11800 SENATE HEALTH,EDUCATION & SOCIAL SERVICES

1963 and 1968 with psychotic symptoms following the use of cannabis. The most common symptoms 'were sudden onset of confusion, generally associated with delusions, hallucinations (usually visual) and emotional lability ... amnesia, disorientation, depersonalisation and paranoid symptoms' (p. 24). Most psychoses were preceded by the use of large doses of cannabis. Chopra and Smith argued that heavy cannabis use was not a sign of pre-existing disorders because a third of their cases had no prior psychiatric history, the symptoms were remarkably uniform regardless of prior psychiatric history, and those who used the most potent cannabis preparations experienced psychoses after the shortest period of use.

The findings of Chopra and Smith have received some support from other smaller case series that suggest that large doses of potent cannabis products can be followed by a 'toxic' psychotic disorder with 'organic' features of amnesia and confusion. These disorders have been reported from the Caribbean (12), New Zealand (13), Scotland (11), South Africa (10), Sweden (8), the United Kingdom (14) and the United States (15).

These disorders have been attributed to cannabis use for the following reasons: the onset of the symptoms followed closely the ingestion of large quantities of cannabis; the affected individuals often exhibited 'organic' symptoms, such as confusion, disorientation and amnesia; some had no personal or family history of psychoses before using cannabis; their symptoms rapidly remitted after abstinence from cannabis use, usually within several days to several weeks; recovery was usually complete with the person having no residual psychotic symptoms; and the disorder only recurred if the individual resumed cannabis use (16).

Sceptical author (2, 17) have criticized the poor quality of information in these studies on: cannabis use; its relationship to the onset of psychosis; the person's premorbid adjustment; and their family history of psychosis. They also emphasize the variety of clinical pictures of 'cannabis psychoses' reported by different observers. These weaknesses impair the value of these case series.

10.1.1 Controlled studies

A small number of controlled studies have been conducted over the past 20 years (18–22). Some studies have either compared persons with 'cannabis psychoses' with persons who have schizophrenia, or compared psychoses occurring in persons who do and do not have biochemical evidence of cannabis use prior to presenting for treatment. Their results have been mixed, in part because of the small sample sizes in studies that have failed to replicate positive findings, and because of variations in the research methods (16).

Several studies have examined the relationship between cannabis use and psychotic symptoms in the general population. Tien and Anthony (23) used data from the Epidemiologic Catchment Area study to examine the relationship between drug use and reports of one or more of 11 'psychotic experiences' during a twelve-month period (4 types of hallucinations and seven types of delusional belief). They compared 477 cases who reported one or more psychotic symptoms with 1818 controls who did not. Cases and controls were matched for age and social and demographic characteristics. Daily cannabis use was found to double the risk of reporting a psychotic symptom (after statistical adjustment for alcohol use and psychiatric diagnoses at baseline).

Thomas (24) reported the prevalence of psychotic symptoms among cannabis users in a random sample of people in a large city in the North Island of New Zealand. One in seven (14%) cannabis users reported 'strange, unpleasant experiences such as hearing voices' or 'becoming convinced that someone is trying to harm you or that you are being persecuted' after using cannabis.

The National Survey of Mental Health and Well-being (NSMHWB) conducted in Australia in 1997 included a screening questionnaire for the presence of psychotic symptoms (25). Among those under 55 years of age who screened positive for a psychotic disorder, 8% (n = 27) met criteria for cannabis dependence in the past 12 months. This was 17% of all persons diagnosed with cannabis dependence (26). After adjusting for demographics, affective and anxiety disorders, smoking status and alcohol dependence, a diagnosis of cannabis dependence doubled the odds of reporting psychotic symptoms (27).

10.1.2 Overall evaluation

The hypothesis that there is a 'cannabis psychosis' is still contentious. In its favour are the equivocal evidence from the case series and the small number of positive controlled studies. Critics of the hypothesis emphasize the poor quality of the clinical judgments about aetiology, the poorly specified criteria used in diagnosing these psychoses, the dearth of controlled studies, and the striking variations in the clinical features of these 'cannabis psychoses'.

It is a plausible hypothesis that high doses of cannabis can produce psychotic *symptoms* but the evidence for a 'cannabis psychosis' as a specific clinical syndrome is much less compelling because the symptoms reported by different observers have been so mixed (28). If cannabis-induced psychoses exist, they are either rare or they only rarely receive medical intervention in Western societies. The total number of cases of putative 'cannabis psychoses' in the 12 case series reviewed in Hall (16) was 397 and 200 of these came from a single series collected over 6 years from a large geographic area in which heavy cannabis use was endemic (9).

10.2 Cannabis use and schizophrenia

10.2.1 Clinical studies

In case-control studies (29, 30), schizophrenic patients are more likely to have used psychotomimetic drugs such as amphetamines, cocaine, and hallucinogens than other psychiatric patients, normal controls or the general population (31). Variations in rates of use between studies reflect differences in the sampling of patients, with younger patients reporting higher rates than older persons with chronic disorders. Studies have also differed in the criteria for diagnosing schizophrenia and the manner in which substance use has been assessed (32).

Alcohol use, abuse and dependence are probably more common in the schizophrenic population than in the general population (33, 34) but findings on cannabis use have been more mixed (16). Generally, cannabis is the most commonly used drug after

alcohol and tobacco, and it is often used with alcohol (32, 35, 36). An Australian study of a clinical sample of persons with schizophrenia (37) has broadly confirmed the pattern of substance use and abuse in American studies, finding alcohol the most commonly abused substance (18% abuse or dependence in the past 6 months), followed by cannabis (13% abuse or dependence in the past 6 months).

The controlled clinical studies disagree about the correlates of substance abuse in schizophrenia. Most have found that young males are over-represented among cannabis users (16), as they are in the general community (38). In some studies, substance users have been reported to have an earlier onset of psychotic symptoms, a better premorbid adjustment, more episodes of illness, and more hallucinations (36, 39, 40) but other well controlled studies have failed to replicate some or all of these findings (41–43).

10.2.2 Population studies

Surveys of psychiatric disorders in the community have reported higher rates of substance abuse disorders among persons with schizophrenia. In the ECA study (44) nearly half of the patients identified as schizophrenic had a diagnosis of substance abuse or dependence (34% for an alcohol disorder and 28% for another drug disorder) (45). These rates were higher than the rates in the general population, namely, 14% for alcohol disorders (46) and 6% for drug abuse (44). Cuffel et al (42) reported that the most commonly used substances among persons with schizophrenia in the ECA study were alcohol (37%) and cannabis (23%), followed by stimulants and hallucinogens (13%). The most common combination was alcohol and cannabis (31%). These findings have also been replicated in a similar survey in Edmonton, Alberta (47).

In the Australian National Survey of Mental Health and Well-Being (NSMHWB), cannabis use and a positive screen for psychosis were associated. Among those under 50 years of age who reported that they had received a diagnosis of schizophrenia, 12% met ICD-10 criteria for a cannabis use disorder in the past 12 months and 21% met criteria for an alcohol use disorder. After adjusting for other disorders and unemployment status, those who met criteria for ICD-10 cannabis dependence were 2.9 times more likely to report that they had been diagnosed with schizophrenia than those without cannabis dependence (26).

A high rate of cannabis use was also reported in the Low Prevalence Study (LPS) of psychoses in the Australian cities of Perth, Melbourne, Brisbane and Canberra (48). In this study persons with a suspected psychotic disorder were assessed by experienced clinicians using ICD-10 criteria, (48) including significant proportions who were not in domestic dwellings (which was a limitation of the NSMHWB sample) (26). One in four (24%) were daily cannabis users, 50% met lifetime criteria for alcohol abuse or dependence and 25% met lifetime criteria for cannabis abuse or dependence (48).

10.3 Explaining the association

One hypothesis is that cannabis use precipitates schizophrenic disorders in vulnerable persons. Its supporters cite the earlier age of onset of psychotic symptoms among persons with schizophrenia who use cannabis and reports that they have better premorbid adjustment, fewer negative symptoms, and a better treatment response (49).

A second possibility is that the association between cannabis use and an acute onset of schizophrenia is spurious. It may be, Arndt et al (39) argue, that schizophrenics with a better premorbid personality are more likely to be exposed to illicit drug use than persons with schizophrenia who are socially withdrawn. There is supportive evidence (50) that persons with acute onset psychoses usually have a better premorbid adjustment and a better prognosis. They also have greater opportunities to use cannabis and other illicit drugs than persons who are socially withdrawn.

A third possibility is that cannabis use is a consequence (rather than a cause) of schizophrenia. For example, cannabis and other drugs may be used to medicate the unpleasant symptoms of schizophrenia (51), such as depression, anxiety, lethargy, and anhedonia, or the unpleasant side effects of the neuroleptic drugs that are often used to treat the disorder (40).

10.3.1 Precipitation of schizophrenia

The most convincing evidence that cannabis use may precipitate schizophrenia comes from a 15-year study of cannabis use and schizophrenia in 50,465 Swedish conscripts (52). This study investigated the relationship between self-reported cannabis use at age 18 and receiving a diagnosis of schizophrenia in the next 15 years (as indicated by the Swedish psychiatric case register). Andreasson et al found that those who had tried cannabis by age 18 were 2.4 times more likely to be diagnosed with schizophrenia than those who had not. The more often cannabis had been used by age 18 the more likely they were to receive this diagnosis. The rate of a schizophrenia diagnosis was 1.3 times higher among those who had used cannabis one to ten times, 3 times higher among those who had used cannabis between one and fifty times, and 6 times higher among those who had used cannabis more than fifty times.

These risks were substantially reduced after statistically adjusting for variables that were related to the risk of developing schizophrenia, namely, having a psychiatric diagnosis at conscription, and having parents who had divorced (as an indicator of parental psychiatric disorder). Nevertheless, the relationship remained statistically significant. The risk of a diagnosis of schizophrenia was still 1.5 times greater for those who had smoked cannabis from one to ten times, and 2.3 times greater for those who had used ten or more times. Andreasson et al (52) and Allebeck (49) have argued that this indicates that cannabis use precipitates schizophrenia in vulnerable individuals.

A number of alternative explanations have been offered of the Swedish finding. First, there was a large gap between self-reported cannabis use at age 18 and the development of schizophrenia over the next 15 years (53). The diagnosis of schizophrenia was based upon a case register so there was no data on how many individuals were using cannabis at the time that their schizophrenia was diagnosed. Andreasson et al argued that cannabis use persisted because use at age 18 was strongly related to a diagnosis of drug abuse.

A second possibility is that schizophrenia was misdiagnosed. On this hypothesis, the higher rate of 'schizophrenia' among the heavy cannabis users was due to cannabis-induced psychoses that were misdiagnosed as schizophrenia (53). Andreasson et al (54) tested this possibility by examining 21 cases of schizophrenia among conscripts in the case register (8 of whom had used cannabis and 13 of whom had not). They found that

80% of these cases met the DSM-III requirement that the symptoms had been present for at least six months, thereby excluding the diagnoses of transient drug-induced psychotic symptoms.

A third hypothesis is that the relationship between cannabis use and schizophrenia is explained by the use of other drugs. Studies show (see chapter 5) that heavy cannabis users in late adolescence are more likely to use other illicit drugs, including amphetamine, which can produce an acute psychosis (55). Amphetamines were the most commonly used illicit drugs in Sweden during the late 1960s and early 1970s (56). On this hypothesis, amphetamine-induced psychoses would produce a spurious association between cannabis use and schizophrenia. The evidence that psychotic symptoms persisted beyond 6 months (54) also makes this an unlikely hypothesis.

A fourth hypothesis is that early cannabis use was a symptom of emerging schizophrenia. Andreasson et al (54) rejected this hypothesis, noting that the cannabis users who developed schizophrenia had better premorbid personalities, a more abrupt onset, and more positive symptoms than the non-users of cannabis. Moreover, there was still a dose-response relationship between cannabis use and schizophrenia among those who had no previous psychiatric history. The persuasiveness of this evidence depends upon whether a *failure* to identify a psychiatric disorder at conscription meant that no disorder was present.

A fifth hypothesis depends upon under-reporting of cannabis use at conscription. Andreasson et al (52) acknowledged that cannabis use was probably under-reported because this information was not collected anonymously. They argued, however, that under-reporting would *under*-estimate the relationship between cannabis use and schizophrenia. This is true if the schizophrenic and non-schizophrenic conscripts were equally likely to under-report. If, for example, pre-schizophrenic subjects were more candid about their drug use, then the apparent relationship between cannabis use and schizophrenia could be spurious (53). This seems unlikely, however, in view of the relationship between the *frequency* of cannabis use by age 18 and the risk of a schizophrenia diagnosis among heavy users.

10.3.2 Exacerbation of schizophrenia

Clinical reports suggest that schizophrenic patients who continue to use cannabis experience more psychotic symptoms (57), respond poorly to neuroleptic drugs (58), and have worse clinical outcomes than those patients who do not (59). These reports have been supported by controlled studies.

Negrete et al (60) conducted a retrospective study of the relationship between self-reported cannabis use and symptoms in the clinical records of 137 schizophrenic patients who had the disorder for at least six months. They found higher rates of hallucinations and delusions and more hospitalisations among patients who were cannabis users. These relationships persisted after statistical adjustment for age and sex. Similar findings have been reported by Cleghorn et al (61) who found that cannabis was the most heavily used drug, and drug abusers had higher rates of hallucinations, delusions and positive symptoms than those who did not abuse drugs. DeQuardo et al (62) reported similar findings in a retrospective study of 67 schizophrenic patients.

Jablensky et al (63) reported a two year follow-up of 1202 first episode schizophrenic patients enrolled in 10 countries as part of a WHO Collaborative study. They found that the use of 'street drugs', including cannabis and cocaine, was associated during the follow up period with more psychotic symptoms and hospitalisation. Martinez-Arevalo et al (64) reported in a study of 62 schizophrenic patients that those who used cannabis during a one-year follow up were more likely to relapse and comply poorly with drug treatment. Caspari (65) reported similar findings in a six year follow up study of 39 schizophrenic patients with a history of cannabis abuse and 39 schizophrenic patients without such a history.

Linszen et al (66) reported a prospective study of 93 psychotic patients whose symptoms were assessed monthly over a year. Twenty-four of these patients were cannabis abusers (11 were less than daily users and 13 were daily cannabis users). The cannabis users relapsed to psychosis sooner, and had more relapses in the year of follow up, than the patients who had not used cannabis. Daily users relapsed earlier, and more often, than the less than daily users who, in turn, relapsed sooner, and more often, than the patients who did not use cannabis. These relationships persisted after statistically controlling for premorbid adjustment, and alcohol and other drug use.

Two uncertainties remain. First, it may be that schizophrenia patients who do and do not use cannabis differ in premorbid personality, family history, and other characteristics. This explanation is unlikely in the WHO schizophrenia study (63) and the Linszen et al study (66), both of which used statistical methods to adjust for these confounders. The second difficulty is separating the contributions that cannabis and other drugs make to the exacerbation of schizophrenic symptoms. Heavy alcohol use is common among persons with schizophrenia, and the heavier their cannabis use, the more likely the person is to use psychostimulants and hallucinogens (32). Only Linszen et al statistically adjusted for the effects of concurrent alcohol and drug use. Our confidence that the effect is attributable to cannabis will increase with replications of the Linszen et al study.

10.3.3 Intervention studies

If cannabis use exacerbates schizophrenia then patients who reduce their cannabis use should have fewer symptoms and lower relapse rates. The major difficulty with testing this prediction is getting persons with schizophrenia to reduce their cannabis use. Dependence on alcohol and other drugs is difficult to treat (67), and persons with schizophrenia often have characteristics that predict a poor treatment outcome, namely, they lack social support, they may be cognitively impaired, they are often unemployed, and they may comply poorly with treatment (32, 68).

There are very few controlled outcome studies of substance abuse treatment in schizophrenia (69). Few of these have produced large enough benefits of treatment, or treated a large enough number of patients, to provide an adequate chance of detecting any positive impacts of abstinence on the course of disorders. The few that have been large enough (70) have not reported results separately by diagnosis. Better designed intervention studies should help to clarify the relationship between cannabis use and schizophrenia.

10.3.4 Self-medication

The evidence for the self-medication hypothesis (that persons with schizophrenia use cannabis to avoid unpleasant symptoms of the illness) is not very compelling. Persons with schizophrenia report that they use alcohol, cannabis and other illicit drugs for similar reasons to persons who do not have schizophrenia, namely, to relieve boredom, to provide stimulation, to feel good, and to socialize with peers (32, 37, 71, 72). The drugs that are most often used by schizophrenic patients are also those that are most readily available in the general population, namely, tobacco, alcohol, and cannabis.

In favour of the self-medication hypothesis is the evidence that some schizophrenic patients report using cannabis for its euphoric effects and to relieve negative symptoms and depression (e.g. (29, 40, 73)). Dixon et al (40), for example, surveyed 83 patients with schizophrenia who reported that cannabis reduced anxiety and depression, and increased a sense of calm, but at the cost of making them feel more suspicious.

Hamera et al (74) examined correlations over 84 consecutive days between self-reported psychotic symptoms, licit and illicit drug use, and medication use in 17 persons with schizophrenia. They found relationships between nicotine and prodromal psychotic symptoms and between caffeine use and symptoms of anxiety and depression but there were no relationships between psychotic symptoms and alcohol or cannabis use. This study does have limitations. The difficulty of the self-monitoring task probably selected patients who were more compliant than a representative sample of schizophrenics and they reported low rates of drug use. It is also possible that the time period of 84 days was too short to fully examine the relationship between drug use and major exacerbations of the illness.

10.4 Summary

Evidence supports the hypothesis that cannabis use exacerbates the symptoms of schizophrenia. This evidence comes from a number of retrospective and prospective studies that have controlled for confounding variables. This hypothesis is also biologically plausible: psychotic disorders involve disturbances in the dopamine neurotransmitter systems (75) and cannabinoids, such as THC, increase dopamine release (76).

It is also possible that cannabis use precipitates schizophrenia in persons who are vulnerable because of a personal or family history of schizophrenia. This hypothesis is consistent with the stress-diathesis model of schizophrenia (50, 77) in which schizophrenia is the result of stress acting upon a genetic 'diathesis' to develop schizophrenia. The only direct evidence for it comes from a study by McGuire et al (21) which reported that schizophrenic patients with a history of heavy cannabis use were 10 times more likely to have a family history of schizophrenia than persons with a psychosis who had not used cannabis.

It remains uncertain whether cannabis use can cause schizophrenia that would not have occurred in its absence (78). If it can, it is unlikely to account for more than a minority of cases. Most of the 274 conscripts in the Andreassen et al study who developed

schizophrenia had not used cannabis (54) and only 21 of those who did were heavy cannabis users. The *treated* incidence of schizophrenia has not increased during the 1970s and 1980s (79), despite very substantial increases in cannabis use among young adults in Australia and North America (38). Although there are complications in interpreting such trends (80), the debate has been about whether the incidence of schizophrenia has *declined* or remained *stationary* rather than *increased* (81).

10.5 References

1. Brill, H. & Nahas, G. (1984) Cannabis intoxication and mental illness, in: Nahas, G. (Ed.) *Marihuana in Science and Medicine*, pp. 263–305 (New York, Raven Press).
2. Thornicroft, G. (1990) Cannabis and psychosis: Is there epidemiological evidence for association, *British Journal of Psychiatry*, 157, 25–33.
3. Georgotas, A. & Zeidenberg, P. (1979) Observations on the effects of four weeks of heavy marijuana smoking on group interaction and individual behavior, *Comprehensive Psychiatry*, 20, 427–432.
4. National Academy of Science (1982) *Marijuana and Health* (Washington, DC, National Academy Press).
5. Ghodse, A. (1986) Cannabis psychosis, *British Journal of Addiction*, 81, 473–487.
6. Hall, W., Solowij, N. & Lemon, J. (1994) The health and psychological consequences of cannabis use (Canberra, Australian Government Publishing Service).
7. Hall, W. (1987) A simplified logic of causal inference, *Australian and New Zealand Journal of Psychiatry*, 21, 507–513.
8. Bernardson, G. & Gunne, L. (1972) Forty-six cases of psychosis in cannabis abusers, *International Journal of the Addictions*, 7, 9–16.
9. Chopra, G. & Smith, J. (1974) Psychotic reactions following cannabis use in East Indians, *Archives of General Psychiatry*, 30, 24–27.
10. Solomons, K., Neppe, V. & Kuyl, J. (1990) Toxic cannabis psychosis is a valid entity, *South African Medical Journal*, 78, 476–481.
11. Wylie, A., Scott, R. & Burnett, S. (1995) Psychosis due to "skunk", *British Medical Journal*, 311, 125.
12. Harding, T. & Knight, F. (1973) Marijuana-modified mania, *Archives of General Psychiatry*, 29, 635–637.
13. Eva, J. (1992) Cannabis psychosis, *Psychiatric Bulletin*, 16, 310–311.
14. Carney, M., Bacelle, L. & Robinson, B. (1984) Psychosis after cannabis use, *British Medical Journal*, 288, 1047.
15. Tennant, F. & Groesbeck, C. (1972) Psychiatric effects of hashish, *Archives of General Psychiatry*, 33, 383–386.
16. Hall, W. (1998) Cannabis use and psychosis, *Drug and Alcohol Review*, 17, 433–444.

17. Gruber, A. & Pope, H. (1994) Cannabis psychotic disorder. Does it exist?, *American Journal of the Addictions*, 3, 72–83.

18. Thacore, V. & Shukla, S. (1976) Cannabis psychosis and paranoid schizophrenia, *Archives of General Psychiatry*, 33, 383–386.

19. Rottanburg, D., Robins, A., Ben-Aire, O., Teggin, A. & Elk, R. (1982) Cannabis-associated psychosis with hypomanic features, *Lancet*, 2, 1364–1366.

20. Imade, A. & Ebie, J. (1991) A retrospective study of symptom patterns of cannabis-induced psychosis, *Acta Psychiatrica Scandinavica*, 83, 134–136.

21. McGuire, P., Jones, R., Harvey, I., Williams, M., McGuffin, P. & Murray, R. (1995) Morbid risk of schizophrenia for relatives of patients with cannabis-associated psychosis, *Schizophrenia Research*, 15, 277–281.

22. McGuire, P., Jones, R., Harvey, I., Bebbington, P., Toone, B., Lewis, S. & Murray, R. (1994) Cannabis and acute psychosis, *Schizophrenia Research*, 13, 161–168.

23. Tien, A. Y. & Anthony, J. C. (1990) Epidemiological analysis of alcohol and drug use as risk factors for psychotic experiences, *Journal of Nervous & Mental Disease*, 178, 473–80.

24. Thomas, H. (1996) A community survey of adverse effects of cannabis use, *Drug & Alcohol Dependence*, 42, 201–207.

25. Hall, W., Teesson, M., Lynskey, M. & Degenhardt, L. (1998) The prevalence in the past year of substance use and ICD-10 substance use disorders in Australian adults. Findings from the National Survey of Mental Health and Well-Being. NDARC Technical Report No. 63 (Sydney, National Drug and Alcohol Research Centre, UNSW).

26. Hall, W. & Degenhardt, L. (2000) Cannabis use and psychosis: A review of clinical and epidemiological evidence, *Australian and New Zealand Journal of Psychiatry*, 34, 26–34.

27. Degenhardt, L. & Hall, W. (2001) The association between psychosis and problematical drug use among Australian adults: Findings from the National Survey of Mental Health and Well-Being, *Psychological Medicine*, in press.

28. Poole, R. & Brabbins, C. (1996) Drug induced psychosis, *British Journal of Psychiatry*, 168, 135–138.

29. Schneier, F. & Siris, S. (1987) A review of psychoactive substance use and abuse in schizophrenia: Patterns of drug choice, *Journal of Nervous and Mental Disorders*, 175, 641–652.

30. Smith, J. & Hucker, S. (1994) Schizophrenia and substance abuse, *British Journal of Psychiatry*, 165, 13–21.

31. Warner, R., Taylor, D., Wright, J., Sloat, A., Springett, G., Arnold, S. & Weinberg, H. (1994) Substance use among the mentally ill: Prevalence, reasons for use and effects on illness, *American Journal of Orthopsychiatry*, 74, 30–39.

32. Mueser, K., Bellack, A. & Blanchard, J. (1992) Comorbidity of schizophrenia and substance abuse: Implications for treatment, *Journal of Consulting and Clinical Psychology*, 60, 845–856.

33. Batel, P. (2000) Addiction and schizophrenia, *European Psychiatry*, 15, 115–122.

34. Cuffel, B. (1992) Prevalence estimates of substance abuse in schizophrenia and their correlates, *Journal of Nervous and Mental Disease*, 180, 589–592.

35. Mueser, K., Yarnold, P., Levinson, D., Singh, H., Bellack, A., Kee, K., Morrison, R. & Yadalam, K. (1990) Prevalence of substance abuse in schizophrenia: Demographic and clinical correlates, *Schizophrenia Bulletin*, 16, 31–56.

36. Hambrecht, M. & Hafner, H. (1996) Substance abuse and the onset of schizophrenia, *Biological Psychiatry*, 40, 1155–1163.

37. Fowler, I., Carr, V., Carter, N. & Lewin, T. (1998) Patterns of current and lifetime substance use in schizophrenia, *Schizophrenia Bulletin*, 24, 443–455.

38. Donnelly, N. & Hall, W. (1994) Patterns of cannabis use in Australia, NCADA Monograph Series No. 27, (Canberra, Australian Government Publishing Service).

39. Arndt, S., Tyrell, G., Flaum, M. & Andreasen, N. (1992) Comorbidity of substance abuse and schizophrenia: The role of premorbid adjustment, *Psychological Medicine*, 22, 379–388.

40. Dixon, L., Haas, G., Weiden, P., Sweeney, J. & Frances, A. (1990) Acute effects of drug abuse in schizophrenic patients: Clinical observations and patients' self-reports, *Schizophrenia Bulletin*, 16, 69–79.

41. Zisook, S., Heaton, R., Moranville, J., Kuck, J., Jernigan, T. & Braff, D. (1992) Past substance abuse and clinical course of schizophrenia, *American Journal of Psychiatry*, 149, 552–553.

42. Cuffel, B., Heithoff, K. & Lawson, W. (1993) Correlates of patterns of substance abuse among patients with schizophrenia, *Hospital and Community Psychiatry*, 44, 247–251.

43. Kovasznay, B., Bromet, E., Schwartz, R. & Myers, C. (1993) Substance abuse and onset of psychotic illness, *Hospital and Community Psychiatry*, 44, 567–571.

44. Anthony, J. & Helzer, J. (1991) Syndromes of drug abuse and dependence, in: Robins, L. & Regier, D. (Eds.) *Psychiatric Disorders in America* pp. 117–154 (New York, Macmillan Free Press).

45. Regier, D., Farmer, M., Rae, D., Locke, B., Keith, S., Judd, L. & Goodwin, F. (1990) Comorbidity of mental disorders with alcohol and other drug abuse: Results from the Epidemiological Catchment Area (ECA) Study, *Journal of the American Medical Association*, 264, 2511–2518.

46. Helzer, J., Burnham, A. & McEvoy, L. (1991) Alcohol abuse and dependence, in: Robins, L. & Regier, D. (Eds.) *Psychiatric Disorders in America*, pp. 81–115 (New York, Free Press, Macmillan).

47. Bland, R., Newman, S. & Orn, H. (1987) Schizophrenia: lifetime comorbidity in a community sample, *Acta Psychiatrica Scandinavica*, 75, 383–391.

48. Jablensky, A., McGrath, J., Herman, H., Castle, D., Gureje, O., Evans, M., Carr, V., Morgan, V., Korten, A. & Harvey, C. (2000) Psychotic disorders in urban areas: an overview of the Study on Low Prevalence disorders, *Australian and New Zealand Journal of Psychiatry*, 43, 221–236.

49. Allebeck, P. (1991) Cannabis and schizophrenia: Is there a causal association?, in: Nahas, G. & Latour, C. (Eds.) *Physiopathology of Illicit Drugs: Cannabis, Cocaine, Opiates*, pp. 23–31 (Oxford, Pergamon Press).

50. Bromet, E., Dew, A. & Eaton, W. (1995) Epidemiology of psychosis with special reference to schizophrenia, in: Tsuang, M., Tohen, M. & Zahner, G. (Eds.) *Textbook in Psychiatric Epidemiology* (New York, Wiley and Sons).

51. Khantzian, E. (1997) The self-medication hypothesis of substance use disorders: A reconsideration and recent applications, *Harvard Review of Psychiatry*, 4, 231–244.

52. Andreasson, S., Allebeck, P., Engstrom, A. & Rydberg, U. (1987) Cannabis and schizophrenia: A longitudinal study of Swedish conscripts, *Lancet*, 2, 1483–1486.

53. Negrete, J. (1989) Cannabis and schizophrenia, *British Journal of Addiction*, 84, 349–351.

54. Andreasson, S., Allebeck, P. & Rydberg, U. (1989) Schizophrenia in users and non-users of cannabis, *Acta Psychiatrica Scandinavica*, 79, 505–510.

55. Bell, D. (1973) The experimental reproduction of amphetamine psychosis, *Archives of General Psychiatry*, 29, 35–40.

56. Inghe, G. (1969) The present state of abuse and addiction to stimulant drugs in Sweden, in: Sjoqvist, F. & Tottie, M. (Eds.) *Abuse of Central Stimulants*, pp. 187–214 (New York, Raven Press).

57. Weil, A. (1970) Adverse reactions to marihuana, *New England Journal of Medicine*, 282, 997–1000.

58. Bowers, M. B., Mazure, C. M., Nelson, J. C. & Jatlow, P. I. (1990) Psychotogenic drug use and neuroleptic response, *Schizophrenia Bulletin*, 16, 81–85.

59. Turner, W. & Tsuang, M. (1990) Impact of substance abuse on the course and outcome of schizophrenia, *Schizophrenia Bulletin*, 16, 87–372.

60. Negrete, J., Knapp, W., Douglas, D. & Smith, W. (1986) Cannabis affects the severity of schizophrenic symptoms: Results of a clinical survey, *Psychological Medicine*, 16, 515–520.

61. Cleghorn, J., Kaplan, R., Szechtman, B., Szechtman, H., Brown, G. & Franco, S. (1991) Substance abuse and schizophrenia: Effects on symptoms but not on neurocognitive function, *Journal of Clinical Psychiatry*, 52, 26–30.

62. DeQuardo, J. R., Carpenter, C. F. & Tandon, R. (1994) Patterns of substance abuse in schizophrenia: Nature and significance, *Journal of Psychiatric Research*, 28, 267–275.

63. Jablensky, A., Sartorius, N. & Emberg, G. (1991) Schizophrenia: Manifestations, incidence and course in different cultures. A World Health Organization Ten-Country Study, *Psychological Medicine Supplement No. 20*.

64. Martinez-Arevalo, M., Calcedo-Ordonez, A. & Varo-Prieto, J. (1994) Cannabis consumption as a prognostic factor in schizophrenia, *British Journal of Psychiatry*, 164, 769–681.

65. Caspari, D. (1999) Cannabis and schizophrenia: results of a follow-up study, *European Archives of Psychiatry & Clinical Neuroscience*, 249, 45–9.

66. Linszen, D. H., Dingemans, P. M. & Lenior, M. E. (1994) Cannabis abuse and the course of recent-onset schizophrenic disorders, *Archives of General Psychiatry*, 51, 273–279.

67. Heather, N. & Tebbutt, J. (1989) *An Overview of the Effectiveness of Treatment for Drug and Alcohol Problems. NCADA Monograph Series No. 11* (Canberra, Australian Government Publishing Service).

68. Kavanagh, D. (1995) An intervention for substance abuse in schizophrenia, *Behaviour Change*, 12, 20–30.

69. Lehman, A., Herron, J., Schwartz, R. & Myers, C. (1993) Rehabilitation for adults with severe mental illness and substance use disorders, *Journal of Nervous and Mental Disease*, 181, 86–90.

70. Jerrell, J. & Ridgeley, M. (1995) Comparative effectiveness of three approaches to serving people with severe mental illness and substance abuse disorders, *Journal of Nervous and Mental Disease*, 183, 566–576.

71. Noordsy, D., Drake, R., Teague, G., Osher, F., Hulbut, S., Beaudett, M. & Paskus, T. (1991) Subjective experiences related to alcohol use among schizophrenics, *Journal of Nervous and Mental Disease*, 179, 411–414.

72. Test, M., Wallisch, L., Allness, D. & Tripp, K. (1989) Substance use in young adults with schizophrenic disorders, *Schizophrenia Bulletin*, 15, 465–476.

73. Peralta, V. & Cuesta, M. (1992) Influence of cannabis abuse on schizophrenic psychopathology, *Acta Psychiatrica Scandinavica*, 85, 127–130.

74. Hamera, E., Schneider, J. & Deviney, S. (1995) Alcohol, cannabis, nicotine and caffeine use and symptom distress in schizophrenia, *Journal of Nervous and Mental Disease*, 183, 559–565.

75. Stahl, S. (1996) *Essential psychopharmacology* (Cambridge, Cambridge University Press).

76. Adams, I. & Martin, B. (1996) Cannabis: Pharmacology and toxicology in animals and humans, *Addiction*, 91, 1585–1614.

77. Cottesman, I. (1991) *Schizophrenia Genesis: The origins of madness* (New York, Freeman and Co).

78. McKay, D. R. & Tennant, C. C. (2000) Is the grass greener? The link between cannabis and psychosis, *Medical Journal of Australia*, 172, 284–6.

79. Der, G., Gupta, S. & Murray, R. (1990) Is schizophrenia disappearing?, *Lancet*, 1, 513–516.

80. Kendell, R., Malcolm, D. & Adams, W. (1993) The problem of detecting changes in the incidence of schizophrenia, *British Journal of Psychiatry*, 162, 212–218.

81. Jablensky, A. (1999) Schizophrenia: Epidemiology, *Current Opinion in Psychiatry*, 12, 19–28.

11 Is cannabis a gateway drug?

Adolescent cannabis use is an understandable concern to the community. This is because adolescents' decisions about whether or not to use drugs are not as informed as those of adults (1) and regular cannabis use may complicate the transition from childhood to adulthood by interfering with school performance, interpersonal relationships with parents and peers, and limiting important life choices, such as whom and when to marry, and what occupation to pursue (2, 3). Young people who start using cannabis in adolescence are more likely to become regular users and are therefore more likely to experience any adverse health effects caused by chronic cannabis use (e.g. (1, 3). Adolescence is also a time of risk-taking when the use of an intoxicant, such as alcohol or cannabis, while driving a car may increase the risk of accidental injury and premature death (1).

One concern about adolescent cannabis use has dominated the cannabis policy debate. This is that adolescent cannabis use may increase the chance that young people will use other more dangerous illicit drugs, such as cocaine and heroin (4–6). This is known as the 'gateway hypothesis'.

In deciding whether cannabis is a gateway drug the first question that needs to be answered is whether cannabis users are more likely to use other illicit drugs. If so, we need to ask whether the relationship is explained by other factors. One possibility is that individuals who use cannabis are more likely to use other illicit drugs for other reasons. We can test this by seeing whether rates of illicit drug use among cannabis users change when we take account of the characteristics of young people who are the most likely to use cannabis.

If there is a relationship between cannabis and other illicit drug use, we have to explain it. The two main explanations that feature in the public debate are: (1) that cannabis users are more likely to use other illicit drugs because of the pharmacological and other effects that cannabis has; and (2) that cannabis users are more likely to use other illicit drugs because the same black market supplies cannabis and other illicit drugs, so cannabis users are more likely to have access to other illicit drugs.

11.1 Is there a relationship between cannabis use and other drug use?

There is abundant evidence from surveys of adolescent drug use in the United States and elsewhere that *regular* cannabis use and the use of cocaine and heroin are associated (7). From the late 1970s to the 1990s in the United States, there was a strong relationship between regular cannabis use and the later use of heroin and cocaine. Kandel (8), for example, found that only 7% of American adolescents who had not used cannabis reported using another illicit drug. By contrast, 33% of those who reported using cannabis had used another illicit drug. Most (84%) daily cannabis users had done so and they had also used many more types of illicit drugs than their peers who had not used cannabis or who were not daily users of cannabis (8).

The same relationship has been observed in Australian surveys of drug use (9). In the 1993 National Campaign Against Drug Abuse (NCADA) survey of drug use in Australia, for example, even though 96% of cannabis users had *not* used heroin, the odds of using heroin were approximately 30 times higher among those who have used cannabis than those who had not (9). In the 1998 National Drug Strategy Household Survey, there was an even stronger relationship: those who reported that they had ever used cannabis were 78 times more likely to report having used heroin. The association is so strong because so few persons who have used heroin had not used cannabis (only 4 out of 276 in the 1998 survey).

Kandel and colleagues have described a typical sequence of involvement with licit and illicit drugs among American adolescents during the 1970s and 1980s. Almost all adolescents who have tried cocaine and heroin, had used alcohol, tobacco and cannabis in that order (10). Those who began to use alcohol and tobacco at an early age, and those who became regular smokers and drinkers, were the ones who were most likely to use cannabis. In turn, it was cannabis users who began use at an early age who were the most likely to become regular cannabis users and the most likely to use hallucinogens, amphetamines and tranquillisers. The heaviest users of these drugs were, in turn, more likely to use cocaine and heroin. Kandel and her colleagues have confirmed these results in longitudinal studies of adolescent drug use in this age cohort (11) and in later cohorts with high rates of crack cocaine use (12, 13).

Generally, the earlier the age at which a young person used any drug in the sequence, and the more regular their use of it, the more likely they were to use the next drug in the sequence (14–16). This sequence of drug involvement has largely been confirmed by other US researchers (7, 17). Longitudinal studies of drug use in Australia (18), Germany (19), New Zealand (20–23), and Sweden (24, 25) have broadly confirmed US findings on sequences of drug involvement and predictors of progression to cannabis and other illicit drug use.

11.2 Is the relationship between cannabis and other drug use spurious?

One explanation of the relationship between daily cannabis use and the use of other drugs is that it is due to the type of person who uses cannabis. According to this 'selective recruitment' hypothesis, the relationship is explained by the recruitment to cannabis use of deviant and nonconformist young persons who have a predilection to use a range of intoxicating drugs like alcohol, cannabis, cocaine and heroin (22). On this hypothesis, the order in which these drugs are tried simply reflects their availability and the societal disapproval of their use (7, 17). That is, alcohol and tobacco use precede cannabis use because alcohol and tobacco are readily available to adolescents, and cannabis use precedes heroin and cocaine use because cannabis is the much commonly used illicit drug and it is more readily available than cocaine and heroin. On this hypothesis, cannabis use is not a cause of the use of other illicit drugs. Rather, cannabis and other illicit drug use are common consequences of pre-existing social deviance and nonconformity (26, 27).

The selective recruitment hypothesis is supported by the substantial correlations between various types of nonconforming adolescent behaviour, including high school drop out, early sexual experience and unplanned pregnancy, delinquency, and alcohol and illicit drug use (28, 29). All of these behaviours are correlated with nonconformist and rebellious attitudes and antisocial conduct in childhood (30) and early adolescence (27, 28).

Regular cannabis users are more likely than their peers: to have a history of antisocial behaviour (23, 31); to be nonconformist and alienated (30–32); to perform more poorly at school (33–35); and to use drugs to deal with personal distress (30, 36). In general, the more of these risk factors that adolescents have, the more likely they are to use cannabis daily, and to use other illicit drugs (31, 37, 38).

The selective recruitment hypothesis can be tested in longitudinal studies by examining whether cannabis use still predicts the use of heroin and cocaine after statistically controlling for pre-existing differences between cannabis users and nonusers in social deviance and non-conformity (22). A number of studies have used this strategy to test the selective recruitment hypothesis.

Yamaguchi (39) tested whether the relationship between cannabis use and 'harder' illicit drug use persisted after statistically controlling for pre-existing adolescent behaviours and attitudes, interpersonal factors, and the age of initiation into drug use. They found that the relationship between cannabis use and the use of other illicit drugs was not explained by these factors or by friends' cannabis use. The same finding has emerged in several other studies (11, 40, 41). In these studies, the relationship between cannabis and heroin use has been reduced but not eliminated by statistically controlling for differences between users and non-users of cannabis.

O'Donnell and Clayton (40) have argued that this is strong evidence in favour of a causal connection between cannabis and heroin use. The strength of their argument depends on whether the most important characteristics of cannabis users have been statistically controlled for in these studies. It would be difficult to argue that this was true in the early studies. Kandel et al. (11), for example, were unable to measure the users' attitudes and family characteristics at the time of drug initiation. In the O'Donnell and Clayton (40) and Robins et al. (41) studies, deviance 'prior' to drug use was assessed retrospectively, with unknown validity. Baumrind (42) argued that 'in the absence of evidence of external validity' of these measures it is 'safer' to assume that the relationship between cannabis use and heroin use is spurious.

Two studies by Fergusson and Horwood (20, 22) address many of the weaknesses in the earlier studies. These report data from a prospective study of 990 New Zealand children who were followed from birth to age 21 years and assessed on a wide range of psychosocial variables that potentially explain the relationship between cannabis use and the use of other illicit drugs. These included: family background (socio-economic status, parental conflict and divorce, childhood sexual abuse, parental punishment and parental attachment); parental adjustment (parental alcohol and drug problems, criminality and illicit drug use); individual characteristics of the young person (gender, intelligence, novelty seeking); early adolescent development (cigarette smoking, frequency of alcohol

use, juvenile offending, school drop out, conduct problems and attitudes towards drug use); peer affiliations (peer use and problems with alcohol and other drug use); and personal history of risk taking. These factors were statistically controlled for in analyses of relationships between cannabis use and use of other illicit drugs.

Fergusson and Horwood (20) reported on the relationship between the use of cannabis by age 16 and the use of other illicit drugs by the age of 18 years. They found a strong relationship between the frequency of cannabis use by age 16 and development of a problem with cannabis, alcohol or other substances by age 18. Early cannabis users came from lower socio-economic status families with a history of parental conflict, parental criminality and alcohol and drug use and low parental attachment. They also had a personal history of conduct problems, low self-esteem, high novelty seeking, and high affiliation with delinquent peers. Adjustment for these factors reduced but did not eliminate the relationship between early cannabis use and the use of other illicit drugs.

Fergusson and Horwood (22) reported a later follow up of the cohort. They found that 69% of their sample reported using cannabis by age 21, and 26% had used one or more other illicit drugs, with 4% having used cocaine or an opiate. In 99% of cases, cannabis use preceded the use of other illicit drugs. They found a strong relationship between level of cannabis use at any age and the use of another illicit drug. Compared to those who had never used cannabis, the risk of using another illicit drug was around 4 times higher among those who had used cannabis once or twice, 12 among those who had used 3 to 11 times, 41 times higher among those who had used 12 to 49 times and 143 times greater among those who had used 50 times or more. The relationships were reduced but remained substantial when other psychosocial factors were controlled for statistically. Compared to non-users of cannabis, the risks (after statistical adjustment) were 3 greater for those who had used once or twice, 8 greater for those who had used 3 to 11 times, 21 greater for those who had used 12 to 49 times and 59 greater for those who had used for 50 times or more.

The results of the Fergusson and Horwood studies make it unlikely that selective recruitment wholly explains the relationship between cannabis use and other illicit drug use. But its findings do not, as Fergusson and Horwood acknowledge, rule out other explanations. Among these is the possibility that there is a shared genetic vulnerability to use and become dependent on cannabis and other illicit drugs.

Studies of alcohol, tobacco and other drug use in identical and non-identical twins indicate that there is a genetic vulnerability to developing dependence on alcohol (43), cannabis (44) and tobacco (45). More importantly, a component of the genetic vulnerability to dependence on these three drug classes is shared or common (46). So too are the shared family and environmental factors that influence alcohol and cannabis dependence (46). The contribution of genes to dependence on other illicit drugs is less certain because rates of use in these twin studies have been too low to provide a powerful test of this hypothesis. The hypothesis of common genes for regular use of cannabis and other illicit drugs has not been directly tested in any of the cohort studies, including that of Fergusson and Horwood. The identification of specific candidate genes for vulnerability to drug dependence will enable this hypothesis to be tested in future studies.

11.3 Explaining the association between cannabis and other drug use

If the association between cannabis and heroin use is not explained by pre-existing differences between cannabis users and nonusers, how might cannabis use 'cause' heroin and cocaine use? The two main competing explanations differ in whether they attribute the relationship to the pharmacological effects of cannabis or to the social context within which cannabis is obtained and used.

One hypothesis is that the pharmacological effects of cannabis use predispose regular cannabis users to use other intoxicating drugs (47, 48). Nahas (47) has hypothesised that 'the biochemical changes induced by marijuana in the brain result in a drug-seeking, drug-taking behaviour, which in many instances will lead the user to experiment with other pleasurable substances' (p xxiii).

Recent studies in animals (e.g. 49) have been interpreted as supporting a pharmacological explanation of the association between regular cannabis use and other drug use (50). These studies indicate that common biochemical pathways underlie the rewarding effects of cannabis, cocaine, heroin and nicotine (51). All these drugs appear to act on dopaminergic neurotransmitter systems that are involved in the 'reward centres' in an area of the midbrain, the nucleus accumbens (52). However, there is as yet no direct evidence from animal studies that administration of THC to animals increases their risk of using other illicit drugs (53).

Pharmacological explanations of the relationship between cannabis and other drug use also have difficulty explaining a number of facts about their relationship. First, there are relatively low rates of progression from cannabis use to the regular use of other illicit drugs; experimentation and discontinuation of cannabis use is the norm (54). Those heavy cannabis users who do use other illicit drugs also continue to use cannabis, as well as the new illicit drugs. As Donovan and Jessor (17) have noted: '...'harder' drugs do not serve as substitutes for 'softer' drugs. Rather, a deepening of regular substance use appears to go along with a widening of experience in the drug domain' (p. 548–549). This pattern of involvement is more consistent with a genetic vulnerability to drug dependence than the hypothesis that cannabis use is a stepping-stone to experimentation with other drugs.

Third, the pattern of progression in drug use among American adolescents in the 1970s was affected by drug availability (14). Among cohorts of heroin users in the 1950s and 1960s, cannabis use was confined to those geographic areas of the US in which it was readily available (5). Research on African-American adolescents also showed a variation in the sequence of drug use. In African-American communities cocaine and heroin were more readily available than hallucinogens so cocaine and heroin use often preceded the use of hallucinogens (14). Similarly, American soldiers in Vietnam used heroin before they used alcohol because heroin was cheaper and more freely available in Vietnam than was alcohol (since many of the American troops were under the legal drinking age of 21) (55).

The historical and geographical variations in sequences of drug use suggest sociological explanations of the use of heroin among heavy cannabis users. One hypothesis is that regular cannabis use predicts an increased use of other illicit drugs because regular cannabis users have an increased contact with other drug users and drug sellers and hence more opportunities to use other illicit drugs than peers who do not use cannabis regularly. Regular cannabis use thereby increases involvement in a drug using subculture which, in turn, exposes cannabis users to peers who have used other illicit drugs, who approve of such drug use, and who provide more opportunities to use other illicit drugs because of their increased availability within their social circle (5, 56).

Although plausible, there is little direct evidence on the drug subculture hypothesis. Goode (5) presented data from the late 1960s indicating that the number of friends who used heroin was a stronger predictor of heroin use than was frequency of cannabis use, arguing that the 'correlation between frequency of use and the use of dangerous drugs ... [is] the result of interaction and involvement with others who use' (p. 332). These observations have been supported by Kandel's (8) finding that the strongest predictor of continued cannabis use in early adulthood was the number of friends who were cannabis users.

Fergusson and Horwood's (22) analysis of the Christchurch Child Development Study was able to examine the contribution of affiliation with drug using peers to the relationship between cannabis and other illicit drug use. They included self-reported peer use of alcohol, cannabis and other illicit drugs in their statistical analyses. Their inclusion reduced but did not eliminate the relationship between cannabis and other illicit drug use, indicating that while peer drug use made a contribution to the association, it did not fully explain it.

The role of socialisation in a drug-using subculture and involvement in drug markets has not been directly tested in the important cohort studies (22). It is nonetheless a plausible hypothesis. Regular cannabis users are distinguished from non-users by their extensive social relationships with other drug users and often by buying and selling cannabis and other illicit drugs to finance their own drug use (5).

11.4 Summary

Research on adolescent use of cannabis and other illicit drug use has revealed a number of consistent findings about the relationship between cannabis and other illicit drug use. First, among American adolescents in the 1970s the use of alcohol and tobacco preceded use of cannabis, which in turn, preceded the use of hallucinogens and 'pills', and the use of heroin and cocaine. Generally, the earlier the age of initiation into drug use, and the greater the involvement with any drug in the sequence, the more likely a young person was to use the next drug in sequence. Similar sequences have been observed in a variety of societies, including Australia.

The explanation of the role of cannabis in the sequence of illicit drug use remains controversial. The relationship does not appear to be spurious. The hypothesis that the sequence of drug use represents a direct pharmacological effect of cannabis use upon the

use of later drugs in the sequence is not compelling. It also seems unlikely that the association between regular cannabis use and the use of other illicit drugs is *wholly* the result of shared risk factors or common causes. Selective recruitment of socially deviant adolescents to cannabis use, plays some role but it also does not explain the relationship. A shared genetic vulnerability to alcohol, tobacco and cannabis dependence is a plausible explanation that cannot be excluded on the available evidence.

If there is a causal relationship between cannabis and other illicit drug use the explanation is more likely to be a sociological than a pharmacological one. The fact that cannabis use predicts an increased chance of using other illicit drugs reflects a combination of: (1) the selective recruitment to heavy cannabis use of persons with pre-existing personality and attitudinal traits (possibly genetic in origin) that predispose to the use of other intoxicants; (2) their affiliation with drug using peers; (3) socialisation into an illicit drug subculture in which there is an increased opportunity and encouragement to use other illicit drugs; (4) increased access to opportunities to purchase and use other illicit drugs because of involvement in illicit drug markets as buyers and sellers; and possibly (5) a shared genetic vulnerability to use and become dependent on a range of different drugs.

11.5 References

1. Kleiman, M. (1989) *Marijuana: Costs of Abuse, Costs of Controls* (New York, Greenwood Press).
2. Baumrind, D. & Moselle, K. (1985) A developmental perspective on adolescent drug abuse, *Advances in Alcohol and Substance Abuse*, 5, 41–67.
3. Polich, J., Ellickson, P., Reuter, P. & Kahan, J. (1984) *Strategies for Controlling Adolescent Drug Use* (Santa Monica, CA, The RAND Corporation).
4. DuPont, R. (1984) *Getting Tough on Gateway Drugs* (Washington, DC, American Psychiatric Press).
5. Goode, E. (1974) Marijuana use and the progression to dangerous drugs, in: Miller, L. (Ed.) *Marijuana: Effects on Human Behavior*, pp. 303–338 (New York, Academic Press).
6. Kleiman, M. (1992) *Against Excess: Drug Policy for Results* (New York, Basic Books).
7. Merrill, J. C., Kleber, H. D., Shwartz, M., Liu, H. & Lewis, S. R. (1999) Cigarettes, alcohol, marijuana, other risk behaviors, and American youth, *Drug and Alcohol Dependence*, 56, 205–212.
8. Kandel, D. B. (1984) Marijuana users in young adulthood, *Archives of General Psychiatry*, 41, 200–209.
9. Donnelly, N. & Hall, W. (1994) Patterns of cannabis use in Australia. NCADA Monograph Series No. 27, (Canberra, Australian Government Publishing Service).
10. Kandel, D. & Faust, R. (1975) Sequence and stages in patterns of adolescent drug use, *Archives of General Psychiatry*, 32, 923–932.

11. Kandel, D. B., Davies, M., Karus, D. & Yamaguchi, K. (1986) The consequences in young adulthood of adolescent drug involvement. An overview, *Archives of General Psychiatry*, 43, 746–54.

12. Kandel, D. & Yamaguchi, K. (1993) From beer to crack: Developmental patterns of drug involvement, *American Journal of Public Health*, 83, 851–5.

13. Kandel, D. B. & Davies, M. (1996) High school students who use crack and other drugs, *Archives of General Psychiatry*, 53, 71–80.

14. Kandel, D. B. (1978) Convergences in prospective longtudinal surveys of drug use in normal populations, in: Kandel, D. B. (Ed.) *Longitudinal Research on Drug Use: Empirical Findings and Methodological Issues*, pp. 3–38 (New York, John Wiley and Sons).

15. Kandel, D. B. & Logan, J. A. (1984) Patterns of drug use from adolescence to young adulthood: I. Periods of risk for initiation, continued use and discontinuation. *American Journal of Public Health*, 74, 660–666.

16. Kandel, D. (1988) Issues of sequencing of adolescent drug use and other problem behaviors, *Drugs and Society*, 3, 55–76.

17. Donovan, J. E. & Jessor, R. (1983) Problem drinking and the dimension of involvement with drugs: A Guttman Scalogram analysis of adolescent drug use, *American Journal of Public Health*, 73, 543–552.

18. Coffey, C., Lynskey, M., Wolfe, R. & Patton, G. C. (2000) Initiation and progression of cannabis use in a population-based Australian adolescent study, *Addiction*, 95, 1679–1690.

19. Hoefler, M., Lieb, R., Perkonigg, A., Schuster, P., Sonntag, H. & Wittchen, H.-U. (1999) Covariates of cannabis use progression in a representative population sample of adolescents: A prospective examination of vulnerability and risk factors, *Addiction*, 94, 1679–1694.

20. Fergusson, D. M. & Horwood, L. J. (1997) Early onset cannabis use and psychosocial adjustment in young adults, *Addiction*, 92, 279–296.

21. Fergusson, D. M. & Horwood, L. J. (1999) Prospective childhood predictors of deviant peer affiliations in adolescence, *Journal of Child Psychology and Psychiatry*, 33, 1059–1075.

22. Fergusson, D. M. & Horwood, L. J. (2000) Does cannabis use encourage other forms of illicit drug use?, *Addiction*, 95, 505–520.

23. McGee, R. & Feehan, M. (1993) Cannabis use among New Zealand adolescents, *New Zealand Medical Journal*, 106, 345.

24. Stenbacka, M., Allebeck, P., Brandt, L. & Romelsjo, A. (1992) Initiation into drug abuse: The pathway from being offered drugs to trying cannabis and progression to intravenous drug abuse, *Scandinavian Journal of Social Medicine*, 20, 94–101.

25. Stenbacka, M., Allebeck, P. & Romelsjo, A. (1993) Initiation into drug abuse: The pathway from being offered drugs to trying cannabis and progression to intravenous drug abuse, *Scandinavian Journal of Social Medicine*, 21, 31–39.

26. Kaplan, H., Martin, S. & Robbins, C. (1982) Pathways to adolescent drug use: Self-derogation, peer influence, weakening of social controls, and early substance use, *Journal of Health and Social Behavior*, 25, 270–289.

27. Newcomb, M. D. & Bentler, P. (1988) *Consequences of adolescent drug use* (California, Sage Publications).

28. Jessor, R. & Jessor, S. L. (1977) *Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth* (New York, Academic Press).

29. Osgood, D. W., Johnston, L. D., O'Malley, P. M. & Bachman, J. G. (1988) The generality of deviance in late adolescence and early adulthood, *American Sociological Review*, 53, 81–93.

30. Shedler, J. & Block, J. (1990) Adolescent drug use and psychological health: A longitudinal inquiry, *American Psychologist*, 45, 612–630.

31. Brook, J., Cohen, P., Whiteman, M. & Gordon, A. (1992) Psychosocial risk factors in the transition from moderate to heavy use or abuse of drugs, in: Glantz, M. & Pickens, R. (Eds.) *Vulnerability to Drug Abuse*, pp. 359–388 (Washington, American Psychological Association).

32. Jessor, R. & Jessor, S. L. (1978) Theory testing in longitudinal research on marihuana use, in: Kandel, D. B. (Ed.) *Longitudinal Research on Drug Use: Empirical Findings and Methodological Issues*, pp. 41–71 (New York, John Wiley and Sons).

33. Bailey, S. L., Flewelling, J. V. & Rachal, J. V. (1992) Predicting continued use of marijuana among adolescents: The relative influence of drug-specific and social context factors, *Journal of Health and Social Behaviour*, 33, 51–66.

34. Hawkins, J. D., Catalano, R. F. & Miller, J. Y. (1992) Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention, *Psychological Bulletin*, 112, 64–105.

35. Kandel, D. & Davies, M. (1992) Progression to regular marijuana involvement: Phenomenology and risk factors for near daily use, in: Glantz, M. & Pickens, R. (Eds.) *Vulnerability to Drug Abuse*, pp. 211–253 (Washington, DC, American Psychological Association).

36. Kaplan, H. B. & Johnson, R. J. (1992) Relationships between circumstances surrounding initial drug use and escalation of drug use: Moderating effects of gender and early adolescent experiences, in: Glantz, M. & Pickens, R. (Eds.) *Vulnerability to Drug Abuse* (Washington, American Psychological Association).

37. Newcomb, M. (1992) Understanding the multidimensional nature of drug use and abuse: The role of consumption, risk factors and protective factors, in: Glantz, M. & Pickens, R. (Eds.) *Vulnerability to Drug Abuse*, pp. 255–296 (Washington, American Psychological Association).

38. Scheier, L. M. & Newcombe, M. D. (1991) Psychosocial predictors of drug use initiation and escalation: An expansion of the multiple risk factors hypothesis using longitudinal data, *Contemporary Drug Problems*, 18, 31–73.

39. Yamaguchi, K. & Kandel, D. B. (1984) Patterns of drug use from adolescence to adulthood. III Predictors of progression, *American Journal of Public Health*, 74, 673–681.

40. O'Donnell, J. A. & Clayton, R. R. (1982) The stepping stone hypothesis—marijuana, heroin and causality, *Chemical Dependencies*, 4, 229–241.

41. Robins, L., Darvish, H. S. & Murphy, G. E. (1970) The long-term outcome for adolescent drug users: A follow-up study of 76 users and 146 nonusers., in: Zubin, J. & Freedman, A. M. (Eds.) *The Psychopathology of Adolescence*, pp. 159–180 (New York, Grune and Stratton).

42. Baumrind, D. (1983) Specious causal attribution in the social sciences: The reformulated stepping stone hypothesis as exemplar, *Journal of Personality and Social Psychology*, 45, 1289–1298.

43. Heath, A. (1995) Genetic influences on alcoholism risk: A review of adoption and twin studies, *Alcohol Health and Research World*, 19, 166–171.

44. Kendler, K. S. & Prescott, C. A. (1998) Cannabis use, abuse, and dependence in a population-based sample of female twins, *American Journal of Psychiatry*, 155, 1016–22.

45. Han, C., McGue, M. & Iacono, W. (1999) Lifetime tobacco, alcohol and other substance use in adolescent Minnesota twins: Univariate and multivariate behavioral genetic analyses, *Addiction*, 94, 981–993.

46. True, W. R., Heath, A. C., Scherrer, J. F., Xian, H., Lin, N., Eisen, S. A., Lyons, M. J., Goldberg, J. & Tsuang, M. T. (1999) Interrelationship of genetic and environmental influences on conduct disorder and alcohol and marijuana dependence symptoms, *American Journal of Medical Genetics*, 88, 391–397.

47. Nahas, G. (1990) *Keep Off the Grass* (Middlebury, VT, Paul Eriksson).

48. Walters, E. (1993) *Marijuana: An Australian crisis* (Malvern, Victoria, Elaine Walters).

49. Tanda, G., Pontieri, F. & Di Chiara, G. (1997) Cannabinoid and heroin activation of mesolimbic dopamine transmission by a common mu1 opioid receptor mechanism, *Science*, 276, 2048–2050.

50. Wickelgren, I. (1997) Marijuana, Harder than thought?, *Science*, 276, 1967–1968.

51. MacCoun, R. (1998) In what sense (if any) is marijuana a gateway drug?, *FAS Drug Policy Analysis Bulletin*, 4.

52. Gardner, E. L. (1999) Cannabinoid interaction with brain reward systems, in: Nahas, G., Sutin, K., Harvey, D. & Agurell, S. (Eds.) *Marihuana and Medicine*, pp. 187–205 (Towa, New Jersey, Humana Press).

53. Zimmer, L. & Morgan, J. (1997) *Marijuana myths, marijuana facts* (New York, The Lindesmith Center).

54. Chen, K. & Kandel, D. B. (1995) The natural history of drug use from adolescence to the mid-thirties in a general population sample, *American Journal of Public Health*, 85, 41–7.

55. Robins, L. (1993) Vietnam veterans' rapid recovery from heroin addiction: A fluke or normal expectation?, *Addiction*, 88, 1041–1054.

56. Cohen, S. (1972) Drug use: Religion and secularization, *American Journal of Psychiatry*, 129, 97.

12 Effects on adolescent psychosocial development

There have been two dominant concerns about the effects of adolescent cannabis use on psychosocial development. One is that adolescent cannabis use may adversely affect educational outcomes. The other is that cannabis use may adversely affect other psychosocial outcomes, such as employment, involvement in crime, and mental health. The evidence relevant to these concerns is discussed in this chapter.

12.1 Adolescent cannabis use and educational performance

It is reasonable to suspect that adolescent cannabis use may impair educational performance and increase the chances that a student will discontinue their education (1). Cannabis use acutely impairs memory and attention and, if used regularly, it could impair learning and school performance, thereby increasing the chance of a student dropping out of school. If the adolescent's school performance was marginal to begin with, as research suggests it is among regular cannabis users, then cannabis use could increase the risk of school failure. Since high school education is so important to occupational choice, this potential effect of adolescent cannabis use could flow through the individual's life.

A number of cross-sectional surveys have examined relationships between cannabis use and educational attainment among school children and youth. The measures of educational outcome have only rarely included school grades and examination performances. Instead these studies have measured truancy and early school leaving, perhaps because confidentiality and privacy preclude access to school grades and performance in external examinations.

Resnick et al (2) reported that a low grade point average was associated with cannabis use in a national sample of 12,118 adolescents in the USA. Brook et al (3) reported that among 1,687 Colombian adolescents those who were dissatisfied with school were more likely to use cannabis. In an Australian study of 199 high school students aged 13–16 years, Jones and Heaven found that young people who were regular cannabis users had a more negative attitude toward school and a poorer record of school attendance than those who were not (4). Lifrak et al reported a negative correlation between cannabis use and scholastic competence for boys (but not for girls) in a sample of 271 seventh and eight grade students (5). Novins & Mitchell (6) also reported a significant association between poor school performance and cannabis use for males (but not females) in a sample of 1464 Native American adolescents.

A number of studies have shown that rates of cannabis and other illicit drug use are higher among young people who either no longer attend school or who are absent from school on any given day. For example, Lynskey et al (7) found that young people in the

Australian School Students' Alcohol and Drugs Survey who reported being away from school the day before the survey had higher rates of cannabis use than students who attended school on that day. Similarly, Fergusson, Lynskey and Horwood (8) found that truancy was more common among cannabis users in a sample of nearly 1,000 16 year old New Zealanders.

Mensch and Kandel (9) examined relationships between educational achievement and cannabis use in the US National Longitudinal Survey of Young Adults. They found that high school graduates reported significantly more cannabis use during adolescence than college graduates, even after controlling for socio-demographic factors, and differences in academic ability, self-esteem and delinquency. The value of this study was compromised by a reliance on retrospective reports of cannabis use, the reliability and validity of which have been questioned (10).

12.2 Explaining the relationship

Four broad explanations of the relationship between cannabis use and educational outcome need to be considered. The first and simplest explanation of the association is that early cannabis use causes poor educational outcomes. Kandel, Davies, Karus and Yamaguchi (11) argued that early cannabis use encourages continued use of the drug, and that cannabis and other illicit drug use encourages anti-conventional behaviours including early school leaving, delinquency, employment problems and difficulties in interpersonal relationships.

A second alternative explanation is that heavy cannabis use is a *consequence* of poor educational attainment. There is some support for this hypothesis in that poor educational performance is a risk factor that precedes cannabis use (12–16). The hypotheses that cannabis use is a cause of poor school performance and that poor school performance is a cause of cannabis use are not mutually exclusive. Both processes could be at work (17) if poor school performance increased the risks of using cannabis, which in turn worsened school performance.

A third possible explanation is that cannabis use and poor educational attainment are reflections of a common syndrome of problem behaviour (18). A wide range of problem behaviours in adolescence are manifestations of a common syndrome of problem behaviours (19).

The final possibility is that the associations between early cannabis use and poor educational outcomes are not causal but the result of common factors that increase the likelihood of both early cannabis use and poor educational performance. There is evidence that the risk factors and life pathways for early cannabis use overlap considerably with those for poor educational performance. These risk factors (see reviews by (15, 20, 21) include: the extent to which the norms and attitudes of the wider community encourage or discourage the use of drugs; social disadvantage and family dysfunction; individual factors including personality and an individual's propensity to violate norms; and the extent to which an individual affiliates with delinquent and drug using peers.

12.3 Longitudinal studies of cannabis use and educational outcomes

These four explanations can only be distinguished by prospective longitudinal studies in which a large representative group of young people is assessed over time on their cannabis use, educational attainment and other potentially confounding factors, such as family and social circumstances, personality characteristics and delinquency. These studies have the following strengths (22). First, they enable us to tell which comes first, cannabis use or poor educational performance. Second, they reduce the effects of bias in retrospective reports of cannabis use and behaviour. Third, they enable us to test causal hypotheses about cannabis use and educational outcomes by statistically adjusting for confounding variables. That is, they allow us to answer the question: do young people who use cannabis have poorer educational outcomes than those who do not, when we allow for the fact that cannabis users are more likely to perform poorly in school before they used cannabis?

Newcomb and Bentler (23) followed a sample of 654 high school students over 8 years to assess the impact of early substance use on educational outcomes at ages 19 to 24 years. They used statistical methods to examine the extent to which cannabis and other drug use were associated with adverse outcomes in young adulthood, after taking account of the effects of confounding factors. Their analyses indicated that early substance users were more likely to abandon a college education.

The results of this study have been supported by Fergusson, Lynskey and Horwood (24) who examined the extent to which cannabis use before the age of 15 years predicted regular drug use, criminal offending, poor mental health and reduced life opportunities at age 16, after adjusting for a range of potentially confounding factors. The sample consisted of 990 young people who had been followed from birth to age 16 years. They were assessed on cannabis use at age 15 and on cannabis use and a wide range of other health and psychological outcomes at age 16.

The ten percent of the sample who had used cannabis by the age of 15 had elevated risks of school problems at age 16. Specifically, 22.5% had left school before age 16 (the minimum school leaving age in New Zealand) compared with only 3.5% of those who had not used cannabis. The frequency of truancy between 15 and 16 years was also higher among those who had used cannabis before the age of 15 years (31.5%) than those who had not used cannabis (4.7%). The relationship between early cannabis use and early school leaving persisted after statistical adjustment for pre-existing differences between early cannabis users and their peers. In a later follow-up of the same birth cohort, Fergusson and Horwood (25) reported that those who had used cannabis before the age of 16 years were more likely to leave school without formal qualifications. This relationship also persisted after control for a wide range of confounding variables.

Duncan et al (12) examined the factors that predicted escalation of substance use in 664 adolescents who were assessed at three time points. They found that academic failure predicted higher levels of substance use (including cannabis use) at the initial time period. Deteriorating academic performance over the course of the study was also associated with increasing substance use.

Ellickson et al assessed cannabis use and a range of other factors in seventh graders who were followed up five years later (26). Cannabis use predicted early school leaving among Latino students, even after controlling demographic variables, family structure, academic orientation and early dev ce. Young Latinos who were heavy cannabis users were more likely to leave school before graduating. After controlling for these confounding factors, cannabis use did not predict early school leaving for Asians, Blacks or Whites.

Garnier, Stein and Jacobs (27) conducted a long-term prospective study of early high school drop-out. They reported that early school leaving was determined by multiple factors, which included adolescent drug use. They found that, after taking account of a range of other determinants of early school leaving, there was still a significant association between drug use assessed at age 17 years and early school leaving.

Krohn, Lizotte and Perez (17) reported that the use of alcohol and other drugs during adolescence increased the risks of precocious transitions to a range of adult roles, including leaving school early. They used longitudinal data from a sample of 775 high-risk adolescents studied from age 13 to 20 years. Early substance use, measured by frequency of alcohol, cannabis and other illicit drug use, predicted early school leaving for males but not for females.

Tanner, Davies and O'Grady (28) used data from the National Longitudinal Study of Youth to examine the influence of drug use (assessed between 14 and 17 years) on social outcomes assessed between the ages of 25 to 30 years. These included educational outcomes (highest grade completed, graduation from high school, college degree) and employment variables (occupational status, unemployment). They found that (after controlling for socio-demographic background, cognitive skill and educational expectations) early drug use predicted early school drop out, failure to graduate from high school and failure to obtain a college degree in males and females. Among males early drug use was also related to lower occupational status and unemployment.

Similar findings have been reported by Brook, Balka and Whiteman (29) in a sample of 1182 Puerto Rican and African American students who were followed over a five year period. Young people who reported using cannabis once a month or more often at age 14 were more likely to leave high school before completing 12th grade, even after controlling for a range of factors assessed at age 14. Young people who used cannabis at least monthly at age 14 were also more likely to report delinquency, other drug related problems, sexual risk taking and to have more friends who exhibited deviant behaviour.

In summary, a number of longitudinal research studies have generally shown that early cannabis use is a risk factor for poor educational outcomes and, in particular, early school leaving. A causal interpretation of the link between early cannabis use and subsequent educational performance has been supported by the fact that many of these studies have statistically controlled for a wide range of variables on which cannabis users and non-users differ. In these studies early cannabis use predicts an increased risk of early school leaving and making precocious transitions to adult roles by: engaging in early sexual activity (30), unplanned pregnancy during adolescence (17, 31), unemployment (25), and leaving the family home (17).

12.4 Explaining the association between cannabis use and early school leaving

In the better longitudinal studies statistical methods have taken account of a wide range of potential explanations of the association between cannabis use and early school leaving. Perhaps the most comprehensive effort was the study by Fergusson et al (24). Their results, and those of other studies, indicate that, even though statistical control substantially reduces the associations between cannabis use and early school leaving, a significant association remains.

It is still possible that the association between cannabis use and early school leaving arises from the effects of factors that were not measured in the studies, such as neighbourhood effects (32) and genetic vulnerability (33). The difficulty in making a causal inference is not peculiar to the relationship between cannabis use and early school leaving. A number of studies, for example, have found a relationship between cigarette smoking and early school leaving which remains after extensive statistical control for confounding factors (25, 26). There is no obvious biological explanation of the relationship so it is more likely to reflect uncontrolled factors that are associated with tobacco use and early school leaving. Although a similar possibility cannot be excluded with respect to cannabis, a number of explanations have been suggested of the relationship between cannabis use and early school leaving.

12.5 Does cannabis use produce an 'amotivational' syndrome?

Daily cannabis use over months and years has been reported to impair motivation and social performance in users in Egypt and the Caribbean (34) (see chapter 6). The existence of an 'amotivational syndrome' among chronic heavy cannabis users has not been supported by the results of a number of field studies conducted in societies where heavy cannabis use is widespread, including Jamaica (35) and Costa Rica (36) (see chapter 6). Evidence reviewed in chapter 6 suggests that an amotivational syndrome is rare, if it exists (37, 38) and 'it may be more parsimonious to regard impaired motivation as a symptom of chronic cannabis intoxication' (p.277) (39). Hence, it appears unlikely that 'amotivation' explains poor school performance.

12.6 Does cannabis use produce cognitive deficits?

A third explanation is that cannabis use causes cognitive impairment, which increases the likelihood of leaving school early. The evidence (as reviewed in chapter 8) indicates that long-term cannabis use does not produce marked impairments in thinking and memory that are as easily detected as those found in long-term heavy alcohol consumers (40). Solowij has argued that daily or near cannabis use over periods of three or more years does produce subtle impairment in selective attention in adults.

These deficits are of doubtful relevance to adolescent cannabis users because few would have used cannabis intensively or long enough to produce the effects found in adults.

The adults in the studies reviewed by Solowij, for example, used cannabis daily for an average of 10 years. By contrast, in the study reported by Fergusson and Horwood (25) the 'heavy' cannabis use group included those who had smoked cannabis on at least ten occasions. There is no evidence in the scientific literature on adults that such low levels of use are associated with any lasting cognitive impairment.

This does not mean that acute cognitive impairment is irrelevant in adolescents. Rather it suggests that any cognitive impairment in cannabis using adolescents is more likely to result from the *acute* effects of cannabis use rather than the effects of long-term use. If cannabis intoxication became an everyday occurrence in the life of an adolescent, their school performance would suffer, especially if it was poor to begin with.

12.7 Does early cannabis use lead to the precocious adoption of adult roles?

Fergusson and Horwood (25) have argued that the effects of early adolescent cannabis use on later development can be attributed to the social setting in which adolescents use cannabis, namely within a group of delinquent and substance using peers. Their views are in agreement with those of Kandel et al (11) who argued that early substance use sets in train a cascade of events that increases later psychosocial risk. On Fergusson and Horwood (25)'s hypothesis, the important causal factors is that cannabis use occurs in a peer group that rejects conventional values, such as high educational achievement and social conformity, and which instead encourages non-conformist behaviour and a premature transition to adulthood.

12.8 Other effects of adolescent cannabis use

12.8.1 Occupational performance

Among young cannabis users who enter the work-force the continued use of cannabis and other illicit drugs in young adulthood might impair job performance for the same reasons that it may impair school performance, namely, that chronic intoxication impairs cognitive and psychomotor performance. There is some support for this expectation in that cannabis users report higher rates of unemployment than nonusers (e.g. (41, 42) but this comparison is confounded by the different educational qualifications of the two groups.

Mensch and Kandel (9) examined cross-sectional relationships between alcohol, tobacco and cannabis use and performance in a range of occupations in a nationally representative sample of Americans. Apart from tobacco use there were only modest associations between cannabis use and occupation. There were very weak negative correlations between job satisfaction and tobacco smoking and cannabis use. Workers in occupations that were lacking in 'complexity, intellectual flexibility and variety' were more likely to smoke cannabis at work, perhaps because heavier cannabis users seek or are forced to accept less challenging jobs. Cannabis use and tobacco smoking were associated with 'lack of conformity or attachment to social institutions, such as having dropped out of school, having participated in delinquent activities, or not being married' (p 181).

Longitudinal studies have suggested that there is a relationship between adolescent cannabis use and job instability among young adults that is not explained by differences in education and other characteristics which precede cannabis use (e.g. (11). Newcomb and Bentler (23) examined the relationships between adolescent drug use and income, job instability, job satisfaction, and resort to public assistance in young adulthood, while controlling for differences between users and nonusers in social conformity, academic potential and income in adolescence. Their findings supported those of Kandel who found that adolescent drug users had a larger number of changes of job than nondrug users. Newcomb and Bentler conjectured that this reflects impaired work performance, or a failure of illicit drug users to develop responsible employment behaviour such as conscientiousness, thoroughness, and reliability.

Fergusson and Horwood (25) included unemployment for 3 months or more as one of their early outcomes in the follow up of their cohort at age 18 years. There was a relationship between how often cannabis had been used by age 16 and being unemployed for 3 months or longer. The rate of unemployment among those who had never used cannabis was 9.5% compared to rates of 18.9% and 37.5% among those who had used 1–9 times and 10 or more times respectively. After adjusting for covariates, the strength of the association was reduced but still significant (namely, 10.5%, 17.3% and 26.9% respectively). After adjustment for peer affiliations, the relationship was no longer statistically significant (12.2%, 13.4% and 14.6% respectively).

One longitudinal study (43) found more mixed evidence of an association between adolescent cannabis use in a sample of 785 young people followed from late high school in 1971–1973 until early adulthood in 1981. They found that adolescent cannabis use was weakly correlated with poor job performance, low job satisfaction or adverse job terminations. The correlations between cannabis use and these indices of job performance were 0.07, 0.07, and 0.17 respectively. These weak relationships between adolescent drug use and adult occupational performance were explained as the result of cannabis use persisting into adult life where it was associated with poor job performance, low job satisfaction, and adverse job termination.

12.8.2 Interpersonal relationships

There are good reasons for suspecting that cannabis use may adversely affect interpersonal relationships. Heavy adolescent drug use may produce a developmental lag, entrenching adolescent styles of thinking and coping which impair the ability to form adult relationships (1). There are also strong correlations between drug use and precocious sexual activity, and early marriage which in turn predicts a high rate of relationship failure (23).

Cross-sectional studies of drug use in young adults have indicated that a high degree of involvement with cannabis predicts a reduced probability of marriage, an increased rate of cohabiting, an increased risk of divorce or failed de facto relationships, and a higher rate of unplanned pregnancy and pregnancy termination (41, 42). These findings have been confirmed in analyses of the longitudinal data from a cohort of young adults (11).

Newcomb and Bentler (23) found similar relationships between drug use and early marriage in their analysis of the data from young adults in Los Angeles. Drug use in adolescence predicted an increased rate of early family formation in late adolescence and

of divorce in early adulthood. They interpreted this as evidence that: 'early drug involvement leads to early marriage and having children which then results in divorce' (p. 97). Newcomb and Bentler argued that this finding provided evidence for their theory of 'precocious development', according to which drug use accelerates development and drug users 'bypass or circumvent the typical maturational sequence of school, work and marriage and become engaged in adult roles of jobs and family prematurely without the necessary growth and development to enhance success with these roles ... [thereby developing] a pseudomaturity that ill prepares them for the real difficulties of adult life' (pp. 35–36).

12.8.3 Mental health

A number of cross-sectional studies of the association between cannabis use and poor mental health in young adults have produced mixed findings. The US National Longitudinal Alcohol Epidemiologic Survey (NLAES), a nationally representative survey of US adults (44) found that persons with DSM-IV major depression in the past 12 months were 6.4 times more likely to have DSM-IV cannabis abuse or dependence than those without major depression (6% vs. 1% respectively)(44).

A study of cannabis use and depressive symptoms did *not* find that frequency of cannabis use was associated with depression in young adult males (45). A weak association observed between early initiation of cannabis use and depression was not significant after controlling for educational attainment, marital status, and alcohol and tobacco use (45).

A study of male army draftees using cannabis but no other illicit drugs found that more problematic cannabis users had a higher rate of DSM-III-R psychiatric disorders and higher scores on the Beck Depression Inventory (BDI) (46). A study of adolescents cannabis users found that frequent users of cannabis had higher levels of depression on the Brief Symptom Inventory than abstainers or recreational users (47). 'Heavy' users were defined as those using cannabis at least 40 times *and at least one other illicit drug*.

Degenhardt et al. (48) examined relationships between cannabis use and mental health using data from the Australian National Survey of Mental Health and Well-Being (NSMHWB), a survey of a nationally representative sample of 10,641 Australian adults aged 18 years and over. There was an association between cannabis use in the past 12 months and affective and anxiety disorders. Among those with cannabis dependence, 14% had an affective disorder and 17% had an anxiety disorder, compared with rates of 6% and 5% respectively in non-users. Heavier cannabis users also reported greater levels of psychological distress (as measured by Kessler's Psychological Distress scale).

The results of a number of longitudinal studies have provided more mixed evidence of the relationship between cannabis use and mental health. Kandel (41) found a cross-sectional study found an association between level of cannabis use and dissatisfaction with life, having consulted a mental health professional, and having been hospitalised for a psychiatric disorder (41). Longitudinal analyses of this cohort, however, found only weak associations between adolescent drug use and adult mental health; the strongest relationship was between cigarette smoking in adolescence and symptoms of depression in adulthood (11).

The cross sectional adult data in Newcomb and Bentler's (23) study also showed strong relationships between adolescent drug use and emotional distress, psychoticism and lack of a purpose in life. Emotional distress in adolescence predicted emotional distress in young adulthood but there were no relationships between adolescent drug use and adult emotional distress, depression and lack of a sense of purpose in life. Adolescent drug use predicted psychotic symptoms in young adulthood, and hard drug use in adolescence predicted increased suicidal ideation in young adulthood, after controlling for general drug use and earlier emotional distress. Newcomb and Bentler interpreted this as evidence that adolescent drug use 'interferes with organised cognitive functioning and increases thought disorganisation into young adulthood' (p 180).

Fergusson and Horwood (25) found a dose response relationship between frequency of cannabis use by age 16 and the likelihood of meeting DSM-IV criteria for an anxiety and depressive disorder and reporting a suicide attempt. These relationships were no longer statistically significant, however, after controlling for confounding factors.

Brook, Cohen and Brook (3) reported a longitudinal study of the relationship between alcohol, tobacco and cannabis use and mental health among 975 adolescents followed from age 13.7 years until 22.1 years in New York state. They found that early cannabis use predicted later antisocial behaviour after controlling for earlier antisocial behaviour. It did not predict an increased risk of anxiety and affective disorders. The strongest relationships between adolescent drug use and adult mental disorders were between cigarette smoking, illicit drug use (other than cannabis) and depression.

McGee, Williams, Poulton and Moffit (49) reported a longitudinal study of the relationships between cannabis use and mental health in a Dunedin, New Zealand, birth cohort between the ages of 15 and 21 years. They found that rates of cannabis use were higher among young people with mental disorders at 15, 18 and 21 years and that cannabis use was predicted by social disadvantage in childhood and low parental attachment. Cannabis use at age 15 did not predict mental health problems at age 18 but having mental health problems at age 15 (primarily alcohol dependence and conduct disorder) modestly predicted cannabis use at age 18. Cannabis use at age 18 also predicted alcohol dependence and conduct disorders at age 21. McGee et al argued that the lack of a relationship between cannabis use and anxiety and affective disorders suggests that cannabis use is not a form of 'self-medication in anxious and depressed individuals but rather reflects a 'willingness to contravene the law'.

12.8.4 Suicide

A small number of studies have examined the relationship between cannabis use and suicide among adolescents (see Hillman et al (50) for a review). Several have found an association but it remains unclear whether it is explained by other factors. An analysis of cross-sectional data from the US National Comorbidity Survey found an association between self-reported suicide attempts and the dependence on a number of drugs, including alcohol, sedatives, stimulants, cannabis, and inhalants (51). The risk for cannabis dependence was still significant after adjusting for socio-demographic factors and the presence of other psychiatric disorders, such as depression and alcohol dependence (odds ratio of 2.4).

Beautrais, Joyce and Mulder (52) reported a case-control study of the role of cannabis and other drug use in serious suicide attempts that resulted in hospitalisation. They compared rates of cannabis use among 302 consecutive hospital cases treated for serious suicide attempts with that in a random sample of 1,028 people in the community. They found that 16% of the suicide attempters had a cannabis use disorder (cannabis abuse or dependence) compared with 2% of the controls. Controlling for social disadvantage and having a diagnosis of depression or alcohol dependence substantially reduced the association but did not eliminate the association (reducing it from an odds ratio of 10 to 2).

The evidence from a small number of prospective studies is also mixed. Fergusson and Horwood (25) also found a dose response relationship between frequency of cannabis use by age 16 and the likelihood of reporting a suicide attempt, but it did not remain statistically significant after controlling for confounding factors. Patton et al (53) reported a longitudinal study on suicide attempts and self-harm in a cohort of 2066 Victorian secondary school students followed from age 15 to 16 to age 21. They found that cannabis was associated with self-harmful behaviour among females but not males, after controlling for depression and alcohol use.

Andreasen and Allebeck (54) reported an association between cannabis use and suicide deaths in their follow up of 50,465 conscripts. They found a fourfold increased risk of suicide among heavy cannabis users. A more detailed analysis of predictors of suicide in this cohort reported by Allebeck and Algulander (55) found that inpatient psychiatric hospitalisation by age 18 was the strongest predictor of suicide risk (OR = 11.3). Use of 'narcotics' (which includes cannabis) did not predict suicide independently of a psychiatric diagnosis (OR =1.3) but a diagnosis of alcohol dependence (OR = 4.3) and drug dependence (OR = 3.6) did.

12.8.5 Delinquency and crime

Cannabis and other illicit drug use are related to social nonconformity (23, 56, 57) so it is unsurprising that there is a relationship between the extent of cannabis use and lifetime delinquency among adult drug users (41, 42), having been convicted of an offence, and having had a motor vehicle accident while intoxicated (41). Surveys of drug use in young people in the juvenile justice system also find high rates of regular cannabis use and a relationship between level of cannabis use and frequency of offending (58, 59).

Longitudinal studies reveal an interesting pattern of relationships between cannabis use and crime. Johnston et al. (60) analysed the relationship between drug use and delinquency in two waves of interviews of adolescent males. In their cross-sectional data, rates of delinquent activity increased steadily with increasing rates of drug use. However, analyses of changes in drug use and crime over time indicated that heavy drug users groups had much higher rates of delinquent acts *before* using drugs. The onset of illicit drug use (including cannabis) had little effect on delinquent acts, except among those who used heroin, whose rates of delinquency increased.

Newcomb and Bentler (23) reported a positive relationship between drug use and criminal involvement in adolescence, but found more mixed results in the relationship between adolescent drug use and criminal activity in young adulthood. Adolescent drug use predicted *drug* crime involvement in young adulthood; but after controlling for other

variables, it was *negatively* correlated with violent crime, and general criminal activities in young adulthood. Newcomb and Bentler argued that these negative correlations indicated that the correlation between different forms of delinquency in adolescence decreases with age, as criminal activities become differentiated into drug-related and non drug-related offences.

White (61) reported a follow up study of the relationship between cannabis use and delinquency in 1892 New Jersey youth followed from age 12 to age 18. He found modest correlations between cannabis use and delinquency at age 15 and age 18 and evidence that there were separate groups of adolescents who either engaged in cannabis use or in delinquent acts. These groups were distinguished by which of these two behaviours was most common among their immediate peers.

Fergusson and Horwood (25) included four measures of delinquency in their analysis of the consequences of adolescent cannabis use. These were: three or more violent offences, three or more property offences, arrested by police, and convicted of an offence in court by age 16. There was a dose-response relationship between each of these outcomes and frequency of cannabis use by age 16. This persisted after adjustment for covariates, suggesting that it was not wholly explained by the characteristics of adolescents who become regular cannabis users by age 16. It also persisted after adjustment for drug use and criminal behaviour in the users peer group, indicating that it was not explained by affiliating with delinquent and drug using peers.

Brook et al (29)'s longitudinal study of 695 African-American and 637 Puerto Rican youth in New York City also assessed self-reported violence towards others. They found that early cannabis use predicted a doubling of the risk of self-reported violence towards others, after adjusting for other covariates (but not for a history of delinquency and violence prior to using cannabis).

Arsenault, Moffit, Caspi and Taylor (62) reported a longitudinal study of the relationships between mental disorders and violence in a cohort of 961 youth studied from birth to age 21 in Dunedin, New Zealand. They assessed psychiatric disorders, including alcohol and cannabis dependence and asked about alcohol and other drug use prior to self-reported violence. Violence was assessed using self-report and police records of convictions for violence. They found that 7.6% of the sample reported engaging in violence in the past year and 4% had been convicted of violent offences. They found strong associations between self-reported and officially recorded violence and alcohol dependence, cannabis dependence and schizophrenia. Controlling for a history of conduct disorder in childhood (prior to using cannabis) substantially reduced the association between cannabis dependence and violence. The authors argued that the relationship reflected the heavy involvement of cannabis dependent and conduct disordered adolescents in the drug market where violence was used to resolve disputes.

12.9 Summary

Cross-sectional and prospective research indicates that young people who use cannabis are at increased risk of adverse psychosocial outcomes including criminal behaviour, poor mental health, impaired educational achievement and reduced life opportunities. The longitudinal studies suggest that a large part of these associations arise because the factors that predispose young people to use cannabis overlap with the factors that predict these outcomes. In ordinary language, the young people who are most likely to use cannabis in early adolescence are the same young people who were at greatest risk of using other drugs, engaging in delinquency, having poorer mental health, attempting suicide, and doing poorly at school *before they began to use cannabis*.

However, not all of the relationships between cannabis use and these poorer social outcomes can be wholly explained this way. There is evidence that early cannabis use further impairs the school performance of adolescents whose performance was poor before they began to use cannabis. It may also predict involvement in criminal behaviour after controlling for a history of conduct disorder, perhaps by exacerbating pre-existing anti-social behaviour. It may possibly increase the risk of suicide but this remains to be clarified by better designed studies.

Plausible mechanisms that may explain these associations have been suggested by Fergusson and Horwood (25), namely, that adolescents who are socially disadvantaged and have conduct problems as children are more likely to become early cannabis users, and early cannabis use increases the chances of an unconventional lifestyle. The latter occurs as a result of affiliating with delinquent and substance using peers and disengaging from conventional social roles such as completing education and obtaining a job. The acute effects of cannabis intoxication may also play a role by encouraging impulsive behaviour and impairing perceptions of risk among the minority of students who are daily cannabis users.

12.10 References

1. Baumrind, D. & Moselle, K. (1985) A developmental perspective on adolescent drug abuse, *Advances in Alcohol and Substance Abuse*, 5, 41–67.
2. Resnick, M. D., Bearman, P. S., Blum, R. W., Bauman, K. E., Harris, K. M., Jones, J., Tabor, J., Beuhring, T., Sieving, R. E., Shew, M., Ireland, M., Bearinger, L. H., & Udry, J. R. (1997) Protecting adolescents from harm: Findings from the National Longitudinal Study on Adolescent Health, *Journal of the American Medical Association*, 278, 823–832.
3. Brook, J. S., Brook, D. W., De La Rosa, M., Duque, L. F., Rodriguez, E., Montoya, I. D. & Whiteman, M. (1998) Pathways to marijuana use among adolescents: Cultural/ecological, family, peer, and personality influences, *Journal of the American Academy of Child & Adolescent Psychiatry*, 37, 759–766.
4. Jones, S. P. & Heaven, P. C. L. (1998) Psychosocial correlates of adolescent drug-taking behaviour, *Journal of Adolescence*, 21, 127–134.

5. Lifrak, P. D., McKay, J. R., Rostain, R., Alterman, A. I. & O'Brien, C. P. (1997) Relationship of perceived competencies, perceived social support, and gender to substance use in young adolescents, *Journal of the American Academy of Child and Adolescent Psychiatry*, 36, 933–940.

6. Novins, D. K. & Mitchell, C. M. (1998) Factors associated with marijuana use among American Indian adolescents, *Addiction*, 93, 1693–1702.

7. Lynskey, M., White, V., Hill, D., Letcher, T. & Hall, W. (1999) Prevalence of illicit drug use among youth: Results from the Australian school students' alcohol and drugs survey, *Australian and New Zealand Journal of Public Health*, 23, 519–524.

8. Fergusson, D. M., Lynskey, M. T. & Horwood, L. J. (1995) Truancy in adolescence, *New Zealand Journal of Educational Studies*, 30, 25–38.

9. Mensch, B. S. & Kandel, D. B. (1988) Dropping out of high school and drug involvement, *Sociology of Education*, 61, 95–113.

10. Brewin, C. R., Andrews, B. & Gotlib, I. H. (1993) Psychopathology and early experience: A reappraisal of retrospective reports, *Psychological Bulletin*, 113, 82–98.

11. Kandel, D. B., Davies, M., Karus, D. & Yamaguchi, K. (1986) The consequences in young adulthood of adolescent drug involvement. An overview, *Archives of General Psychiatry*, 43, 746–54.

12. Duncan, S. C., Duncan, T. E., Biglan, A. & Ary, D. (1998) Contributions of the social context to the development of adolescent substance use: A multivariate latent growth modeling approach, *Drug and Alcohol Dependence*, 50, 57–71.

13. Hundelby, J. D. & Mercer, G. W. (1987) Family and friends as social environments and their relationship to young adolescents' use of alcohol, tobacco, and marijuana, *Journal of Clinical Psychology*, 44, 125–134.

14. Kelly, D. H. & Balch, R. W. (1971) Social origins and school failure: A reexamination of Cohen's theory of working-class delinquency, *Pacific Social Review*, 14, 413–439.

15. Hawkins, J. D., Catalano, R. F. & Miller, J. Y. (1992) Risk and protective factors for alcohol and other drug problems in adolescence and early adulthood: Implications for substance abuse prevention, *Psychological Bulletin*, 112, 64–105.

16. Jessor, R. (1976) Predicting time of onset of marijuana use: A developmental study of high school youth, *Journal of Consulting and Clinical Psychology*, 44, 125–134.

17. Krohn, M. D., Lizotte, A. J. & Perez, C. M. (1997) The interrelationship between substance use and precocious transitions to adult statuses, *Journal of Health and Social Behavior*, 38, 87–103.

18. Jessor, R. & Jessor, S. L. (1977) *Problem Behavior and Psychosocial Development: A Longitudinal Study of Youth* (New York, Academic Press).

19. Donovan, J. E. & Jessor, R. (1985) Structure of problem behavior in adolescence and young adulthood, *Journal of Consulting and Clinical Psychology*, 53, 890–904.

20. Newcomb, M. D. & Bentler, P. M. (1989) Substance use and abuse among children and teenagers, *American Psychologist*, 44, 242–248.

21. Kandel, D. B. (1980) Drug and drinking behavior amo youth, *Annual Review of Sociology*, 6, 235–285.

22. Rutter, M. (1988) *Longitudinal data in the study of causal processes: Some uses and some pitfalls* (Cambridge, Cambridge University Press).

23. Newcomb, M. D. & Bentler, P. (1988) *Consequences of adolescent drug use* (California, Sage Publications).

24. Fergusson, D. M., Lynskey, M. T. & Horwood, L. J. (1996) The short-term consequences of early onset cannabis use, *Journal of Abnormal Child Psychology*, 24, 499–512.

25. Fergusson, D. M. & Horwood, L. J. (1997) Early onset cannabis use and psychosocial adjustment in young adults, *Addiction*, 92, 279–296.

26. Ellickson, P., Bui, K., Bell, R. & McGuigan, K. A. (1998) Does early drug use increase the risk of dropping out of high school?, *Journal of Drug Issues*, 28, 357–380.

27. Garnier, H. E., Stem, J. A. & Jacobs, J. K. (1997) The process of dropping out of high school: A 19-year perspective, *American Educational Research Journal*, 34, 395–419.

28. Tanner, J., Davies, S. & O'Grady, B. (1999) Whatever happened to yesterday's rebels? Longitudinal effects of youth delinquency on education and employment, *Social Problems*, 46, 250–274.

29. Brook, J. S., Balka, E. B. & Whiteman, M. (1999) The risks for late adolescence of early adolescent marijuana use, *American Journal of Public Health*, 89, 1549–1554.

30. Rosenbaum, E. & Kandel, D. B. (1990) Early onset of adole nt sexual behavior and drug involvement, *Journal of Marriage and the Family*, 52, 783–798.

31. Mensch, B. & Kandel, D. B. (1992) Drug use as a risk factor for premarital teen pregnancy and abortion in a national sample of young white women, *Demography*, 29, 409–429.

32. Ensminger, M. E., Lamkin, R. P. & Jacobson, N. (1996) School leaving: A longitudinal perspective including neighborhood effects, *Child Development*, 67, 2400–2416.

33. Plomin, R. & Craig, I. (1997) Human behavioural genetics of cognitive abilities and disabilities, *Bioessays*, 19, 1117–1124.

34. Brill, H. & Nahas, G. (1984) Cannabis intoxication and mental illness, in: Nahas, G. (Ed.) *Marihuana in Science and Medicine*, pp. 263–305 (New York, Raven Press).

35. Rubin, V. & Comitas, L. (1975) *Ganja in Jamaica: A Medical Anthropological Study of Chronic Marihuana Use* (The Hague, Mouton).

36. Carter, W., Coggins, W. & Doughty, P. (1980) *Cannabis in Costa Rica: A study of chronic marihuana use* (Philadelphia, Institute for the Study of Human Issues).

37. Halikas, J., Weller, R., Morse, C. & Shapiro, T. (1982) Incidence and characteristics of motivational syndrome, including associated findings, among chronic marjiuana users, in: National Institute on Drug Abuse (Ed.) *Marijuana and Youth: Clinical Observations on Motivation and Learning*, pp. 11–26 (Rockville, MD, National Institute on Drug Abuse).

38. Mendelson, J., Rossi, A. & Meyer, R. (1974) *The Use of Marihuana: A Psychological and Physiological Inquiry* (New York, Plenum Press).

39. Channabasavanna, S. M., Paes, M. & Hall, W. (1999) Mental and behavioural disorder due to cannabis, in: Kalant, H., Corrigal, W., Hall, W. & Smart, R. (Eds.) *The health effects of cannabis*, pp. 276–290 (Canada, Centre for Addiction and Mental Health).

40. Solowij, N. (1998) *Cannabis and cognitive functioning*, (Cambridge, Cambridge University Press.

41. Kandel, D. B. (1984) Marijuana users in young adulthood, *Archives of General Psychiatry*, 41, 200–209.

42. Robins, L., Darvish, H. S. & Murphy, G. E. (1970) The long-term outcome for adolescent drug users: A follow-up study of 76 users and 146 nonusers., in: Zubin, J. & Freedman, A. M. (Eds.) *The Psychopathology of Adolescence*, pp. 159–180 (New York, Grune and Stratton).

43. Stein, J. A., Smith, G. M., Guy, S. M. & Bentler, P. M. (1993) Consequences of adolescent drug use on young adult job behavior and job satisfaction, *Journal of Applied Psychology*, 78, 463–474.

44. Grant, B. F. (1995) Comorbidity between DSM-IV drug use disorders and major depression: Results of a national survey of adults, *Journal of Substance Abuse*, 7, 481–97.

45. Green, B. E. & Ritter, C. (2000) Marijuana use and depression, *Journal of Health and Social Behavior*, 41, 40–49.

46. Troisi, A., Pasini, A., Saracco, M. & Spalletta, G. (1998) Psychiatric symptoms in male cannabis users not using other illicit drugs, *Addiction*, 93, 487–492.

47. Milich, R., Lynam, D., Zimmerman, R., Logan, T., Martin, C., Leukefield, C., Portis, C., Miller, J. & Clayton, R. (2000) Differences in young adult psychopathology among drug abstainers, experimenters, and frequent users, *Journal of Substance Abuse*, 11, 69–88.

48. Degenhardt, L., Hall, W. & Lynskey, M. (2001) The relationship between cannabis use, depression and anxiety among Australia adults: Findings from the National Survey of Mental Health and Well-Being, *Social Psychiatry and Psychiatric Epidemiology*, in press.

49. McGee, R., Williams, S., Poulton, R. & Moffitt, T. (2000) A longitudinal study of cannabis use and mental health from adolescence to early adulthood, *Addiction*, 95, 491–503.

50. Hillman, S. D., Silburn, S. R., Green, A. & Zubrick, S. R. (2000) Youth Suicide in Western Australia Involving Cannabis and Other Drugs (Perth, Western Australian Drug Abuse Strategy Office).

51. Borges, G., Walters, E. E. & Kessler, R. C. (2000) Associations of substance use, abuse and dependence with subsequent suicidal behavior, *American Journal of Epidemiology*, 151, 781–789.

52. Beautrais, A. L., Joyce, P. R. & Mulder, R. T. (1999) Cannabis abuse and serious suicide attempts, *Addiction*, 94, 1155–1164.

53. Patton, G. C., Harris, J. B., Schwartz, M. & Bowes, G. (1997) Adolescent suicidal behaviors: A population-based study of risk, *Psychological Medicine*, 27, 715–724.

54. Andreasson, S. & Allebeck, P. (1990) Cannabis and mortality among young men: A longitudinal study of Swedish conscripts, *Scandinavian Journal of Social Medicine*, 18, 9–15.

55. Allebeck, P. & Allgulaner, C. (1990) Suicide among young men: psychiatric illness, deviant behaviour and substance abuse, *Acta Psychiatrica Scandinavica*, 86, 565–570.

56. Donovan, J. E. & Jessor, R. (1983) Problem drinking and the dimension of involvement with drugs: A Guttman Scalogram analysis of adolescent drug use, *American Journal of Public Health*, 73, 543–552.

57. Polich, J., Ellickson, P., Reuter, P. & Kahan, J. (1984) *Strategies for Controlling Adolescent Drug Use* (Santa Monica, CA, The RAND Corporation).

58. Salmelainen, P. (1995) The correlates of offending frequency: A study of juvenile theft offenders in detention (Sydney, New South Wales Bureau of Crime Statistics and Research).

59. Trimboli, L. & C., C. (1998) Cannabis and crime: treatment programs for adolescent cannabis use, *Crime and Justice Bulletin*, 41, 1–16.

60. Johnston, L. D., O'Maley, P. M. & Eveland, L. K. (1978) *Drugs and delinquency: A search for causal connections* (New York, John Wiley and Sons).

61. White, H. R. (1991) Marijuana use and delinquency: A test of the 'independent cause' hypothesis, *Journal of Drug Issues*, 21, 231–256.

62. Arseneault, L., Moffitt, T. E., Caspi, A., Taylor, P. J. & Silva, P. A. (2000) Mental disorders and violence in total birth cohort: Results from the Dunedin study, *Archives of General Psychiatry*, 57, 979–986.

13 Therapeutic uses of cannabis

Cannabis has had a long history of medical and therapeutic use in India and the Middle East (1–3), where it was used to treat pain, convulsions, spasm, nausea and to induce sleep. Cannabis was introduced to Britain in the mid-nineteenth century by O'Shaughnessy (4) who had used the drug while an Army surgeon in India (2, 3). He recommended its use for the relief of pain, muscle spasms, and convulsions occurring in tetanus, rabies, rheumatism and epilepsy (3). Cannabis was widely used as an analgesic, anticonvulsant and antispasmodic in Britain and the USA during the latter half of the nineteenth and the early part of the twentieth centuries.

Medical uses of cannabis declined after the turn of the twentieth century because natural cannabis preparations varied in potency and effectiveness. Cannabis was largely supplanted by pharmaceutically pure drugs, such as the opiates, aspirin, chloral hydrate, and the barbiturates, all of which could be given in standard doses to produce more predictable effects (2, 3). Many of these drugs could also be injected to provide rapid relief of symptoms whereas cannabis extracts had to be given orally (5). After the introduction of international drug control agreements in the early part of the 20th century, the medical use of cannabis preparations was discouraged by laws that treated cannabis as a 'narcotic' drug. Cannabis disappeared from the American pharmacopoeia in the early 1940s after the passage of the Marijuana Tax Act (1), although it continued to be used in Australia into the 1960s (6).

The isolation of THC in 1964 (7) occurred shortly before cannabis became widely used as a recreational drug by American youth. Its illegality and recreational use hindered pharmaceutical research, so the rediscovery of its therapeutic uses was serendipitous. Its value as an anti-emetic agent in treating nausea caused by cancer chemotherapy was discovered by young adults who had used cannabis recreationally while undergoing chemotherapy for leukemia (8).

From the mid 1970s until the early 1980s clinical research was undertaken on the therapeutic value of cannabis and cannabinoids. On the whole, however, this research was very thin and uneven, and, consequently, many of the claims for the therapeutic efficacy of cannabinoids rely on the reports of individuals who have derived medical benefit from its use (e.g. (1, 9). When cannabinoids and cannabis are advocated for medical uses it is primarily for relief of symptoms rather than to cure any underlying disease. The conditions for which cannabis is most commonly advocated are for symptomatic relief of nausea, vomiting, appetite loss, and chronic pain (10).

13.1 Cannabinoids as anti-emetic agents

Severe nausea and vomiting may prompt patients to discontinue life-saving chemotherapy and radiotherapy for cancer (10). Anti-emetic drugs (e.g. the phenothiazines) are effective in controlling nausea and vomiting in cancer patients

undergoing chemotherapy but a substantial minority of patients do not benefit from these drugs. The incomplete success of existing treatments prompted oncologists in the late 1970s and early 1980s to study the anti-emetic properties of cannabinoids (10).

One of the earliest trials studied the effects of THC on nausea and vomiting (11) in 22 patients with a variety of cancers, 20 of whose nausea and vomiting had proven resistant to existing anti-emetic drugs. Patients were randomly assigned to receive oral THC and placebo in one of four different orders. Outcome was assessed by patients' self-reports of nausea and vomiting after THC and placebo into three categories: complete response if there was vomiting after placebo but not after THC; partial response if there was a greater than 50% reduction in nausea and vomiting after THC compared to placebo; and no response if there was a less than 50% reduction in nausea and vomiting.

There were 29 trials, 14 of placebo and 15 of THC. There was no anti-emetic response in any of the 14 placebo trials. There were 5 successes, 7 partial responses, and 3 no responses in the 15 THC trials. Most patients (13/16) reported a 'high' after receiving THC, an experience which was correlated with the anti-emetic effect. The most common side-effect was sleepiness. Two patients experienced visual illusions and hallucinations and depression lasting several hours. Several patients reported that smoking cannabis had the same anti-emetic effects as oral THC.

A trial by Chang et al (12) largely supported the findings of Sallan et al (11). In this study 15 patients with osteogenic sarcoma receiving monthly high dose methotrexate therapy served as their own controls. They were assigned to receive three THC and three placebo trials in randomised order during six treatment sessions. If the patients vomited, the remaining doses of either THC or placebo were administered by smoking a cigarette. The effect of THC and placebo on vomiting and retching episodes were assessed by nursing staff who graded response into three categories: excellent (greater then 80% reduction after THC by comparison with placebo in each of these endpoints); fair (greater than 30% and less than 80% reduction), and no response (less than 30% reduction).

Eight patients had an excellent response, 6 a fair response, and one had no response. On all outcomes THC produced a statistically greater reduction in nausea and vomiting than placebo. There was a relationship between blood levels of THC and reports of nausea and feeling 'high'. Higher THC blood levels were achieved when cannabis was smoked than when THC was taken orally. There were few side effects, sedation being the most common (12/15 patients). Four patients experienced 5 dysphoric reactions in the course of 281 THC drug doses (2%). None of these lasted more than 30 minutes, and all were successfully managed by reassurance.

Since these early studies, a number of controlled clinical trials have compared the effectiveness of THC with a placebo or another anti-emetic drug (see (13–15) for reviews). Studies comparing oral THC with existing anti-emetic agents have had less consistent results than comparisons with placebo but the results have generally indicated that THC is as effective as the anti-emetic drug prochlorperazine (13, 15). The equivalence of THC and prochlorperazine was reported in one of the largest and best conducted studies (16).

Although cannabinoids showed *some* anti-emetic efficacy by comparison with prochlorperazine they typically failed to stop nausea in two thirds of patients. In one controlled study, THC produced complete control of emesis in only 13% of cases as against 47% who received metoclopramide. It achieved 'major control' of vomiting (two or fewer episodes) in 27% as against 73% in the comparator (10). The same has been true of the anti-emetic effects of nabilone and levonantradol (10).

Since these trials were conducted much more effective anti-emetic drugs than prochlorperazine have become available (10). These newer agents have dramatically reduced nausea and vomiting. The selective serotonin type 3 receptor agonists, such as ondansetron, have achieved complete control over nausea induced by cisplatin in 75% of cases and up to 90% for less emetogenic chemotherapy (10). Side effects include headache and constipation but these are generally well tolerated. These drugs have reduced the demand for THC as an anti-emetic drug.

13.2 Cannabinoids and HIV-related wasting

Cannabis has also been used therapeutically as an anti-nausea agent, an appetite stimulant and an analgesic in patients with HIV-related wasting (10). HIV/AIDS patients often experience nausea and weight loss, either while receiving antiviral drugs to suppress HIV, or as a direct effect of the AIDS-related diseases. Wasting syndrome in HIV/AIDS has been defined by the US Centers for Disease Control and Prevention as 'the involuntary loss of more than 10% of baseline average body weight in the presence of diarrhoea or fever of more that 30 days that is not attributable to other disease processes' (10).

In animal studies cannabinoids have been shown to act on brain centres that control appetite (17), supporting reports of benefits in patients with AIDS. Few controlled trials have been published on the effectiveness of cannabis or cannabinoids for this purpose. Oral THC has been shown to be of benefit in short–term trials (18, 19) and it has been registered for this purpose in the US. Some patients do not like dronabinol because of its psychoactive side effects, the difficulty in controlling their dose, the delayed onset of effects, and the prolonged effects when it is taken orally (10). There are anecdotal reports that smoked cannabis is effective for the treatment of HIV/AIDS-associated anorexia and weight loss (1, 20). There have not been any controlled studies on smoked cannabis but one is underway in California.

A major concern with HIV-infected patients smoking cannabis for medical purposes is that it might have immunosuppressive effects or infectious organisms in cannabis plant material may produce opportunistic infections. Recent epidemiological evidence does allay this concern to a degree in that a large prospective cohort study of HIV/AIDS in homosexual and bisexual men recently failed to find any relationship between cannabis use, or any other psychoactive drug use, and the development of clinical AIDS (21). Nonetheless, the immunosuppressive effects of THC and smoked cannabis need to be investigated in any research on the therapeutic uses of cannabinoids in the treatment of HIV-related wasting.

13.3 Cannabinoids as anti-glaucoma agents

Glaucoma is the leading cause of blindness in the United States, causing 300,000 new cases each year (22). It is caused by a gradual increase in pressure within the eye, 'intraocular pressure' (IOP). If untreated, IOP may damage the optic nerve, leading to blindness. Its incidence increases over the age of 35, especially among individuals who are short-sighted. Many drugs that reduce IOP have unwanted side-effects and patients may become tolerant to their therapeutic effects.

The effects of cannabis on IOP were discovered serendipitously by researchers and patients in the early and middle 1970s. Hepler and his colleagues (23–25) demonstrated that cannabis and oral THC substantially reduced IOP in normal volunteers and in patients with glaucoma (23–25). Subsequent research indicated that THC produced this effect (22).

Although there have been a number of case reports of the successful use of cannabis in the management of glaucoma (e.g. (1, 9), there have not been any controlled clinical studies of its effectiveness and safety. Although THC reduces IOP acutely there are doubts about its long-term effectiveness because tolerance develops to this effect (26). The US Institute of Medicine concluded that there was no evidence to support the use of THC in glaucoma (10). It argued that the effects of cannabis and THC on IOP are too short-lived, and the high oral doses that were required produced side effects that precluded its long-term use (10). The harmful effects of chronic cannabis smoking, it argued, outweighed its modest medical benefits. A cannabinoid drug with longer lasting effects on IOP and fewer psychoactive effects than THC could be of greater use (10).

13.4 Cannabinoids and epilepsy

Animal studies have provided some support for the historical use of cannabis preparations to control seizures in epilepsy, tetanus and rabies (3). Cannabidiol (CBD) appears to be a potent anticonvulsant in animals (27–29). There is very limited evidence on the therapeutic effects of cannabinoids in humans with epilepsy. There are a small number of case studies of individuals with epilepsy in which the use of cannabis appeared to enhance the anticonvulsant effects of more traditional anticonvulsant medication (e.g. (1, 30).

There is one randomised placebo controlled study of CBD in 15 patients whose epilepsy was not controlled by conventional anti-convulsants. Four of the eight patients who received CBD in addition to their usual anti-convulsant drugs were free of seizures throughout the study period, and three were improved. By contrast, only 1 out of 7 patients in the placebo condition showed any clinical improvement (31). Despite this suggestive evidence of efficacy there has been no further research on the anticonvulsant properties of CBD (3). This may be because more effective anticonvulsant drugs exist and pharmaceutical companies have no interest in marketing a naturally occurring substance that cannot be patented.

13.5 Cannabinoids and muscle spasticity

Muscle spasticity is the increased resistance to passive stretch of muscles. Involuntary contractions may occur which can be painful and debilitating. About 90% of MS patients eventually develop muscle spasticity, in the form of stiffness, spasms, cramps, aches or pain. Recent animal research has found that THC reduces both tremor and spasticity among diseased mice, suggesting that the cannabinoid system may be involved in control of these functions (32). A survey of 112 MS patients (33) supported the use of cannabis for MS, and some open studies have suggested it is of benefit (34–36).

Clinical studies have not supported the anecdotal evidence, but this may be due to the studies' limitations (10). The survey results suggest that it would be useful to investigate the potential therapeutic value of cannabinoids in relieving symptoms associated with MS (37). The regular use of *smoked* cannabis is not advisable in a chronic illness such as MS.

Muscle spasticity is also common among patients with spinal cord injuries, 60% of whom are younger than 35 years and need long-term care. As with MS, surveys of these patients suggest that cannabis reduces spasticity, nausea and insomnia. Carefully designed clinical trials of THC should be conducted, and have been proposed in the UK (38).

13.6 Cannabinoids and movement disorders

Movement disorders are caused by abnormalities in brain areas that control motor functions. They result in abnormal skeletal muscle movements in the face, limbs and trunk that may occur in patients with dystonia, Huntington's disease, Parkinson's disease and Tourette's syndrome (10). There is limited research that cannabis is useful for treating movement disorders.

There is some evidence that the muscle spasms or 'tics' experienced by patients with Gilles de la Tourette Syndrome are relieved by THC (e.g. (39). Since stress often transiently exacerbates movement disorders, the anxiety-relieving effects of cannabis or cannabinoids might help patients with movement disorders. However, regular cannabis smoking would be a risk for persons already suffering from chronic health conditions (10).

The evidence that cannabinoids have therapeutic effects in patients with movement disorders is largely anecdotal (e.g. (1, 40). Grinspoon and Bakalar (1), for example, presented four case histories of individuals with multiple sclerosis whose condition improved while they smoked cannabis, and deteriorated after they stopped smoking.

There has been one controlled study by Clifford (34) who examined the effects of THC on tremor in 8 patients (4 male and 4 female) with advanced multiple sclerosis. Five patients reported subjective benefit from THC and there was objective evidence of benefit in two of these cases. There was also evidence that their clinical condition deteriorated when they were given placebo and that it improved with the reinstatement of THC.

Grinspoon and Bakalar (1) also described several patients with paraplegia and quadriplegia who reported that cannabis use helped to reduce muscle spasm. The experiences of these individuals were supported by reports in a survey of 43 individuals with spinal cord injuries, 22 of whom reported that they used cannabis to control their muscle spasm.

One controlled trial has evaluated the effects of CBD on chorea in 19 patients with advanced Huntington's disease (41). In this study patients received CBD or placebo for six weeks under double blind conditions in a crossover design. There was no evidence of improvement in chorea on any of the clinical, self-report or motor measures.

13.7 Cannabinoids as anti-asthmatic agents

Smoked cannabis and oral THC dilate the bronchial tubes in normal persons and persons with asthma (42, 43), that is, they increase the lung's capacity to absorb oxygen. Tashkin and colleagues (43), for example, found that smoking a 2% THC cannabis cigarette produced a bronchodilator effect nearly equivalent to that of a clinical dose of isoproterenol, an anti-asthmatic medication.

A major obstacle to the therapeutic use of cannabinoids in asthma is the fact that oral THC produces a much smaller bronchodilator effect and after a substantial delay, than smoked cannabis (44). Attempts to give THC as an inhalant produce irritation and reflex bronchoconstriction (44). Smoking cannabis is the most dependable way of delivering an effective dose of THC but this is an inappropriate way to administer a drug to patients with asthma because it would also deliver other noxious substances that would nullify its therapeutic effects and increase the risk of other respiratory diseases, including cancer in the long-term (44). The unwanted psychotropic effects from cannabis smoking have also been a barrier to its use as an anti-asthmatic drug.

13.8 Cannabinoids as analgesics

Animal studies suggest that cannabinoids may be useful as analgesics. The CB_1 receptor acts on pathways that partially overlap with those affected by opioids like morphine but it acts through pharmacologically distinct mechanisms. This means that cannabinoids and opioids probably have different side effects and may have additive or synergistic analgesic effects.

The few controlled studies of the analgesic efficacy of cannabinoids in humans have been inconclusive. Three experimental pain studies in humans produced mixed results (45–47), but they were poorly controlled (10). More encouraging results have come from three clinical studies of the effects of cannabinoids in patients with severe cancer pain that was persistent and had resisted traditional analgesics (48–50). These studies, which were all double blind and placebo controlled, demonstrated that cannabinoids had analgesic effects equivalent to those of codeine, without its severe side effects, while improving mood, well-being, and appetite.

13.9 The limitations of anecdotal evidence

With the exception of its anti-emetic, anti-nausea and appetite stimulating effects, much of the case for the therapeutic uses of cannabis and cannabinoids is based upon anecdotal evidence. Such evidence is distrusted in clinical medicine. This is especially so in chronic conditions which have a fluctuating course of remission and exacerbation because it is difficult in these diseases to exclude alternative explanations of improvements in a patient's condition that follow their use of THC. It is difficult to exclude the possibility of simple coincidence: that is, THC preceded an improvement in the patient's condition that would have occurred in its absence. It is for these reasons that this review has relied upon evidence from controlled clinical trials in appraising the therapeutic uses of cannabinoids.

13.10 The risks of therapeutic cannabinoid use

For most people the primary adverse effect of acute cannabis use is impaired psychomotor performance. This makes it inadvisable for anyone under the influence of cannabis or THC to operate machinery that might put the user or others in danger, such as driving a car or operating equipment. Most people can be expected to show impaired performance of complex tasks, and a minority experience dysphoria. People who have psychiatric disorders (including substance dependence) may be vulnerable to cannabis dependence, and so sustained therapeutic cannabis use would be contraindicated for them. The short-term immuno-suppressant effects are not well established; if they exist, they are probably not large enough to preclude legitimate medical use. The US Institute of Medicine concluded that the acute effects of cannabis use were 'within the risks tolerated for many medications' (10).

The chronic effects of cannabis are of greater concern for medical use. They fall into two categories: the effects of chronic smoking, and the possibility of dependence on cannabis or THC. Cannabis smoke like tobacco smoke is a risk factor for cancer, lung damage, and poor pregnancy outcome. Smoked cannabis is therefore unlikely to be a safe medication for any chronic medical condition that requires daily use over a period of years. The risk of developing dependence on cannabis is highest in adolescents, particularly those with conduct disorders, and people with psychiatric disorders, or problems with substance abuse (10).

13.11 Obstacles to therapeutic cannabinoid use

Despite their comparative safety, and the evidence for the therapeutic effects of cannabinoids as anti-emetics and appetite stimulants, they have not been widely used clinically. Nor has pharmacological research developed synthetic cannabinoids for medical use. There are two main reasons for this. One is the lack of incentives for pharmaceutical companies to develop and market cannabinoid drugs; the other is the politics of recreational cannabis use.

13.11.1 The market outlook for therapeutic cannabinoids

The decision to develop and conduct clinical trials on a new drug is based upon a drug company's judgment that there is likely to be an adequate return on investment. The research and development costs of cannabinoids are likely to be similar to those of neuropharmaceuticals and anti-inflammatory drugs (10). In the case of the cannabinoids, there are the additional costs of meeting regulatory requirements for drugs derived from a prohibited plant.

The potential market for cannabinoids is determined by the current and projected number of patients who may use the drug, the sales of existing drugs for the indication, the availability of competing products, and the duration of disease (e.g. disease with an early age of onset and a need for long term use). Factors that affect market return include the company's ability to patent the drug, the availability of other forms of market protection, access to health insurance reimbursements, restrictions on access because of drug scheduling, social attitudes towards the drug, its adverse effect profile, and its interactions with other drugs. Naturally occurring substances such as THC cannot be patented; only newly synthesized or derived cannabinoid drugs can be patented.

13.11.2 The politics of therapeutic cannabinoids

Research on the therapeutic use of cannabinoids in the USA has become a casualty of the debate about the legal status of recreational cannabis use. For example, some of the groups advocating the therapeutic use of cannabis have also been proponents of cannabis legalisation (e.g. NORML), thereby fuelling the fears of opponents of cannabis use that success in the campaign for marijuana rescheduling will be the thin edge of a wedge to legalise cannabis. Other proponents of legalisation (e.g. 1) have argued for the legalisation of cannabis as a way of making cannabis available for therapeutic purposes.

On the other side of the argument are those opponents of cannabis use who fear that the admission that cannabis, or any of its constituents, may have a therapeutic use will send the 'wrong message' to youth. This has led to the denial that cannabinoids have any therapeutic effects, and to attempts to prevent all scientific inquiry into any such effects (Bernstein, 1989 cited (52) (p.395).

It is unfortunate that a connection has been forged between the debates about the legal status of cannabis as a recreational drug and the use of cannabinoids for therapeutic use. There is a world of difference between the use of controlled doses of a purified drug under medical supervision and the recreational use of crude preparations of a drug. In a rational world, clinical decisions about whether to use pure cannabinoid drugs should not be abrogated because crude forms of the drug may be abused by those who use it recreationally. We do not allow this type of thinking to deny us the use of opiates for analgesia. It should not deny patients access to any therapeutic uses of cannabinoids derivatives that may be revealed by pharmacological research.

13.12 Summary

The following provisional conclusions can be drawn on the therapeutic uses of cannabis. First, there is sufficient evidence that THC is an anti-emetic agent to justify it being made available in pure synthetic form to cancer and AIDS patients. In the light of the recent development of more effective anti-emetic agents, it remains to be seen how widely used THC will be for this purpose. Second, there is also reasonable evidence for the efficacy of THC in the treatment of AIDS-related wasting. Third, the suggestive evidence of the usefulness of cannabinoids as analgesic and anti-spasmodic agents warrants further pharmacological and experimental investigation, and perhaps clinical research into their effectiveness.

Despite the basic and clinical research work which was undertaken in late 1970s and early 1980s the cannabinoids have not been widely used therapeutically or extensively investigated. This seems largely attributable to the disincentives pharmaceutical companies have to develop cannabinoid drugs and the regulatory obstacles to their registration. The discouragement of therapeutic research also derives from the fact that THC, the most therapeutically effective cannabinoid, has the psychoactive effects sought by recreational users. The discovery of the cannabinoid receptor may help to overcome some of the resistance to research into the therapeutic uses of cannabinoids by holding out the prospect that the psychoactive effects of the cannabinoids can be disengaged from their other therapeutically desirable effects.

13.13 References

1. Grinspoon, L. & Bakalar, J. (1993) *Marihuana, the forbidden medicine* (New Haven, Yale University Press).
2. Mechoulam, R. (1986) The pharmacohistory of cannabis sativa, in: Mechoulam, R. (Ed.) *Cannabinoids as Therapeutic Agents*, pp. 1–20 (Boca Raton, FL, CRC Press).
3. Nahas, G. (1984) Toxicology and pharmacology, in: Nahas, G. (Ed.) *Marihuana in Science and Medicine*, pp. 109–246 (New York, Raven Press).
4. O'Shaughnessy, W. (1842) On the preparation of the Indian hemp, or gunjah (cannabis indica) and their effects on the animal system in health and their utility in the treatment of tetanus and other convulsive disorders, *Transcripts of the Medical Physicians' Society of Calcutta*, 8, 421–461.
5. Iversen, L. (2000) *The Science of Marijuana* (Oxford, Oxford University Press).
6. Casswell, A. (1992) Marijuana as medicine, *Medical Journal of Australia*, 156, 497–498.
7. Gaoni, Y. & Mechoulam, R. (1964) Isolation, structure and partial synthesis of an active constituent of hashish, *Journal of the American Chemistry Society*, 86, 1646–1647.
8. Grinspoon, L. (1990) Testimony in the Matter of Marihuana Rescheduling, in: Randall, R. C. (Ed.) *Cancer Treatment and Marijuana Therapy*, pp. 5–12 (Washington DC, Galen Press).

9. Randall, R. C. (1990) *Cancer Treatment and Marijuana Therapy* (Washington DC, Galen Press).

10. Institute of Medicine (1999) *Marijuana and Medicine: Assessing the Science Base* (Washington, DC, National Academy Press).

11. Sallan, S. E., Zinberg, N. E. & Frei, E. (1975) Antiemetic effect of delta-9-tetrahydrocannabinol in patients receiving cancer chemotherapy, *New England Journal of Medicine*, 293, 795–797.

12. Chang, A. E., Shiling, D. J., Stillman, R. C., Goldberg, N. H., Seipp, C. A., Barofsky, I., Simon, R. M. & Rosenberg, S. A. (1979) Delta-9-tetrahydrocannabinol as an antiemetic in cancer patients receiving high-dose methotrexate, *Annals of Internal Medicine*, 91, 819–824.

13. Carey, M. P., Burish, T. G. & Brenner, D. E. (1983) Delta–9-tetrahydrocannabinol in cancer chemotherapy: Research problems and issues, *Annals of Internal Medicine*, 99, 196–114.

14. Poster, D. S., Penta, J. S., Bruno, S. & Macdonald, J. S. (1981) Delta-9-tetrahydrocannabinol in clinical oncology, *Journal of the American Medical Association*, 245, 2047–2051.

15. Levitt, M. (1986) Cannabinoids as antiemetics in cancer chemotherapy, in: Mechoulam, R. (Ed.) *Cannabinoids as Therapeutic Agents*, pp. 71–83 (Boca Raton, FL, CRC Press).

16. Ungerleider, J. T., Andrysiak, T., Fairbanks, L., Goodnight, J., Sarna, G. & Jamison, K. (1982) Cannabis and cancer chemotherapy: A comparison of oral delta-9-THC and prochlorperazine, *Cancer*, 50, 636–645.

17. DiMarzo, V., Goparaju, S., Wang, L., Liu, J., Batkai, S., Zoltan, J., Fezza, F., Miura, G., Palmiter, R., Sugiura, T. & Kunos, G. (2001) Leptin–regulated endocannabinoids are involved in maintaining food intake, *Nature*, 410, 822–825.

18. Beal, J., Olson, R., Morales, J., Bellman, P., Yangco, B., Lefkowitz, L., Plasse, T. & Shepard, K. (1995) Dronabinol as a treatment for anorexia associated with weight loss in patients with AIDS, *Journal of Pain and Symptom Management*, 10, 89–97.

19. Beal, J., Olson, R., Lefkowitz, L., Laubenstein, L., Bellman, P., Yangco, B., Morales, J., Murphy, R., Powderly, W., Plasse, T., Mosdell, K. & Shepard, K. (1997) Long-term efficacy and safety of dronabinol for acquired immunodeficiency syndrome-associated anorexia, *Journal of Pain and Symptom Management*, 14, 7–14.

20. Clarke, R. (1995) *Marijuana Botany—an advanced study: The propagation and breeding of distinctive cannabis* (Berkeley, CA, Ronin Publishing).

21. Kaslow, R., Blackwelder, W., Ostrow, D., Yerg, D., Palenick, J., Coulson, A. & Valdiserri, R. (1989) No evidence for a role of alcohol or other psychoactive drugs in accelerating immunodeficiency in HIV-1-positive individuals: A report from the Multicenter AIDS Cohort Study, *Journal of the American Medical Association*, 261, 3424–3429.

22. Adler, M. W. & Geller, E. B. (1986) Ocular effects of cannabinoids, in: Mechoulam, R. (Ed.) *Cannabinoids as Therapeutic Agents* (Boca Raton, FL, CRC Press).

23. Hepler, R. S. & Petrus, R. J. (1971) Marihuana smoking and intraocular pressure, *Journal of the American Medical Association*, 217, 1392.

24. Hepler, R. S. & Petrus, R. J. (1976) Experiences with administration of marihuana to glaucoma patients, in: Cohen, S. & Stillman, R. C. (Eds.) *The Therapeutic Potential of Marihuana*, pp. 63–75 (New York, Plenum Medical Book Company

25. Hepler, R. S., Frank, I. M. & Petrus, R. (1976) Ocular effects of marihuana smoking, in: Braude, M. C. & Szara, S. (Eds.) *The Pharmacology of Marihuana*, pp. 815–824 (New York, Raven Press).

26. Jones, R., Benowitz, N. & Herning, R. (1981) The clinical relevance of cannabis tolerance and dependence, *Journal of Clinical Pharmacology*, 21, 143S–152S.

27. Chesher, G. B. & Jackson, D. M. (1974) Anticonvulsant effects of cannabinoids in mice: drug interactions within cannabinoids, and cannabinoid interactions with phenytoin, *Psychopharmacologia*, 37, 255–264.

28. Consroe, P. & Snider, S. R. (1986) Therapeutic potential of cannabinoids in neurological disorders, in: Mechoulam, R. (Ed.) *Cannabinoids as Therapeutic Agents*, pp. 21–50 (Boca Raton, FL, CRC Press).

29. Institute of Medicine (1982) *Marijuana and Health* (Washington, DC, National Academy Press).

30. Consroe, P. F., Wood, G. C. & Buchsbaum, H. (1975) Anticonvulsant nature of marihuana smoking, *Journal of the American Medical Association*, 234, 306–307.

31. Cunha, J. M., Carlini, E. A., Pereira, A. E., Ramos, O. L., Pimentel, C., Gagliardi, R., Sanvito, W. L., Lander, N. & Mechoulam, R. (1980) Chronic administration of cannabidiol to healthy volunteers and epileptic patients, *Pharmacology*, 21, 175–185.

32. Baker, D. (2000) Reply: A sanguine approach to cannabis, *Trends in Pharmacological Sciences*, 21, 197–197.

33. Consroe, P., Musty, R., Rein, J., Tillery, W. & Pertwee, R. (1997) The perceived effects of smoked cannabis on patients with multiple sclerosis, *European Neurology*, 38, 44–48.

34. Clifford, D. B. (1983) Tetrahydrocannabinol for tremor in multiple sclerosis, *Annals of Neurology*, 13, 669–671.

35. Petro, D. & Ellenberger, C. (1981) Treatment of human spasticity with delta-9-hydrocannabinol, *Journal of Clinical Pharmacology*, 21, 413s–416s.

36. Ungerleider, J., Andrysiak, T., Fairbanks, L., Ellison, G. & Myers, L. (1987) Delta-9-THC in the treatment of spasticity associated with multiple sclerosis, *Advances in Alcohol and Substance Abuse*, 7, 39–50.

37. Achirona, A., Mirona, S., Lavieb, V., Margalitc, R. & Biegonb, A. (2000) Dexanabinol (HU-211) effect on experimental autoimmune encephalomyelitis: Implications for the treatment of acute relapses of multiple sclerosis, *Journal of Neuroimmunology*, 192, 26–31.

38. House of Lords Select Committee on Science and Technology (1998) Cannabis: The Scientific and Medical Evidence (London, House of Lords, The Stationary Office).

39. Muller-Vahl, K., Kolbe, H., Schneider, U. & Emrich, H. (1999) Cannabis in movement disorders, *Research in Complementary Medicine (Cannabis and Cannabinoid Medicine)*, 6.

40. Meinck, H. M., Schonle, P. W. & Conrad, B. (1989) Effect of cannabinoids on spasticity and ataxia in multiple sclerosis, *Journal of Neurology*, 236, 120–122.

41. Consroe, P., Laguna, J., Allender, J., Snider, S., Stern, L., Sandyk, R., Kennedy, K. & Schram, K. (1991) Controlled trial of cannabidiol in Huntington's disease, *Pharmacology, Biochemistry and Behavior*, 40, 701–708.

42. Tashkin, D. P., Shapiro, B. J., Ramanna, L., Taplin, G. V., Lee, Y. E. & Harper, C. E. (1976) Chronic effects of heavy marihuana smoking on pulmonary function in healthy young males, in: Braude, M. & Szara, S. (Eds.) *Pharmacology of marihuana*, pp. 291–5 .

43. Tashkin, D. P., Shapiro, B. J. & Lee, Y. E. (1975) Effects of smoked marijuana in experimentally induced asthma, *American Review of Respiratory Disease*, 112, 377–386.

44. Tashkin, D. (1993) Is frequent marijuana smoking harmful to health?, *Western Journal of Medicine*, 158, 635–637.

45. Clark, W., Janal, M., Zeidenberg, P. & Nahas, G. (1981) Effects of moderate and high doses of marihuana on thermal pain: A sensory decision analysis, *Journal of Clinical Pharmacology*, 21, 299s–301s.

46. Hill, S., Schwin, R., Goodwin, D. & Powell, B. (1974) Marihuana and pain, *Journal of Pharmacology and Experimental Therapeutics*, 188, 415–418.

47. Libman, E. & Stern, M. (1985) The effects of delta-9-tetrahydrocannabinol on cutaneous sensitivity and its relation to personality, *Personality, Individuality and Difference*, 6, 169–174.

48. Noyes, R., Brunk, F., Avery, D. H. & Canter, A. (1975) The analgesic properties of delta-9-tetrahydrocannabinol and codeine, *Clinical Pharmacology and Therapeutics*, 18, 84–89.

49. Noyes, R., Brunk, S., Baram, D. & Canter, A. (1975) Analgesic effect of delta-9-tetrahydrocannabinol, *Journal of Clinical Pharmacology*.

50. Staquet, M., Gantt, C. & Machin, D. (1978) Effect of a nitrogen analog of tetrahydrocannabinol on cancer pain, *Clinical Pharmacology and Threapeutics*, 23, 397–401.

51. Randall, R. C. (1988) *Marijuana, Medicine and the Law* (Washington DC, Galen Press).

52. Randall, R. C. (1989) *Marijuana, Medicine and the Law, Volume II* (Washington DC, Galen Press).

53. Mechoulam, R. (1988) Direct testimony, in: Randall, R. C. (Ed.) *Marijuana, Medicine and the Law*, pp. 319–330 (Washington DC, Galen Press).

14 A comparison of the health effects of cannabis with alcohol and tobacco

This chapter compares the most probable harms caused by cannabis use with those caused by alcohol and tobacco, two commonly used psychoactive substances in Western societies. A number of issues arise in comparing the health effects of cannabis with those of these two drugs. The first are difficulties in making causal inferences about the connections between cannabis use and the adverse health and psychological consequences which have been attributed to it (1). The second is lack of information about the risks of cannabis use for users. Both of these problems arise from the scarcity of epidemiological studies of the health risks of cannabis use by comparison with such studies of alcohol and tobacco use.

A third set of difficulties arise in measuring the public health impact of these risks. The methods used to date have typically involved comparisons of the numbers of deaths, persons years of life lost, and hospital bed days attributable to conditions caused by each type of drug (e.g. English et al. (2)). The most recent innovation has been to use a combination of Life Years Lost (YLL) and Disability Adjusted Life Years (DALYs) to estimate the total burden of disease attributable to alcohol, tobacco and illicit drug use (3, 4).

14.1 The probable adverse health effects of cannabis

The following are the major adverse health and psychological effects of acute and chronic cannabis use, classified by the degree of confidence in the relationship.

14.1.1 Acute effects

The major acute psychological and health effects of cannabis intoxication are:

- anxiety, dysphoria, panic and paranoia, especially in naive users;
- cognitive impairment, especially of attention and memory while intoxicated;
- psychomotor impairment, and probably an increased risk of accidental injury or death if an intoxicated person attempts to drive a motor vehicle or operate machinery;
- an increased risk of experiencing psychotic symptoms among those who are vulnerable because of a personal or family history of psychosis;
- an increased risk of low birth weight babies, and possibly of birth defects, if used during the first trimester of pregnancy.

14.1.1 Chronic effects

The major health and psychological effects of chronic cannabis use, especially daily use over many years, remain uncertain but the major **probable** adverse effects appear to be:

- respiratory diseases caused by smoking cannabis, such as chronic bronchitis, and changes in lung tissue that are precursors of malignancy;
- development of a cannabis dependence syndrome, characterised by an inability to abstain from or to control cannabis use;
- an increased risk of developing cancers of the aerodigestive tract, i.e. oral cavity, pharynx, and oesophagus.

The following are the major **possible** adverse effects of chronic, heavy cannabis use which remain to be confirmed by controlled research:

- subtle forms of cognitive impairment, most particularly of attention and memory, which persist while the user remains chronically intoxicated, and may or may not be reversible after prolonged abstinence from cannabis.
- a decline in occupational performance marked by underachievement in adults in occupations requiring high level cognitive skills, and impaired educational attainment in adolescents.

High risk groups

A number of groups are at increased risk of experiencing some of these adverse effects.

Adolescents

- Adolescents with a history of poor school performance whose educational achievement may be further limited by the cognitive impairments produced by chronic intoxication with cannabis;
- Adolescents who initiate cannabis use in the early teens are at higher risk of progressing to heavy cannabis use and other illicit drug use, and to the development of dependence on cannabis.

Women of childbearing age

- Babies born to women who continued to smoke cannabis may have a slightly lower birth weight.

Persons with pre-existing diseases

Persons with a number of pre-existing diseases who smoke cannabis are probably at an increased risk of exacerbating symptoms of their diseases. These include:

- individuals with cardiovascular diseases, such as coronary artery disease, cerebrovascular disease and hypertension;
- individuals with respiratory diseases, such as asthma, bronchitis, and emphysema;
- individuals with schizophrenia who are at increased risk of precipitating or of exacerbating schizophrenic symptoms;
- individuals who are or have been dependent upon alcohol and other drugs are probably at an increased risk of developing dependence on cannabis.

14.2 The implications of increased potency of cannabis

It has been claimed that a substantial increase in the average THC content of cannabis has 'made obsolete' much of what we once knew about the risks and consequences of cannabis use (5) because most of this was based on research on cannabis with low levels of THC. This argument is unconvincing for two reasons. First, as discussed in chapter 2, the evidence does not support claims that the average THC content of cannabis products has increased substantially in recent decades. Second, it is untrue that the research literature on the adverse health effects is based on studies of populations consuming cannabis with low levels of THC. The field studies in Costa Rica, Greece, Jamaica and Egypt examined very heavy, long term cannabis users and laboratory studies conducted in the USA involved subjects consuming 30 mg THC per day for periods of a month.

The claim about increased potency is popular because it appears to explain an apparent increase in the adverse effects of cannabis use. There probably has been some increase in the prevalence of some of these effects, most notably dependence, although this is uncertain because of limitations with the available data (6). There are, however, two more plausible alternative explanations for any increase in adverse effects of cannabis use: (1) cannabis markets have increased the availability of more potent forms of cannabis; and (2) changes in the patterns of cannabis use have increased the prevalence of harmful patterns of cannabis use (6).

The effect of using more potent cannabis products will depend upon the type of health effect in question, and the user's experience with cannabis. Higher average doses of THC will probably increase the risk of adverse psychological effects of cannabis use, an effect likely to be most obvious among naive or first time cannabis users. This effect may discourage further experimentation with the drug among these users. Risks of increased THC exposure among regular cannabis users possibly include an increased risk of accidents among those who drive while intoxicated, especially if cannabis use is combined with alcohol, and an increased risk of regular cannabis users developing dependence. If the THC content of the most commonly used cannabis products has increased, the net adverse effects of cannabis use may have marginally increased. Respiratory risks may be marginally decreased if cannabis smokers are able to titrate their doses of THC.

14.3 A comparison of the health risks of alcohol, cannabis and nicotine

We have used the following as authorities on the health risks of alcohol and tobacco: Anderson et al. (7); English et al (2); the Institute of Medicine (8); the International Agency for Research into Cancer (9); Mathers, Vos and Stephens (4); Roselle et al (10); and the Royal College of Physicians (11).

14.3.1 Acute effects

Alcohol

Some of the acute risks of cannabis use are similar to those of alcohol. Both drugs cause psychomotor and cognitive impairment, especially of memory and planning. In the case of alcohol these impairments increase the risks of motor vehicle and other accidents (2, 8). While cannabis intoxication probably increases the accident risks in hazardous situations, it remains to be determined whether it increases risky behaviour.

However, alcohol and cannabis differ in their relation to intentional injuries. First, alcohol intoxication is strongly associated with aggressive and violent behaviour. The relationship is complex, and the nature and extent of alcohol's causal role is controversial (12–14), but changes in the level of alcohol consumption appear to affect the incidence of violent crime (15–17). There is also increasing evidence that alcohol plays a role in suicide (18). Although cannabis and violence may be correlated among adolescents (see chapter 6), it remains to be clarified whether the relationship is causal because persons with a history of violence are more likely to become heavy cannabis users.

Second, substantial doses of alcohol taken during pregnancy can produce a Foetal Alcohol Syndrome (2). There is weak evidence that cannabis can adversely affect the development of the foetus when used during pregnancy (19), but there is no equivalent for cannabis of the foetal alcohol syndrome.

Third, acute alcohol use has one health risk that is not shared with cannabis. In large doses alcohol can cause death by asphyxiation, alcohol poisoning, cardiomyopathy and cardiac infarct. There are, by contrast, no recorded overdose fatalities from cannabis.

Tobacco

Cannabis and tobacco share acute irritant effects of smoke upon the respiratory system and THC and nicotine both stimulate the cardiovascular system. Smoking cannabis and tobacco can adversely affect persons with cardiovascular and respiratory diseases. In both cases, these effects arise from the fact that the drug is smoked.

14.3.2 Chronic effects

Alcohol

There are a number of risks of chronic alcohol use, which may be shared by chronic cannabis use. First, daily use of both increases the risk of developing dependence. There is strong evidence of such a syndrome for alcohol and reasonable evidence for cannabis. One difference is that withdrawal symptoms are mild in dependent cannabis users who abruptly stop using cannabis, whereas the abrupt cessation of alcohol use in severely dependent drinkers can produce a severe withdrawal syndrome that can be fatal in a small proportion of cases, if untreated (20).

Second, there is reasonable evidence that chronic heavy alcohol use can produce psychotic symptoms and psychoses in some individuals, either during acute intoxication or during withdrawal. There is suggestive evidence that chronic heavy cannabis use may

produce a toxic psychosis, some epidemiological evidence that heavy cannabis use may precipitate schizophrenia in individuals with a personal or a family history of psychiatric disorder, and stronger evidence that cannabis use worsens the course of schizophrenia.

Third, there is good evidence that chronic heavy alcohol use can indirectly cause brain injury—the Wernicke-Korsakov syndrome—with symptoms of severe memory defect and an impaired ability to plan and organise. With continued heavy drinking, and in the absence of vitamin supplementation, this injury may produce severe and irreversible cognitive impairment. Chronic cannabis use does not produce cognitive impairment of comparable severity. There is suggestive evidence that chronic cannabis use may produce subtle deficits in cognitive functioning that may or may not be reversed by abstinence.

Fourth, there is reasonable evidence that chronic heavy alcohol use impairs occupational performance in adults and educational achievement in adolescents. There is suggestive evidence that chronic heavy cannabis use produces similar, albeit less marked, impairments in the occupational and educational performance of adolescents and adults.

Fifth, there is good evidence that chronic, heavy alcohol use increases the risk of premature mortality from accidents, suicide and violence. There is no comparable evidence for chronic cannabis use, although it is likely that dependent cannabis users who frequently drive while intoxicated with cannabis would be at greater risk of accidental injury or death.

Sixth, alcohol use has been accepted as a contributory cause of cancer in various tissues and organs of the digestive system and breast cancer in women. There is suggestive evidence that chronic cannabis smoking may be a cause of cancers of the aerodigestive tract.

Seventh, heavy alcohol use is a major cause of liver cirrhosis and is also implicated in gastritis, high blood pressure, stroke, cardiac arrhythmias, cardiomyopathy, pancreatitis, and polyneuropathy. On the other hand, alcohol use is also associated with a reduction in the risk of heart disease that is of public health significance in societies with high rates of heart disease (18). No equivalent adverse or protective effects have been reported for cannabis. There is some evidence that THC may be therapeutically useful for appetite stimulation and as anti-emetics in patients undergoing cancer therapy.

Tobacco

The major adverse health effects shared by chronic cannabis and tobacco smokers are chronic bronchitis, and probably, cancers of the aerodigestive tract (i.e. the mouth, tongue, throat, oesophagus, lungs). The increased cancer risk is a consequence of the fact that both drugs are smoked. It is possible that chronic cannabis smoking also shares the cardiotoxic properties of tobacco smoking but this remains to be investigated. These respiratory risks could be avoided by a change to the oral route of administration which would also reduce but not eliminate the cardiovascular risks.

Tobacco smoking is associated with a wide variety of other chronic health conditions for which cannabis smoking has not so far been implicated. These include cancer of the cervix, stomach, bladder and kidney, coronary heart disease, peripheral vascular disease, and stroke, as well as cataracts and osteoporosis (2).

14.4 Comparing the magnitude of risks

Many of the quantitative risks of cannabis use can only be guessed at in the absence of studies of the dose-response relationship between cannabis use and adverse health effects. The following are guesstimates of the risks of cannabis use for the most probable adverse health effects. When in doubt we have assumed that the relative risks of cannabis use are similar to the risks of alcohol or tobacco.

Motor Vehicle Accidents: If we assume that driving while intoxicated with cannabis produces a comparable increase in the risk of accidents to that produced by driving while intoxicated with alcohol (say with a blood alcohol level of 0.05% to 0.10%), then the RR of an accident while intoxicated would be in the range of 2 to 4. The fact that alcohol and cannabis are often used in combination makes it difficult to estimate the relative risk of having an accident when using cannabis alone.

Respiratory Diseases: If we assume that a daily cannabis user who smokes 5 or more joints per day faces a comparable risk of respiratory disease to that of a 20 cigarette a day tobacco smoker, then the RR of developing chronic bronchitis would be 6 or greater for those who had ever smoked cannabis, and substantially higher among those who had been daily cannabis smokers over many years and those who also smoked tobacco (2). Recent research suggests that the risk of daily cannabis smoking is more like that of smoking 10–15 cigarettes per day (21), so the relative risks may be smaller.

Respiratory Tract Cancers: If we make the same worst case assumptions about daily cannabis smoking then the relative risks of various cancers of the respiratory tract would be of the order of: 5 for oropharyngeal cancer, 4 for oesophageal cancer, and 7 for lung cancer (2). Again these risks would be substantially higher among cannabis smokers who also smoked tobacco. The recent case control study of head and neck cancer suggested a relative risk of 2 for cannabis smoking, after adjustment for tobacco use (22).

Low Birthweight Babies: Making a worst-case assumption, a woman who smokes cannabis during pregnancy may double her chance of giving birth to a low birthweight baby (2). The average size of the effect is smaller than that for tobacco smoking (19).

Schizophrenia: This is one of the few health consequences for which there is a quantitative estimate of relative risk. If we use the estimated RR from the study by Andreasson et al (23) after adjustment for confounding variables, then an adolescent who had smoked cannabis 50 or more times by age 18 would have a 2 to 3 times higher risk of developing schizophrenia than an adolescent who had not used cannabis.

Dependence: The risk of cannabis dependence is estimated by the proportion of those who have ever used cannabis, or have had a history of daily use, who become dependent on the drug. The best estimates from US data in the late 1970s and early 1980s is that 10% of those who have ever used cannabis (24), and between 33% and 50% of those who have had a history of daily cannabis use, will become dependent on cannabis (see Hall et al (25)). The comparable risks among those who had ever used tobacco (32%), opiates (23%) and alcohol (15%) were higher than the risk for cannabis users (24).

14.5 Public health significance

14.5.1 Motor vehicle accidents

The epidemiological studies indicate that in its own right, cannabis makes at most a very small contribution to motor vehicle accidents, and so, on the whole, it may seem be a minor road safety problem by comparison with alcohol. Its public health significance for road safety may be in amplifying the adverse effects of alcohol in the majority of drivers who drive when intoxicated by alcohol and cannabis.

14.5.2 Respiratory diseases

Respiratory diseases, such as bronchitis, caused by cannabis smoking are likely to have greater public health significance than respiratory cancers. This is for two reasons. First, respiratory cancers require a greater length of exposure to cigarette smoke (15 to 20 years) than does chronic bronchitis. Second, there are very few cannabis users who use the drug for more than 5 years (26). On current patterns of use, cannabis smoking is more likely to produce respiratory disease than it is to cause premature deaths from cancers of the respiratory tract.

14.5.3 Respiratory tract cancers

Even if we make the worst case assumption that the risks of cancer are comparable among daily tobacco and cannabis smokers then cannabis smoking will make a small contribution to the occurrence of these cancers, on current patterns of use in developed societies (1). Only a minority of those who ever use cannabis become daily users, and a much smaller proportion of these use cannabis beyond their middle twenties by comparison with the high proportions of tobacco smokers who do so (26). Among this minority, concurrent cannabis and tobacco use may exacerbate the adverse respiratory effects of each.

14.5.4 Low birthweight babies

If cannabis smoking during pregnancy doubles the risks of a low birthweight baby, its public health significance will be much less than that of tobacco smoking, because the prevalence of cannabis use is much lower than that of tobacco smoking. The risks of a low birthweight baby will be higher among women who also smoke tobacco, as do many of those who smoke cannabis during pregnancy.

14.5.5 Schizophrenia

If the relationship between cannabis use and schizophrenia is causal, cannabis use would account for less than 10% of new cases of schizophrenia. Even this figure seems unlikely, however, since the incidence of schizophrenia has probably declined during the period when cannabis use among adolescents and young adults has increased (27).

14.5.6 Dependence

Cannabis dependence is potentially a more prevalent outcome than any of the other potentially adverse health effects of cannabis. On the ECA estimates, approximately 4% of the adult US population met diagnostic criteria for cannabis abuse or dependence in their lifetime and 2% in the past year. This compares with 14% who met diagnostic criteria for alcohol abuse and dependence at some time in their lives. This is a substantial proportion of the population but there may be a high rate of remission of symptoms in the absence of treatment.

14.6 Overall public health significance

Overall, the relative risks of adverse health effects for cannabis are small to moderate and the proportion of users who use regularly is much smaller than the proportions of alcohol and tobacco users who do so (28). In aggregate, then, the public health problems caused by cannabis *on current patterns of use* are modest compared with those of alcohol and tobacco.

A number of attempts have been made to directly compare the effects of alcohol, tobacco and illicit drugs on mortality, morbidity and societal costs. One of the earliest was an Australian study by Holman et al (29) which estimated the number of deaths, person years of life lost and number of hospital bed days that could be attributed to the use of alcohol, tobacco and illicit drugs. According to Holman et al, in Australia in 1986 there were 23,639 deaths attributable to these three classes of drugs. Of these 17,800 were attributed to tobacco, 5,360 to alcohol and 479 to illicit drugs, of which 289 (60%) were due to opiate use. There was a similar rank ordering of person years of life lost (92,023 for tobacco, 66,034 for alcohol and 16,438 for illicit drugs) and bed days (1,014,336 for tobacco, 1,009,591 for alcohol and 57,361 for illicit drugs). No deaths were attributed to cannabis use and cannabis made no contribution to morbidity. The authors concluded 'that apart from dependence, abuse and withdrawal, no other adverse health effect of cannabis is sufficiently substantiated or quantified to enable an analysis of resultant morbidity or mortality' (p. 377).

English et al (2) updated the Holman et al estimates of drug-caused mortality and morbidity in Australia in 1992. Unlike Holman et al, English et al included estimates of the protective effects of moderate alcohol consumption on mortality from cardiovascular disease. The inclusion of a protective effect for alcohol reduced the number of deaths attributed to alcohol from 5,360 in 1986 to 3,660 in 1992 and person years of life lost declined from 66,034 to 55,540. The contributions of tobacco and illicit drugs to mortality did not change much from the earlier estimates (18,290 and 488 respectively). Opiates were responsible for 92% of illicit drug deaths and no deaths were attributed to cannabis. Cannabis contributed to hospital bed days through treatment of cannabis dependence and abuse (1% of all bed days attributed to illicit drug use).

More recently, Ridolfo and Stevenson (30) updated the English et al estimates for Australia in 1998 using a different method to take account of the protective effect of alcohol on cardiovascular deaths. In their analysis alcohol produced a *net reduction* of 2371 deaths because the number of deaths averted by moderate alcohol use exceeded the number of deaths that alcohol caused. The number of deaths attributed to tobacco marginally increased from 18,290 to 19,019 and the number of deaths attributed to illicit drugs increased from 488 to 1,023 because of a substantial increase in the number of opioid overdose deaths.

14.6.1 Burden of disease estimates

A different approach to estimating the public health impact of alcohol, tobacco and cannabis was adopted in the Global Burden of Disease (GBD) Study (3, 33). In this study, an estimate of the years of life lost (YLL) as a result of the use of drugs was added to the disability caused by diseases to estimate the number of Disability-Adjusted Life-

Years (DALYs) for each type of drug use. This enabled an estimate of the proportion of global burden of disease that was accounted for by different types of drug use.

Murray and Lopez estimated that 3.5% of global DALYs was attributable to alcohol, 2.6% to tobacco, and 0.6% to illicit drugs (3). In six of the eight world regions, tobacco and alcohol outranked illicit drugs in DALYs. Illicit drugs outranked alcohol in the Middle Eastern region, and tobacco in the Latin American region. The authors caution that 'because of the great difficulty in reliably estimating prevalence of illicit drug use, and of reliably quantifying its health effects, the estimates for this risk factor may well be too low' (p. 310). The illicit drug that made the largest contribution to the global burden was heroin.

The Australian Burden of Disease and Injury (ABDI) (4) adapted the approach of Murray and Lopez to estimate the contribution that alcohol, tobacco and illicit drugs made to the burden of disease and injury in Australia. The ABDI study used the comprehensive data collected on mortality and morbidity in Australia which includes surveys of the health of nationally representative samples of Australians. Their findings differed from those of the GBD study in the rank ordering of alcohol and tobacco because the Australian study included an estimate of the burden of disease that was averted by moderate alcohol use. Tobacco accounted for 9.7% of the total burden of disease in Australia, alcohol accounted for 2.2% and illicit drugs for 1.8%. Among illicit drugs, the overwhelming majority of the burden was due to heroin dependence, which accounted for 1.2% of total burden. Cannabis dependence and abuse accounted for 0.2% of all disability. No deaths were attributed to cannabis use (4).

14.6.2 Summary of public health impact

Studies of mortality and morbidity and disease burden attributable to alcohol, tobacco and illicit drugs differ in their rankings of impact depending upon whether the mortality benefits of moderate alcohol use are included or not. They leave little doubt, however, that *on current patterns of use*, alcohol and tobacco are much more damaging to public health in developed societies than illicit drugs. Among illicit drugs, cannabis makes no known contribution to mortality and a minor contribution to morbidity and disability.

14.6.3 Predicting the effects of changes in the prevalence of cannabis use

These estimates of the public health impact of cannabis use are based on *current patterns of use*. They cannot be used to predict what would happen if there was a major change in the prevalence of cannabis use, as may happen if cannabis were to become as freely available and as heavily promoted as alcohol and tobacco. Although in principle, it may seem simple to predict the public health consequences of increased cannabis use (e.g. by multiplying its harms at present by the increased number of users), such a calculation would assume that the risks of cannabis use did not change with the characteristics of the user, or the legal regime under which the drug was used.

Both assumptions are questionable. Cannabis is likely to be used by a different population when its use is illegal and prevalence of use is lower than would be the case if it were legal and more people used it. This has been reported with alcohol, for example, with different patterns of alcohol consumption and alcohol-related problems in

'dry' (non-drinking) and 'wet' (high level of drinking) cultures. If adult cannabis use were legalised, it might also be easier to reduce some of these health risks, for example, by encouraging cannabis users to ingest rather than to smoke the drug, or by reducing the tar content of cannabis that is smoked. Decriminalising cannabis for adult use would probably also increase use by adolescents, the health effects of which would be very difficult to predict. Estimating the net effects of harm reduction efforts in adults and a likely increase in adolescent use is therefore difficult.

For these reasons we have not attempted to predict the health risks of cannabis use if it became as widely used as alcohol and tobacco. All that can be said with confidence is that if its rate of use increased to the levels of cigarette smoking and alcohol use, its adverse impact on public health would increase. It is impossible to say precisely by how much.

14.7 Summary

Cannabis use can harm health when it is used daily over years or decades. Considerable uncertainty remains about whether some of these effects are attributable to cannabis use alone or to tobacco and alcohol. There is too little data on the relationship between frequency, quantity and duration of cannabis use, and the risks of many of these effects. Using estimates of the known effects of alcohol and tobacco, the most probable adverse effects of chronic heavy cannabis use over a period of years are: the development of a dependence syndrome; an increased risk of motor vehicle accidents; an increased risk of chronic bronchitis; an increased risk of respiratory cancer; an increased risk of giving birth to low birth weight babies when used during pregnancy; and perhaps, an increased risk of developing schizophrenia among those who are vulnerable. Many of these risks are shared with alcohol and tobacco, which is unsurprising given that cannabis is an intoxicant, like alcohol, that is usually smoked, like tobacco.

On *current patterns of use*, cannabis poses a much less serious public health problem than alcohol and tobacco in Western societies. This is no cause for complacency as the public health significance of alcohol and tobacco are substantial, and the public health impact of cannabis would probably increase if the prevalence of heavy daily cannabis use were to approach that of heavy alcohol use, or that of daily cigarette smoking among adults.

14.8 References

1. Hall, W., Johnston, L. & Donnelly, N. (1999) Assessing the health and psychological effects of cannabis use, in: Kalant, H., Corrigall, W., Hall, W. & Smart, R. (Eds.) *The health effects of cannabis*, pp. 1–17 (Toronto, Canada, Centre for Addiction and Mental Health).

2. English, D., Holman, C., Milne, E., Winter, M., Hulse, G., Codde, S., Corti, B., Dawes, V., De Klerk, N., Knuiman, M., Kurinczuk, J., Lewin, G. & Ryan, G. (1995) *The quantification of drug caused morbidity and mortality in Australia, 1995* (Canberra, Commonwealth Department of Human Services and Health).

3. Murray, C. J. L. & Lopez, A. D. (1996) Quantifying the burden of disease and injury attributable to ten major risk factors, in: Murray, C. J. L. & Lopez, A. D. (Eds.) *The Global Burden of Disease: A Comprehensive Assessment of Mortality and Disability from Diseases, Injuries, and Risk Factors in 1990 and Projected to 2020*, pp. 295–324 (Cambridge, MA, Harvard School of Public Health).

4. Mathers, C., Vos, T. & Stevenson, C. (1999) *The Burden of Disease and Injury in Australia* (Canberra, Australian Institute of Health and Welfare).

5. Gold, M. S. (1991) Marijuana, in: Miller, N. S. (Ed.) *Comprehensive Handbook of Alcohol and Drug Addiction*, pp. 353–376 (New York, Dekker).

6. Hall, W. & Swift, W. (2000) The THC content of cannabis in Australia: Evidence and implications, *Australian and New Zealand Journal of Public Health*, 24, 503–508.

7. Anderson, P., Cremona, A., Paton, A., Turner, C. & Wallace, P. (1993) The risk of alcohol, *Addiction*, 88, 1493–1508.

8. Institute of Medicine (1987) *Causes and Consequences of Alcohol Problems: An Agenda for Research* (Washington DC, National Academy Press).

9. International Agency on Cancer (1990) *Cancer: Causes, Occurrence and Control* (Lyon, International Agency on Cancer).

10. Roselle, G., Mendenhall, C. L. & Grossman, C. J. (1993) Effects of alcohol on immunity and cancer, in: Yirmiya, R. & Taylor, A. N. (Eds.) *Alcohol, Immunity and Cancer*, pp. 4–21 (Baton Rouge, CRC Press).

11. Royal College of Physicians (1987) *A Great and Growing Evil: The medical consequences of alcohol abuse* (London, Tavistock).

12. Pernanen, K. (1991) *Alcohol in Human Violence* (New York and London, Guilford).

13. Martin, S. E. (1993) Alcohol and Interpersonal Violence: Fostering Multidisciplinary Perspectives (NIH, Department of Health and Human Services).

14. Pohorecky, L. A., Brick, J. & Milgram, G. G. E. (1993) Alcohol and aggression, *Journal of Studies on Alcohol*, Supplement No. 11, September.

15. Room, R. (1983) Alcohol and crime: Behavioral aspects, in: Kadish, S. H. (Ed.) *Encyclopaedia of Crime and Justice*, pp. 35–44 (New York, Free Press).

16. Lenke, L. (1990) *Alcohol and Criminal Violence — Time Series Analyses in a Comparative Perspective* (Stockholm, Almquist and Wiksell).

17. Cook, P. J. & Moore, M. J. (1993) Economic perspectives on reducing alcohol-related violence, in: Martin, S. E. (Ed.) *Alcohol and interpersonal Violence: Fostering Multidisciplinary Perspectives*, pp. 193–212 (NIAAA Research Monograph 24, NIH Publication No. 93–3496, Department of Health and Human Services).

18. Edwards, G., Anderson, P., Babor, T., Casswell, S., Ferrence, R., Giesbrecht, N., Godfrey, C., Holder, H., Lemmens, P., Makela, K., Midanik, L., Norstrom, T., Osterberg, E., Romelsjo, A., Room, R., Simpura, J. & Skog, O. (1994) *Alcohol*

policy and the public good (Oxford, Oxford University Press).

19. English, D., Hulse, G., Milne, E., Holman, C. & Bower, C. (1997) Maternal cannabis use and birth weight: A meta-analysis, *Addiction*, 92, 1553–1560.

20. Hall, W. & Zador, D. (1997) The alcohol withdrawal syndrome, *Lancet*, 349, 1857–1860.

21. Taylor, D. R., Poulton, R., Moffitt, T., Ramankutty, P. & Sears, M. (2000) The respiratory effects of cannabis dependence in young adults, *Addiction*, 95, 1669–1677.

22. Zhang, Z.-F., Morgenstern, H., Spitz, M., Tashkin, D., Yu, G.-P., Marshall, J., Hsu, T. & Schantz, S. (1999) Marijuana use and increased risk of squamous cell carcinoma of the head and neck, *Cancer Epidemiology, Biomarkers and Prevention*, 8, 1071–1078.

23. Andreasson, S., Allebeck, P., Engstrom, A. & Rydberg, U. (1987) Cannabis and schizophrenia: A longitudinal study of Swedish conscripts, *Lancet*, 2, 1483–1486.

24. Anthony, J. C., Warner, L. & Kessler, R. (1994) Comparative epidemiology of dependence on tobacco, alcohol, controlled substances, and inhalants: Basic findings from the National Comorbidity Survey, *Experimental and Clinical Psychopharmacology*, 2, 244–268.

25. Hall, W., Solowij, N. & Lemon, J. (1994) The health and psychological consequences of cannabis use (Canberra, Australian Government Publishing Service).

26. Bachman, J. G., Wadsworth, K. N., O'Malley, P., Johnston, L. & Schulenberg, J. (1997) *Smoking, drinking and drug use in young adulthood: The impacts of new freedoms and responsibilities* (Mahwah, NJ, Lawrence Erlbaum).

27. Der, G., Gupta, S. & Murray, R. (1990) Is schizophrenia disappearing?, *Lancet*, 1, 513–516.

28. Hall, W. (1995) The public health implications of cannabis use, *Australian Journal of Public Health*, 19, 235–242.

29. Holman, C., Armstrong, B. K., Arias, L. N., Martin, C. A., Hatton, W. M., Hayward, L. D., Salmon, M. A., Shean, R. E. & Waddell, V. P. (1988) The quantification of drug caused morbidity and mortality in Australia 1988 (Part I and Part II) (Canberra, Commonwealth Department of Community Services and Health).

30. Ridolfo, B. & Stevenson, C. (2001) *The quantification of drug-caused mortality and morbidity in Australia, 1998* (Canberra, Australian Institute of Health and Welfare).

31. Collins, D. & Lapsley, H. (1991) Estimating the economic costs of drug abuse in Australia (Canberra, Australian Government Publishing Service).

32. Collins, D. & Lapsley, H. (1996) The social costs of drug abuse in Australia in 1988 and 1992 (Canberra, Australian Government Publishing Service).

33. Murray, C. & Lopez, A. (1997) Global mortality, disability and the contribution of risk factors: Global Burden of Disease Study, *Lancet*, 349, 1436–1442.

SB

75

STATE OF ALASKA

DEPT. OF HEALTH AND SOCIAL SERVICES

OFFICE OF THE COMMISSIONER

FRANK H. MURKOWSKI, GOVERNOR

P.O. BOX 110601
JUNEAU, ALASKA 99811-0601
PHONE: (907) 465-3030
FAX: (907) 465-3068

January 24, 2004

Honorable Fred Dyson, Chairman
Senate Health, Education and
Social Services Committee
Alaska State Capitol; Rm. 121
Juneau, AK 99801

Dear Senator Dyson,

The Department of Health and Social Services respectfully requests a hearing in the Senate Health, Education, and Social Services Committee on Senate Bill 75 "An Act relating to public health and public health emergencies and disasters; relating to duties of the public defender and office of public advocacy regarding public health matters; relating to certain claims for public health matters; making conforming amendments; and providing for an effective date."

This bill is the culmination of a number of years work to develop a modern public health statute for Alaska.

A copy of Governor Murkowski's transmittal letter providing additional information on the bill and the associated fiscal note should be on file with the committee. The department is preparing a detailed sectional analysis of the bill that will be provided to your committee staff within the next several days.

Your favorable consideration of this request will be appreciated.

Sincerely,

Sherry Hill, Special Assistant
Office of the Commissioner

cc: Kevin Jardell, Legislative Director
Office of the Governor

Dr. Richard Mandsager Director
Division of Public Health

SB75

FRANK H. MURKOWSKI
GOVERNOR

GOVERNOR@GOV.STATE.AK.US

STATE OF ALASKA
OFFICE OF THE GOVERNOR
JUNEAU

P.O. BOX 110001
JUNEAU, ALASKA 99811-0001
(907) 465-3500
FAX (907) 465-3532
WWW.GOV.STATE.AK.US

January 20, 2005

The Honorable Ben Stevens
President of the Senate
Alaska State Legislature
Sta Capitol, Room 111
Juneau, AK 99801-1182

Dear President Stevens:

Under the authority of art. III, sec. 18, of the Alaska Constitution, I am transmitting a bill relating to public health and public health emergencies and disasters; relating to duties of the public defender and Office of Public Aadvocacy regarding public health matters; relating to certain claims for public health matters; and making conforming amendments.

Alaska's disease control laws were originally adopted by the Territorial Legislature in 1949. Some changes have been made to the laws since statehood. However, the recent severe acute respiratory syndrome crisis demonstrated the need to modernize them. Alaska is no longer protected from world disease outbreaks by geographical isolation. Modern air links rapidly put Alaskans at risk from infectious diseases originating on the other side of the globe. In a recent study, Alaska was noted as the only state in the nation with inadequate legal authority to respond to a public health emergency.

The Department of Health and Social Services (department) routinely uses the traditional public health disease control tools of epidemiological surveillance and investigation, and historically has used isolation and quarantine to stop the spread of disease in the rare times it has been warranted. Today, new global health threats, coupled with heightened expectations in the modern American social and legal environment for protection of individual rights, require the department to have more clearly defined legal authorities to act to protect the public while protecting the due process rights of infected individuals. This bill would give the department the needed flexibility to protect Alaskans from public health threats. The department would be authorized to offer medication to infected individuals who wish to take it. However, the department would not have authority to force medication upon infected individuals.

COMMITTEE COPY

The bill also would provide for powers to deal with public health issues that could arise in a declared disaster emergency.

I urge your support of this important bill.

Sincerely yours,

Frank H. Murkowski
Governor

Enclosure

FISCAL NOTE

STATE OF ALASKA
2005 LEGISLATIVE SESSION

Fiscal Note Number: 1
Bill Version: SB 75
(S) Publish Date: 1/21/05
Dept. Affected: Health & Social Services

Revision Date/Time (Note if correction):

Title: RELATING TO PUBLIC HEALTH AND PUBLIC HEALTH EMERGENCIES

RDU: Public Health

Component: Public Health Admin Svcs

Sponsor: (RLS) BY REQUEST OF THE GOVERNOR

Requester: GOVERNOR

Component No: 292

Expenditures/Revenues (Thousands of Dollars)

Note: Amounts do not include inflation unless otherwise noted below.

OPERATING EXPENDITURES	FY 2006	FY 2007	FY 2008	FY 2009	FY 2010	FY 2011
Personal Services						
Travel						
Contractual						
Supplies						
Equipment						
Land & Structures						
Grants & Claims						
Miscellaneous						
TOTAL OPERATING	0.0	0.0	0.0	0.0	0.0	0.0
CAPITAL EXPENDITURES						
CHANGE IN REVENUES (0)						

FUND SOURCE (Thousands of Dollars)

1002 Federal Receipts						
1003 GF Match						
1004 GF						
1037 GF/Mental Health						
Other(Specify Type-do not abbreviate)						
Other(Specify Type-do not abbreviate)						
TOTAL	0.0	0.0	0.0	0.0	0.0	0.0

Estimate of any current year (FY2005) cost:
Mark this box (X) if funding for this bill is included in the Governor's FY 2006 budget proposal:

POSITIONS

Full-time						
Part-time						
Temporary						

ANALYSIS: *(Attach a separate page if necessary)*

Passage of this legislation is not expected to have a budget impact on the Division of Public Health, as the bill simply clarifies legal authority and provides new due process provisions for programmatic activities already conducted by the Division. The bill does not add new functions or mandates to the Department of Health & Social Services' legal responsibilities.

Prepared by: Richard Mandsager, M.D. Phone 465-3090
Division: Public Health Date/Time 01/05/2005
Approved by: Joel S. Gilbertson, Commissioner Date 01/06/2005
Agency: Department of Health and Social Services

(Revised 9/23/2004 OMB)

COMMITTEE COPY

Sectional Analysis of HB 95/SB 75 (Public Health)

(Prepared by the Department of Law and the Department of Health and Social Services, January 25, 2005)

HB 95/SB 75 would clarify the Department of Health and Social Services' legal authority to detect and respond to a public health threat, including the authority to conduct testing, screening, and examination of individuals, as well as quarantine and isolation powers with court authority; and the authority to collect relevant data; the Department's powers are augmented in conjunction with the Department of Military and Veterans' Affairs when the governor declares a condition of disaster emergency related to public health; and legal representation and court powers are clarified with respect to court proceedings related to conditions of public health importance.

I. Purpose and Intent (Section 1):

Sec. 1: Section 1 sets out the purpose and intent of the bill.

II. Changes to kinds of claims that may not be brought against the state or its agents, officers, or employees (Section 2):

Sec. 2: Types of damage: Section 2 adds acts or omissions related to isolation, quarantine, medical treatment, or other actions taken under the state's public health authority and power to a list of damages for which an action may not be brought against the state or its agents, officers, or employees.

III. Repeal of statutes and changes to citations of repealed statutes (Sections 3, 6, and 12):

Sec. 3: Section 3 deletes a citation to a statute that would be repealed by the bill regarding tuberculosis screening of public school employees.

Sec. 6: Section 6 renumbers citations to reflect statutes that would be repealed by the bill regarding registry of person with impairments.

Sec. 12: Section 12 repeals certain statutes regarding registry of persons with impairments and regarding tuberculosis and other disease control.

IV. Changes to general section regarding the Department of Health and Social Services' administration of public health laws (Sections 4, 5, and 7):

Sec. 4: Section 4 rewrites the section on the administration of public health laws to modernize and more clearly and accurately reflect the Department of Health and Social Services' public health powers.

Sec. 5: Section 5 clarifies the nature of the regulations the Department of Health and Social Services is charged with adopting as regards reporting of conditions of public health importance and confidentiality of information received under provisions regarding public health authority and powers.

Sec. 7: Section 7 adds a definition of "condition of public health importance" to the chapter regarding the administration of public health laws.

V. Updates to the Department of Health and Social Services' public health powers and authority (Section 8):

Sec. 8: Section 8 adds new sections regarding the Department of Health and Social Services' public health authority and powers to the chapter dealing with disease control. These sections replace provisions for two disease-specific conditions (tuberculosis and SARS), repealed under sec. 12, and provide authority that is not specific to a particular disease. The new sections are described as follows:

- prevention and control of conditions of public health importance
- data collection
- requirement to maintain confidentiality of information obtained
- requirement to maintain list of reportable diseases
- power to conduct epidemiological investigation
- medical treatment powers and authority
- isolation and quarantine powers and authority
- powers in a public health disaster
- definitions

Section 8 also balances the state's public health powers with modernized due process provisions for protection of individual rights.

VI. Changes to legal representation and court powers (Sections 9-11):

Sec. 9: Section 9 amends the right of an indigent person to counsel to include when the person is subject to isolation, quarantine, testing, screening, or examination related to disease control. If eligible, such right to counsel may be provided by the Public Defender Agency.

Sec. 10: Section 10 gives magistrates and district court judges the power to issue orders related to testing, screening, and examination of individuals related to disease control.

Sec. 11: Section 11 expands the Office of Public Advocacy's responsibilities to include acting as guardian ad litem for individuals in court proceedings related to testing, screening, examination, isolation, and quarantine related to disease control.

VII. Effective date (Section 13):

Sec. 13: Section 13 sets out an immediate effective date for the bill.

State of Alaska

Department of Health & Social Services

Frank H. Murkowski
Governor
P.O. Box 110001
Juneau, Alaska 99811-0001

FACT SHEET

Joel Gilbertson
Commissioner
907-465-3030
FAX: 907-465-3068
www.hss.state.ak.us

January 21, 2005

Public Health Law Reform in Alaska

This legislation will ensure the Department has the appropriate legal authorities to protect and promote the public's health.

Issues / Background:

- Law is critically important to public health practice as it provides both the statutory framework within which governmental public health agencies operate, and the legal authorities required to monitor health status in communities, identify health threats, and act to control the spread of disease.
- Law is also an important tool of public health, in that it provides the vehicle for certain public policy strategies used to protect and promote health, for example, seat belt laws and tobacco tax laws.
- This public health law reform initiative deals with the first area of public health law only – the provision of the statutory framework and legal authorities for the public health agency to act.
- The Department's legal authorities for public health pose a problem in that they are both:
    - Antiquated – the basic enabling statute for public health (18.05.010) has not been updated since 1949 (10 years prior to statehood); and,
    - Layered – there are disease-specific laws which have been added-on over time to the general enabling statute, causing confusion and concern that the department does not have sufficient authority to detect and respond to future new threats to public health, such as the predicted influenza pandemic.
- A third problem posed by current public health law is that it does not provide for clear protections of individual rights in the event of a public health police power action.
- Twice in the past ten years, the Alaska legislature has been forced to act to fix State laws when public health authorities were questioned:
    - 1st in the mid-90's, when questions in the judicial system led to the addition of a new law detailing tuberculosis control procedures;
    - 2nd in the Spring of 2003 when concerns over the potential lack of quarantine authority for the new public health threat – SARS – resulted in rapid enactment of a new law for that particular disease.

-more-

Details:

- The proposed legislation will ensure that our State public health agency has:
  - a statutory framework that supports their mission, services and role;
  - clear authority for control of conditions of public health importance; and
  - established due process provisions for the protection of individual rights.

- The proposed legislation:
  - Defines "Essential Public Health Services" based on the nationally accepted description developed by the U.S. Public Health Functions Task Force
  - Describes the State's role in protection and promotion of the public's health
  - Provides clear authority for controlling of conditions of public health importance through:
    - Surveillance
    - Epidemiologic Investigation
    - Medical Treatment, Quarantine & Isolation
  - Requires protection of individual rights through modern due process provisions
  - Strengthens requirements for confidentiality and security of health records.
  - Adds new powers for the governor under the Alaska Disaster Act to enforce public health protection measures in the event of a declared disaster.

-30-

Contact: Richard Mandsager at 465-3090 or Deborah Erickson at 465-8615, Director's Office, Division of Public Health. Or email at: Richard_Mandsager@health.state.ak.us or Deb_Erickson@health.state.ak.us

24G-1
1/26/2005
(2:38 PM)

AMENDMENT

OFFERED IN THE SENATE HEALTH, EDUCATION AND SOCIAL SERVICES COMMITTEE

BY _____

TO: SB 75

Page 13, line 28, following "(g)," through page 13, line 31:

Delete all material.

Page 14, line 1:

Delete "the office of public advocacy to provide a guardian ad litem for the individual."

Page 17, following line 14:

Insert the following new material:

"**Sec. 18.15.389. Representation; guardian ad litem.** An individual who is the respondent in proceedings under AS 18.15.355 - 18.15.390 has the right to be represented by counsel in the proceedings. If the individual cannot afford an attorney, the court shall direct the public defender agency to provide an attorney. The court may, on its own motion or upon request of the individual's attorney or a party, direct the office of public advocacy to provide a guardian ad litem for the individual."

STATE OF ALASKA
DEPARTMENT of HEALTH & SOCIAL SERVICES
DIVISION of PUBLIC HEALTH

FRANK H. MURKOWSKI, GOVERNOR

OFFICE OF THE DIRECTOR
P.O. BOX 110610
JUNEAU, AK 99811-0610
PHONE: (907) 465-3090
FAX: (907) 465-4632

February 8, 2005

The Honorable Fred Dyson
Alaska State Senate
State Capitol Room 121
Juneau, AK 99801-1182

Dear Senator Dyson,

Thank you for taking the time to meet with me last week regarding SB 75, the public health bill, and SB 73, the bill that authorizes certificates of participation for the construction of a new virology laboratory in Fairbanks. I appreciated your advice, and also the opportunity to discuss some of the other issues in which we have a common interest.

I have an amendment to propose to SB 75 that clarifies the right counsel and authority to request guardian ad litem services. A description of the proposed amendment (enclosed) follows:

> The current bill provides the right to counsel for a person in a court action when the department asks a court for an order to quarantine or isolate that person to protect the public from substantial risk due to exposure to an infectious disease. Appointed counsel will be provided to the person if he or she can not afford their own. The bill also gives the court in such a proceeding authority to direct the Office of Public Advocacy to provide guardian ad litem services to the person upon the request of the person's attorney. These provisions are found in AS 18.15.385(g).
>
> The amendment would delete these provisions from AS 18.15.385(g) and insert a new statutory provision, AS 18.15.389, devoted solely to the issues of representation and guardian ad litem services. This section would expand the right to counsel to include proceedings brought pursuant to AS 18.15.375(e) (challenging ex parte testing orders) as well as isolation/quarantine provisions. The section would also give the court authority, on its own motion or at the request of a party, to direct the Office of Public Advocacy to provide guardian ad litem services for the person responding to the court proceeding. Current language in the bill would only allow the appointment of a guardian ad litem if requested by the lawyer of the person.

Also enclosed for the committee is a copy of the Sectional Analysis on SB 75 prepared by the Department of Law.

Thank you for your consideration and assistance in advancing the proposed amendment and sharing the Sectional Analysis with the HESS Committee members. Please contact me if you have any questions.

Sincerely,

Richard Mandsager/DLE

Richard Mandsager, M.D.
Director, Division of Public Health

enclosures

24G-1
2/8/05
(9:49 AM)

AMENDMENT

OFFERED IN THE SENATE HEALTH, EDUCATION AND SOCIAL SERVICES COMMITTEE

BY _____

TO: SB 75

Page 13, line 28, following "(g)," through page 13, line 31:

Delete all material.

Page 14, line 1:

Delete "the office of public advocacy to provide a guardian ad litem for the individual."

Page 17, following line 14:

Insert the following new material:

"**Sec. 18.15.389. Representation; guardian ad litem.** An individual who is the respondent in proceedings under AS 18.15.375(e) or 18.15.385 has the right to be represented by counsel in the proceedings. If the individual cannot afford an attorney, the court shall direct the public defender agency to provide an attorney. The court may, on its own motion or upon request of the individual's attorney or a party, direct the office of public advocacy to provide a guardian ad litem for the individual."

AMENDMENT

Offered in the Senate HESS Committee By: Senator
To: SB 75

Amending Section 5:
Page 6, line 17:

(4) the transportation of dead bodies; **except that the commissioner may not require that a dead body be embalmed unless the body is known to carry a communicable disease or embalmment is otherwise required for the protection of the public health or for compliance with federal law**;

...

STATE OF ALASKA
DEPT. of HEALTH and SOCIAL SERVICES
DIVISION of PUBLIC HEALTH

FRANK H. MURKOWSKI, GOVERNOR

P.O. BOX 110610
JUNEAU, AK 99811-0610
PHONE: (907) 465-3090, FAX: (907) 586-1877

February 4, 2005

The Honorable Fred Dyson
Alaska State Senate
State Capitol, Room 121
Juneau, AK 99801

The Honorable Kim Elton
Alaska State Senate
State Capitol, Room 115
Juneau, AK 99801

Dear Senators Dyson and Elton:

Thank you for your letter of January 25 on the issue of transportation of dead bodies in or out of Alaska. I appreciate your proposed legislation to clarify and strengthen the need to honor religious prohibitions regarding embalming. It has been the practice of the Department of Health and Social Services (DHSS) to grant such waivers when requested.

However, as your letter suggests, I believe DHSS can accomplish your goals by changing regulations – and that we already have the necessary statutory authority to do so.

It is my intent to work within DHSS and the Department of Law on a regulatory fix. Division of Public Health staff is checking to see how quickly we can adopt such changes in the Alaska Administrative Code.

I would be happy to meet with either or both of you on this matter, or to provide more information. Please feel free to contact me at any time. My office number is 465-3090. I look forward to working with you.

Sincerely,

Richard Mandsager, M.D
Director
Division of Public Health

Cc: Joel Gilbertson, Commissioner
Department of Health and Social Services

PUBLIC HEALTH

PROTECTING AND PROMOTING THE HEALTH OF ALL ALASKANS

SB 75: An Act Relating to Public Health

Presentation to the Senate HESS Committee

February 9, 2005

Richard Mandsager, M.D., Director
Alaska Department of Health & Social Services
Division of Public Health