

In Search of the Neurobiology of Addiction Recovery: A Brief Commentary on Science and Stigma

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August, 2007

In 1997, Dr Alan Leshner, then Director of the National Institute on Drug Abuse (NIDA) published a seminal article, “Addiction is a Brain Disease, and It Matters,” in one of the world’s leading scientific journals (Leshner, 1997). That event was the opening salvo in a decade-long research and public education campaign to re-educate the public about the nature of addiction. The focus of this campaign has been to move “addiction is a disease” from the status of an ideological proclamation by policy activists and an organizing metaphor for individuals seeking to resolve alcohol and other drug problems to a science-grounded conclusion. The involvement of scientists was, in part, a response to earlier and continuing anti-disease polemics, e.g., *Heavy Drinking: The Myth of Alcoholism as a Disease* (Fingarette, 1989), *The Diseasing of America* (Peele, 1989), *The Myth of Addiction* (Davies, 1992) and *Addiction is a Choice* (Schaler, 2000). In the 1990s, the prolonged debate over disease conceptualizations of alcoholism and drug dependency moved from the philosophy departments to the scientific laboratories with the greatest financial investment in history in genetic and neurobiological studies of addiction. The fruits of that research triggered a campaign to re-educate the public and policy makers about the nature of addiction.

The “addiction is a brain disease” campaign has gained momentum in recent years. In a 2005 special issue of *Nature* entitled Focus on the Neurobiology of Addiction, a distinguished group of scientists assembled the latest evidence that addiction at its most fundamental essence is a neurobiological disorder. This was followed in May 2007, by Dr. Nora Volkov’s¹ historic lecture, “The Neurobiology of Free Will,” at the American Psychiatric Association’s annual conference. This lecture signaled a maturing of the research community’s understanding of addiction as a brain disease. Dr. Volkov described the most complex picture to date of how drugs compromise multiple regions of the brain and how these discrete effects collectively elevate continued AOD use as the supreme priority in personal decision-making—a priority that transcends other needs of the individual, his or her family, and society.

These findings have been communicated to the public via the metaphor of the “hijacked brain” in major media outlets--from Bill Moyers 1998 PBS special *Moyers on Addiction: Close to Home* to the 2007 HBO special *Addiction: Why Can’t They Just Stop?*--and through popular magazines--*Time Magazine*’s July 16, 2007 cover story, “How We Get Addicted”). The National Institute on Drug Abuse has attempted to explain this brain hijacking process to the public as follows:

The initial decision to take drugs is mostly voluntary. However, when drug abuse takes over, a person’s ability to exert self control can become seriously impaired. Brain imaging studies from drug-addicted individuals show physical changes in

¹ The present Director of the National Institute on Drug Abuse.

areas of the brain that are critical to judgment, decisionmaking, learning and memory, and behavioral control. Scientists believe these changes alter the way the brain works, and may help explain the compulsive and destructive behaviors of addiction. (NIDA, 2007, p. 7.)

Many recovery advocates have celebrated these scientific discoveries and have helped promote programs like the HBO special that interpret this science to the public and policy makers. The purpose of this brief commentary is to talk about a crucial missing component in the addiction science agenda and in these public awareness programs. Put simply, what is missing is recovery.

I would suggest the following hypotheses:

- 1) communicating the neuroscience of addiction without simultaneously communicating the neuroscience of recovery and the prevalence of long-term recovery will increase the stigma facing individuals and families experiencing severe alcohol and other drug problems, and
- 2) the longer addiction science is communicated to the public without conveying the corresponding recovery science, the greater the burden of that stigma will be.

Shifting the public view of the etiology of addiction from one of volitional misconduct to a brain disease may not alter social distance between alcohol and drug dependent individuals and the larger citizenry. Campaigns that sought to reduce the stigma of mental illness by educating the public that mental illness was a brain disease inadvertently invoked perceptions that the mentally ill were less than human and invoked harsher behavior toward the mentally ill (Mehta & Farina, 1997; Corrigan & Watson, 2004). While such research has not been directly replicated in the addictions field, Crawford and colleagues (1989) did find that humanitarian attitudes toward the alcoholic (e.g., a sympathetic attitude and belief that treatment should be supported by public funds) were not directly related to whether alcoholism was or was not viewed as a disease.

The vivid brain scan images of the addicted person may make that person's behavior more understandable, but they do not make the person whose brain is being scanned more desirable as a friend, lover, spouse, neighbor, or employee. In fact, in the public's eye, there is short distance between the perceptual categories of brain diseased, deranged and dangerous. We should not forget that a century ago biological models of addiction provided the policy rationale for prolonged sequestration of addicted persons and their inclusion in mandatory sterilization laws (White, 1998). Further, christening addiction a CHRONIC brain disease—as I have done in innumerable presentations and publications, may, without accompanying recovery messages, inadvertently contribute to social stigma from a public that interprets “chronic” in terms of forever and hopeless (“once an addict, always an addict”)(See Brown, 1998 for an extended discussion of this danger).

Conveying that persons addicted to alcohol and drugs have a brain disease that alters emotional affect, compromises judgment, impairs memory, inhibits one's capacity for new learning, and erodes behavioral impulse control are not communications likely to

reduce the stigma attached to alcohol and other drug problems, UNLESS there are two companion communications: 1) With abstinence and proper care, addiction-induced brain impairments rapidly reverse themselves, and 2) millions of individuals have achieved complete long-term recovery from addiction and have gone on to experience healthy, meaningful, and productive lives. Conveying these latter statements may not be as important to changing stigma as personally knowing one or more people in long-term recovery who have achieved such success, but such statements would establish a social climate in which addiction recovery could flourish and recovered and recovering people would have access to the opportunities and relationships available to other citizens.

So why don't the leading addiction scientists communicate findings related to the neurobiology of addiction recovery and the prevalence of long-term recovery? The reason would appear to be that the answers to these questions are not yet known—at least not at the same depth and certainty with which we are unraveling the neurobiology of addiction. There has been no guiding recovery research agenda to answer such questions. Preliminary studies on brain recovery from addiction following abstinence are very promising (e.g., Bartsch, Homola, Biller, et al, 2007) and recovery prevalence studies reveal rates of sustained remission higher than the public or treatment professionals would expect (Dawson, Grant, Stinson, et al, 2005; de Bruijn, van den Brink, Graaf, et al, 2006), but the neurobiology of recovery and the prevalence, pathways, styles and stages of long-term recovery remain the new frontiers of addiction research.

It is time to enter those frontiers. In the neurobiology arena, there are basic questions to be answered, including:

- To what degree does neurobiology influence who recovers from addiction and who does not achieve such recovery?
- What is the extent to which addiction-related brain pathology can be reversed through the long-term recovery process?
- What is the time period over which such pathologies are reversed in recovery—days, months, years?
- What role can pharmacological adjuncts, social support and other services play in extending and speeding this process of brain recovery?
- Are there critical differences in the extent and timing of neurobiological recovery related to age of onset of use, duration of addiction career, problem severity and complexity, age of onset of recovery, gender, genetic load for addiction, developmental trauma, ethnicity, primary drug choice, and other potentially critical factors?

We need a comprehensive recovery research agenda, and that agenda needs a strong component focused on the neurobiology of addiction recovery. The financial investment in a recovery research agenda is unlikely to be forthcoming without concerted advocacy. Every time an addiction scientist presents brain scans illustrating the neurobiology of addiction, a recovery advocate needs to be present to request the brain scans that illustrate the neurobiology of recovery.

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