Cannabis and mental health

More evidence establishes clear link between use of cannabis and psychiatric illness

Papers pp 1195, 1199, 1212

In the 1990s the use of cannabis increased much among young people so that it is now becoming more common than tobacco smoking in some countries.¹⁻² The ready availability of the drug, the increasing social disapproval of cigarette smoking, stern drink driving laws, and perceptions that cannabis is safe or less harmful than cigarettes or alcohol may explain these changes. The increase in use is of concern because cannabis may be a gateway to other drugs,³ and it may cause psychiatric illnesses. The link between cannabis and psychosis is well established, and recent studies have found a link between use of marijuana and depression.⁴⁻⁷ Does cannabis cause these conditions, or do patients use cannabis to relieve their distress?

The explanation most accepted is that cannabis triggers the onset or relapse of schizophrenia in predisposed people and also exacerbates the symptoms generally.^{4 5} Establishing direction of causality is difficult and is most appropriately assessed in non-clinical samples, but a low incidence of the illness and the fact that most drug users take other drugs in addition to cannabis create methodological problems and explain the dearth of reliable evidence.

The study often quoted in support of the causal hypothesis examined the incidence of schizophrenia in more than 50 000 Swedish conscripts followed up for 15 years.⁸ It showed that use of marijuana during adolescence increased the risk of schizophrenia in a doseresponse relation. Questions have, however, remained about the validity of the diagnosis, the possible causal role of other drugs, and prodromal symptoms of schizophrenia that might have led to the use of cannabis, rather than cannabis triggering the psychosis.^{4 5}

A longer follow up and reanalysis of this cohort published in this issue (p 1199) confirms the earlier findings and clarifies that cannabis, and not other drugs, is associated with later schizophrenia and that this is not explained by prodromal symptoms.⁹ In a similar vein, a three year follow up of a Dutch cohort of 4045 people free of psychosis and 59 with a baseline diagnosis of psychotic disorder showed a strong association between use of cannabis and psychosis.¹⁰ Length of exposure to use of cannabis predicted the severity of the psychosis, which likewise was not explained by use of other drugs. Participants who showed psychotic symptoms at baseline and used cannabis had a worse outcome, which also implies an additive effect. In a New Zealand cohort, individuals who had used cannabis three times or more by age 15 or 18 were not more likely to have schizophreniform disorder at age 26 (p 1212), although they showed an increase in "schizophrenia symptoms" (but not schizophrenia).¹¹ The meaning of "schizophrenia symptoms" requires clarification to interpret these results.

The evidence in relation to depression is growing. A 15 year follow up of an adult community sample of 1920 participants in the United States showed that use of cannabis increased the risk of major depression at follow up fourfold.7 Use of cannabis was specifically associated with an increase in suicidal ideation and anhedonia. Similar findings in an Australian study reported in this issue (p 1195) show a dose-effect relation between the use of cannabis and anxiety or depression in a large cohort of 14-15 year olds followed for seven years.¹² This is reflected in higher rates of anxiety or depression according to the frequency with which cannabis was used. The link is stronger for young women than young men in this cohort, although sex differences have not been found in other studies.⁶⁷ Baseline depression did not predict later marijuana use in either study and therefore does not support the self medication hypothesis. The study in the New Zealand cohort did not find an association between cannabis use at age 15 and depressive disorder at age 26. The authors found, however, that young people who had used cannabis three times or more by age 18 were more likely to have a depressive disorder at age 26, even after use of other drugs was controlled for.

Although the number of studies is small, these findings strengthen the argument that use of cannabis increases the risk of schizophrenia and depression, and they provide little support for the belief that the association between marijuana use and mental health problems is largely due to self medication. Whether the use of cannabis triggers the onset of schizophrenia or depression in otherwise vulnerable people or whether it actually causes these conditions in non-predisposed people is not yet resolved. Further, it cannot be assumed that mechanisms are the same for both conditions (cannabinoids have effects on a variety of neurotransmitter systems) or at different developmental stages. For example, although evidence shows that mental disorder leads to the use of cannabis among adolescents, the reverse seems true in early adulthood.¹⁵

The shown dose-response relation for both schizophrenia and depression highlights the importance of reducing the use of cannabis in people who use it. It was estimated that lack of exposure to cannabis would have reduced the incidence of psychosis requiring treatment by as much as 50% in the Dutch cohort,¹⁰ and is similarly reflected in the Swedish cohort, showing that the use of cannabis increased the risk of schizophrenia by 30%.⁹ This large effect is surprising and not yet reflected in an increased incidence of schizophrenia in the population. If true, the use of cannabis will contribute to more episodes or new cases of the illness—food for thought for both clinicians and legislators.

Joseph M Rey professor of child and adolescent psychiatry

University of Sydney, Coral Tree Family Service, PO Box 142, North Ryde, NSW 1670, Australia (jrey@doh.health.nsw.gov.au)

Christopher C Tennant professor of psychiatry

University of Sydney, Royal North Shore Hospital, St Leonard's, NSW 2065, Australia (ctennant@doh.health.nsw.gov.au)

Competing interests: None declared.

 Gledhill-Hoyt J, Lee H, Strote J, Wechsler H. Increased use of marijuana and other illicit drugs at US colleges in the 1990s: results of three national surveys. *Addiction* 2000;95:1655-67.

- 2 Miller P, Plant M. Heavy cannabis use among UK teenagers: an exploration. Drug Alcohol Depend 2002;65:235-42.
- tion. Drug Alcohol Depend 2002;65:235-42.
 Fergusson DM, Horwood LJ. Does cannabis use encourage other forms of illicit drug use? Addiction 2000;95:505-20.
- 4 McKay DR, Tennant CC. Is the grass greener? The link between cannabis and psychosis. *Med J Aust* 2000;172:284-6.
- 5 Hall W, Degenhardt L. Cannabis and psychosis. Aust N Z J Psychiatry 2000;34:26-34.
- Rev JM, Sawyer MG, Raphael B, Patton GC, Lynskey MT. The mental health of teenagers who use marijuana. *Br J Psychiatry* 2002;180: 222-6.
 Bovasso GB. Cannabis abuse as a risk factor for depressive symptoms. *Am*
- 7 Bovasso GB. Cannabis abuse as a risk factor for depressive symptoms. Am J Psychiatry 2001;158:2033-7.
 8 Andreason S Allebeck P Engstrom A Rydberg U Cannabis and schizo.
- 8 Andreasson S, Allebeck P, Engstrom A, Rydberg U. Cannabis and schizophrenia. A longitudinal study of Swedish conscripts. *Lancet* 1987;2:1483-6.
- Zammit S, Allebeck P, Andreasson S, Lundberg I, Lewis G. Self reported cannabis use as a risk factor for schizophrenia in Swedish conscripts of 1969: historical cohort study. *BMJ* 2002;325:1199-201.
- 0 Van Os J, Bak M, Hanssen, M, Bijl RV, de Graaf R, Verdoux H. Cannabis use and psychosis: A longitudinal population-based study. *Am J Epidemiol* 2002;156:319-27.
- 11 Arseneault L, Cannon M, Poulton R, Murray R, Caspi A, Moffit TE. Cannabis use in adolescence and risk for adult psychosis: longitudinal prospective study. BMJ 2002;325:1212-3.
- prospective study. *BMJ* 2002;325:1212-3.
 12 Patton GC, Coffey C, Carlin JB, Degenhardt L, Lynskey M, Hall W. Cannabis use and mental health in young people: cohort study. *BMJ* 2002;325:1195-8.
- 13 McGee R, Williams S, Poulton R, Moffitt T. A longitudinal study of cannabis use and mental health from adolescence to early adulthood. *Addiction* 2000;95:491-503.

Residents' hours of work

We need to assess the impact of the new US reforms

To many, "resident physician" conjures up an image of long hours of work, fuelled by caffeine and adrenaline. This overlooks the reality that residency is an educational experience that completes a physician's preparation for independent practice. About 100 000 resident doctors in the United States—as providers of care and as learners—will be affected by reforms regarding their hours of duty, which were recently announced by the US Accreditation Council for Graduate Medical Education.¹ Under the new rules, set to take effect in July 2003, residents will work no more than 80 hours per week, have shifts that are no longer than 24 hours, and have 10 hours of rest between shifts.

The literature on sleep deprivation supports these reforms. Many articles show that sleep deprivation in laboratory and field studies has shown a negative effect on the performance of residents.²⁻⁴ Reduced performance due to sleep deprivation may be associated with increased errors and contribute to adverse events when fatigued members of staff participate in the care of patients.⁵

Accreditation is a voluntary approach of professional self regulation. The consequences of failed self regulation are often regulatory interventions, which are costly. The United States spends about \$200bn (£128bn; €203bn) annually towards regulations related to health, safety, and the environment. Whether these achieve the desired outcome is quantified for only a third.⁶ The consequence of not assessing outcomes is that we do not know whether regulations have the intended effect. New York State, through its Department of Health, began to limit duty hours for resident doctors in 1989. Fifteen years later, the impact of this

reduction on the safety of patients, education, and the professional lives of residents is still the subject of opinion and guesswork. Articles on New York's experience show the conflicting nature of the reports. Some noted that the limits had no effect on the care of patient and improved welfare of residents.⁷ Others found that they reduced the quality of care in teaching institutions.⁸ Reports that doctors trained under the limits were less familiar with their obligations to patients were countered by findings that residents did not want to leave patients until the process of care was completed.⁹

Absence of comprehensive objective data from the initiatives in New York State has reduced the value of this potential learning laboratory in guiding efforts to champion duty hours of residents. The academic community must not ignore the opportunity to benefit from the natural experiment that will result from the implementation of the new standards for hours of duty.

Beyond showing whether the standards will achieve their intended public goals, the results will help in developing new models for providing care with fewer hours for residents. Many institutions will use night float systems—a separate resident will be assigned to cover all or a part of the on-call hours of rotation and introduce other changes in how work is scheduled—or coverage by non-resident providers to comply with the standards, and some of these interventions, such as increased transfers of care and a growing workload for residents on call, in themselves have the potential to increase errors. Collecting data on costs, outcomes for patients, residents' education, and satisfaction for both groups will not be easy, and exploring the link between errors in health care and

BMJ 2002;325:1184-5