ARTICLE IN PRESS

International Journal of Drug Policy xxx (2016) xxx-xxx

Contents lists available at ScienceDirect



International Journal of Drug Policy

journal homepage: www.elsevier.com/locate/drugpo



Review paper

Alcohol and cannabis: Comparing their adverse health effects and regulatory regimes

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ARTICLE INFO

Article history:
Received 15 June 2016
Received in revised form 4 October 2016
Accepted 28 October 2016
Available online xxx

Keywords: Cannabis Alcohol Adverse health effects Regulatory regime

ABSTRACT

The claim that the adverse health effects of cannabis are much less serious than those of alcohol has been central to the case for cannabis legalisation. Regulators in US states that have legalised cannabis have adopted regulatory models based on alcohol. This paper critically examines the claim about adverse health effects and the wisdom of regulating cannabis like alcohol. First, it compares what we know about the adverse health effects of alcohol and cannabis. Second, it discusses the uncertainties about the long term health effects of sustained daily cannabis use. Third, it speculates about how the adverse health effects of cannabis may change after legalisation. Fourth, it questions the assumption that alcohol provides the best regulatory model for a legal cannabis market. Fifth, it outlines the major challenges in regulating cannabis under the liberal alcohol-like regulatory regimes now being introduced.

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Comparisons of the adverse health effects of alcohol and cannabis have been central to the case for cannabis legalisation (BBC, 2014; Boffey, 2014; Editorial Board of the New York Times, 2014). The four US states that have so far legalised cannabis for adult use – Colorado, Washington State (2012), Alaska and Oregon (2014) – have largely adopted regulatory regimes modelled on those for alcohol (Hall & Lynske, 2016a, 2016b, 2016c; Pardo, 2014).

There are understandable reasons for the comparisons of adverse health effects and the implicit regulatory lessons from alcohol drawn by those advocating for legalisation. First, alcohol and cannabis are used in similar social contexts and for similar reasons, namely to improve mood and to enhance conviviality and the enjoyment of recreational activities (Hall & Pacula, 2010). Second, any comparison of adverse health effects favours cannabis because its adverse health effects are very modest compared with those of alcohol (Hall, Room, & Bondy, 1999; Room, Fischer, Hall, Lenton, & Reuter, 2010). Third, the comparison highlights a major form of societal hypocrisy in most developed countries, namely, that the use of a less harmful drug like cannabis is prohibited (on pain of imprisonment) while a much more dangerous drug like alcohol is freely available, heavily promoted and widely used in ways that cause substantial harm to drinkers and others. Fourth, advocates of reform may want to avoid comparing cannabis with the other widely smoked drug, tobacco, in order to avoid any argument that tobacco control provides a more suitable regulatory model for cannabis than alcohol. Given these factors it may seem a reasonable inference that the most appropriate regulatory approach to a legal cannabis market would be one based on that for alcohol, possibly with less stringent enforcement.

This paper critically examines these assumptions. First, it compares what we know now about the adverse health effects of alcohol and cannabis. Second, it discusses the major uncertainties that remain about the long term health effects of sustained regular cannabis use. Third, it speculates on how the adverse health effects of cannabis may change after the legalisation of recreational use. Fourth, it questions the assumption that alcohol is the best regulatory model for a legal cannabis market by asking how successful alcohol regulation has been in minimising the adverse health effects of alcohol. Fifth, it concludes with an outline of the major challenges in regulating cannabis under a modified alcohol regulatory regime.

The adverse health effects of alcohol

Thanks to over half a century of epidemiological research, the adverse health effects of alcohol are reasonably well understood (Babor et al., 2010; Parry, Patra, & Rehm, 2011; Rehm & Shield, 2013; Shield, Parry, & Rehm, 2013). In large doses alcohol can cause fatal overdoses from respiratory depression and alcohol intoxication, is a major cause of road accidents, and contributes to assaults and suicide (Babor et al., 2010). When consumed heavily and regularly, alcohol can cause a dependence syndrome and other mental disorders such as severe depression and psychosis (Connor,

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http://dx.doi.org/10.1016/j.drugpo.2016.10.021 0955-3959/© 2016 Elsevier B.V. All rights reserved. W. Hall/International Journal of Drug Policy xxx (2016) xxx-xxx

Haber, & Hall, 2016). Sustained heavy use increases the risks of liver cirrhosis, pancreatitis, cancers of the oral cavity, breast, and colon, some types of heart disease and stroke, and neurological diseases such as Wernicke–Korsakov syndrome and dementia (Rehm et al., 2013). For these reasons, alcohol use makes a substantial contribution to the global burden of disease (Forouzanfar et al., 2015; Lim et al., 2012; Naghavi et al., 2015; Rehm et al., 2009, 2010; Vos et al., 2015; Whiteford et al., 2013).

The adverse health effects of cannabis: the standard account

The known adverse effects of using cannabis look very modest by comparison with the manifold and protean adverse health effects of alcohol (Hall, 2015; Hall & Degenhardt, 2009; Hall, Renström, & Poznyak, 2016). As advocates of more liberal cannabis policies stress, cannabis is not known to cause fatal overdoses (Gable, 2004) because it does not have respiratory depressant effects like the opioids or alcohol (Boffey, 2014). Cannabis intoxication only modestly increases road accident risk (roughly two-fold) (Asbridge, Hayden, & Cartwright, 2012) by comparison with alcohol (6-10 fold) (Hall, 2015). There is weak evidence that cannabis use increases depression or suicide risk (Hall et al., 2016). Acutely some cannabis users have very unpleasant experiences, such as, anxiety, paranoia and hallucinations, but, it is usually argued, these symptoms resolve as the effects of intoxication dissipate. It is difficult to establish causality between cannabis use and mental illnesses because of other confounding factors, e.g. alcohol use, that also increase the risks of mental disorders (Hall,

Cannabis dependence can develop in those who engage in sustained daily or near daily use (Anthony, 2006; Anthony, Warner, & Kessler, 1994). The existence of cannabis dependence is often discounted by advocates of more liberal cannabis policies as an artefact of prohibition because, it is argued, cannabis users only seek treatment as a way of avoiding criminal penalties and that social norms will develop among users after legalisation that will discourage this pattern of use (Pacula, Powell, Heaton, & Sevigny, 2015). If the existence of cannabis dependence is conceded, then it is argued that the risk of developing dependence on cannabis is much smaller than the comparable risks for alcohol, nicotine or heroin (Anthony et al., 1994), and that the health and social consequences of cannabis dependence are much less serious than those for alcohol, nicotine and heroin dependence.

A critical analysis of the standard account

Taking cannabis dependence seriously

Cannabis dependence is not an artefact of prohibition. This is clear from the increase in the numbers of persons seeking treatment for problem cannabis use in the Netherlands (EMCDDA, 2013) where cannabis use, possession and small scale retail sales were decriminalised over 40 years ago (Room et al., 2010). The health problems reported by cannabis dependent persons – e.g. bronchitis and impaired memory – are much less serious on average than those reported by persons who are alcohol dependent (Hall, 2015) (e.g. delirium, liver disease, gastritis) but this does not mean that cannabis dependence is a minor problem (Hall, 2015).

First, cannabis dependence is a problem in itself for those who seek help. An inability to control one's cannabis use is a problem if you do not want to spend most of your days intoxicated in ways that interfere with your capacity to perform social roles. It may also require users to spend a substantial proportion of their income on cannabis. Some users simply do not like having impaired control over their drug use.

Second, the widely cited estimates of the risk of dependence among (9% of lifetime cannabis users and 15% of adolescent users) (Anthony et al., 1994) are probably under-estimates derived from population surveys done in the early 1990s. As Caulkins (2016) has pointed out, at this time the great majority of lifetime cannabis users did not use cannabis often enough to put themselves at risk of developing dependence. In US household survey data in 1998, for example, only a third of lifetime cannabis users had used cannabis more than 100 times (a criterion often used to define regular tobacco users). The dependence risk among cannabis users who had used this often was three times higher than that in lifetime users, namely, 27% (Caulkins, 2016). The relevance of these risks to contemporary cannabis users is uncertain because of the substantial increases in the THC content of cannabis over the past two decades (McLaren, Swift, Dillon, & Allsop, 2008).

Third, the outcomes for treatment of cannabis use disorders resemble those for the psychosocial treatment of alcohol dependence, in that a small proportion of treated cases achieve enduring abstinence from any episode of treatment (Martin & Rehm, 2012). In longitudinal studies cannabis dependence has a high rate of remission in the absence of treatment (Sarvet & Hasin, 2016; Heyman, 2013). However, among persons with cannabis dependence who seek treatment, cognitive behavioral treatment produces low abstinence rates six and 12 months after treatment (Gates, Sabioni, Copeland, Le Foll, & Gowing, 2016).

Correlates of cannabis dependence

Cannabis dependence in young adults is correlated with a variety of poor psychosocial outcomes (Hall, 2015; Hall et al., 2016). These include increased risks of tobacco and nicotine dependence (Ramo, Delucchi, Hall, Liu, & Prochaska, 2013; Rubinstein, Rait, & Prochaska, 2014); illicit drug use; developing schizophrenia; leaving school early, and showing poor cognitive performance in mid-adulthood (Hall, 2015). It is often argued that the relationships are not causal because these associations are better explained by a combination of factors that are correlated with regular cannabis use and these outcomes, namely, other drug use (alcohol, tobacco, and stimulants) and poor cognitive ability and greater propensity to take risks among those who are most likely to become regular cannabis users (Macleod et al., 2004). Some of these associations between heavy cannabis use and poor psychosocial outcomes in young adulthood (other illicit drug use; psychosis; poor school outcomes and cognitive impairment) may simply be correlations but it is unlikely that they all are, as the following brief summary of research suggests.

Illicit drug use

There are plausible non-causal explanations of the apparent "gateway effect" of heavy cannabis use (Hall & Lynskey, 2005; Morral, McCaffrey, & Paddock, 2002). One factor undoubtedly is the selective recruitment into regular cannabis use of young people who are at higher risk of using a variety of illicit drugs, independently of the fact that they have used cannabis (Morral et al., 2002). This includes young people who have parental history of drug use disorders, a personal history of conduct disorders in childhood and adolescence, and who have been early and regular tobacco smokers (Fergusson, Boden, & Horwood, 2006; Fergusson, Boden, & Horwood, 2008; Meier et al., 2016). Another factor is that daily cannabis users socialise with peers who are also daily cannabis users, who are more likely to approve of and use other illicit drugs. They are also more likely to be involved in illicit drug markets because they sell cannabis to peers to finance their own cannabis and other drug use (Fergusson et al., 2008). It is also possible that the heavy use of nicotine in adolescence may change brain function in ways that make users more likely to find the

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