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## Addictive Behaviors Reports

journal homepage: [www.elsevier.com/locate/abrep](http://www.elsevier.com/locate/abrep)

## People control their addictions no matter how much the “chronic” brain disease model of addiction indicates otherwise; we *know* that people can quit addictions—With special reference to harm reduction and mindfulness☆

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## ARTICLE INFO

## Article history:

Received 10 March 2016

Received in revised form 6 May 2016

Accepted 19 May 2016

Available online xxxxx

**Epigraph, Free Will in Action:** A philosophy professor of my acquaintance who smoked was arguing with someone in a bar in favor of free will. She self-consciously ground out her cigarette and threw the rest of the pack away. (She never smoked again.) “THAT’S free will,” she practically screamed.

## Keywords:

Chronic brain disease

Free will

Nora volkow

Harm reduction

Mindfulness

Natural recovery

## ABSTRACT

The world, led by the United States, is hell bent on establishing the absence of choice in addiction, as expressed by the defining statement that addiction is a “*chronic relapsing* brain disease” (my emphasis). The figure most associated with this model, the director of the American National Institute on Drug Abuse, Nora Volkow, claims that addiction vitiates free will through its effects on the brain. In reality, while by no means a simple task, people regularly quit their substance addictions, often by moderating their consumption, usually through mindfulness-mediated processes (Peele, 2007).

Ironically, the brain disease model’s ascendance in the U.S. corresponds with epidemic rises in opiate addiction, both painkillers (Brady et al., 2016) and heroin (CDC, n.d.), as well as heroin, painkiller, and tranquilizer poisoning deaths (Rudd et al., 2016). More to the point, the conceptual and treatment goal of eliminating choice in addiction and recovery is not only futile, but iatrogenic. Indeed, the National Institute on Alcohol Abuse and Alcoholism’s epidemiological surveys, while finding natural recovery for both drug and alcohol disorders to be typical, has found a decline in natural recovery rates (Dawson et al., 2005) and a sharp increase in AUDs (Grant et al., 2015).

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Nora Volkow, director of the American National Institute on Drug Abuse, the best-known representative of the model of addiction as a chronic brain disease (see Nature, 2014) declared “Addiction as a disease of free will” (Volkow, 2015) for which we as a society need to:

Understand that addiction is not just a disease of the brain, but one in which the circuits that enable us to exert free will no longer function as they should. Drugs disrupt these circuits. The person who is addicted does not choose to be addicted; it’s no longer a choice to take the drug.

The Surgeon General’s first report on smoking (Department of Health, Education, and Welfare, 1964), “Smoking and Health,” definitively linked smoking to cancer, beginning a cultural process over the following decades in which over half of smokers quit. But a substantial minority didn’t quit.

The Department of Health and Human Services (2002) published a volume entitled, “Those Who Continue to Smoke.” The investigators

imagined those who continued to be addicted as being handicapped in some way. The results were perplexing: “In summary, these trends do not suggest that the population of smokers who remains is more addicted, more resistant to cessation messages, less likely to attempt cessation, or increasingly composed of those with limited activities or poor mental health” (p. 143).

One particularly interesting and surprising finding in the monograph was an interaction between age and degree of dependence in smoking cessation: more dependent younger smokers were less likely to quit than less dependent ones; more dependent older smokers were *more likely* to do so. Jettisoning all assumptions about addiction, a sensible deduction would be that older heavier smokers, sensing their mortality and wanting to delay death, which they knew was more likely to ensue given the severity of their habit, were more motivated to quit and more often did so.

The whole point in smoking cessation efforts was to recognize smoking as addictive, which is now universally accepted (although the 1964 Surgeon General’s report explicitly rejected the idea; cf. Peele, 2010). Yet these results confound our notion of addiction, and certainly the ironclad, neuroscientific, brain disease model of it.

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Despite the consensus around smoking's addictiveness, perhaps these results are limited to nicotine/smoking. No, they are not. A contributor to this issue, Gene Heyman (2013), analyzed the most recent NESARC data according to a timeline of likelihood of quitting a drug dependence:

Although varied [according to the specific substance], the remission results were orderly. An exponential growth curve closely approximated the cumulative frequency of remitting for different drugs and different ethnic/racial groups. Thus, each year a constant proportion of those still addicted remitted, independent of the number of years since the onset of dependence.

In summary, addiction as the idea that people are irresistibly, inexorably, irreversibly stuck in a drug dependence so that no effort of will can extricate them is wrong, as proved by research that directly tests this belief. Yet Volkow has no fear of being contradicted while claiming the counterfactual assertion and, moreover, cloaking it with the mantle of science.

### 1. Reductionism, harm reduction, natural remission

People readily substitute “scientism” for science: that is, being awed by seemingly scientific activity in place of the actual science of testing hypotheses with data.

The *New York Times* offer one of many examples of a clinician coming to grips with harm reduction, or the idea that total abstinence is not the only beneficial outcome.

Can Nicotine Be Good for You? My new patient explained that in her sophomore year at college she had started smoking. The effect, she said, was like “a key that fit perfectly into a lock.” Her brain felt clearer, her thoughts were more coherent, her mood and energy improved. Not wanting to damage her lungs, she soon switched over to nicotine gum and had been taking the same amount of it for well over a decade. (my emphasis) She asked me what I thought of her use of the drug. The short answer was that I didn't know what to make of it.

Ultimately, this clinician could only justify allowing her client to persist in this less harmful form of addiction by concluding that her addiction was “in her brain.”

But as I thought about our conversation later, I found her image of a key in a lock particularly striking; it was the very same one that psychiatrists and neurophysiologists use to describe the interactions in the brain between neurotransmitters and their receptors. And in fact, neurons do have receptors into which nicotine neatly fits, mimicking the actions of the brain's own molecules. (Fels, 2016)

What if the writer were forced to confront the best data, which shows that people regularly overcome substance addictions, including smoking, even after they are notable for failing to do so for years, even decades? She seemingly wouldn't be able to gather sufficient moral commitment to approve of continuing the addiction in a less harmful form. Her justification for proceeding on this basis is, in my wording, “This woman can't quit her addiction. It's the neurochemical key to her brain.”

But what about all of those people who do quit addictions? At some point in my presentations, I ask the audience, “Have any of you quit a smoking addiction?” A third to a half raise their hands. Virtually none of the people in these exercises relies on the nicotine replacement therapy (NRT) used by this woman. This demonstration doesn't prove that this woman can quit chewing nicotine gum. But it is important to establish scientifically accurate parameters for this possibility.

Using this *Times* case in a workshop, I said: “Well, of course, this therapist did a brain scan to show that this particular woman has a

particular neurochemical reaction *proving* nicotine has the key to *her* brain.” No she (the therapist) didn't. She wouldn't know how. Nobody does. There is no such neurochemical key. Nor can a brain scan show that people are able, or on the verge, of quitting or cutting back their addiction. There is some experiential configuration that creates both the addiction and change that can't be broken down into neurochemical, cognitive, and situational components. The residue strongly resembles what might be called free will (Peele and Thompson, 2015).

Therapy is often directed to lowering a person like this woman's anxiety levels and to figuring out her experiential and situational keys—called addictive cues or triggers—for the purpose of assisting her to abstain. But it's the woman's right to refuse to participate in this process, and our obligation to accept her choice. What is wrong, and immoral, is to tell the woman that such change isn't possible. This phenomenon of mislabeling continued addiction as a biological imperative has been imbedded in harm reduction by Dole and Nyswander (1967), the developers of methadone maintenance, through their claim that former heroin users absolutely require substitute narcotics such as methadone or buprenorphine because they suffer from a permanent “metabolic disease.”

But they don't. Maybe people want that substitute (and who is to say this “want” is not a “need”), either currently, for a long time, or forever. It's their right to choose, without added guilt laid on by therapists or would-be helpers. The parallel is inescapable to observers who justify gay lifestyles on the grounds that people's sexuality is genetically determined. Would such defenders of gay rights then arrest people who chose a same-sex mate but who were proved not to have a gay gene (one that does not exist)? Of course, when confronted with bisexuality, such well-meaning advocates for tolerance based on biological imperatives are left hemming and hawing.

### 2. The strange (according to whom?) workings of the human mind

In the area of harm reduction, two landmark studies show that an outcome once claimed by both Alcoholics Anonymous and the journal *Science* (Pendery et al., 1982) to be nigh on impossible occurs regularly—as I, along with another contributor to this volume, Nick Heather, have noted for three decades (Heather and Robertson, 1981; Peele, 1983, 1987b, 2013). Neither study explicitly addresses harm reduction, or what was then called controlled drinking. Yet each offers fundamental insight into its natural occurrence.

A treatment study with alcohol-dependent subjects conducted by the most prestigious pharmacologically-based research center in the United States, at the University of Pennsylvania as led by Charles O'Brien, attempted to establish the benefits of “pharmacogenetic matching” in the case of Naltrexone treatment (NTX) for alcoholism (Oslin et al.,

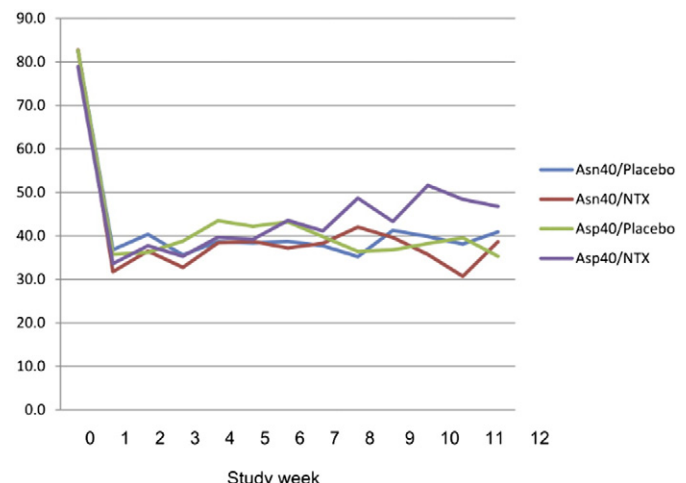


Fig. 1. Percent days of any drinking during the course of treatment.

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