Letters to the Editor

CANNABIS AND PSYCHOSIS: ACCUMULATING EVIDENCE

The report by Stefanis *et al*. (2004) adds to the evidence linking the heavy use of cannabis with increased risks of psychosis or psychotic symptoms. The advantages of this study include: a large and representative sample; measurement of multiple dimensions of psychotic symptoms; statistical control for reverse causality mediated via distress; and adjustment for comorbid depression. The study produces evidence of a dose/response association between cannabis use and psychotic symptoms in which: (a) rates of symptoms increased with increasing use of cannabis; (b) the early use of cannabis appeared to have a greater impact on later risks of psychotic symptoms. An interesting feature is that the study finds this association in a cultural context in which the use of cannabis by young people was low with only 6% reporting cannabis use. The authors suggest that this evidence adds credence to the hypothesis that cannabis contributes to the population expression of psychosis.

Recent reviews of the evidence

There have been two recent reviews that have examined the linkages between cannabis use and the development of psychotic symptoms (Arseneault *et al.* 2004; Smit *et al.* 2004). Both have concluded that the weight of the evidence favors the hypothesis that the use of cannabis may provoke the onset of psychosis or psychotic symptoms. However, this consensus of the psycho-social consequences of cannabis use in young people has been challenged in a recently published review (Macleod *et al*. 2004). This review concluded that 'Available evidence does not strongly support a causal relationship between cannabis use and psychosocial harm in young people . . .'. This review included some of the evidence on the cannabis/psychosis link. What distinguishes the conclusions of the two early reviews on cannabis and psychosis from the more recent review is not differences in reviewing methodology or the evidence but rather differences in the *opinions* of authors about the role of uncontrolled confounding. The reviews by Arseneault *et al.* (2004) and Smit *et al.* (2004) take the evidence at face value and conclude that since an association between cannabis use and psychosis has been found to persist after control for confounding, the weight of the evidence favors the hypothesis. Macleod *et al*. (2004), however, focus more critically on the issue of uncontrolled or residual confounding and suggest that because of the possibility of such confounding, strong evidence for a causal link does not exist. These different ways of describing the same evidence and issues are likely to lead to further confusion in an area already subject to controversy. For example, following the publication of the Macleod article, my local paper published a letter claiming that the review had 'debunked the myth that cannabis causes psychosis'. In fact, it had done nothing of the sort; it had merely drawn attention to the limitations in the existing evidence and phrased conclusions in such a way that a careless reader would be prone to conclude that there was no credible evidence linking cannabis and psychosis.

This debate is, of course, of direct relevance to the study by Stefanis *et al.* In particular, the review by Macleod *et al*. highlights the importance of paying attention to the control of third or confounding factors. Unfortunately, this is the very area in which the article by Stefanis *et al*. has limitations. Although the authors adjust their results for a number of confounding factors (other drug use, gender and school grade) it is clear that there are many other social, family and individual factors that could have confounded the association. It is, thus, entirely possible that the associations reported by Stefanis *et al*. could be ascribed to uncontrolled residual confounding rather than to a causal link between cannabis and psychosis. This difficulty is, in theory at least, redeemable since the longitudinal design used by Stefanis *et al*. has the potential for further adjustments the association between cannabis use and psychosis for wider range of prospectively assessed confounders.

Towards resolving the ongoing debate

The tensions between the conclusions drawn by recent authoritative and peer reviewed assessments of the linkages between cannabis use and psychosis raise important issues about the types of evidence that is needed to reduce the uncertainty and controversy in this area. As I have argued in a previous letter (Fergusson 2004) on this

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topic, two general approaches seem promising. The first approach is through improvements in epidemiological research design and analysis to estimate the effects of non-observed confounding factors. Here several strategies are possible. In longitudinal designs it is possible to use fixed effects regression methods to control non-observed confounders (Duncan *et al.* 1998; Fergusson *et al.* 2002). Twin designs may be used to control non-observed genetic and common environmental factors through the use of the discordant twins design (Lynskey *et al*. 2003). In their review Macleod *et al*. point to the use of genetic markers and the principle of Mendelian Randomisation as a promising approach to controlling confounding factors (Davey Smith & Ebrahim 2003). Although each of these approaches has limitations, each may add a different perspective on the issue of uncontrolled confounding.

The second route to clarifying this issue is through more basic science research into the neuro-chemistry, biology and physiology of the effects of cannabis and the development of psychosis. As Stefanis *et al*. point out, there is suggestive but by no means conclusive evidence that the sensitization of the mesolimbic dopaminergic system may be one pathway by which the repeated use of cannabis may lead to the onset of psychotic symptoms. Better evidence on the underlying neuro-chemistry and biology of the effects of cannabis and the origins of psychosis is likely to play an important role in furthering our understanding of the extent to which statistical linkages between cannabis and psychosis reflect underlying causal processes.

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CANNABIS AND PSYCHOSIS: EXTENDING THE DEBATE

Four recent prospective studies in three countries (Israel, New Zealand, Sweden) have found relationships between the frequency with which cannabis had been used and the risk of receiving a diagnosis of schizophrenia or of reporting psychotic symptoms (Zammit *et al.* 2002; Arseneault *et al.* 2002; Van Os *et al.* 2002; Fergusson *et al.* 2003). The studies show that: cannabis use precedes psychosis; and that the risk of psychosis is higher for those who begin use at an earlier age, are regular cannabis users, and have a history of psychotic symptoms (Hall *et al.* in press). The importance of these studies is that they have substantially reduced our uncertainty about the relationship between cannabis use and psychosis. Indeed, the fact that the relationships persist after controlling for confounding factors, makes it more likely than not that cannabis use plays a causal role in the onset of psychosis (Hall & Pacula, 2003).

The paper by Stefanis *et al*. (2004) adds to the debate. They investigate the effect of exposure to cannabis early in adolescence on subclinical positive and negative symptoms of psychosis. Their findings from a large Greek cohort indicate that cannabis may drive the population risk of psychosis at the level of subtle alterations in mental states that form the dimensions of positive and negative psychotic experiences. This is important as one of the factors which limits the certainty around the relationship between cannabis and psychosis is the absence of a correlation between population cannabis consumption and incidence of psychosis.

It is uncertain whether cannabis use primarily precipitates psychosis in those who are at increased risk for a variety of other reasons or whether cannabis use causes psychotic disorders in people who would not have developed a disorder in the absence of cannabis use. Recent modelling indicates that it is not easy to choose between these possibilities using epidemiological data (Degenhardt *et al*. 2003). The absence of any change in the incidence of schizophrenia in countries where cannabis use has increased (e.g. Australia) makes it unlikely that cannabis use can produce psychoses that would not have occurred in its absence. It seems more likely that cannabis use can precipitate schizophrenia in vulnerable individuals.

Stefanis *et al*. argue that the effects of cannabis are detectable beyond conventional criteria and may act at the level of population risk of psychosis. While I would argue that the relationship should still be evident in the epidemiological based data, their interesting findings alert us to the need for further well controlled research in this area.

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EXPLORING THE RELATIONSHIP BETWEEN CANNABIS USE AND PSYCHOSIS

Evidence for an association between cannabis use and psychosis continues to accumulate. A number of population-based longitudinal studies have found that cannabis use is associated with the later development of psychotic symptoms or psychotic diagnoses [1–6]. Studies of subjects with schizophrenia show associations between cannabis use and increased levels of positive symptoms [7,8] as well as increased number of relapses [9,10]. Additional lines of evidence support a causal role for cannabis in the development of psychosis. In experimental studies administration of cannabinoids can induce transient psychotic experiences, whilst molecular studies suggest that cannabis can increase meso-limbic dopaminergic transmission as well as inhibit glutamatergic release, in keeping with current theories of schizophrenia aetiology [11–13].

The problem, of course, is that it is very difficult to establish causality from observational studies [14]. The most likely alternative explanation for the association between cannabis use and psychosis is that personality traits or other confounders predispose individuals both to using cannabis and to developing psychotic symptoms. An association reported between tobacco use and *reduced* risk of schizophrenia argues against confounding by factor such as personality traits influencing non-specific substance use, but cannot rule it out [15].

There is increasing interest in the possibility that psychotic phenomena exist as a continuum that extends into the general population [16,17]. Stefanis *et al*. [18] examine the relationship between cannabis use and psychotic symptom dimensions in a cross-sectional survey of a population-based birth cohort. They report associations between cannabis use and the presence of subclinical levels of both positive and negative dimensions of psychosis. The association with negative symptoms persists after adjustment for depressive symptoms, suggesting that this association is not simply due to the rating scales inadvertently measuring depressive symptoms. A similar finding was reported in another non-clinical sample that used the same rating scales [19].

In contrast, people with schizophrenia who use cannabis report fewer negative symptoms [8,20,21]. This has been one argument in support of the self-medication hypothesis of cannabis use [20], though in fact there is little evidence that cannabis use alleviates symptoms of psychosis. A possible explanation for these apparently contradictory results is confounding by premorbid levels of functioning, such that individuals with the propensity for developing a more severe illness characterised by marked negative symptomatology may

be less likely to seek out and partake in substance misuse [19]. This could lead to an apparent association between cannabis use and reduced levels of negative symptoms in clinical samples if this effect were stronger than any effect of cannabis on increasing negative symptoms.

Stefanis *et al.* also report a stronger association between cannabis use and positive symptoms in subjects first using cannabis before age 15; results similar to those reported from the New Zealand Dunedin cohort [4]. These findings are of great interest as they suggest that early adolescence, when the brain is still undergoing maturational processes such as myelination, may be a critical period in terms of vulnerability to the effects of exposure to cannabis. If this is indeed the case, this raises important public health concerns given the trends over time for more frequent use and earlier age of first use of cannabis during adolescence.

The Stefanis *et al.* study is not able to address the issue of whether cannabis increases the risk of psychosis or psychotic symptoms. However, it points to a promising method of studying the aetiology of psychotic symptoms in the community. Future longitudinal research of psychotic symptoms in the community could lead to an improved understanding of the relationship between cannabis and psychosis and whether there are critical periods of brain development when cannabis is particularly harmful.

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OPIATE AGONIST MAINTENANCE TREATMENT FOR INJECTING DRUG USER PEER EDUCATORS

HIV is spreading rapidly among injecting drug users (IDUs) in many areas of the world, particularly in parts of Eastern Europe and South-east Asia (UNAIDS 2003). There are highly effective programs to reduce HIV transmission among IDUs, including peer education, syringe exchange, and methadone maintenance treatment (National Institute of Health (NIH) 1997). Despite its effectiveness, methadone maintenance treatment is available in only a limited number of countries (see http://www.indro-online.de/nia.htm), and it has been difficult to implement even pilot methadone treatment programs in many of the developing/transitional countries where HIV is spreading among IDUs.

There have also been major problems in initiating peer education programs in most countries, because of one issue in particular. These programs utilize peers, usually former or current IDUs, to do HIV education and promote risk reduction. In our experiences in China and Vietnam, serving as a peer educator creates an enhanced sense of self-worth that motivates the educators to stop using drugs themselves. The challenge is that their positions require them to work in an environment in which there are many stimuli likely to evoke drug cravings. Work in such an environment, combined with their own histories of drug addiction, leads many peer educators to relapse back to drug use despite their best efforts to stop using (Peer Educators, personal communication).

Providing agonist maintenance treatment to peer educators with histories of heroin addiction should greatly reduce their tendencies to continue heroin use. Agonist treatment would both reduce drug cravings and, if taken at a sufficient dosage, produce a cross-tolerance to heroin, such that if the person did use heroin, he or she would not feel a drug effect from the heroin.

We would like to suggest that all peer educators with a history of heroin use be offered opiate agonist (either methadone or buprenorphine) treatment as part of their employment. This would not only serve to help protect peer educators from drug use and make them more effective in their work, but it would also create a method for obtaining experience with opiate agonist treatment in countries that currently do not provide such treatment. Accepting the treatment should be entirely voluntary on the part of the peer educators. Such pilot treatment programs should be rigorously evaluated, though pre-versus post comparisons should be used, as it would be unethical to deny agonist treatment to a control group. The most appropriate comparison may be to the previous detoxification treatments that many of the peer educators have undergone. The goals of this treatment would be to greatly reduce illicit drug use and to increase psychological and social functioning. While providing agonist treatment would be an increased cost, we suspect it may be highly cost effective in terms of increased effectiveness and reduced turnover among peer educators. Like all current forms of drug abuse treatment, however, it would be unrealistic and inappropriate to expect complete cessation of illicit drug use among all persons who received treatment.

We urge funding agencies such as the Global Fund, the World Bank, USAID, and the Gates Foundation to consider requiring the availability of opiate agonist treatment (either methadone or buprenorphine) as a component of the ethical operation of peer education programs for heroin users. We recognize that the resistance many countries have to providing opiate agonist treatment can be formidable, that there are important logistical issues, and that it is critical to have appropriately trained clinical staff. However, we believe that providing this type of treatment to peer educators would be an excellent starting point for gaining positive experiences with these treatments.

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LOW INCOME AND VULNERABILITY FOR GAMBLING PROBLEMS

In their editorial that appeared in the June 2004 issue of *Addiction*, Rehm *et al.* (2004) cite poverty as an important environmental modifier of the relationship between substance use and harm (the topic of their editorial). For example they cite Harrison & Gardiner (1999) who state that men in the lower socio-economic status (SES) category in their study had a much increased risk of alcohol-related mortality compared to those in the highest SES category, despite the fact that volume of drinking was greater for those in higher SES categories.

Readers may like to know that the same effect appears to be evident in the case of gambling and gambling problems according to a secondary analysis of the data from the first British Gambling Prevalence Survey (Orford *et al.* 2003). The rate of participation in any gambling in the last 12 months and the number of different gambling activities engaged in showed little variation with household income, although both were lowest in the group on very small incomes (less than £5200 per annum). Nor did average total stake on activities such as the national and other lotteries, football pools and bingo or average sum of losses on activities such as playing fruit machines, betting on horse or dog races or playing casino games, vary very much by income (questioning people about expenditure on gambling is fraught with difficulties, and preliminary work suggested that people thought in terms of stakes for certain activities and in terms of losses for others). When stakes and losses were calculated as percentages of income the picture was much clearer, with the highest averages in the lowest of three income categories (less than £15 600 a year in 1999–2000) and lowest in the highest income group (£31 200 or more). That may go a long way towards explaining one of the main findings of the survey, that household income was one of a small number of variables significantly associated with problem gambling in a logistic regression analysis. Controlling for other socio-demographic variables, those in the lowest of the three income categories were nearly three times as likely as the average person to score above the threshold on a problem gambling screening measure.

Income appears to be an important modifier of the relationship between extent of engagement in gambling and gambling-related problems.

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ALCOHOL POISONINGS, DRINKING BEHAVIOR AND VIOLENCE IN RUSSIA: A REPLY TO ROSSOW

In her editorial to my study of weekend effects on binge drinking and homicide in Russia (Pridemore 2004), Rossow (2004) cites two studies (Poikolainen *et al.* 2002; Simpura & Paakkanen 1997) that she argues contradict my main methodological assumption and she states correctly that violation of this assumption would call into question the finding that alcohol poisoning deaths peak on weekends in Russia. I thank Dr Rossow for providing a critical and thought-provoking introduction, and while I suspect that she and I agree on many of the issues involved, it is necessary to respond to her specific points.

Measurement issue 1: The timing of the recording of alcohol poisoning deaths

The main assumption in my study was that alcohol deaths recorded on a particular day partially represent the spillover effects of drinking the previous evening. For example, a death due to alcohol poisoning recorded on Sunday may actually result from drinking on Saturday night. This might occur because respiratory depression can continue for several hours before breathing stops completely, or simply because the death may not be discovered until the next morning.

Rossow challenges this assumption by referencing two studies. The first is Poikolainen *et al's* (2002) analysis of alcohol poisoning deaths on holidays in Finland. Rossow states that their results show that alcohol poisonings peak on specific holidays but not the day after, leading the reader to believe that the day after registers a normal number of deaths. Rossow also points out that if the spillover assumption is incorrect the number of alcohol poisoning deaths on Fridays and Saturdays in my study is less than 4% higher than expected. She argues that this finding of no weekend effect on *binge drinking* agrees with a Simpura & Paakkanen (1997) survey of Muscovites that found the distribution of *drinking occasions* by day to be remarkably even.

First, while the Poikolainen *et al*. study should be commended, the authors do not address recording issues of alcohol poisonings in Finland or Russia. Second, an examination of their data (see Figure 1a and b in their study) reveals Rossow's statement to be inaccurate. The Poikolainen *et al*. data show that 'the number of alcohol poisonings on the day following holidays is significantly higher than expected'. To be certain I contacted Dr Poikolainen, who stated that alcohol poisoning deaths *commonly take place the day after drinking bouts* and that 'the peaks have slopes' (Personal E-mail: communication with Dr Poikolainen, July and August 2004). By the latter statement Dr Poikolanen meant that while the peak occurs the day of the holiday, the days adjacent to the holidays also experience significantly more alcohol poisoning deaths than expected, which is why the authors used the term 'celebration periods' in their article.

Third, in studies of Lithuania and Moscow, Chenet *et al*. (2001, 1998) found similar patterns of alcohol poisoning deaths as in my data. The Moscow study employed 1993–95 mortality data, which are obviously a better indicator of alcohol poisoning deaths in 1994 Moscow than the number of 'drinking occasions' referred to by Rossow. It should be stressed that Simpura and Paakkanen are themselves very cautious throughout their paper and that they explicitly recognize the difference between routine 'drinking occasions' and bouts of heavy alcohol consumption. For example, Rossow tries to make her point by quoting Simpura and Paakkanen (p. 114) when they state that '[t]he weekend effect [on drinking occasions] is hardly visible at all.' But in the very next sentence the authors say that 'Calculating the distribution of alcohol intake instead of the distribution of occasions gives a stronger weekend peak.'

Measurement issue 2: Limitations of the Simpura and Paakkanen survey

Since Rossow refers to the results of the Simpura and Paakkanen survey to question my assumption, its limitations must be mentioned. This is not an indictment of Simpura and Paakkanen's work. Not only are they pioneers in carrying out alcohol research in Russia given the lack of previous studies and reliable data and the inability to survey the Russian population until the collapse of the Soviet Union, but they are extremely careful to point out the limitations of their survey. For example, they make it clear that 'an unrecoverable technical error' (p. 110) negatively affected their ability to carry out certain aspects of their analysis related to drinking occasions and consumption, and that 'radical operations were needed to transform the data to represent "typical Russian drinking"' (p. 120). They are also candid in their assessment of the issues of reliability of sample and validity of measures that I will briefly mention here.

First, while a worthwhile case study, Moscow is not representative of Russia. It is a sprawling metropolis and capital city. Its economic, demographic, and health characteristics (including the rate of deaths due to alcohol poisoning and to homicide) are considerably different from Russia as a whole. Second, the problems associated with surveys of drinking (both in terms of occasions and amount) are well-known. As pointed out by Simpura and Paakkanen, the most marginalized problem drinkers (i.e. those with whom we are most concerned when discussing binge drinking and alcohol poisoning deaths) are likely to be disproportionately absent from such surveys. These problems are especially likely to show up in surveys of the Russian population, where (1) per capita alcohol consumption is the highest in the world, (2) nearly one-third of adult Russian males admit to binge drinking at least once per month (Bobak *et al*. 1999) and, (3) the population is unused to being surveyed. Indeed, the severe limitations in measuring consumption via surveys in Russia even when employing a more rigorous, better-funded, and ongoing survey (i.e. the Russian Longitudinal Monitoring Survey) than that which Simpura and Paakkanen were able to undertake given their limited resources, have been criticized in this journal (Nemtsov 2003). These difficulties were carefully studied by Laatikainen *et al*. (2002), whose comparison of self-reports and biological markers of

consumption led them to conclude 'that alcohol consumption especially in Russia may not be reliably estimated by self-reporting' (p. 282).

CONCLUSION

There are meaningful limitations to my study that I discussed in the original article. Further, even though I carry out research on alcohol and violence, I am sceptical of a direct causal relationship and believe that careful research is necessary to understand the mechanisms through which alcohol may serve to increase the risk of violent offending and victimization. Most importantly, I am not arguing here that my assumption that many alcohol poisoning deaths are actually recorded a day later than the drinking behavior that led to them is correct. Rossow's contention of the merit of this assumption may be accurate and I thank her for bringing a critical eye and more attention to this issue. Instead I wish simply to make it clear that a genuine assessment of the two studies Dr Rossow referenced do not marshal the evidence against this assumption that she claims. The point under debate is a measurement and recording issue and my hypothesis of spillover effects is easily testable. Thus I encourage further research on the matter since the validity, or at least the accuracy of the estimates, of several of the studies mentioned here rest on the outcome.

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