century, addicts could seek medical treatment that included prescriptions of maintenance doses. But beginning with the Harrison Narcotics Act in 1914, nonmedical use or purchase of cocaine and opiates was restricted and all narcotics sold or prescribed were required to be registered. As a result, physicians were no longer able to treat addicts through maintenance and ceased treating them altogether. This shift, writes Acker, transformed both the context and meaning of opiate use and led to 'a new form of addict identity as the behaviors to maintain addiction were criminalized' (2002, p. 167).

Courtwright has a slightly different take. With the decline of medical (iatrogenic) addiction in the late nineteenth century, 'opiate addiction … began to assume a new form: it ceased to be concentrated in upper-class and middle-class white females and began to appear more frequently in lower-class urban males, often neophyte members of the underworld. By 1914 the trend was unmistakable'. For Courtwright, 'the trend toward criminalization … was well underway before the basic narcotic statutes were enacted' (2001a, p. 3).

In her contribution to this volume, Acker goes further, adopting an ecological approach that she tentatively labels as a 'historical epidemiology of drug use'. Acker 'builds on epidemiology and emphasizes the need to consider the structural and social contexts in which groups of people encounter and use drugs'. From this context she examines how the increasing impoverishment and physical segregation of the Hill district of Pittsburgh framed an epidemic of crack cocaine. As Acker persuasively argues, community substance use and addiction cannot be understood nor can effective interventions be developed if the economic and social context in which they arose is ignored (Acker, 2010).

While Acker's addicted Hill District crack users are treated as criminals, the wealthier addicted Pittsburghers, who purchase their cocaine in the Hill District, are generally treated as 'chemically dependent' and sent to rehab centers rather than to prisons. According to historian Timothy Hickman, this 'double meaning of addiction', in which some of the addiction was attributed to disease and some to hedonism and antisocial behavior, has long historical roots (Hickman, 2004, pp. 185–186). Although Hickman does not make the connection, his essay provides a context for the emergence of the psychoanalytic construct of the 'addicted personality', which first appeared in Lawrence Kolb's 1925 article, 'Types and Characteristics of Drug Addicts', and in his subsequent work (Kolb, 1925, 1962). Despite Kolb's insistence that addiction was a medical issue, federal officials adopted Kolb's construct as evidence of the general character defects of addicts and as justification to extend the criminalization of drug use (Courtwright, 2001a; Tracy and Acker, 2004).

As medical treatment for alcohol addiction became the norm in the mid-twentieth century, maintenance clinics for the treatment of narcotics addiction became illegal. From 1923 to the opening of the first methadone treatment center in 1965 in New York City, writes Jim Baumohl, 'addicts were demonized, hounded, subjected to draconian criminal penalties, and never treated except in the confines of a hospital or jail'. Aside from a very few wealthy private patients, 'abstinence was the only legitimate goal of treatment' (Baumohl, 2004). By the 1930s, even the supporters of maintenance programs 'believed most addicts to be incurable' (p. 228).

It was in this context that in 1935 the US Public Health Service established the Center for Drug Addiction (CDA) at the federal prison hospital at Lexington, Kentucky (Campbell, 2007). Informally labeled as 'Narco', the facility, which continued its addiction research until 1979, was designed to be a treatment hospital for incarcerated addicts. In 1948, the research unit became the first basic research laboratory of the newly formed National Institute of Mental Health, Addiction Research Center (ARC). Inmates became voluntary participants in ARC experiments that tested reactions to a wide variety of substances including alcohol, barbiturates, heroin, methadone, major and minor tranquilizers, and psychedelics.

The CDA's benign approach to addicts was an exception, but the venue for its research, a federal prison, reflected the policies of Henry Anslinger, the influential director of the Federal Bureau of Narcotics (1930–1962). Anslinger's role in shaping and extending the criminalization of drug use policy cannot be over-estimated (Carroll, 2004). Anslinger 'influenced Americans' attitudes toward narcotic drugs and drug users and sellers, depicting both users and sellers as criminals'. This is evident in Anslinger's 1937 Congressional testimony in which he claimed that marijuana 'is dangerous to the mind and body, and particularly dangerous to the criminal type, because it releases all of the inhibitions'. It causes some individuals to 'have an increased feeling of physical strength and power', which is dangerous because they 'fly into a delirious rage, and they are temporarily irresponsible and may commit violent crimes' (Anslinger, 1937).

Although a number of influential experts, including leaders of the American Medical Association and the American Bar Association, argued for the medicalization and clinical treatment of addicts, Anslinger stifled their voices (Toby, 2005). In 1944, at the urging of New York City Mayor Fiorella La Guardia, the New York Academy of Medicine conducted a study of the effects of marijuana, whose findings contradicted Anslinger's claims. The Commission found that cannabis did not cause violence and despite Anslinger's insistence otherwise, they concluded that marijuana could be medically beneficial. Anslinger denounced the report and instructed the Bureau of Narcotics agents to investigate the commission members' own drug use. Further, he threatened prison sentences for anyone carrying out independent research on cannabis.

In the postwar era, Anslinger altered his views of the effect of marijuana on its users but not his policy toward its use. Testifying in Congress in 1948, Anslinger claimed that cannabis caused the user to become peaceful and pacifistic; thus, the Communists were recruiting Americans into cannabis use as part of their plot to weaken the will to fight (Toby, 2005).

Like Anslinger, those who continue criminalizing marijuana use in the United States today claim to base their views on scientific research, but also like Anslinger, antipathy toward

marijuana use reflects deeper cultural values rather than robust science. A similar claim can probably be made about those who support unrestricted availability of marijuana. The point here, as much of recent addiction history reveals, is that the classification of substances as licit or illicit has less to do with science than politics and culture.

Licit Mind-Altering Drugs

An examination of the history of the introduction of a new class of antidepressants in the late 1980s called selective serotonin reuptake inhibitors, or SSRIs (Prozac®, Paxil® and Zoloft®), illustrates the extent to which science and culture become inextricably bound. Spurred on by massive advertising efforts in the late 1990s and Peter Kramer's best-selling book, *Listening to Prozac* (Kramer, 1993), SSRIs, according to psychiatrist Nicholas Weiss, have become 'consumer products appropriate for wide usage or general lifestyle enhancement'. SSRIs' predecessors, monoamine oxidase inhibitors (MAOIs) and tricyclics, were viewed as 'disease therapies to be kept strictly in the medical domain' (Weiss, 2004). Why, asks Weiss, had 'no one listened to [the tricyclic] Imipramine?' (p. 329). His answer, like so much else connected to addiction, lies in the history of alcoholism.

The definition of 'alcoholism' as a distinct disease affecting only a minority of drinkers, writes Weiss, has removed the blame for alcohol-related social problems from the substance to a subgroup of susceptible individuals. Thus alcohol use, though not abuse (drunkenness), is socially acceptable. 'This enabled the alcohol beverage industry to sell its product, despite widespread concerns about the dangers and evils of alcohol, as long as drinking was officially proscribed for that susceptible population' (p. 349). The diagnosis of depression, according to Weiss, 'functioned in an analogous, though inverse manner'. A diagnosis of depression identified a susceptible group 'who *should* become users, those with a current or potential medical depression' (p. 349, italics added). Therefore, dependence on SSRIs is authorized, even though they are mind-altering (and often addictive) substances because depression has been constructed as a disease. The risks of SSRI use are downplayed because the condition they treat is defined as illness, despite a spate of warnings about the hazards associated with SSRIs (Breggin and Breggin, 1994; Healy, 1997; Glenmullen, 2000).

Similarly, although Weiss does not make this connection, a diagnosis of attention deficit hyperactivity disorder (ADHD) authorizes placing patients (mainly children) on addictive stimulant medications, such as Ritalin® (methylphenidate). According to historian Nicholas Rasmussen, the current amphetamine epidemic should be viewed in the context of the medical use of stimulants to treat depressive disorders and how this resulted in a wider epidemic of stimulant use by mid-twentieth century. Building on this history, Rasmussen connects the present methamphetamine epidemic to the earlier iatrogenic epidemic (Rasmussen, 2008a, 2008b). This history appears to be repeating itself as Ritalin® and other stimulants prescribed for the treatment of ADHD become widely used as recreational drugs on American college campuses and beyond (DeGrandpre, 1999; Mayes *et al*, 2009).

Recognizing how prescription drugs once again have morphed into recreational and self-medicating substance use and abuse has important implications for those who wish to

understand and treat the current wave of addiction and substance abuse. For Rasmussen these evolutions have resulted as much from changing populations who use stimulants, as from the biological actions of these drugs. A similar argument has recently been made by psychologist Richard DeGrandpre (DeGrandpre, 2006).

In fact, for most illicit addictive substances there is a companion licit substance, like methylphenidate, whose action mirrors that of the proscribed drug. As DeGrandpre points out, although Ritalin® and cocaine act similarly on the brain, the former is widely prescribed for children, while the use of cocaine is a felony. Similarly, the street drug, Ecstasy, acts on the same serotonin receptors as SSRIs. Although far from controversial, the risk of addiction to mind-altering pharmaceuticals has been justified because of the putative benefit conferred by their consumption. Ironically, the effectiveness of many SSRIs and atypical antipsychotics is tied to their permanent alteration of brain structures and functions, albeit in putatively desirable ways.

Rhetoric and Biology

If current popular rhetoric characterizing substances such as caffeine, chocolate, carbohydrates, not to mention addictive behaviors including gambling, sex and shopping are addictive, we either inhabit the most addictive society that ever existed or have failed to notice retrospectively how addictive human behaviors are. Or, as the logic of the histories reviewed here suggests, a wide range of human consumption and behaviors have been (re)constructed as addictions.

Speaker asks to what extent the 'characteristic rhetoric' toward addictive substances is a 'reflection of genuine drug problems … and to what extent it is an expression of various social tensions – class struggles, demographic changes, racial and ethnic conflicts, etc. – or an expression of particular values and ideologies?' She also wonders 'what accounts for the persistent use of these themes and images', and 'to what extent … this popular rhetoric not only reflected but shaped public perceptions and drug policy itself during this century' (Speaker, 2004, p. 219). To these questions we may add what the histories of addiction reveal about the biological effects on the human brain and what these biological mechanisms reveal about the histories of addiction.

For many cultural theorists, portraying biology and the past sciences of addiction as culturally constructed has authorized ignoring current science altogether. But the fact that science, like everything else, is socially constructed in no way diminishes its explanatory power any more than it limits the value of historical interpretations, such as those examined in this essay, which like all historical research and writing are socially constructed and contingent (Kushner, 1998). In any case, an increasing number of scholars of addiction, including those whose articles are included in this volume, have begun to engage rather than ignore current addiction science.

One concrete example of the value of such an integrated approach is found in the history of the identification of cigarette smoking as addictive behavior. As Alan Brandt writes, although the addictive nature of nicotine may today be seen as an undisputed fact of its chemical properties, nicotine's classification as an addictive substance is rooted more in the history of attitudes toward smoking than in its neurochemical mechanisms (Brandt, 2004).

Brandt's claim is elaborated by Mark Parascondola, Nicolas Rasmussen and Virginia Berridge. They demonstrate that the focus on nicotine as an addictive substance came rather late in the identification of the health hazards of smoking (Berridge and Mars, 2004; Rasmussen, 2008a, 2010; Berridge, 2009; Parascondola, 2009).

As Brandt points out, although the addictive potential of the nicotine in tobacco was often noted long before the 1988 Surgeon General's report on nicotine and addiction (Office of Surgeon General, 1988), attitudes toward cigarette smoking have a complex history. As late as 1964, the Surgeon General's advisory committee on the health consequences of smoking concluded that 'the evidence indicates this dependence to be psychogenic in origin' and 'the biological effects of tobacco, like coffee … are not comparable to those produced by morphine, alcohol, barbiturates, and many other potent addicting drugs' (Office of Surgeon General, 1964). As a result of the dramatic decline of smoking because of its associated health risks, its recategorization as addictive in the 1980s was, according to Brandt, 'far less problematic than would have been the case a decade earlier'. This was particularly so because smoking increasingly had become 'associated with certain social groups – generally those less educated and of lower socioeconomic status' and notes Brandt, 'in a culture prone to stigmatize its poor and disfavored, changing perceptions about the "average smoker" eased the growing attribution of addiction' (2004, p. 391).

Despite the powerful social forces and public health efforts that have created an almost universal awareness of the adverse health consequences associated with smoking, many persist in the habit (Brandt, 2007; CDC, 2009; Prevention, 2009). The addictive properties of nicotine are generally portrayed as the culprit. However, given that more than 50 per cent of smokers have, albeit with great difficulty, managed to cease smoking, coming to terms with persistent smoking requires a more nuanced analysis, one that takes into account the interaction of culture and biology.

Part of the reason for this persistence, according to Keane, is evident if one contrasts the immediate rewards of smoking with its long-term consequences (Keane, 2002). As Piazza *et al* pointed out, for many persons 'compulsive drug intake is a symptom of a larger addiction disorder' in which a substance or substances are employed to mitigate an underlying condition (Piazza *et al*, 1998, p. 106). This is particularly true of persistent smokers where smoking prevalence has shown to be significantly and positively associated with co-morbid psychiatric disorders such as major depressive disorders, schizophrenia, ADHD, Tourette syndrome, conduct disorder, anxiety disorders, and alcohol and substance abuse (Glassman, 1993; Levin *et al*, 1996; Dalack *et al*, 1998; Tizabi *et al*, 1999; Lasser *et al*, 2000; Mihailescu and Drucker-Colin, 2000; Kumari, 2005; Wilens *et al*, 2007; Tizabi *et al*, 2009).

Nicotine is a powerful drug that operates on a number of neurotransmitters including dopamine, GABA and nicotinic subtype acetylcholine (ACh), a neurotransmitter associated with attention (Parascandola, 2003). Stimulant medications, such as Ritalin®, are effective in treating attentional disorders such as ADHD because they increase the nicotinic transmission through ACh receptors. Smokers suffering from inattention may turn to nicotine to help manage their symptoms because it mimics the effects of ACh (Mihailescu and Drucker-Colin, 2000; Levin, *et al*, 2006; Changeux and Taly, 2008).

Lawn and colleagues have affirmed an association between these mental health issues and difficulty in quitting (Lawn *et al*, 2002). Studies suggest that many individuals who are

unable to quit smoking are more nicotine dependent and more likely to suffer from psychiatric comorbidity than former smokers and never smokers (Fagerstrom *et al*, 1996). Thus, a recent study following more than 20 000 smokers for 10 years found that few factors are as highly associated with persistent smoking as 'depressive symptoms' that 'are uniquely associated with lifetime and current dependence' (Hu *et al*, 2006, p. 299; Ischaki and Gratziou, 2009). Nicotine improves attentiveness in smokers and attenuates attentional deficits in adults with schizophrenia, ADHD and Alzheimer's (Mihailescu and Drucker-Colin, 2000; McEvoy and Allen, 2002; Levin *et al*, 2006; Changeux and Taly, 2008). Similarly, recent research has shown that nicotine is an effective tic reducer among some persons afflicted with Tourette syndrome (Howson *et al*, 2004; Orth *et al*, 2005).

Syndromes of Dependence

Like persistent smoking, all addictions operate through a combination of cultural and social constraints as they interact with biological mechanisms of substances and behavior. Thus Edwards argues that the seemingly contradictory social constructionist and biologically reductionist claims can best be integrated through the histories of addictions. Accepting the social constructionist critique of the diseasing of alcoholism, Edwards finds that what is called alcoholism is 'best approached through a framework of the dependence-syndrome concept', where 'the dependent state is not a matter of all or nothing (addict or not addict), but something which can be experienced in varied and measurable degrees (more or less dependent)' (Edwards, 2002, p. 162). Edwards' insistence on the distinction between syndrome and disease is not trivial. Measles, polio and Huntington's are diseases because a tentative diagnosis based on signs and symptoms is confirmed or rejected through a laboratory test indicating infection by a pathogen or the presence of a genetic mutation. In contrast, the cause of a syndrome, such as schizophrenia, Tourette syndrome or affective disorders (depressions), remains unknown (Sutter, 1996; Kushner *et al*, 2004). The diagnosis of syndromes depends on the identification of a list of possible combinations of signs and symptoms displayed by a patient within a certain time period. This list of signs and symptoms is tentative and disagreement often surfaces over which signs and symptoms are crucial to authorize a diagnosis (Hacking, 1995; Kushner, 1999). As a result, identification of a syndrome often varies over time and by geographic location (Ziporyn, 1992).

As with pneumonia, a variety of routes can lead to alcohol dependence. Unlike pneumonia, but like most psychiatric syndromes, these include both cultural and/or biological factors in the enabling spectrum. Those who meet the criteria (in terms of signs and symptoms) for alcohol dependence experience real illness, even if the etiology and level of distress and particular path to dependence are not the same for every alcohol-dependent person. Recognition of the many routes to an alcohol dependence syndrome sanctions researchers and clinicians to craft a variety of interventions and policies that consider a spectrum of cultural and biological triggers. Such recognition must include, no matter what the trigger, the biological and social effects on the individual. This requires engagement with the accumulating evidence from recent research that substance dependence, including alcohol dependence, alters brain reward and sensitization mechanisms, including brain

architecture and neurochemistry, sometimes permanently (Braun, 1996; Wilson and Kuhn, 2005). This seems true even when the addiction, such as gambling, is not attached to a substance. The question remains whether labeling nonsubstance behaviors as addictions is justified because they impact and alter the same brain reward systems (the ventral tegmental area or VTA) as do cocaine and heroin (Blaszczynski and Nower, 2002; Nestler and Malenka, 2004; Wilson and Kuhn, 2005).

As most behaviors have an impact on brain chemistry, how do we decide which of these are addictions and which are not? Many of the histories of addiction discussed in this article and in this volume agree that what is considered an addiction, and what is not, reflects social and cultural values as much as it tells us a truth about the mechanisms of the brain. But admitting that does not, however, trivialize the importance of biology to addiction. Any understanding of the history of addictions requires such an integrative approach (Berridge, 2004). Addictions are syndromes of dependence, informed and 'enabled' by an interaction of culture and biology. That may be why psychologist Clinton Kilts and his colleagues have found that the targeted partial agonist D-cycloserine, nevertheless, requires continued cognitive behavioral therapy in order for treatment to be effective and sustained (Kilts, 2009).

Similarly, Scott Vrecko examines the success of the anti-craving drug, Naltrexone®, for the treatment of a variety of compulsive behaviors. However, Vrecko resists the claim that anti-craving drugs represent 'a new treatment for a new kind of addiction'. Rather, he sees the process as seeking neurobiological interventions that achieve social outcomes. 'We are producing more responsible individuals', writes Vrecko, 'who are better able to adhere to the duties, expectations, and obligations of their families and society'. If Vrecko's argument seems to parallel Foucault's claims that psychiatry aims at creating a shared consciousness, it differs in that for Vrecko, this is a socially desirable goal, because it produces 'better citizens' (Vrecko, 2010).

Sometimes, argues psychiatrist Sally Satel, an addicted patient comes face to face with the adverse consequences of addiction. This, she reports, is often the most important trigger to recovery. Satel's clinical observation seems to suggest that, as nineteenth century observers insisted, even an addict can summon the will to break their addiction (Satel, 2009). Such reports serve as additional evidence that addiction is best understood as a syndrome rather than a disease.

The Cultural Biology of Addiction

This returns us to Edwards' view that addictions are actually syndromes of dependence that have multiple triggers and pathways, ranging from the cultural to organic, but are probably informed by a combination that we might label as 'cultural biology'. This cultural biology of substance dependence is based on centuries of observations. The science of each era has attempted to identify the mechanisms that underlay the observed behaviors. That in retrospect these attempts reflect the dominant scientific paradigm of each era is neither surprising, nor does it undercut the evidence that there are organic triggers for and biological effects from substance dependence. That these interact with cultural and social forces would not surprise any serious neuroscientist. Like Edwards, they would concede that current neurobiological hypotheses are by definition tentative.

As Edwards reminds us, and as the smoking discussion above elaborates, for much of human history, including our own era, most mind-altering substances have been initially consumed as a means of self-medication for a variety of ills, not least of all for disorders of consciousness, including major and minor psychiatric disorders (Edwards, 2002; Courtwright, 2001b). That self-medication plays an important role in persistent substance use and abuse, despite awareness of potential harm, is a fertile ground for further historical research (Allen *et al*, 1998; Felitti, 2003; Murphy *et al*, 2003). Self-medication, like the conditions it aims to treat, is rooted in culture and biology and cannot be understood apart from that interaction. Like all culturally mediated biological phenomena, each society responds to these human behaviors within the context and confines of larger social, political and cultural constraints. From this perspective, addiction is one possible outcome of humans' drive to alter consciousness; what we label 'addiction' might be understood as a *possible* consequence of the human desire to alter consciousness.

Although a majority of addiction researchers and scientists have questioned the effectiveness of abstinence policies, governments at all levels continue to make abstinence the centerpiece of all addiction policies. In this debate, a cultural biology of addiction can make important contributions to official addiction policy because it provides a context for the failure of the abstinence policy. First and foremost, abstinence is a failed policy because it denies the historical evidence that humans in all societies and cultures have and continue to rely on substances to alter their consciousness. Addictive behaviors, rather than diminishing, have increased, spurred on in part by industries that manufacture and market consciousness-altering commodities and, in part, as Vrecko suggests, by widening the behaviors that are classified as addictive. In the face of persistent human drives to alter consciousness and markets that cater to them, abstinence appears unattainable as a universal goal. The histories of addiction indicate that official abstinence is also a failed policy because as both addiction scholars and brain researchers recognize, addiction is a chronic relapsing/remitting syndrome. From that perspective as well, any successful policy or intervention must include harm reduction. Anthropologists, sociologists and historians of addiction along with addiction scientists can jointly play a crucial role in moving drug policy toward harm reduction. Indeed, this is a project that, like so many others related to addictions, requires collaboration if it is to be successful. For, as the scholarship and research of this volume indicate, what we label as addictions are truly a bio-cultural phenomenon.

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