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ABSTRACT

Neuroscience research on animals has identified the neurochemical circuitry on which psychoactive drugs of dependence act and produced models of the development of tolerance, elicitation of withdrawal symptoms, and the rapid reinstatement of drug use after abstinence. Human neuroimaging studies have provided support for the hypothesis that similar neurobiological processes are at work in addicted humans. Leading proponents of neurobiological addiction research have argued that it provides strong support for the view that addiction is a chronic brain disease. They also argue that broad acceptance of the model will improve societal attitudes and policies towards addicted persons. We briefly examine the research evidence for the claim that addiction is a chronic relapsing brain disease and discuss the promised advantages of the brain disease model of addiction and the likelihood of their realization. We also discuss the potential costs of social policies advocated on the basis of addiction neuroscience research, namely, advocacy of ‘high risk’ social policies that are congenial to legal industries that promote the use of addictive commodities; renewed advocacy of legally coerced addiction treatment; and the promotion of research into expensive, high technology, biomedical interventions that aim to treat addiction by directly intervening in ‘addicted brains’.

Introduction

A minority of regular drug users persist in using drugs in the face of problems caused by their use (Gerstein & Harwood, 1990), a pattern of drug use that earns a diagnosis of addiction. There has been a long running debate between the dominant commonsense understanding of this behaviour – that drug users simply choose to behave in this way – and a medical model of addiction that sees this behaviour as a symptom of a biologically-based illness caused by changes in brain function that chronic drug use produces that makes it difficult for addicted drug users to desist from using drugs (Carter & Hall, 2012).

The commonsense, moral model of addiction explains addicted patterns of drug use in much the same way as any other socially disapproved behaviour, that is, as a behaviour in which the drug user chooses to engage. ‘Addiction’ is often dismissed as a self-interested excuse used by drug users to evade moral and legal responsibility for their behaviour. On this view, drug users who break the criminal law (e.g. to fund their drug use) should be prosecuted and punished if found guilty in the same way as other offenders (Szasz, 1975).

A medical model of addiction, by contrast, argues that a minority of chronic drug users develop a mental or medical disorder – an addiction – that requires treatment if the sufferer is to become and remain abstinent (e.g. Leshner, 1997; Volkow & Li, 2004). The idea that addiction is a disease was first advocated at the end of the eighteenth and beginning of the nineteenth centuries in order to explain the chronicity of problem alcohol use. In the last three decades, neuroscience research has provided renewed support for a medical model by providing more detailed analyses of brain processes that, its advocates claim, explain the difficulties that addicted persons have in quitting and avoiding a return to drug use if they do.

According to its proponents, broad acceptance of the brain disease model will produce major, beneficial policy changes for assisting addicted individuals (Leshner, 1997; Volkow & Li, 2004).
Specifically, they claim that it will encourage more humane therapeutic responses by ensuring the provision of more accessible and effective treatment of addiction that is either publicly funded or covered by health insurance (Dackis & O'Brien, 2005; McLellan, Lewis, O'Brien, & Kleber, 2000).

In this paper we briefly summarize the evidence offered for the claim that addiction is a brain disease and discuss the proposed policy benefits that would flow from its wider social acceptance. We first summarize and briefly examine the evidence offered in support of this view of addiction. We then outline the claims made for the beneficial social impacts of its acceptance. We next consider more critical reviews of the evidence for the model and discuss some possibly less welcome potential policy uses that may be made of the brain disease model of addiction (BDMA) (Carter & Hall, 2012).

We briefly outline three specific policy implications that some have drawn from the brain disease model, namely, that we should trial deep brain stimulation (DBS) as a treatment for addiction (e.g. Luigjes et al., 2012; Stephen et al., 2012); that we should compel some persons to be treated for their addiction for their own good (Caplan, 2008); and that we should attempt to identify persons at high social and genetic risk of developing an addiction and intervene to prevent them from doing so (e.g. Hutchison, 2010; Singh & Rose, 2009).

Evidence for a BDMA

Animal models of drug self-administration
Addiction neuroscience models of the brain processes underlying both acute drug effects and chronic drug use rely on studies of drug self-administration studies in rats (Koob, 2006). These models possess considerable face validity for human addiction in that rats will self-administer psychoactive drugs (by injection in the case of cocaine, heroin and nicotine and by ingestion in the case of alcohol) at high frequencies (Koob, 2006). These models also possess predictive validity in that the drugs that animals will most readily self-administer are also those that are most addictive in humans. The fact that self-administration of these drugs is reduced by direct electrical stimulation of ‘reward centres’ in rats’ brains adds weight to the view that self-administration of these drugs is rewarding (Koob, 2006).

Animal models have identified the brain circuitry on which the major drugs of addiction act (Koob & Volkow, 2010). For example, they have shown that ablating or chemically blocking dopamine receptors in brain reward circuits abolishes self-administration of these drugs. This work has also identified a critical role for the dopaminergic ‘mesolimbic brain reward system’. This consists of the ventral striatum, nucleus accumbens, amygdala, frontal cortices and other interconnected structures that respond to rewards in which the neurotransmitter dopamine plays a key role (Koob & Le Moal, 2006).

Animal models of chronic drug self-administration also appear to reproduce key features of human addiction, such as drug tolerance and dose escalation, withdrawal and reinstatement following abstinence (Koob & Le Moal, 2006). Researchers have shown, for example, that animals given free access to drugs will increase the frequency and amount of drug that is self-administered and they will work increasingly harder to obtain drugs, suggesting the development of tolerance. Animals also persist in performing the behavior rewarded by self-administration (e.g. a bar press) long after drug delivery has ceased and will often do so in the face of aversive stimuli (e.g. electrical foot shock). They will also rapidly resume self-administration if given a priming dose of the drug, or if presented with painful stimuli or cues (e.g. a light) that were previously associated with drug delivery (Koob & Le Moal, 2006).

Human neuroimaging studies
Over the past two decades findings from animal studies of addiction have received support from functional human neuroimaging studies of the effects of psychoactive drugs on the functioning of ‘normal’ and ‘addicted’ brains (Koob & Le Moal, 2006; Koob & Volkow, 2010). Radioactively labelled tracers have enabled researchers to identify the molecular sites of action in the brain of most
major drugs of addiction in humans. Functional magnetic resonance imaging has identified differences in the responses of specific brain regions to the effects of drugs and the presentation of drug-related cues to addicted and non-addicted persons (Volkow, Fowler, Wang, Teland, & Baler, 2010).

Neuroimaging studies also support the hypothesis that drugs of addiction act on the dopaminergic mesolimbic reward system in similar ways in humans and animals. In addition, human neuroimaging studies have identified dopamine-mediated changes in key cortical areas (especially the orbitofrontal cortex) that are correlated with impaired decision-making and impulse control (Koob & Le Moal, 2006). Many of these changes persist after sustained periods of abstinence, suggesting that these brain changes underlie the high rates of relapse to drug use in addicted persons who become abstinent (Reske & Paulus, 2011).

Human genetics of addiction
Twin and adoption studies of addictive disorders have shown that genetic susceptibility makes a substantial contribution to the risk of developing most common forms of human addiction (Ball, 2008; Kendler et al., 2012). Twin studies estimate that the heritability (i.e. percentage of variation explained by genetic factors) of alcohol, nicotine and cannabis dependence is typically in the range of 40–60% (Ball, 2008). These studies have prompted searches for specific alleles that influence drug metabolism and levels of brain neurotransmitters and transporters. Large-scale Genome Wide Association Studies (GWAS) have found correlations between genetic markers (single nucleotide polymorphisms) and addiction risk. The identification of risk alleles that influence function in the mesolimbic reward system seems to fit well with the hypothesis that addiction is a brain disease caused by the chronic use of addictive substances in vulnerable individuals.

Clinical evidence of the chronicity of addiction
The chronic and relapsing nature of addiction is central to the brain disease model. Indeed, chronicity was the major reason for the hypothesis that addiction was a brain disease in the late nineteenth century (Courtwright, 2010). Physicians who treated alcoholism and opiate dependence in the second half of the nineteenth century reported that their patients often returned to alcohol and opiate use after lengthy periods of abstinence. They hypothesized that chronic drug use had changed the brains of ‘inebriates’ in ways that made it difficult for them to desist from using drugs. Neuroscience research in the late twentieth century claims to explain the brain mechanisms that underlie these high rates of relapse in individuals treated for addiction.

Addiction neuroscience research also provides explanations for the effectiveness of pharmacological addiction treatments such as methadone maintenance, nicotine replacement, varenicline and naltrexone (Koob, Lloyd, & Mason, 2009). All these drugs act on the same brain receptors as drugs of dependence, enabling addicted persons to remain abstinent by blocking or attenuating the rewarding effects of drugs of dependence.

Recent clinical observations of Parkinson's patients have provided novel support for the claim that the chronic use of drugs that act on the mesolimbic dopaminergic reward system can produce addictive behaviour. A substantial minority of patients treated for Parkinson's disease with dopamine replacement therapy (DRT) – perhaps as many as one in six – develop compulsive disorders, such as compulsive gambling, sexual behaviours and overeating (Weintraub et al., 2010). The causal relationship between these disorders and DRT is supported by the fact that they develop after DRT is initiated and usually remit upon cessation or substantial reductions in the dose of DRT medication (Ambermoon, Carter, Hall, Dissanayaka, & O'Sullivan, 2011; Carter, Ambermoon, & Hall, 2011).

Criticism of the BDMA
Recent critics of the BDMA focus on contesting the central claim that addiction is a chronic relapsing disorder that overwhelms an individual's ability to control or reduce their drug use. They primarily
cite findings from epidemiological surveys of mental disorders showing that the majority of addicted persons recover without treatment (e.g. Heyman, 2009; Kincaid & Sullivan, 2010). Advocates of the BDMA use the same survey data to claim that addiction is a highly prevalent disorder that causes serious social and economic problems (Koob & Le Moal, 2006). Yet, as Heyman (2009) points out, most of the persons who have at some time in their lives met diagnostic criteria for dependence in these surveys are not dependent at the time of interview. Most have ceased using their drug of dependence years, often decades, before, and usually without any professional assistance.

These survey data have several limitations: reports of past symptoms of addictive disorders are subject to selective recall, while persons with more severe addictive disorders are often not well represented in surveys (Degenhardt & Hall, 2012). Nonetheless, similar results have been reported in studies of representative samples of addicted persons in the population who have been followed over time. These include longitudinal studies of persons with what is regarded as one of the more severe forms of addiction, namely heroin addiction. Robins and colleagues, for example, followed up US servicemen who had used heroin in Vietnam three years after their return to the USA (Robins, 1993; Robins, Helzer, Hesselbrock, & Wish, 2010). Most of those who had regularly used heroin in Vietnam ceased use before they returned to the USA and most did not become re-addicted after their return. Similar results have been reported in longitudinal studies of adolescent drug users followed into adulthood. Substantial proportions of young people in these surveys meet criteria for dependence on alcohol, cannabis and other drugs in late adolescence and early adulthood (e.g. Fergusson, Boden, & Horwood, 2008) but cessation of drug use has been the norm, usually in the absence of treatment, as a result of life events, such as securing paid employment, marrying and having children (Bachman, Wadsworth, O’Malley, Johnston, & Schulenberg, 1997).

Critics of the BDMA also cite evidence on the efficacy of psychological treatments that seem incompatible with strong forms of the BDMA (Heyman, 2009). Foremost among these is contingency management in which small incentives (e.g. financial rewards for providing clean urine samples) substantially reduce drug use in addicted persons (Heyman, 2009; Higgins et al., 2010). The responsiveness of addicted drug users to small changes in the consequences of their drug use seems hard to reconcile with a brain disease model in which addictive drug use is seen as a compulsive form of behaviour driven by impaired brain reward and cognitive control systems over which the individual has little or no control.

Reconciling competing views of addiction

These seemingly contradictory views of addiction embodied in the BDMA and the counterviews of the sceptics can potentially be reconciled if we accept that addictive disorders vary in severity, and that milder forms are much more common in the population. The milder disorders occur in young adults in whom most remit without treatment as drug use changes in response to life events such as marriage, mortgages and children (Bachman et al., 1997). Such disorders can nonetheless harm young drug users (e.g. via deaths, injuries, suicides, mental disorders and impaired educational achievement) and therefore require policy attention, such as public education and self-help advice delivered via the internet, rather than specialist addiction treatment (Hall & Swift, 2006; Hall & Teesson, 1999).

The natural history of problem drug use in young adults also suggests that a minority of those who do develop an addictive pattern of drug use will continue to do so well into their early 30s. The disorders in this group of chronic drug users most clearly resemble the chronic relapsing disorders seen in treated populations. It is arguably this group of addicted persons in whom persistent alterations in brain function may plausibly play a role in perpetuating drug use (Kincaid & Sullivan, 2010).

Acknowledging these features of the epidemiology of addictive behaviours undermines the strongest form of the model as encapsulated in the unqualified claim that ‘addiction is a chronic relapsing brain disease’ (Koob & Le Moal, 2006; Koob & Volkow, 2010). It would be more accurate to say that: ‘a minority of persons with more severe forms of addiction can develop chronic relapsing disorders’.
This weaker claim means that population estimates of the lifetime prevalence of addictive disorders are not the same as the prevalence of cases of addiction severe enough to warrant the diagnosis of a chronic and relapsing brain disorder.

Epidemiological research has identified who is most at risk of developing these chronic addictive disorders. Foremost among these are males who perform poorly in primary school and engage in antisocial behaviour in their teens, initiate all types of drug use at a young age, preferentially affiliate with other drug using peers, have few relationships with non-drug using peers and whose life choices in adulthood are restricted by their failure to complete secondary education or any post-secondary training (Fergusson et al., 2008). We also know the characteristics of the persistent heavy drug users who receive specialist treatment for addiction (Hall, Teesson, Lynskey, & Degenhardt, 1999). They are heavy users in their mid-30s, who have often made multiple failed attempts to quit, and often enter treatment under social pressure from their partners and families, employers or the courts for convictions for drink-driving, assault or drug-related property crimes (Hall & Lucke, 2010). The BDMA provides a better fit for this minority of chronically addicted persons than it does to the much larger proportion of young adults who report problem drug use in population surveys.

If we accept this analysis, how useful is it for public policy purposes to think of chronically addicted individuals as suffering from a brain disease? What are the implications for public policy in deciding to treat severe forms of addiction as chronic, relapsing brain diseases?

Potential benefits of the BDMA

Improved addiction treatment
A central claim made by advocates of the BMDA is that it will lead to more effective, biologically based, treatments of addiction (Leshner, 1997; Volkow & Li, 2004). These may include drugs that will directly target underlying neural mechanisms of addiction, new biological approaches to reduce relapse, such as drug vaccines, implantable agonists and antagonists, and using genomic information to match patients to the treatment that is most likely to be successful.

So far these benefits remain promissory. The number of new drug treatments approved for addiction over the past several decades has been modest (e.g. acamprosate, buprenorphine, naltrexone and varenicline) and their efficacy while superior to placebo is slight (Kalant, 2010; Koob et al., 2009). There remain major challenges in developing better drug treatments, namely, the long lead time between identifying promising new therapeutic agents and delivering them into routine care because of the substantial costs and time required to undertake clinical trials. Pharmaceutical companies have also been reluctant to invest in developing new drugs for addiction because of the stigma associated with these disorders and doubts about whether these treatments will be profitable. They may also not wish to develop treatments for addiction if the same drugs have potentially more profitable uses, such as the treatment of chronic pain (Koob et al., 2009).

DBS for intractable addiction
DBS is a targeted form of neurosurgery that has recently been advocated for the treatment of addiction (Luigjes et al., 2012; Stephen et al., 2012). DBS involves inserting microelectrodes into specific regions of the brain and modulating neural activity by applying an electrical current. It was first used to treat movement disorders in Parkinson's disease (Benabid et al., 1993), but is now being investigated to treat intractable psychiatric disorders, such as Tourette's syndrome (Servello, Porta, Sassi, Brambilla, & Robertson, 2008), obsessive compulsive disorder (FDA, 2009; Lipsman, Neimat, & Lozano, 2007), and depression (Schlaepfer et al., 2008).

Advocates of trialling DBS to treat drug addiction (Bauer, Pohl, Klosterkotter, & Kuhn, 2008; Lu, Wang, & Kosten, 2009; Luigjes et al., 2012) cite evidence from animal studies in which lesions in the dopaminergic reward pathway reduce drug self-administration (e.g. Knapp, Tozier, Pak, Ciraulo, & Kornetsky, 2009; Rouaud et al., 2010; Vassoler et al., 2008). They also cite case reports that DBS has
reduced co-morbid addictive behaviour in patients treated for other psychiatric conditions (see Carter & Hall, 2011 for a review).

We and others have criticized the evidence base for trials in addiction (Carter, Bell, Racine, & Hall, 2011; Carter & Hall, 2011; Synofzik & Schlaepfer, 2008). DBS is an invasive intervention that carries significant short- and long-term risks in 1–2% of patients. It costs over US$50,000 for the surgery, with ongoing maintenance costs of over US$10,000 every few years (Baltuch & Stern, 2007). Given the enormous shortfall in access to less expensive forms of addiction treatment, DBS will, at best, only be available to the very few patients who can afford it. The opportunity costs of providing DBS, even if it proves safe and effective, arguably make such trials a very low priority for public funding (Carter & Hall, 2011).

Compulsory addiction treatment for paternalistic reasons

In paternalistically motivated compulsory addiction treatment, an addicted individual is compelled to undergo treatment for their own good. A disease model of alcoholism was the rationale for passage of Inebriates Acts in Australia in the late nineteenth century. Under this legislation, magistrates could sentence an ‘inebriate’, typically an alcohol dependent male, to residential ‘treatment’ for up to six months (Lewis, 1992).

A specific form of mandatory treatment for heroin addiction has recently been advocated by a leading bioethicist (Caplan, 2008). Caplan has justified this proposal by appeal to a BDMA, arguing that the autonomy of addicted persons is impaired in ways that make them unable to act in their own best interests, so the state should compel them to undergo treatment in their own best interests (Caplan, 2008). He proposed that opioid addicted persons should be mandated by courts to use naltrexone implants (Caplan, 2008; Sullivan et al., 2008). He argues that opioid addiction robs individuals of their autonomy and that providing them with implantable naltrexone restores their autonomy by removing their cravings for heroin and blocking its euphoric effects should they succumb to temptation. We have criticized this superficially plausible approach in detail elsewhere (Hall, Capps, & Carter, 2008).

Potential public policy uses of the BDMA

The BDMA has not replaced the moral view of addiction in the country where it has been most heavily promoted: the USA. There has arguably been limited legislative support for this model in the USA despite over 15 years’ advocacy by successive directors of the leading NIH body that funds research on addiction, the National Institute on Drug Abuse (NIDA) (Leshner, 1997; Volkow & Li, 2004). NIDA has expended considerable reputational capital and resources in promoting this view (e.g. Dackis & O'Brien, 2005; Leshner, 1997; Volkow & Li, 2004).

In speculating about what may happen over the next several decades, it is safe to predict that the policy implications drawn from addiction neuroscience will be refracted through pre-existing beliefs about addictive drugs and those who use them. Our moral views and biases are notoriously difficult to shift (Kahneman, 2011). Neuroscientists of more liberal views see neuroscience research on addiction as providing support for the decriminalization, if not the legalization, of cannabis and MDMA (e.g. Nutt, 2009). We agree with Courtwright (2010) that those of more conservative views regarding addiction neuroscience see it as strengthening the case for the status quo, or possibly justifying even more punitive policies in order to prevent an epidemic of an acquired ‘brain disease’.

Legal drugs: an overinvestment in high risk strategies?
A major concern is that neurobiological explanations of addiction will shift the policy focus to the biomedical treatment of severely addicted persons and away from social policies that aim to reduce problematic drug use. It may also be more attractive for governments to fund research that promises to provide a ‘cure’ for a ‘disease of addiction’ than it is to fund social policies that aim to address the social drivers of addiction, such as poverty, lack of education and social isolation (Kleiman, 1992). Governments also find enthusiastic and well-resourced support for such policies from the alcohol, tobacco and gambling industries that profit from selling addictive commodities (Miller, Carter, & De Groot, 2012).

Predictive genomic medicine and neurobiological treatments of addiction promise to deliver high-technology solutions to addiction by targeting interventions at individuals who are at higher risk of developing addiction (Collins, 1999). Social scientists and public health professionals have expressed concerns that the premature adoption of these approaches may be at the expense of more broadly effective population health policies (Carter & Hall, 2012; Rose, 1992).

Population health policies affect the whole community, not just those who are drug dependent or at the risk of becoming so. Population-based tobacco control strategies, such as taxing cigarettes, banning advertising and restricting the areas where people can smoke, have halved cigarette smoking in Australia (White, Hill, Siahpush, & Bobevski, 2003) and the US (Pierce et al., 1998) over the past three decades. These strategies are much more effective and efficient than strategies that focus only on addicted persons, or persons at high risks of developing addiction (Rose, 1992; Vos et al., 2010). The reason is simple: fewer social resources are needed to increase taxes, ban advertising and restrict opportunities to smoke than are needed to screen whole populations to identify and intervene with the minority who are at high genetic risk of dependence, if they smoke tobacco (Hall, Madden, & Lynskey, 2002). There is similarly strong evidence for the greater effectiveness and efficiency of population-based strategies than high risk strategies in reducing risky alcohol use (Doran, Hall, Shakeshaft, Vos, & Cobiac, 2010).

The concept of addiction as a brain disease lends itself to a seductive policy simplification that is promoted by the alcohol and tobacco industries (and more recently the gambling industry), namely, that the sole policy goal should be to identify the minority of drinkers, smokers and gamblers who are biologically at the risk of developing addiction and treat them, leaving the rest of the population to drink, smoke and gamble with impunity (Hall et al., 2002; Miller et al., 2012). This view ignores the adverse public health effects of alcohol intoxication, cigarette smoking and problem gambling; it is also at odds with the dimensional nature of problem drug use; and it greatly oversimplifies the findings of genetic and neuroscientific research (Gartner, Barendregt, & Hall, 2009; Hall et al., 2002).

These policies are much more attractive to the alcohol, tobacco and gambling industries than population-level approaches that reduce per capita consumption of their products. We should accordingly be concerned about the misuse of neuroscience research by these industries. The tobacco industry, for example, has a history of promoting research on genetic susceptibility to tobacco-related disease (Brandt, 2007; Gundle, Dingel, & Koenig, 2010). Analyses of tobacco industry documents show that it funded genetic research on smoking in the 1970s and 1980s (Gundle et al., 2010) in the hope that this would locate the risks of smoking in the genome of smokers rather than in the product itself. The alcohol industry has also promoted the idea that alcohol-related problems are confined to a minority of genetically and neurobiologically vulnerable drinkers (Midanik, 2006; Miller et al., 2012). The alcohol industry's preferred policy approach to advocate interventions for problem drinkers rather than increasing alcohol taxes and reducing alcohol availability (Babor, Miller, & Edwards, 2010). The gambling industry, in the face of threats of restrictive regulation (e.g. mandatory precommitment and maximum betting limits), has funded research into the genetics and neurobiology of problem gambling (Vrecko, 2008), presumably for similar strategic reasons.
One can only speculate about the possible effects that addiction neuroscience will have on illicit drug policy. Some neuroscientists have argued that there is no neurobiological justification for the fact that alcohol and tobacco are legal while heroin, cocaine and cannabis are not (Nutt, King, Saulsbury, & Blakemore, 2007). The legal status of these drugs, they argue, is not correlated in any simple way with their effects on brain neurochemistry (Ashcroft, Campbell, & Capps, 2007; Nutt et al., 2007). These neuroscientists (e.g., Iversen, 2002; Nutt et al., 2007) have argued that policies towards licit and illicit drugs should better reflect their comparative harmfulness to users.

Despite this, policies towards licit and illicit drugs have moved in different directions and for reasons that bear little relationship to findings on the neurobiological bases of addiction, tobacco policies in developed countries have become more like policies towards illicit drugs: restricting promotion, sale, opportunities to smoke and increasing tobacco taxes. This has taken a half-century of anti-smoking efforts in the face of concerted opposition from the tobacco industry (Brandt, 2007; Proctor, 2012).

Alcohol policy, by contrast, has been deregulated in most developed countries. Trading hours have increased, alcohol is readily available in supermarkets and corner stores, and it is heavily discounted and promoted to young adults (Room, 2007). Government policies have similarly expanded access to gambling (Orford, 2011; Productivity Commission, 2010).

By contrast there has been limited success in attempts to liberalize drug laws over the last half-century. The most widely advocated form of liberalization has been the decriminalization of cannabis. Under this policy, it remains illegal to produce or supply cannabis but fines or other noncriminal penalties are imposed for possession and/or use of quantities of cannabis up to a specified maximum amount. Persons possessing larger quantities of cannabis may still face criminal charges. The main argument offered in favour of decriminalization has been that it reduces the societal costs of cannabis prohibition while retaining most of its benefits (Hall & Pacula, 2003; Room, Fischer, Hall, Lenton, & Reuter, 2010).

There are good reasons to doubt that neuroscience will resolve policy debates in such a politically and morally saturated area as illicit drug policy. We are inclined to agree with David Courwright's hypothesis about the likely impact of NIDA's brain disease model on US drug policy: that addiction neuroscience research is more likely to be used in justifying more restrictive policies towards illicit drugs in order to prevent adolescents from acquiring a ‘chronic brain disease’.

Conclusions

The most direct potential benefit from an improved understanding of the neurobiology of addictive disorders is improved treatment. But major challenges remain to be overcome before these benefits are realized. The impact of the BDMA on policies towards addiction is much less certain. It could encourage research into expensive biological interventions to treat addiction and give renewed support to more coercive approaches to treating addiction. It may also be used by industries that market addictive commodities to undermine public health drug control policies. This is despite the fact that it is much simpler, cheaper and more efficient to use social policies to discourage the whole population from smoking tobacco, drinking heavily or engaging in problem gambling.

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REFERENCES


Australia, 192, 468–470.


Hutchison, K.E. (2010). Substance use disorders: Realizing the promise of pharmacogenomics and personalized medicine.
research ethics requires more than informed consent.

In A. Carter, W. Hall, & J. Illes (Eds.), Addiction neuroethics: The ethics of addiction research and treatment (pp. 278–301). New York: Elseveir.


