

POSITION PAPER

Respiratory health effects of cannabis: Position Statement of The Thoracic Society of Australia and New Zealand

D. R. TAYLOR¹ and W. HALL^{2,3}

¹*Department of Medical and Surgical Sciences, Dunedin School of Medicine, University of Otago, Dunedin, New Zealand,* ²*National Drug and Alcohol Research Centre, University of New South Wales, Sydney, New South Wales* and ³*Office of Public Policy and Ethics, Institute of Molecular Bioscience, University of Queensland, Brisbane, Queensland, Australia*

Abstract

Both the gaseous and the particulate phases of tobacco and cannabis smoke contain a similar range of harmful chemicals. However, differing patterns of inhalation mean that smoking a 'joint' of cannabis results in exposure to significantly greater amounts of combusted material than with a tobacco cigarette. The histopathological effects of cannabis smoke exposure include changes consistent with acute and chronic bronchitis. Cellular dysplasia has also been observed, suggesting that, like tobacco smoke, cannabis exposure has the potential to cause malignancy. These features are consistent with the clinical presentation. Symptoms of cough and early morning sputum production are

common (20–25%) even in young individuals who smoke cannabis alone. Almost all studies indicate that the effects of cannabis and tobacco smoking are additive and independent. Public health education should dispel the myth that cannabis smoking is relatively safe by highlighting that the adverse respiratory effects of smoking cannabis are similar to those of smoking tobacco, even although it remains to be confirmed that smoking cannabis alone leads to the development of chronic lung disease. (*Intern Med J* 2003; 33: 310–313)

Key words: cannabis, lung function, respiratory disease.

INTRODUCTION

There is considerable debate about the legal status of cannabis. In most countries the cultivation, sale and use of cannabis is illegal and carries criminal penalties. More recently, others have decriminalized but not legalized its use. The debate focuses on discrepancies between the legal status of cannabis and both alcohol and tobacco; whether it is a 'gateway' drug leading to use of other illicit substances; and associations between cannabis use and mental illness and criminal behaviour.

Tetrahydrocannabinol (THC) has widespread pharmacological effects, notably in the central nervous system.¹ Less attention is given to the adverse effects of cannabis smoking on the lungs, where the effects of THC are perhaps of less importance than the numerous products of combustion to which smokers are exposed. There is a widespread perception that compared to tobacco, smoking cannabis is relatively free from harm.² This myth has gained ground for a number of reasons. First, compared to tobacco smoking there are fewer individuals who smoke cannabis, although this pattern has changed: in New Zealand, 9.7% of 21-year-olds were

smoking cannabis regularly compared to 28.1% who were smoking tobacco.³ The figures are similar for Australia.⁴ The rates for older adults are probably lower (1–3%),⁵ and whether younger individuals will continue to smoke cannabis into and beyond mid-adult life is not clear. For these reasons, diagnosing patients with cannabis-related pulmonary pathology is still uncommon. Second, compared to the large body of evidence for the adverse effects of tobacco smoking, there have been few studies of sufficient duration to assess the long-term effects of cannabis smoke exposure. Third, the effects of cannabis smoking are frequently confounded: a significant proportion of cannabis smokers are also concurrent tobacco smokers (69% in one study³), making it difficult to separate the independent effects of cannabis and tobacco smoking.

Such evidence as does exist strongly suggests that smoking cannabis will result in a similar range of adverse effects in the lungs to that of tobacco smoking. This is the basis for informing health professionals, parents, teachers and politicians as well as groups such as 'risk-taking' adolescents that the respiratory hazards of smoking cannabis are significant.

The Thoracic Society of Australia and New Zealand is committed to promoting a smoke-free society so that the adverse effects of tobacco smoking will eventually be reduced. The purpose of the present paper is to summarize the evidence that, after taking differing levels of consumption into account, regular cannabis smoking also poses risks to respiratory health.

Correspondence to: D. Robin Taylor, Dunedin School of Medicine, PO Box 913, Dunedin, New Zealand.

Email: robin.taylor@stonebow.otago.ac.nz

Received 11 February 2002; *accepted* 22 July 2002.

Funding: None

Conflicts of interest: None

RECOMMENDATIONS

1 *The constituents of cannabis and tobacco smoke include a similar range of pro-inflammatory and carcinogenic substances (Level I)*

Analysis of the constituents of mainstream cannabis and tobacco smoke reveals that both the gaseous and the particulate phases contain a range of harmful chemicals. Although the pharmacologically active ingredients (nicotine and cannabinoids) differ, the 'tar content', including carcinogens such as vinyl chloride, dimethyl- and methyl-ethylnitrosamine, benzathracene and benzpyrene, is comparable for both types of smoke.⁶ This is the primary reason for asserting that the effects of inhaling the products of combustion of the two materials are likely to be similar.

Assessing the cumulative exposure of cannabis smokers to these toxins is difficult. Tobacco cigarettes are of a relatively standard size but this is not the case for cannabis. Second, the deposition and absorption of the products of cannabis combustion in the lung depends on patterns of inhalation and breath holding time. For cannabis, the depth of inhalation is greater and the breath holding time is longer. In one study, carbon monoxide levels and tar deposition were five times and four times greater, respectively, after smoking a single cannabis 'joint' than after a tobacco cigarette.⁷ Thus consumption as measured by frequency of exposure (numbers of cigarettes or 'joints' smoked) does not necessarily reflect the extent of exposure to noxious materials.

2 *The histopathological effects of cannabis smoke inhalation are similar to those of tobacco smoke (Level 1); although premalignant histological changes may also occur, there are few data as yet to confirm that cannabis smoking causes malignancy in the respiratory tract (Level 3)*

Studies have demonstrated that, even after limited exposure to cannabis smoke, airway inflammation develops.⁸⁻¹¹ Two studies have evaluated the histopathological features associated with cannabis smoke exposure using mucosal biopsy and bronchoalveolar lavage; both have yielded similar results.^{10,11} Features consistent with chronic bronchitis including oedema, vascular hyperplasia, inflammatory cell infiltration and goblet cell hyperplasia occurred. These changes were comparable to those that occurred with tobacco smoke. Strikingly, the degree of abnormality was similar among cannabis and tobacco smokers despite the fact that tobacco smokers' frequency of exposure was four to fivefold more than that of cannabis smokers. This emphasizes that the method of inhalation is critical in enhancing the relative injury caused by cannabis smoke.¹¹

These studies also found evidence of cellular dysplasia, raising the possibility that cannabis smoking may result in malignancy in the longer term. Cannabis smoke is also mutagenic.^{12,13} There have been case reports that heavy cannabis use is associated with cancer of the tongue and lung.^{14,15} More recently, case control¹⁶ and cohort studies¹⁷ have yielded conflicting

data regarding the risk of malignancy with cannabis, probably because of selection bias and age discrepancies.¹⁸ On balance, data to date suggest that cannabis smokers may be at increased risk of developing malignancy particularly if they also smoke tobacco cigarettes. Given the long latency between the onset of exposure and the advent of malignancy for tobacco smoke, the risks are likely to be similar for those who continue to smoke cannabis regularly.

3 *Acute exposure to cannabis smoke results in small decrements in lung function accompanied by respiratory symptoms (Level 2)*

Early studies confirm that even after short-term but fairly heavy exposure to cannabis smoke, small but significant changes in lung function occur.¹⁹ There was a mean decline in forced expiratory volume in 1 s (FEV₁) of 3%, and there were dose-dependent changes in maximum mid-expiratory airflow and airway conductance in a group of 28 healthy men who smoked approximately five joints daily for 6-8 weeks. The changes in lung function were reversible after cessation.

Symptoms of cough and sputum production occur in approximately 20-25% of cannabis smokers. Wheeze, exercise-related dyspnoea and nocturnal symptoms occur more frequently than in non-users.²⁰⁻²³ These symptoms develop even in young individuals after a short period of exposure, and probably reflect acute rather than chronic histopathological effects. Lung function is usually normal at this stage.²³

4 *With longer term exposure, tobacco and cannabis smoking have additive effects on lung function and respiratory symptoms (Level 2)*

There are now sufficient data to conclude that the effects of chronic cannabis exposure in the lungs are independent of tobacco smoking. Cross-sectional studies record small but significant negative changes in airway function with regular cannabis smoking.^{20,22-24} Almost all these studies indicate that cannabis exposure and tobacco smoking have additive effects.

There are fewer longitudinal data, and here the evidence is conflicting. In one study the small but significant decrement in FEV₁ in cannabis users was approximately twice that attributable to tobacco smoking.²¹ In contrast, in a study comparing heavy cannabis-only smokers (>3 joints per day) and tobacco-only smokers with non-smoking controls, only tobacco smoking was associated with decline in FEV₁ over an 8-year interval.²⁵ The discrepancy between these results may be due to differences in the populations studied, and clearly further longitudinal data are required. It is possible that, as for tobacco smoking, a small but significant proportion of chronic cannabis smokers will exhibit decline in lung function in association with the development of chronic obstructive pulmonary disease. This seems likely given that the short- to medium-term effects of cannabis and tobacco exposure are so similar. Again, longitudinal data are needed to address this issue.

It has been reported that the frequency of respiratory infections is significantly higher among cannabis users

than non-smoking controls.²⁶ Two mechanisms have been adduced to explain this finding. First, it may be due to impairment of mucociliary clearance associated with the histopathological changes in airway mucosa discussed previously. Second, there are potential changes in local immunological competence which predispose to bacterial invasion.^{27,28} These hypotheses remain to be tested.²⁹

OTHER ISSUES

It is claimed that inhalation of cannabis smoke via a water pipe ('bong') may reduce the exposure to potentially toxic materials. However, there appears to be no significant reduction in risk with this modified inhalation technique. Indeed, the relative amount of tar being delivered to the airways (expressed as a proportion of inhaled THC) is similar.³⁰

It is recognized that patients with a history of psychiatric problems are much more likely to be tobacco smokers than subjects without such a history,³¹ and they are at greater risk of smoking-related respiratory disease. In addition, there is also a link between psychiatric illness and cannabis use,¹ indicating that this particular subgroup may be at particular risk of respiratory disease with prolonged exposure to both tobacco and cannabis smoke.

CONCLUSIONS AND RECOMMENDATIONS

All the available evidence suggests that the risks of regular cannabis smoking are similar to those of regular tobacco smoking. Similarities between the combustion products of cannabis and tobacco, as well as their acute and subacute pathological effects in regular smokers, make this biologically plausible. The fact that the long-term sequelae of cannabis smoking have not been clearly documented is largely due to the decline in cannabis use that has hitherto occurred during early adult life, possibly as a result of its illegal status in most countries. This pattern may change if its legal status changes.

The Thoracic Society of Australia and New Zealand actively promotes a smoke-free environment as an important public health measure to improve and maintain respiratory health. Legislation and policies towards cannabis should be formulated in the light of our knowledge about its harmful effects on respiratory health. The burden of respiratory ill health will inevitably increase if consumption increases particularly in the longer term. As for tobacco, every step should be taken to discourage cannabis smoking. To this end, public health education should include the message that the respiratory effects of smoking cannabis are similar to those of tobacco.

REFERENCES

- Hall W, Degenhardt L, Lynskey M. The health and psychological effects of cannabis use. In: National Drug Strategy. Monograph Series no. 25. Canberra: Commonwealth Department of Health and Aged Care, Commonwealth of Australia, 2001.
- Anonymous. Marijuana; special report. *New Scientist* 1998; 2122.
- Poulton RG, Brooke M, Moffitt TE, Stanton WR, Silva PA. Prevalence and correlates of cannabis use and dependence in young New Zealanders. *NZ Med J* 1997; 110: 68–70.
- Hall W, Johnston L, Donnelly N. Epidemiology of cannabis and its consequences. In: Kalant H, Corrigall W, Hall W, Smart R, eds. *The Health Effects of Cannabis*. Toronto: Centre for Addiction and Mental Health, 1999; 78–9.
- Hall W. The public health implications of cannabis use. *Aust J Public Health* 1995; 19: 235–42.
- Institute of Medicine. *Marijuana and Medicine: Assessing the Science Base*. Washington DC: National Academy Press, 1999.
- Wu TC, Tashkin DP, Djahed B, Rose J. Pulmonary hazards of smoking marijuana as compared with tobacco. *N Engl J Med* 1988; 318: 347–51.
- Gong H, Fligiel S, Tashkin DP, Barbers RG. Tracheobronchial changes in habitual heavy smokers of marijuana with and without tobacco. *Am Rev Respir Dis* 1987; 136: 142–9.
- Fligiel SEG, Venkat H, Gong H, Tashkin DP. Bronchial pathology in chronic marijuana smokers: a light electron microscopy study. *J Psychoactive Drugs* 1988; 20: 33–42.
- Fligiel SEG, Roth MD, Kleerup EC, Barsky SH, Tashkin DP. Tracheobronchial histopathology in habitual smokers of cocaine, marijuana and/or tobacco. *Chest* 1997; 112: 319–26.
- Roth MD, Arora A, Barsky SH, Kleerup EC, Simmons M, Tashkin DP. Airway inflammation in young marijuana and tobacco smokers. *Am J Respir Crit Care Med* 1998; 157: 928–37.
- Leuchtenberger C. The effects of cannabis smoke on cellular biochemistry of in vitro test systems. In: Fehr KO, Kalant H, eds. *Cannabis and Health Hazards*. Toronto: Addiction Research Foundation, 1983.
- Marselos M, Karamanakis P. Mutagenicity, developmental toxicity and carcinogenicity of cannabis. *Addiction Biol* 1999; 4: 5–12.
- Caplan GA, Brigham BA. Marijuana smoking and carcinoma of the tongue. Is there an association? *Cancer* 1989; 66: 1005–6.
- Sridar KS, Raub WA, Weatherby NL. Possible role of marijuana smoking as a carcinogen in the development of lung cancer at an early age. *J Psychoactive Drugs* 1994; 26: 285–8.
- Sidney S, Beck JE, Tekawa IS, Quesenbery CP, Friedman GD. Marijuana use and mortality. *Am J Public Health* 1997; 87: 585–90.
- Zhang ZF, Morgenstern H, Spitz M. Marijuana use and increased risk of squamous cell carcinoma of the head and neck. *Cancer Epidemiol Biomarkers Prev* 1999; 8: 1071–8.
- Hall W, MacPhee D. Cannabis use and cancer. *Addiction* 2002; 97: 243–7.
- Tashkin DP, Shapiro BJ, Lee YE, Harper CE. Subacute effects of heavy marijuana smoking on pulmonary function in healthy men. *N Engl J Med* 1976; 294: 125–9.
- Bloom JW, Kaltenborn WT, Paoletti P, Camilli A, Lebowitz MD. Respiratory effects of non-tobacco cigarettes. *BMJ* 1987; 295: 1516–18.
- Sherrill DL, Kryzanowski M, Bloom JW, Lebowitz MD. Respiratory effects of non-tobacco cigarettes; a longitudinal study in the general population. *Int J Epidemiol* 1991; 20: 132–7.
- Tashkin DP, Coulson AH, Clark VA, Simmons M, Bourque L, Duann S *et al.* Respiratory symptoms and lung function in habitual heavy smokers of marijuana alone, smokers of marijuana and tobacco, smokers of tobacco alone and non-smokers. *Am Rev Respir Dis* 1987; 135: 209–16.
- Taylor DR, Poulton RG, Moffitt TE, Ramankutty P, Sears MR. The respiratory effects of cannabis dependence in young adults. *Addiction* 2000; 95: 1669–77.

- 24 Tashkin DP, Calvarese BM, Simmons MS, Shapiro BJ. Respiratory status of seventy four habitual marijuana smokers. *Chest* 1980; 78: 699–706.
- 25 Tashkin DP, Simmons MS, Sherrill DL, Coulson AH. Heavy marijuana smoking does not cause an accelerated decline in FEV₁ with age. *Am J Respir Crit Care Med* 1997; 155: 141–8.
- 26 Polen MR, Sidney S, Tekawa IS, Sadler M, Friedman GD. Health care use by frequent marijuana smokers who do not smoke tobacco. *West Med J* 1993; 158: 596–601.
- 27 Baldwin GC, Tashkin DP, Buckley D. Habitual smoking of marijuana and cocaine impairs alveolar macrophage function and cytokine production. *Am J Respir Crit Care Med* 1997; 156: 1606–13.
- 28 Sherman MP, Campbell LA, Gong H. Antimicrobial and respiratory burst characteristics of pulmonary alveolar macrophages recovered from smokers of marijuana alone, smokers of tobacco alone, smokers of marijuana and tobacco, and non-smokers. *Am Rev Respir Dis* 1991; 144: 1351–6.
- 29 Hall W, Solowij N. Adverse effects of cannabis. *Lancet* 1998; 352: 1611–16.
- 30 Doblin R. The MAPS/California NORMAL marijuana waterpipe/vaporiser study. *Newsletter Multidisciplinary Assoc Psychedelic Studies* 1994; 5: 19–22.
- 31 Lasser K, Boyd JW, Woolhandler S, Himmelstein DU, McCormick D, Bor D. Smoking and mental illness: a population based prevalence study. *JAMA* 2000; 284: 2606–10.