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Open Peer Commentaries

Singing the Brain Disease Blues

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Well over a decade ago, the National Institute on Drug Abuse began advancing the idea that addiction is a "brain disease." Over the years, the concept has become orthodoxy—a dubious achievement that has justifiably prompted Buchman and his colleagues (2010) to call for a more nuanced perspective on addiction. In this peer commentary we challenge the validity of the brain disease model of addiction and discuss its adverse implications for treatment.

Let us begin with the concept of brain disease. "That addiction is tied to changes in brain structure and function is what makes it, fundamentally, a brain disease," wrote a former director of the National Institute of Drug Abuse in a seminal 1997 *Science* article (Leshner 1997). What does this statement really mean? Surely, drugs operate at the level of the brain (Hyman 2007). No dispute there. Regular, heavy use of alcohol, nicotine, heroin, cocaine, and other substances produces brain changes (reward centers are "hijacked," as it is commonly put) that, in turn, influence the urge to use drugs and the struggle to quit. Brain-related differences among users influence the rapidity with which they develop addiction, their subjective experience of the drug, the potency of their craving, and the severity of their withdrawal symptoms.

That said, why should this make addiction a brain disease as opposed to, say, a molecular disease, a psychological disease, or a sociocultural disease? All are equally valid perspectives for different purposes. As psychologist Nick Heather wisely asserted, "Addiction can be defined in any way the definer thinks fit. . . . The crucial issue is how useful the definition is for specific purposes" (Heather 1998). So, for example, if one's purpose is to investigate dopamine circuitry, then viewing addiction as as brain-based phenomenon makes sense. But if one's purpose is providing psychosocial treatment and devising policy, then the "neurocentric" view doesn't help much.

The mechanical simplicity of the "brain disease" rhetoric has a seductive appeal that obscures the considerable degree of choice in addiction, as Buchman and colleagues note. Consider the daily routine of addicts. They rarely spend all of their time in the throes of an intense neurochemical siege. Most heroin addicts, for example, perform some kind of gainful work between administrations of the drug. In the days between binges, cocaine addicts make many decisions that have nothing to do with drug-seeking. Should they try to find a different job? Kick that freeloading cousin off their couch? Attend a Narcotics Anonymous meeting, enter treatment if they have private insurance, or register at a public clinic if they don't? These decisions are often based on personal meaning. Many autobiographical accounts by former addicts reveal that they were startled into quitting by a spasm of self-reproach (Lawford 2008): "My God, I almost robbed someone!" or "What kind of mother am I?"

Most important, knotty philosophical issues of free will versus determinism aside, addiction is an activity whose course can be altered by its foreseeable consequences. No amount of reinforcement or punishment can alter the course of an entirely autonomous biological condition. Imagine bribing an Alzheimer's patient to keep her dementia from worsening, or threatening to impose a penalty on her if it did. It won't work. But incentives do work in addicted patients, as clinical trials of a strategy called "contingency management" show. The standard trial compares addicts who know they will receive a reward for submitting drug-free urines with matched addicts not offered rewards (Silverman et al. 2001). In general, the groups that are eligible to be rewarded with, for example, cash, gift certificates, or services are about two to three times more likely to turn in drug-free urines compared with similar counterparts who were not able to work for such incentives. In drug courts (a jail-diversion treatment program for nonviolent drug offenders), offenders are sanctioned for continued drug use (perhaps a night or two in jail) and rewarded for cooperation with the program. The judge holds the person, not his or her brain, accountable for setbacks and progress.

Even without formal incentives or sanctions, users perform their own mental calculations all the time. Repetitive drug use is reduced or stopped altogether when the adverse consequences of drug use exceed its rewards. An addict might reason, for example, "Heroin quells my psychic pain and soothes withdrawal, but it is costing my family

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too much." In a choice model, according to psychologist Gene Heyman (2009), addiction is the triumph of feel-good local decisions ("I'll use today") over punishing global anxieties ("I don't want to be an addict tomorrow"). As the relative value of costs and benefits of addiction change over time (yes, benefits: people use drugs and maintain addictions for psychological reasons—a reality obfuscated by the brain disease model), users become less ambivalent about quitting.

Whether powered by changes in meaning or incentives, the capacity for self-governance is the key to the most promising treatments for addiction. This fact is often obscured, however, by a semantic glitch whereby the state of "addiction" is taken to mean that the desire to use is unmalleable and beyond the reach of environmental contingencies. In circular fashion, then, addicted individuals are believed to be helpless to change their behavior. And, if so, it is wrong to expect them to respond to sanctions.

This was the destructive logic employed by the opponents of Proposition 36 in California, a 2001 referendum on the state's jail diversion program for nonviolent drug offenders (Urada et al. 2008). They prevailed and within a few years treatment program staff began clamoring for permission to use modest penalties and incentives—without them, the staff had no leverage. Similarly, drug courts and probationary programs have been hampered by ideological resistance to imposing consequences for positive urine tests because, after all, victims of a brain disease cannot be held accountable for their behavior; what's more, they are "supposed" to relapse.

The brain disease model implies erroneously that the brain is necessarily the most important and useful level of analysis for understanding and treating addiction. Like Buchman and colleagues, we believe that it is far more productive to view addiction as a behavior that operates on several levels, ranging from molecular function and structure to brain physiology to psychology to psychosocial environment and social relations (see also Kendler 2005; Lilienfeld 2007). The lower levels of explanation, particularly the brain, are merely among them—and not necessarily the most informative for practical purposes. Indeed, an "eliminative" or "greedy" (to use philosopher Daniel Dennett's apt term) reductionistic view—which posits that lower levels of analysis render higher levels superfluous—leads to ambivalence about the importance of holding addicts accountable and, in turn, undermines the most effective behavioral treatments available (Dennett 1995). Fortunately for addicts, their behavior can be modified by contingencies. Official rhetoric does them a disservice when it implies they are merely helpless victims of their own hijacked brains.

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