



Figure 1 Mean scores on the Alcohol Use Disorders Identification Test: University of Otago students (2002) vs. general population peers (New Zealand Health Survey 2002/3)

tional policies that do not adequately discourage drunkenness, and inadequate enforcement of the intoxication provisions of liquor legislation.

International evidence supports a reduction in liquor outlet densities around campuses, increased prices via taxation, better enforcement of liquor laws, restrictions on advertising and promotion, a minimum purchase age of 20 or 21, stricter controls over the service of alcohol at student events, and screening and brief intervention in student health services [14,15].

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THE CANNABIS AND PSYCHOSIS CONNECTION QUESTIONED: A COMMENT ON FERGUSSON ET AL. 2005

Fergusson and colleagues assert that their results from a New Zealand cohort ‘add to the growing body of evidence that regular cannabis use may increase risks of psychosis’ [1]. In fact, their study raises more questions than it answers.

One obvious question is raised by their use of ten items from Symptom Checklist 90 as the only assessment tool for symptoms of psychosis. The items assessed focus heavily on paranoid ideation, e.g. ‘feeling other people cannot be trusted’, ‘feeling you are being watched or talked about by others’, and ‘having ideas or beliefs that other do not share.’ This is of concern because it is well known—widely reported in the literature [2] and commonly referenced in popular culture for decades [3,4]—that paranoid feelings are a relatively frequent effect of acute marijuana intoxication.

Fergusson *et al.* give no indication that respondents were asked to distinguish between feelings experienced while intoxicated and feelings experienced at other times. Thus, we are left with no clue as to whether these are

long-term effects actually indicative of mental illness or simply the normal, passing effects of acute intoxication. This is akin to reporting that people who go to bars are more erratic drivers than people who don't, without bothering to look at whether they'd been drinking at the time their driving skills were assessed. In addition, Fergusson *et al.* seem not to have considered that what might be an indication of psychosis in other circumstances could be an entirely normal reaction for people who use marijuana. Someone using a substance that is both illegal and socially frowned-upon almost by definition has 'ideas or beliefs that others do not share.' This is not a sign of mental illness, but rather an indication of a rational, thinking person realistically assessing his or her situation. Considering the widespread use of undercover officers in drug stings, the same can be said for 'feeling other people cannot be trusted'.

Fergusson does not report which symptoms appeared most often, or whether the differences in average levels of symptoms between users and non-users came from a few people having many symptoms or many people having a few symptoms. This raises yet more questions, as the daily user group, with the highest levels of supposed psychosis, reported an average of less than two symptoms each. Based on the data reported, it is entirely possible that the case for marijuana 'causing' mental illness is based solely on marijuana smokers having the completely reasonable feelings that they have beliefs different from mainstream society and thus should be a tad suspicious of others.

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MIRKEN REFUTED: REASONS FOR BELIEVING THAT THE ASSOCIATION BETWEEN CANNABIS USE AND RISK OF PSYCHOSIS IS PROBABLY CAUSAL

In his comment on our research, Mirken [1] suggests that our conclusions that 'our findings add to the growing body of evidence that regular cannabis use may increase risks of psychosis' can be explained by the fact that 'the case for marijuana causing mental illness is based solely on marijuana smokers having the completely reasonable feelings that they have different beliefs from mainstream society . . .'. These arguments succeed only by virtue of Mirken ignoring most of the body of evidence to which we refer. In particular, it has been well established by longitudinal studies that the heavy use of cannabis is associated with increased rates of both psychosis [2,3] and psychotic symptoms [4–8]. This consistent finding using different approaches to assessing psychosis and psychotic symptoms makes it very difficult to claim that the link between cannabis and psychosis/psychotic symptoms simply reflects the fact that cannabis users have different beliefs from the rest of society.

In his specific critique of our research Mirken raises two general issues. First, it is proposed that any increase in paranoid symptoms amongst cannabis users may reflect the effects of legislation which justifies cannabis users being 'a tad suspicious of others' or 'feeling other people cannot be trusted'. However this argument is not consistent with the evidence on this topic, since linkages between cannabis and psychosis/psychotic symptoms have been found in societies which have both liberal and conservative policies towards cannabis. Thus, findings from New Zealand [4–6] where cannabis use is illegal have been similar to those from Holland [8], which has more liberal legislation. These comparisons suggest that it is implausible to propose that increased rates of psychotic symptoms amongst cannabis users reflect the impacts of cannabis legislation on the belief system of users.

Second, it is suggested that the associations between cannabis and psychosis/psychotic symptoms may simply reflect reports of the acute effects of cannabis use. This argument has merit given the evidence that acute cannabis intoxication may mimic the symptoms of psychosis [9]. Mirken claims that it was necessary for our research to distinguish these short-term effects from longer-term effects by ascertaining whether the symptoms occurred in the context of cannabis use. A little reflection on our

Table 1 Mean psychotic symptoms at 18, 21 and 25 years (number of subjects) by frequency of cannabis use at age 17–18 years

Age (Years)	Frequency of Cannabis Use (17–18 years)					P
	Never	Less than monthly	At least monthly	At least weekly	Daily	
18	0.64 (598)	0.95 (242)	1.07 (82)	1.93 (70)	1.64 (33)	<0.0001
21	0.74 (577)	0.96 (232)	1.31 (80)	1.67 (61)	1.06 (33)	<0.0001
25	0.70 (574)	0.80 (229)	1.08 (77)	1.13 (60)	1.03 (32)	<0.01

research analysis reveals the difficulties of this proposal. In particular, the focus of the analysis was on weekly and daily users of cannabis. Given this high frequency of use it would have been difficult, if not impossible, for the respondents to identify the times of the day in which they were experiencing acute symptoms and the times of the day they experienced longer-term symptoms. However, the power of a longitudinal design is that it has the capacity to examine both short- and long-term associations. This capacity is used in Table 1 below which examines the linkages between cannabis use at ages 17–18 and levels of psychotic symptoms at ages 18, 21 and 25. This table shows that linkages between cannabis use and psychotic symptoms persist for up to seven years. In passing, we note that a recently published paper by Henquet *et al.* [7] reports associations over a four year period. This association between cannabis use and long-term psychotic symptoms clearly argues against the view that the association is due to cannabis users reporting acute symptoms of cannabis intoxication.

In summary, the arguments proposed by Mirken fail on three grounds. First, these arguments disregard the growing body of evidence showing linkages between cannabis use and psychosis/psychotic symptoms assessed in a variety of ways and in a range of social contexts. Second, cross-cultural comparisons suggest that it is highly unlikely that these associations reflect the responses of cannabis users to legislation, since the associations exist in societies with both liberal and conservative attitudes. Finally, evidence from recent studies suggests linkages between cannabis use and psychosis/psychotic symptoms persist over a lengthy period of time implying that it is highly unlikely that these associations reflect reports of the acute effects of cannabis intoxication.

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ESTABLISHMENT OF A MOO JOOSE ARCHIVE

In response to Virginia Berridge's comment (Berridge 2004) that all the documents pertaining to the *Moo Joose* alcoholic milk case (Munro 2004) should be avail-