Editorial

What are the public policy implications of a neurobiological view of addiction?

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This editorial discusses the limitations of equating addiction with neurobiological processes and the potential benefits and risks of this approach for public health policy.

Neuroscience research has provided a greater understanding of the neurochemical mechanisms underpinning drug use and addiction. Alteration of gene regulation and functioning appears to explain many of the persistent changes in brain structure and function that are observed after chronic substance use. These neurobiological changes may explain why relapse is common, even after years of abstinence [1]. This research also indicates that there are molecular, cellular and other system-level mechanisms common to both drug addiction and other compulsive behaviours, such as gambling, hypersexuality and compulsive over-eating [2–4].

How should these findings affect the way in which we deal with addiction to illicit drugs, tobacco smoking, alcohol consumption and 'behavioural addictions'? Should we view addiction primarily as a disease, like other physical ailments, and treat it medically? This view, often described as the 'brain disease' model of addiction, is promoted strongly by prominent public health researchers, neuroscientists and addiction physicians [5,6]. Advocates believe that it will produce a range of clinical and social benefits that include: reduced stigma towards addicted individuals, increased treatment-seeking and compliance with medical treatment regimens, less reliance on imprisonment and other punitive responses to addictive drug use and greater investment in addiction treatment and research [5,6].

The public health consequences of the brain disease view of addiction may be complex and unexpected. A complete analysis of its potential impact needs to consider adverse consequences as well as benefits.

ADDICTION AS A 'BRAIN DISEASE': A REASON TO QUIT OR AN EXCUSE FOR FAILURE?

Confidence in one's ability to remain abstinent—abstinence self-efficacy—reliably predicts future abstinence from smoking and other drug use [7]. Acceptance of the 'brain disease' view could increase abstinence self-efficacy if smokers or drug-dependent people believe that addiction can be overcome with medical assistance; for example, pharmaceutical treatments such as nicotine replacement therapy, buproprion and varenicline for smoking cessation [8]. These pharmacological therapies have demonstrated efficacy and improve smoking cessation outcomes. However, they currently have little impact on population smoking prevalence because of low uptake by smokers [9]. Increasing the proportion of quit attempts that are assisted could help to reduce smoking prevalence if a sufficient number of smokers used these methods.

It is also possible that the 'brain disease' view may erode confidence in one's ability to achieve and maintain abstinence and discourage quit attempts if a brain disease was seen as inherently unchangeable. Some commentators worry that handing the responsibility for quitting to medical professionals could lead addicted individuals to absolve themselves of responsibility for their drug taking. This could reduce self-efficacy and motivation to help themselves [9] and adversely affect their ability to maintain abstinence in the long term. Studies have shown that smokers who attribute a failure to quit to unchangeable intrinsic factors, such as the 'type of person', have lower personal quitting intentions and lower quitting self-efficacy [10]. While some addicted individuals do develop a chronic and relapsing disorder consistent with the 'brain disease' view of addiction, the vast majority of addicted

individuals mature out of their addiction and recover naturally without requiring medical treatment [11]. In the case of tobacco smoking, ex-smokers outnumber current smokers, and most of these quit without any formal cessation assistance [9]. Convincing smokers that they have a 'brain disease' could reduce unassisted or natural recovery by deterring smokers from attempting to quit or reducing their chances of success if they do make an attempt.

NEUROBIOLOGY AND NOVEL TREATMENTS FOR ADDICTION

A major criticism of the 'brain disease' model of addiction is that it medicalizes human behaviour and overemphasizes the biological bases of behaviour at the expense of social and psychological influences [12]. These critics argue that depicting addiction as a 'brain disease' could privilege the development and use of expensive and sometimes risky medical interventions, such as drug vaccines [13] and deep brain stimulation [14], to the neglect of proven social policies such as taxation and regulation [9,15]. The idea that addiction is a 'brain disease' might also lend itself to the view that we should identify the minority of people who are most susceptible and subject them to individually focused preventive measures (e.g. vaccines) [16] rather than using strategies that target the entire population.

THE IMPORTANCE OF POPULATION-WIDE MEASURES

A near universal hope in scientific reports on the neurobiological basis of addiction is that this research will lead to more effective new treatments for addiction. However, as a recent commentary noted, very few effective treatments exist despite decades of research into the neurobiological processes underlying addiction [17]. Research into the environmental and social factors that contribute to drug addiction, or emerging issues such as obesity, is likely to produce policy interventions that reach a greater proportion of the population, and are faster and cheaper to implement. The prevalence of tobacco smoking continues to fall in high-income countries, despite a lack of treatment breakthroughs, thanks to effective public health policies such as increased tobacco taxation, public smoking bans, advertising bans and counter-advertising. New expensive pharmacological treatments for tobacco smoking are of limited relevance to the majority of the world's current smokers, who now reside in low- and middle-income countries [18].

A major risk of focusing on neurobiological mechanisms of addiction is that it perpetuates a view of addiction as a problem of individuals that is best addressed by medical treatment. This shifts the attention away from societal and environmental contributions to drug use and addiction. There is strong evidence that environmental approaches to reducing substance use are the most effective in reducing population-level harm. Vested interests such as the alcohol, tobacco and gambling industries have demonstrated their willingness to misuse neurobiological research to shift attention away from their role in establishing and maintaining addiction by marketing addictive products [19]. It is important that policy makers and government officials are not seduced by the allure of neuroscience to neglect population-level approaches that target the causes of addiction that are the most amenable to change and have the greatest chance of reducing substance use and related harm.

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