Letters to the Editor

DO UNIVERSITY STUDENTS DRINK MORE HAZARDOUSLY THAN THEIR NON-STUDENT PEERS?

University students suffer from and inflict high levels of alcohol-related harm [1,2]. Folklore depicts drunkenness as integral to the student experience, and the 'drunken student rampage' is staple fare for the news media. Students are perceived to be among the heaviest young drinkers. However, to date, there are no empirical data from outside North America to support that perception. We sought to compare university student drinking with that of the same age group in the general population in New Zealand.

We compared scores on the Alcohol Use Disorders Identification Test (AUDIT) [3] attained by 17–24 year-olds in two surveys: an internet-based survey of University of Otago students in 2002 (n = 1424, 82% response) [4], and the 2002/3 New Zealand Health Survey (NZHS; n = 1406, 72% response), based on a stratified random sample of households and conducted by face-to-face interview [5]. The University of Otago is the third largest (approximately 17,000 students) of eight universities in New Zealand. National aggregate consumption is approximately 9 litres of pure alcohol per person aged 15 years and older, a level similar to that in other English-speaking countries, including the USA [6].

The differences, particularly among 18–23 year-olds, are startling: students' scores were, on average, 50–60% higher than those of their peers (Fig. 1). Furthermore, the scale is not linear, i.e. the risk indicated by a score of 12 is more than 50% higher than that for a score of 8. The prevalence of hazardous drinking (AUDIT score ≥ 8), was almost twice as high among students (65% vs. 36%; 95% CI for the difference: 28%, 35%), while harmful drinking (AUDIT score ≥ 15) was three times as prevalent (31% vs. 9%; 95% CI for the difference: 18%, 24%).

Limitations of the comparison include the fact that some university students would be included in the NZHS figures. Around 15% of 17–24 years-olds in New Zealand were full-time university students in 2002 [7,8]. However, it should be noted that the NZHS sampling frame excluded university halls of residence [5], which house a fair proportion of students in the age group of interest. This feature of the comparison would tend to underestimate differences in drinking between students and non-students, although probably to a small extent. A second limitation was the difference in survey modality: computerised for the students and face-to-face for the NZHS. This might overstate differences in the estimates, on account of the higher reporting of health risk behaviours in computerised methods vs. traditional survey technologies [9]. The net effect of these two factors would be difficult to estimate but is probably small.

Strengths of the comparison include the reliance on a common measure, the similar timing of the surveys, and high response rates. It may be tempting to dismiss this as a problem unique to the University of Otago, an old institution in a part of the country with a heavy drinking tradition. However, at the University of Waikato, a newer institution in a demographically distinct region of the country, researchers arrived at similar estimates of AUDIT scores as recently as 2000 [10].

A recent US study comparing 18–29 year-olds in the general population with college students, showed that monthly heavy drinking (5 + drinks per occasion for men, 4 + for women) was significantly more common in college students than in the general population sample (24% vs. 20%). A diagnosis of alcohol dependence was also significantly more common among college students than among their non-student peers (15% vs. 12%) [11].

Is student hazardous drinking a cause for concern? It is, after all, a centuries-old tradition [12]. Anecdote suggests that students moderate their drinking after leaving university, but evidence shows that a sizeable portion does not [13]. The significantly increased likelihood of acute harms caused by a few years’ regular exposure to the intoxicating effects of alcohol should not be dismissed lightly. It behoves health authorities and the education sector to consider the implications of exposing a large proportion of our young to environments that facilitate or encourage hazardous drinking.

These 'alcogenic' environments consist of high concentrations of licensed premises, events that have a primary focus on drinking, intense advertising, promotion, and aggressive pricing by the liquor industry, institu-
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].

KYP KYPRI
School of Medical Practice and Population Health University of Newcastle, Australia
and
Injury Prevention Research Unit
Department of Preventive and Social Medicine
University of Otago, New Zealand
E-mail: kypri@tpg.com.au

MATTHEW CRONIN & CRAIG S. WRIGHT
Public Health Intelligence
Ministry of Health
New Zealand

References

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 different beliefs from mainstream society and thus should be a tad suspicious of others.

BRUCE MIRKEN
Director of Communications
Marijuana Policy Project
Box 77492
Capitol Hill
Washington, DC 20013
USA
E-mail: Bruce@mpp.org

MITCH EARLEYWINE
Associate Professor of Psychology
Department of Psychology
University of Southern California
Los Angeles
CA 90089-1061
USA
E-mail: earleyw@usc.edu

References

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
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.

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

<table>
<thead>
<tr>
<th>Age (Years)</th>
<th>Never</th>
<th>Less than monthly</th>
<th>At least monthly</th>
<th>At least weekly</th>
<th>Daily</th>
<th>P</th>
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<tr>
<td>18</td>
<td>0.64</td>
<td>0.95</td>
<td>1.07</td>
<td>1.93</td>
<td>1.64</td>
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<td></td>
<td>(598)</td>
<td>(242)</td>
<td>(82)</td>
<td>(70)</td>
<td>(33)</td>
<td></td>
</tr>
<tr>
<td>21</td>
<td>0.74</td>
<td>0.96</td>
<td>1.31</td>
<td>1.67</td>
<td>1.06</td>
<td>&lt;0.0001</td>
</tr>
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<td></td>
<td>(577)</td>
<td>(232)</td>
<td>(80)</td>
<td>(61)</td>
<td>(33)</td>
<td></td>
</tr>
<tr>
<td>25</td>
<td>0.70</td>
<td>0.80</td>
<td>1.08</td>
<td>1.13</td>
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<td>(574)</td>
<td>(229)</td>
<td>(77)</td>
<td>(60)</td>
<td>(32)</td>
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</tbody>
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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-
able to inquirers independent of any actor in the case, I am pleased to report that the library of the Australian Drug Foundation has established a *Moo Joose* archive. The archive contains all the published and unpublished materials collected by ADF staff that bear upon the case and were available to the author. The collection is open to any person who has an interest in the *Moo Joose* case or in a related subject. Inquiries about materials held in the archive should be directed to: The Librarian, Australian Drug Foundation, PO Box 818, North Melbourne, Victoria, Australia 3051 or by e-mailing library@adf.org.au

I am grateful to Professor Berridge for her advice in this matter.

GEOFFREY MUNRO
Director
Community Alcohol Action Network

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**References**
