The impact of marijuana smoking on lung function

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As marijuana becomes more widely used, and is legalised in several jurisdictions, the question of whether regular marijuana smoking has a deleterious effect on respiratory health, similar to tobacco smoking, has been a major topic of investigation. While multiple studies indicate the potential for development of respiratory symptoms reflecting airway inflammation and chronic bronchitis (dominantly cough, wheeze, and sputum production), studies reporting detailed longitudinal lung function assessments have been substantially fewer.

A systematic review of 22 studies concluded that there is low-strength evidence that smoking marijuana is associated with cough, sputum production and wheezing, but evidence on the association between marijuana use and chronic obstructive lung disease and pulmonary function was judged to be insufficient [1]. However, there is little doubt that marijuana use sufficient to bring users into contact with healthcare providers is associated with respiratory symptoms. A patient-centred observational study using electronic health records of 8932 patients with cannabis abuse or dependence or at least two cannabis positive urine drug screens, matched with non-cannabis-using patients, found that cannabis use was associated with a greater risk for asthma, COPD and pneumonia, regardless of whether or not they had a concomitant tobacco-use disorder [2].

With respect to lung function, Ribiero and Ind [3] reviewed 19 studies conducted among marijuana smokers, involving from 23 to over 7000 individuals. While the findings for airflow obstruction vary, with some showing lower forced expiratory volume in 1 s (FEV_1) to forced vital capacity (FVC) ratios among marijuana smokers compared with controls, other studies have not found changes to the FEV_1/FVC ratio, despite symptoms of bronchitis. The explanation for this appears to be an effect on FVC: several studies show that marijuana is associated with a higher FVC, little effect on FEV_1 and as a consequence a lower ratio of FEV_1 to FVC. A tendency to higher total lung capacity and greater airway resistance and lower conductance has also been noted in several studies, including the Dunedin longitudinal cohort study [4]. Airway responsiveness to methacholine or histamine has been examined in a few studies but no significant effect found.

In the Coronary Artery Risk Development (CARDIA) study of over 5000 individuals, marijuana smoking was non-linearly associated with lung function, unlike tobacco smoking. They reported an increase in FEV_1 over time up to 7 joint-years, and then a tendency to decline, while FVC increased even in heavy users up to 20 joint-years [5]. In another large population-based study from NHANES, an association with

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airflow obstruction (FEV_1/FVC <70%) was found only in those with over 20 joint-years exposure [6]. The SPIROMICS study of over 2000 subjects, of whom 43% were former and 8% current marijuana smokers, reported a higher FEV_1 and FVC but no relation overall with symptoms apart from more symptoms of chronic bronchitis in heavy users [7]. On the other hand, a cross-sectional study in general practice in Scotland of some 500 individuals found a 0.3% increase in the prevalence of COPD for every joint year of marijuana smoking [8].

Given this relative lack of data, and conflicting results among the larger studies, the report in this issue using data from a Canadian study of lung health is of considerable interest. Tan *et al.* [9] present data collected in two phases of a population-based study examining symptoms and lung function in smokers and non-smokers of cigarettes and marijuana. A cross-sectional population-based study (the Canadian Obstructive Lung Disease (COLD) study) involved over 5200 randomly selected participants aged over 40 years (mean 59 years), from which 1285 selected subjects (enriched for COPD, mean age 65 years) entered a subsequent longitudinal follow-up period (the Canadian Cohort of Obstructive Lung Disease (CanCOLD) study) with assessments every 18 months for 3 years. Smoking prevalence rates in the two studies were similar: 36–44% smoked tobacco only, only 3% in each study smoked marijuana only, and 16–17% had smoked both tobacco and marijuana. Of all marijuana smokers, over 80% had also smoked tobacco. The analyses of the effects of marijuana therefore rely heavily on statistical adjustments for categories of tobacco consumption.

Table 1 of the paper of Tan *et al.* [9] suggests no adverse effect of marijuana-only smoking on lung function or symptoms compared with non-smokers of either substance, with slightly higher FEV_1 and FVC values, and a virtually identical FEV_1/FVC ratio to non-smokers. Table 2, reporting on subjects in the longitudinal study, similarly shows no effect in marijuana-only smokers but the expected lung function impairments in tobacco smokers. Figures 2a and 2b showing the dose responses to smoking of marijuana and tobacco are of particular interest; while there is a clear dose–response reduction in lung function with tobacco smoking, this is not evident for marijuana where light smoking (<5 joint-years) apparently was associated with a modest decline in FEV_1, moderate use (5–20 joint years) appears to be beneficial with an FEV_1 trajectory equal or better than in non-smokers, whereas heavy marijuana smoking (over 20 joint-years) was deleterious.

There are a number of important limitations to the study, including significant differences in age, sex distribution, and education between marijuana smokers and tobacco smokers which contribute to some of the difficulties in interpretation of their findings. The very small number of marijuana-only smokers (181 in the baseline cohort and only 33 at the onset of follow-up of the longitudinal cohort) and the substantial dropout rate of over 45% in the longitudinal study are of concern. There were only three heavy smokers in the marijuana only group. Marijuana and dual marijuana and tobacco smokers were younger than tobacco-only smokers, and most marijuana smokers had already quit by the time that the baseline cohort was assessed. Interpreting the associations noted as "effects" of marijuana on lung function requires us to speculate that marijuana use has long-term effects on lung function that persist or even accelerate long after quitting use. Such an effect may be possible, but is inconsistent with evidence that the airway symptoms related to marijuana use resolve on quitting [10, 11].

Could there be a healthy smoker effect which allows those who do not develop symptoms from smoking marijuana to smoke moderately and yet have preserved lung function, while those with light consumption were more susceptible, developed symptoms and reduced lung function, which limited their consumption or influenced the decision to quit? This question cannot be answered from these data but could be assessed in future, more detailed studies.

Cohort studies with more robust designs and follow-up data are mostly restricted to younger populations and the effects of marijuana smoking on lung function in older adults are largely unexplored, in part because adults tend to quit marijuana use before middle age. Whether this pattern of marijuana use will change with legalisation remains to be seen. Extrapolating findings from younger adults into the risk of COPD in older populations is difficult because of the demonstrated effect of marijuana smoking on FVC: for a given percent predicted FEV_1 value, marijuana smokers are more likely to meet criteria for COPD because they have higher FVC values, and hence a lower FEV_1/FVC ratio [3, 4, 12]. We do not yet know what happens to FVC (or FEV_1) values when marijuana smokers quit. The CanCOLD study raises the possibility that the long-term consequences may be clinically important and this needs urgent investigation.

While the study of Tan *et al.* [9] confirms that heavy tobacco smoking is harmful, and suggests that heavy current marijuana smoking adds to the decline in function, there are a number of uncertainties that make quantification of the lung function effects of marijuana alone difficult to judge. In common with many previous studies, the great majority of marijuana smokers were also tobacco smokers, making statistical adjustments to control for this combined effect both necessary, but also problematic. Given the increasing

use of marijuana with legalisation in many jurisdictions, more research is needed to fully evaluate the effects of marijuana smoking on lung function.

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