REVIEW

Cannabis use by children and young people

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Arch Dis Child 2006;**91**:692–695. doi: 10.1136/adc.2005.071860

A popular internet site¹ describes cannabis as “a leafy plant, the leaves and flowering tops (buds) of which may be either smoked or eaten. It also comes in a more concentrated resinous form called hashish, and as a sticky black liquid called hash oil”. It is said that users often report a pleasant “subjective enhancement of visual and auditory perception, sometimes with synaesthesia (sounds take on visual colourful qualities)” and the sense that time passes more quickly than real time, a “fatuous euphoria”, as well as relaxation and relief from stress.²

As with other drugs of abuse, this pleasant effect is likely to be mediated and reinforced through the release of dopamine in “an evolutionarily ancient” reward circuit comprising the subcortical ventral tegmentum, nucleus accumbens, striatum, and the mesial prefrontal cortex.^{2–3} Cannabinoid receptors in the brain imply the existence of endogenous cannabinoids, which may modulate basal motor activity.² Because of receptor density in basal ganglia and cerebellum, cannabis is said to be associated with psychomotor effects on balance and fine motor control. Iverson² suggests that these motor effects may be implicated in the anecdotal accounts of relief from muscle pain in multiple sclerosis.

The rate of cannabis use by young people approximately doubled during the 1990s, perhaps tracking a more general rise in youth disaffection and disturbance.⁴ It may now have reached a plateau in Western Europe and North America (30–40% will have used it by mid-adolescence).⁵ Although it remains illegal, occasional cannabis use has been considered a normal activity of youth and is not strongly associated with emotional or behavioural disorder. However, although the exact location of the threshold is not clear, approximately half of those who use cannabis more than monthly exhibit behavioural or emotional difficulties.⁶ Also, some argue that adverse effects linked to cannabis have increased in recent times. This perception may relate to earlier onset of use, the use of water pipes or bongs to achieve more efficient delivery, preferential use by chronic users of the stronger preparations available, or use by more vulnerable individuals.^{7–8}

Since occasional use does not appear to be damaging, in the absence of any other difficulties enquired for, the clinician’s role may include reassurance of anxious teachers or parents. However, early cannabis use may represent a “gateway” to other illicit substances.^{9–10} Also,

more intensive use, or use by very young or pre-adolescents may be associated with a range of antecedent and concurrent difficulties.^{11–12} This “misuse”¹³ tends to occur in the context of antecedent behaviour disorder, substance using parents, family breakdown, and loss of trusting attachments to key adults. Among those with complex difficulties, the history of adversity may extend to abuse and neglect. Hence, clarifying the potential harm associated with cannabis use requires distinguishing between the effects of the drug itself from other co-occurring antecedent or persistent adversities. Indeed, regular use “...could be a marker, rather than a cause, of a life trajectory more likely to involve adverse outcomes”.¹⁴ Links between cannabis and anxiety or depressive symptoms¹⁵ or risk taking behaviours^{16–18} may represent this type of non-causal association.

However, cannabis misuse may itself be associated with harm. For instance, in keeping with frontal cortex effects, intoxication is linked with impaired vigilance, ability for “complex thought”,² memory,¹⁹ and other changes.²⁰ In keeping with these neuropsychological effects, among older adolescents, compared to measurements prior to initiation of use, there is evidence of a small drop in overall IQ among current but not previous heavy users. This may be due to the long half-life of cannabis and persistent subacute intoxication apparent for days after cessation.^{21–22} The impact of this may be most apparent among those whose performance was already marginal.²³ Also, prenatal exposure to cannabis may be associated with subtle lasting decrements in the cognitive performance of offspring, apparent well into adolescence, that appear to be specific to cannabis.^{24–25} Consequently, at a key time in their development, significant numbers²⁶ of often already disadvantaged urban youth across the world²⁷ may be coping with periods of subtle but sustained intellectual impairment of practical significance.²³ Indeed, it may be this rather than the more dramatic psychotic reaction which is the most important population effect of cannabis misuse.

In addition, approximately 10% of those who use cannabis appear ultimately to become dependent.^{28–29} This is linked with a withdrawal syndrome comprising mainly behavioural and emotional symptoms but without most of the physical effects associated with alcohol, sedative, or opiate withdrawal.³⁰ The syndrome requires near daily use to develop, and is more likely among those with other behaviour difficulties (who may be most likely to use heavily), but is less severe than its adult counterpart.³¹ Most symptoms are now said to onset within 24 hours of abstinence, peak within the first week, and

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Accepted 10 April 2006

last approximately 1–2 weeks, a time course apparently not dissimilar to other withdrawal syndromes.³² Although less intense, the symptomatic overlap with other withdrawal syndromes possibly reflects the shared underlying neuro-anatomy of addiction.³ Hence, together with association with antisocial peers, and financing difficulties, a degree of addiction may hinder the natural desistance of antisocial behaviour that usually occurs in early adulthood.³³

Higher mortality among current adult male users compared to non- or occasional users has been reported. This appears to be associated with HIV infection and may be explained by the higher rate of use by those engaged in risk behaviours.³⁴ The same authors reported increased mortality among female users from accidents which they linked to the psychomotor effects. Although there is low toxicity in overdose, perhaps attributable to the relative absence of cannabinoid receptors from the brain stem,² small children are not immune to accidental overdose from cannabis used in cooking.³⁵

One of the most fascinating recent controversies about cannabis is whether it can cause schizophrenia, a mental illness associated with hallucinations, delusions, and lasting impairment of social and intellectual functioning. Arguing that much of the data concerns symptoms rather than the illness, a recent report⁸ concluded that a causal relationship exists but that “at worst, use of cannabis increases the lifetime risk of schizophrenia by 1%”. This is conservative compared with other reviews^{36–37} or original data,^{38–43} pointing to an approximate doubling of the risk for schizophrenia symptoms, of the illness itself, and a probably even higher risk for younger users.^{38–41} Some argue that it is possible to calculate a “population attributable fraction” of 8% (8% of the cases would not occur if cannabis use was eliminated).³⁶ In light of the (as yet) unchanged epidemiology of schizophrenia, others suggest that cannabis is more likely to precipitate early a syndrome that is already likely or inevitable.⁴⁵ Ultimately, only the large army conscript studies^{42–43} have sufficient statistical power to examine prospectively the antecedents of schizophrenia itself and these trace young people only from 16 years. Nevertheless, from the data, reviews, and clinical experience, it may be realistic to view early onset of frequent use by already vulnerable young people as exposing them to the risk of a severe psychiatric disturbance which although perhaps potentially reversible (i.e. short of schizophrenia), may occur at a crucial time in their development and be potentially gravely disruptive to progress in relationships, education, or work. A so-called “gene environment interaction” whereby certain individuals may be more susceptible, may be implicated in this risk.⁴⁴

Childhood antecedents of schizophrenia include developmental deficits in executive function, language, and social and motor capacities that are more common in boys.^{46–47} These phenomena are also common among children with the early onset behaviour disorders often seen by community paediatricians and others in mental health services for children and young people.⁴⁸ Hence, until much more fine specification of the underlying vulnerability is possible, it appears that the childhood factors that are linked with later risk for cannabis abuse and with schizophrenia are not specific to either and mark high risk for a range of adverse outcomes. Since gender appears not to be an independent predictor of psychosis,³⁹ the fewer girls with these difficulties appear as vulnerable as boys. Hence, children with a range of pre-existing developmental difficulties (e.g. ADHD, learning, language, and social disabilities), or psychotic-like symptoms seem to be at significant risk and those with both could be particularly vulnerable to the effects of cannabis misuse. The one-in-seven young people who have experienced

“unpleasant psychotic like symptoms” (sometimes by early adolescence) (see box 1) should certainly avoid cannabis if they can be persuaded to do so. Even if young people do not experience these effects, “some of their peers may”, and so young people can be advised to avoid putting others under pressure to use.⁴⁵

The effectiveness of brief or minimal interventions in reducing adult alcohol use in at-risk individuals seems now to be established.^{49–50} Extrapolating from these findings, there is a good possibility that opportunistic screening for cannabis use (and other substances too, including tobacco and alcohol) followed by a brief, balanced, and scientifically informed discussion with the young person will reduce likelihood of escalating use. Since parents and carers (if available; their absence may be part of the young person’s predicament) are likely allies of the intervener, it is usually important to engage them too in the discussion, sensitively balancing the need for a degree of confidentiality. Young people may not confess the full extent of their misuse in front of parents, but they often acknowledge that some use has occurred (parents are often themselves partially aware), allowing some common ground. Clearly the ability to connect empathically with the young person, while avoiding the appearance of being yet another disapproving adult is crucial in these interviews.

The current research literature also describes a number of formal family based, cognitive behavioural, or group interventions^{51–52} for those with more complex presentations. The Cannabis Youth Treatment Study, concerning cannabis misusing adolescents who would “typically present to publicly funded outpatient treatment” and the largest and most comprehensive randomised trial to date, examined their relative benefit. However, it unexpectedly showed a broad similarity of effect.⁵³ The authors concluded that effectiveness may not in fact be tied to any specific technique but to “general factors [and]...systematic and structured interventions”. This is in keeping with recent reviews of psychological interventions for distressed and disturbed youth⁵⁴ and adults⁵⁵ and with the conclusions of treatment outcome investigations.^{56–57} These suggest that clinicians’ capacity to engage a young person and retain their active connection with (often an array of) services appears to capture key elements of effective psychosocial intervention.

Clinicians argue that this means working to develop a trusting relationship with the young person and using family and community resources in developmentally informed (by assessment of the young person’s development and circumstances) rehabilitation of affected youth.¹³ Using a carefully constructed and maintained doctor–patient relationship (acknowledging that it may require considerable aptitude to relate to some mistrustful and angry young people), it is often possible to negotiate realistic interim goals that are generic to adolescents in an array of predicaments, and which facilitate the conditions in which development can proceed. These may require “a strategic coordination”⁵⁸ involving

Box 1: Psychotic-like symptoms

“Hearing voices that other people do not hear; the idea that someone else can control your thoughts; other people being aware of your private thoughts; having thoughts that are not your own; having (odd) ideas and beliefs that others do not share; the idea that something is seriously wrong with your body; never feeling close to another person; the idea that something is wrong with your mind; feeling other people cannot be trusted; feeling that you are watched or talked about by others”^{11,39}

Box 2: Symptoms of withdrawal

- Craving
- Anxiety, depressed mood, irritability
- Trouble concentrating, restlessness, impaired sleep, loss of appetite^{30 31}

Box 3: Opportunistic intervention

Establish if use or misuse.

- If "use" only:
 - Offer opportunistic brief intervention based on accurate knowledge
 - Offer accurate information; also to parents if practicable
- If misuse, further screen for child care, child protection, educational and health related problems, and refer as appropriate

dialogue or links with family (balanced with confidentiality) and other services (with which the clinician will have become familiar) aimed at increased contact with non-using peers, ensuring child protection, access to educational, training, or other health (e.g. obstetric, genito-urinary medicine) services, and ultimately at the goal of a productive future. Interestingly, such an approach is reminiscent of the "complex array [of facilities and people]...that must be integrated across multiple venues over time" in the management of chronic disease.⁵⁹ Indeed, some argue that substance misuse in general should be conceived of as a chronic disease, which in severe cases it may well be.⁶⁰ However, unlike orthodox chronic disease, time (and maturation) may be the ally of clinicians and their young patients.

"Physical health has improved...over the last 50 years. Against expectation, psychosocial disorders (crime, suicide, suicide behaviours, depression, eating disorders, alcohol and drug abuse) have become significantly more prevalent".⁶¹ For doctors to remain relevant to youth, these phenomena and their implications should be grasped by medical schools, training programmes, and particularly by those with an interest in community child health or adolescent medicine. All doctors should be introduced to the current epidemiological realities, to the biology of substances, and to the basic skills required to relate to youth and families. Specialists need to have sound knowledge and skills in these areas. They should appreciate that, at least currently, cannabis is the most widely used and misused, and perhaps underestimated of substances of abuse and that this phenomenon is globalising. Faced with any adolescent cannabis use, training programmes should encourage screening for background serious problems impinging on care, education, morale, and mental health, as a routine part of a systems review. Discovering these, a competent doctor should be able to refer, either to a substance service they know to be competent in addressing the complex developmental needs of young people (not all are), or directly, for example to social services or education, acting themselves as a coordinator and advocate. This approach is not a radical departure, especially for paediatricians, but an adaptation of skills that are already in their armamentarium: in development, biology, systematic

understanding, coordination, experience of relating to young people and families in extremis, teamwork, a degree of tenacity, and, often neglected, physical health. In this way they can "...advocate the recognition and understanding of the social, psychological, and biological forces that are particularly impinging on young people and threatening to spoil young lives".⁶¹ They would be very welcome allies for the small group of child psychiatrists attempting to develop NHS services for an often disadvantaged, neglected, and widely disparaged population.

Competing interests: none declared

REFERENCES

- 1 http://www.erowid.org/plants/cannabis/cannabis_basics.shtml.
- 2 Iversen L. Cannabis and the brain. *Brain* 2003;**126**(pt 6):1252–70.
- 3 Nestler EJ, Malenka RC. The addicted brain. *Scientific American* 2004;**290**(3):78–85.
- 4 Prosser J, McArdle P. Secular trends in childhood mental disorder: a review. *Psychol Med* 1996;**26**:715–25.
- 5 Rodham K, Hawton K, Evans E, et al. Ethnic and gender differences in drinking, smoking and drug taking among adolescents in England: a self-report school-based survey of 15 and 16 year olds. *J Adolesc* 2005;**28**:63–73.
- 6 Meltzer H, Gatward R, Goodman R, et al. *Mental health of children and adolescents in Great Britain*. Office of National Statistics, 2000.
- 7 Hall W, Swift W. The THC content of cannabis in Australia: evidence and implications. *Aust N Z J Public Health* 2000;**24**:503–8.
- 8 http://www.drugs.gov.uk/publication-search/acmd/cannabis_reclass_2005.
- 9 Lynskey MT, Heath AC, Bucholz KK, et al. Escalation of drug use in early-onset cannabis users vs co-twin controls. *JAMA* 2003;**289**:427–33.
- 10 Lynskey M, Vink J, Boomsma D. Early onset cannabis use and progression to other drug use in a sample of Dutch Twins. *Behav Genet* (in press).
- 11 McArdle P. Substance abuse by children and young people. *Arch Dis Child* 2004;**89**:701–4.
- 12 McArdle P, Wieggersma A, Gilvarry E, et al. European adolescent substance use: the roles of family structure, function and gender. *Addiction* 2002;**97**:329–36.
- 13 Gilvarry E, Christian J, Crome I, et al. *The substance of young needs*. Health Advisory Service, 2001.
- 14 Macleod J, Oakes R, Copello A, et al. Psychological and social sequelae of cannabis and other illicit drug use by young people: a systematic review of longitudinal, general population studies. *Lancet* 2004;**363**:1579–88.
- 15 Goodwin RD, Fergusson DM, Horwood LJ. Association between anxiety disorders and substance use disorders among young persons: results of a 21-year longitudinal study. *J Psychiatr Res* 2004;**38**:295–304.
- 16 Viner R, Booy R. Epidemiology of health and illness. *BMJ* 2005;**330**:411–14.
- 17 Fergusson DM, Horwood LJ, Swain-Campbell N. Cannabis use and psychosocial adjustment in adolescence and young adulthood. *Addiction* 2002;**97**:1123–35.
- 18 Ramrakha S, Caspi A, Dickson N, et al. Psychiatric disorders and risky sexual behaviour in young adulthood: cross sectional study in birth cohort. *BMJ* 2000;**321**:263–6.
- 19 Solowij N, Stephens RS, Roffman RA, et al. Cognitive functioning of long-term heavy cannabis users seeking treatment. *JAMA* 2002;**287**:1123–31.
- 20 Skosnik PD, Spatz-Glenn L, Park S. Cannabis use is associated with schizotypy and attentional disinhibition. *Schizophr Res* 2001;**48**:83–92.
- 21 Fried P, Watkinson B, James D, et al. Current and former marijuana use: preliminary findings of a longitudinal study of effects on IQ in young adults. *CMAJ* 2002;**166**:887–91.
- 22 Pope HG Jr, Gruber AJ, Hudson JL, et al. Neuropsychological performance in long-term cannabis users. *Arch Gen Psychiatry* 2001;**58**:909–15.
- 23 Fergusson DM, Horwood LJ, Beutrais AL. Cannabis and educational achievement. *Addiction* 2003;**98**:1681–92.
- 24 Smith A, Fried P, Hogan M, et al. Effects of prenatal marijuana on response inhibition: an fMRI study of young adults. *Neurotoxicol Teratol* 2004;**26**:533–42.
- 25 Fried PA. Adolescents prenatally exposed to marijuana: examinations of facets of complex behaviours and comparisons with the influence of in utero cigarettes. *J Clin Pharmacol* 2002;**42**:97s–102s.
- 26 Zoccolillo M, Vitaro F, Tremblay RE. Problem drug and alcohol use in a community sample of adolescents. *J Am Acad Child Adolesc Psychiatry* 1999;**38**:900–7.
- 27 United Nations General Assembly. *Declaration on the guiding principles of drug demand reductions*, A/S-20/II. Vienna: United Nations, 1998.
- 28 Poulton RG, Brooke M, Moffitt TE, et al. Prevalence and correlates of cannabis use and dependence in young New Zealanders. *N Z Med J* 1997;**110**:68–70.
- 29 Fergusson DM, Horwood LJ. Cannabis use and dependence in a New Zealand birth cohort. *N Z Med J* 2000;**113**:156–8.
- 30 Budney AJ, Hughes JR, Moore BA, et al. Review of the validity and significance of cannabis withdrawal syndrome. *Am J Psychiatry* 2004;**161**:1967–77.
- 31 Vandrey R, Budney AJ, Kamon JL, et al. Cannabis withdrawal in adolescent treatment seekers. *Drug Alcohol Depend* 2005;**78**:205–10.

- 32 **Budney A**, Moore B, Vandrey R, *et al*. The time course and significance of cannabis withdrawal. *J Abnormal Psychol* 2003;**112**:393–402.
- 33 **Hussong A**, Curran P, Moffitt T, *et al*. Substance abuse hinders desistance in young adults' antisocial behavior. *Dev Psychopathol* 2004;**16**:1029–46.
- 34 **Sidney S**, Beck J, Tekawa I, *et al*. Cannabis and mortality. *Am J Public Health* 1997;**87**:585–90.
- 35 **Boros CA**, Parsons DW, Zoanetti GD, *et al*. Cannabis cookies: a cause of coma. *J Paediatr Child Health* 1996;**32**:194–5.
- 36 **Arseneault L**, Cannon M, Witton J, *et al*. Causal association between cannabis and psychosis: examination of the evidence. *Br J Psychiatry* 2004;**184**:110–17.
- 37 **Smit F**, Bolier L, Cuijpers P. Cannabis use and the risk of future schizophrenia. *Addiction* 2004;**99**:425–30.
- 38 **Arseneault L**, Cannon M, Poulton R, *et al*. Cannabis use in adolescence and risk for adult psychosis: longitudinal prospective study. *BMJ* 2002;**325**:1212–13.
- 39 **Fergusson DM**, Horwood LJ, Swain-Campbell NR. Cannabis dependence and psychotic symptoms in young people. *Psychol Med* 2003;**33**:15–21.
- 40 **Henquet C**, Krabbendam L, Spauwen J, *et al*. Prospective cohort study of cannabis use, predisposition for psychosis, and psychotic symptoms in young people. *BMJ* 2005;**330**:11.
- 41 **Stefanis N**, Delespaul P, Henquet C, *et al*. Early cannabis exposure and positive and negative dimensions of psychosis. *Addiction* 2005;**99**:1333–41.
- 42 **Zammit S**, Allebeck P, Andreasson S, *et al*. Self-reported cannabis use as a risk factor for schizophrenia in Swedish conscripts of 1969: historical cohort study. *BMJ* 2002;**325**:1199–201.
- 43 **Weiser M**, Reichenberg A, Rabinowitz J, *et al*. Self-reported drug abuse in male adolescents with behavioral disturbances, and follow-up for future schizophrenia. *Biol Psychiatry* 2003;**54**:655–60.
- 44 **Caspi A**, Moffitt T, Cannon M, *et al*. Moderation of the effect of adolescent-onset cannabis use on adult psychosis by a functional polymorphism in the catechol-o-methyltransferase gene: longitudinal evidence of a gene × environment interaction. *Biol Psychiatry* 2005;**57**:1117–27.
- 45 **Hall W**. The psychotogenic effects of cannabis use: challenges in reducing residual uncertainties and communicating the risks. *Addiction* 2004;**99**:511.
- 46 **Sporn A**, Addington A, Gogtay N, *et al*. Pervasive developmental disorder and childhood onset schizophrenia: comorbid disorder or a phenotypic variant of a very early onset disease? *Biol Psychiatry* 2004;**55**:989–94.
- 47 **Gochman P**, Greenstein D, Sporn A, *et al*. Childhood onset schizophrenia: familial neurocognitive measures. *Schizophr Res* 2004;**71**:43–7.
- 48 **McArdle P**. Attention-deficit hyperactivity disorder and life-span development. *Br J Psychiatry* 2004;**184**:468–9.
- 49 **Heather N**. The public health and brief interventions for excessive alcohol consumption: the British experience. *Addictive Behaviors* 1996;**21**:857–68.
- 50 **Crawford MJ**, Patton R, Touquet R, *et al*. Screening and referral for brief intervention of alcohol-misusing patients in an emergency department: a pragmatic randomised controlled trial. *Lancet* 2004;**364**:1334–9.
- 51 **Waldron HB**, Kaminer Y. On the learning curve: the emerging evidence supporting cognitive-behavioral therapies for adolescent substance abuse. *Addiction* 2004;**99**(suppl 2):93–105.
- 52 **Liddle H**, Dakof G, Parker K, *et al*. Multidimensional family therapy for adolescent drug abuse: results of a randomized clinical trial. *Am J Drug Alcohol Abuse* 2001;**27**:651–88.
- 53 **Dennis M**, Godley S, Diamond G, *et al*. The cannabis youth treatment stud: main findings from two randomised trials. *Journal of Substance Abuse Treatment* 2004;**27**:197–213.
- 54 **Jensen PS**, Weersing R, Hoagwood KE, *et al*. What is the evidence for evidence-based treatments? A hard look at our soft underbelly. *Mental Health Services Research* 2005;**7**:53–74.
- 55 **Westen D**, Novotny CM, Thompson-Brenner H. The empirical status of empirically supported psychotherapies: assumptions, findings, and reporting in controlled clinical trials. *Psychol Bull* 2004;**130**:631–63.
- 56 **Hser YI**, Evans E, Huang D, *et al*. Relationship between drug treatment services, retention, and outcomes. *Psychiatric Services* 2004;**55**:767–74.
- 57 **Hser YI**, Grella CE, Hubbard RL, *et al*. An evaluation of drug treatments for adolescents in 4 US cities. *Arch Gen Psychiatry* 2001;**58**:689–95.
- 58 **Royal College of Paediatrics and Child Health**. *Bridging the gaps: health care for adolescents*. Royal College of Paediatrics and Child Health, 2003.
- 59 **Lawrence DM**. Chronic disease care: rearranging the deck chairs. *Ann Intern Med* 2005;**143**:458–9.
- 60 **McLellan AT**, Lewis DC, O'Brien CP, *et al*. Drug dependence, a chronic medical illness: implications for treatment, insurance, and outcomes evaluation. *JAMA* 2000;**284**:1689–95.
- 61 **Royal College of Paediatrics and Child Health**. *Strengthening the care of children in the community*. Royal College of Paediatrics and Child Health, 2002.