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Effects of cannabis on lung function: a population-based cohort study

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ABSTRACT: The effects of cannabis on lung function remain unclear and may be different from those of tobacco. We compared the associations between use of these substances and lung function in a population-based cohort (n=1,037).

Cannabis and tobacco use were reported at ages 18, 21, 26 and 32 yrs. Spirometry, plethysmography and carbon monoxide transfer factor were measured at 32 yrs. Associations between lung function and exposure to each substance were adjusted for exposure to the other substance.

Cumulative cannabis use was associated with higher forced vital capacity, total lung capacity, functional residual capacity and residual volume. Cannabis was also associated with higher airway resistance but not with forced expiratory volume in 1 s, forced expiratory ratio or transfer factor. These findings were similar among those who did not smoke tobacco. In contrast, tobacco use was associated with lower forced expiratory volume in 1 s, lower forced expiratory ratio, lower transfer factor and higher static lung volumes, but not with airway resistance.

Cannabis appears to have different effects on lung function from those of tobacco. Cannabis use was associated with higher lung volumes, suggesting hyperinflation and increased large-airways resistance, but there was little evidence for airflow obstruction or impairment of gas transfer.

KEYWORDS: Cannabis, cohort study, marijuana, respiratory function, smoking, tobacco

The pulmonary effects of smoking cannabis have not been extensively researched. In common with tobacco, smoking cannabis is associated with airway inflammation and symptoms of bronchitis, although the evidence that it causes airflow obstruction is not conclusive [1–5]. Among the reasons for this continuing uncertainty are its illegal status, making it difficult to obtain reliable estimates of cannabis exposure, and the common practice of combining cannabis with tobacco, which makes it difficult to separate the effects of the two substances [6]. Thus, although cannabis is widely used throughout the world, there is a paucity of information on its respiratory effects.

Apart from the respective psychoactive components of cannabinoids and nicotine, cannabis and tobacco smoke contain a similar mix of toxic and irritant chemicals [7]. However, there are reasons to suspect that their effects on the respiratory system may not be the same. Cannabis smokers tend to smoke fewer cigarettes a day than tobacco smokers, but these tend to be packed more loosely and unfiltered. Differences in depth of inhalation, breath-hold time and leaving a shorter butt may increase the deposition of tar and carbon monoxide absorption from cannabis smoke [8–10]. Several case reports of bullous lung disease in young cannabis smokers raise the possibility that cannabis (or the techniques used to smoke it) may have a greater effect on lung parenchyma than tobacco [11–13], although this association has been disputed [14]. A recent report comparing smokers of cannabis and tobacco found that, although both cannabis and tobacco smokers had evidence of airflow obstruction on spirometry, cannabis was associated with more lung hyperinflation on lung volume measurement but a lower risk of emphysema on computed tomography (CT) scanning than tobacco [15]. Although these findings do not support the suggestion that cannabis smokers are more susceptible to emphysema, they do indicate that cannabis and tobacco may have quite different effects on lung function.

We investigated the impact of cannabis and tobacco smoking on lung function in a population-based birth cohort followed to age 32 yrs.

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METHODS

Participants are members of the Dunedin Multidisciplinary Health and Development Study, a longitudinal study of the health and behaviour of a complete cohort of individuals born in Dunedin, New Zealand, in 1972 and 1973 [16]. A total of 1,037 individuals (52% male; 91% of eligible births) participated in the age 3 yrs assessment, forming the base sample for the study. Study members represent the full range of socioeconomic status in the general population of the South Island of New Zealand and are primarily of New Zealand/European ethnicity. The cohort has been assessed at ages 3, 5, 7, 9, 11, 13, 15, 18, 21, 26 and, most recently, at 32 yrs, when we assessed 972 participants (96% of the living cohort). The Otago Ethics Committee approved the study. Written informed consent was obtained for each assessment.

Cannabis smoking history was obtained at ages 18, 21, 26 and 32 yrs [17]. At each assessment participants were asked how many times they had used marijuana in the previous year. Cumulative exposure to cannabis was calculated as the number of ''joint-years'' since age 17 yrs. These estimates assume that the number of times marijuana had been smoked in the previous year was representative of all years since the previous assessment. Where data were not collected at a particular assessment, the amount smoked reported at the next assessment was used to calculate cumulative exposure. 1 joint-yr is defined as the equivalent of one joint a day for 1 yr.

Cumulative tobacco exposure was calculated from the reported number of cigarettes smoked per day up to 18 yrs, 18–21 yrs, 21–26 yrs and 26–32 yrs. Where data were not collected for an assessment, the amount smoked reported at the next assessment was used to calculate cumulative exposure. One pack-year is defined as the equivalent of 20 cigarettes a day for 1 yr. Those who had smoked less than one cigarette a day for 1 yr, and fewer than 20 packets in their lifetime, were regarded as nonsmokers [18].

Spirometry has been measured at each assessment since age 9 yrs. At age 32 yrs a broad range of lung function tests, including spirometry, total lung capacity (TLC), functional residual capacity (FRC), residual volume (RV), airway resistance (R_{aw}), specific airway conductance adjusted for thoracic gas volume (sG_{aw}), transfer factor of the lung for carbon monoxide ($T_{L,CO}$) and alveolar volume (V_A) were measured using the plethysmograph and a Sensormedics Vmax 6200 module (Yorba Linda, CA, USA) [19–21]. This system uses a heated wire mass flow sensor and methane dilution for measurement of alveolar volume and calculation of $T_{L,CO}$. A portable spirometer (Spiropro, Sensormedics) was used to test study members (n=27) who declined to sit in the plethysmograph or were unable to attend the research unit. Spirometry was repeated 10–15 min following inhalation of 200 μg salbutamol *via* a metered dose inhaler and volumatic spacer device (Allen and Hanburys, Stockley Park, UK). Study members were asked to refrain from use of their inhalers and not to smoke on the day of the assessment. All tests were reviewed by a senior technician to ensure that only acceptable and reproducible results were entered for analysis. Equipment was calibrated daily, and weekly quality control measures using biological controls were performed to ensure accuracy and precision of test equipment.

At age 32 yrs, haemoglobin was measured on a Sysmex XE2100 automated haematology analyser (Sysmex Corporation, Kobe, Japan). Exhaled carbon monoxide was measured before $T_{L,CO}$ measurement using a Micro CO monitor (Micromedical, Rochester, UK) and the average of two tests was recorded.

Height without shoes was measured at each age. Questions were asked about current and prior asthma and asthma symptoms using previously developed questionnaires [22]. Current asthma is defined as a reported diagnosis of asthma with symptoms or medication use in the previous 12 months.

Statistical analysis

To assess whether pre-existing differences in lung function influenced the propensity to smoke, regression analyses of cumulative pack-years and joint-years to age 32 yrs were performed using spirometry at age 15 yrs (forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC) and FEV_1/FVC) as the main predictor. These analyses adjusted for sex and height at age 15 yrs.

Initial analyses of sex–cannabis and sex–tobacco smoking interaction terms found no evidence that the effect of smoking either substance was different for males and females for any of the outcomes. The independent associations between lung function measurements at age 32 yrs and cannabis and tobacco smoking were assessed by linear regression using the measurement of lung function as the dependent variable and estimates of both cannabis and tobacco exposure as independent variables. Analyses included terms for height and sex to adjust for differences in predicted lung function, as recommended by VOLLMER *et al.* [23] except for the FEV_1/FVC ratio, which was adjusted for sex only. Analyses of $T_{L,CO}$ also adjusted for pre-test exhaled carbon monoxide and haemoglobin. Analyses of the association of cannabis with lung function were repeated after excluding those with any lifetime history of cigarette smoking.

To assess changes in lung function associated with tobacco and cannabis smoking, regression analyses were repeated for FEV_1, FVC and the FEV_1/FVC ratio using the estimates of both joint-years and pack-years as predictors with adjustment for the measurements obtained at age 15 yrs. These analyses also adjusted for sex, height at age 32 yrs, change in height between ages 15 and 32 yrs, and current asthma diagnosis.

Because pregnancy may affect lung function, pregnant females were excluded (n=31). Visual inspection of the residuals from the regression analyses identified one clear outlier who was also excluded. Lung function measurements were approximately normally distributed, except for R_{aw} and sG_{aw}. Repeat analyses after log-transformations of these variables to approximate normal distributions provided similar results (not shown). Analyses were performed using Stata version 10 (StataCorp, College Station, TX, USA).

RESULTS

Reported cannabis and tobacco use at each age are summarised in the online supplementary material. The number of study members who reported using cannabis was higher at ages 21 and 26 yrs than at ages 18 or 32 yrs (table 1 in the supplementary material). The number of tobacco smokers was similar at all ages, although the number of heavy smokers increased with age.

Cumulative pack-years of tobacco smoking by age 32 yrs correlated with joint-years of cannabis (Spearman's $\rho=0.49$, $p<0.0001$) (table 1). None of the measures of spirometric lung function (FEV1, FVC or FEV1/FVC) at age 15 yrs predicted subsequent pack-years of tobacco consumption or joint-years of cannabis use by age 32 yrs (all p-values $\geqslant 0.3$).

Mean values of lung function according to the categories of cannabis and tobacco use are shown in table 2 in the online supplementary material. When analysed separately, cannabis and tobacco were both associated with a broad range of lung function measures (tables 3 and 4 in the supplementary material). However, when the effects of cannabis and tobacco were considered together (*i.e.* with simultaneous adjustment for exposure to the other substance), different patterns of effects were observed (table 2). After adjusting for tobacco exposure, cannabis was associated with significantly higher FVC values but there was no significant association with FEV1 or FEV1/FVC ratios. In contrast, tobacco was associated with a nonsignificant trend to lower FEV1 values and significantly lower FEV1/FVC ratios, but there was no association with FVC. The findings for post-bronchodilator spirometry were similar, except that in this analysis the association between tobacco smoking and lower FEV1 values was significant (table 5 in the supplementary material). Both cannabis and tobacco were associated with higher values for TLC, FRC and RV, although the association between tobacco and TLC was of borderline statistical significance (table 2). Cannabis was significantly associated with higher R_{aw} and lower sG_{aw}. Tobacco was not associated with differences in R_{aw} but was associated with lower sG_{aw} with borderline statistical significance. Cannabis use was not significantly associated with $T_{L,CO}$, but because of higher values for V_A, transfer factor per unit lung volume ($T_{L,CO}/V_A$) were lower. Tobacco was associated with lower total lung $T_{L,CO}$ and lower $T_{L,CO}/V_A$, but not with V_A.

Associations between cannabis exposure and lung function among non-tobacco smokers are shown in table 3. These show a similar pattern of findings to those shown in table 2. Cannabis use was associated with higher values for TLC and V_A and with trends to higher values for FVC and RV. Cannabis use was not associated with FEV1, FEV1/FVC or $T_{L,CO}$ among these study members, but was associated with higher R_{aw} and lower sG_{aw}.

Associations between cumulative cannabis and tobacco smoking and spirometric lung function after adjustment for spirometry measurements at 15 yrs are shown in table 4. Cannabis use was significantly associated with higher values for FVC, but was not significantly associated with FEV1 or FEV1/FVC ratios. Tobacco smoking was significantly associated with lower FEV1 values and with lower FEV1/FVC ratios. The pattern of findings for cannabis was similar when tobacco smokers were excluded, except that the association between joint-years and FVC was of borderline statistical significance (table 6 in the supplementary material).

TABLE 1 Cumulative cannabis and tobacco use up to age 32 yrs

	Cannabis joint-yrs			Total
	0	⩽1	>1	
Tobacco pack-yrs				
0	226	208	40	474 (49.0)
⩽10	35	171	72	278 (28.8)
>10	23	82	110	215 (22.2)
Total	284 (29.4)	461 (47.7)	222 (23.0)	967

Percentage values are given in parentheses. Chi-squared (4)=242, $p<0.001$.

DISCUSSION

These findings indicate that cannabis is associated with changes in lung function that are independent of the effects of tobacco smoke and appear to be of a different pattern. Both substances were associated with higher values for static lung volumes, indicating a tendency toward hyperinflation and gas trapping, but although cannabis was associated with increased R_{aw}, there was little evidence that it was associated with airflow obstruction (lower FEV1/FVC ratios) once tobacco consumption had been taken into account. Cannabis was also not associated with impairment of the $T_{L,CO}$. By contrast, tobacco smoking was associated with both airflow obstruction and lower $T_{L,CO}$ but not with R_{aw}.

Cannabis was consistently associated with higher lung volumes, whether measured as FVC by spirometry, static lung volumes (TLC, FRC and RV) by plethysmography or as V_A by gas (methane) dilution. This consistency suggests that the findings are unlikely to be an artefact of measurement technique. Moreover, cannabis use was associated with higher values for FVC at age 32 yrs in the analyses that adjusted for FVC at age 15 yrs. It is notable that, although cannabis was not significantly associated with lower $T_{L,CO}$, the higher values for V_A meant that the transfer factor per unit of alveolar volume ($T_{L,CO}/V_A$) was lower in cannabis smokers. The clinical relevance of this is uncertain.

The pattern of lung function changes with cannabis is consistent with a recent report by ALDINGTON *et al.* [15], who compared lung function tests and CT scan findings in a convenience sample of volunteers who were smokers of either cannabis, tobacco, both or neither. They found that cannabis was associated with hyperinflation on both lung function tests and CT scans but that there was little evidence of emphysema. ALDINGTON *et al.* [15] also found that cannabis smokers had evidence of airflow obstruction measured by the FEV1/FVC ratio, although this was of marginal statistical significance and less obvious than in tobacco smokers. In our analysis, and in an earlier report from the Dunedin cohort (up to age 26 yrs), we also found an association between cannabis smoking and lower FEV1/FVC ratios, which was of borderline significance after adjusting for tobacco use [24]. These findings are in keeping with those of a recent meta-analysis that found no consistent association between long-term cannabis use and airflow obstruction [5].

Our findings also confirm two previous reports of decreased sG_{aw} in cannabis users and indicate that cannabis impacts on large airway function despite having little effect on the FEV1/FVC ratio [15, 25]. This finding is not explained by the increase in lung volumes (and therefore the thoracic gas volume used to calculate sG_{aw}) among cannabis smokers since cannabis was

TABLE 2 Associations of cannabis and tobacco use with lung function at age 32 yrs

	Subjects n	Cannabis			Tobacco		
		Coefficient	95% CI	p-value	Coefficient	95% CI	p-value
FEV_1 mL	919	4.0	-3.7–11.8	0.311	-5.0	-10.3–0.2	0.061
FVC mL	919	12.0	3.0–21.0	0.009	0.1	-6.0–6.2	0.968
FEV_1/FVC %	919	-0.08	-0.18–0.02	0.127	-0.11	-0.18– -0.04	0.003
TLC mL	883	25.0	13.9–36.0	<0.001	7.4	-0.2–14.9	0.057
FRC mL	884	15.1	4.8–25.4	0.004	10.5	3.5–17.6	0.003
RV mL	883	12.6	7.0–18.3	<0.001	6.3	2.4–10.1	0.002
R_{aw} $cmH_2O \cdot L^{-1} \cdot s^{-1}$	884	0.014	0.002–0.026	0.024	-0.001	-0.01–0.01	0.827
sG_{aw} $mL \cdot s^{-1} \cdot cmH_2O^{-1} \cdot L^{-1}$	884	-3.3	-5.5– -1.0	0.005	-1.5	-3.0–0.01	0.059
$T_{L,CO}$ $mL \cdot min^{-1} \cdot mmHg^{-1}$	841	-0.019	-0.09–0.05	0.589	-0.130	-0.19– -0.07	<0.001
$T_{L,CO}/V_A$ $mL \cdot min^{-1} \cdot mmHg^{-1} \cdot L^{-1}$	841	-0.016	-0.03– -0.01	0.003	-0.023	-0.03– -0.01	<0.001
V_A mL	894	17.8	6.8–28.9	0.002	2.3	-5.2–9.9	0.545

FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity; TLC: total lung capacity; FRC: functional residual capacity; RV: residual volume; R_{aw}: airway resistance; sG_{aw}: specific airway conductance; $T_{L,CO}$: transfer factor of the lung for carbon monoxide; V_A: alveolar volume. Linear regression analyses of lung function at age 32 yrs using both cannabis and tobacco exposures as predictors. All analyses adjusted for sex and, except for FEV_1/FVC and sG_{aw}, for height. Analyses of $T_{L,CO}$ also adjust for exhaled carbon monoxide and blood haemoglobin. Coefficients represent the difference in lung function associated with each joint-year of cannabis or pack-year of tobacco up to age 32 yrs, adjusted for use of the other substance.

also associated with increased R_{aw} without adjustment for lung volume. This observation is compatible with the high prevalence of bronchitic symptoms and evidence of bronchial epithelial injury among cannabis smokers [3, 4]. Although increased R_{aw} may plausibly contribute to hyperinflation, the increased R_{aw} among cannabis users did not appear to explain the higher lung volumes: adjusting for R_{aw} made no material difference to the association between cannabis and lung volumes (data not shown).

TABLE 3 Association of cannabis use with lung function at age 32 yrs amongst non-tobacco smokers

	Subjects n	Coefficient	95% CI	p-value
FEV_1 mL	449	1.5	-16.0–19.0	0.867
FVC mL	449	17.5	-2.5–37.4	0.087
FEV_1/FVC %	449	-0.19	-0.42–0.04	0.100
TLC mL	433	33.5	9.9–57.1	0.006
FRC mL	434	8.1	-13.1–29.4	0.452
RV mL	433	12.0	-0.3–24.4	0.057
R_{aw} $cmH_2O \cdot L^{-1} \cdot s^{-1}$	434	0.029	0.001–0.057	0.042
sG_{aw} $mL \cdot s^{-1} \cdot cmH_2O^{-1} \cdot L^{-1}$	434	-6.7	-11.8– -1.7	0.010
$T_{L,CO}$ $mL \cdot min^{-1} \cdot mmHg^{-1}$	418	0.032	-0.114–0.178	0.662
$T_{L,CO}/V_A$ $mL \cdot min^{-1} \cdot mmHg^{-1} \cdot L^{-1}$	418	-0.019	-0.042–0.004	0.106
V_A mL	438	28.5	4.3–52.7	0.021

FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity; TLC: total lung capacity; FRC: functional residual capacity; RV: residual volume; R_{aw}: airway resistance; sG_{aw}: specific airway conductance; $T_{L,CO}$: transfer factor of the lung for carbon monoxide; V_A: alveolar volume. Linear regression analyses of lung function at age 32 yrs using cannabis exposure as the predictor. Analyses are restricted to those without a tobacco smoking history. All analyses adjusted for sex and, except for FEV_1/FVC and sG_{aw}, for height. Analyses of $T_{L,CO}$ also adjust for exhaled carbon monoxide and blood haemoglobin. Coefficients represent the difference in lung function associated with each joint-year of cannabis up to age 32 yrs.

Considered separately, cannabis and tobacco smoking were associated with a broader range of lung function findings because most smokers used both substances (tables 3 and 4 in the supplementary material). One potential problem with the combined cannabis–tobacco analyses is whether the regression analyses adequately adjust for the confounding influence of tobacco smoking when assessing the associations with cannabis. The analyses were therefore repeated among those with no tobacco smoking history. Although these analyses also excluded most of the heavy users of cannabis and had smaller sample sizes, the pattern of findings was similar. Among non-tobacco smokers, cannabis was significantly associated with higher values for TLC, R_{aw} and V_A, lower values for sG_{aw}, and with nonsignificant trends to higher values for FVC and RV. There was no significant association with the FEV_1, FEV_1/FVC ratio or $T_{L,CO}$ (table 4).

Why smoking cannabis might have different effects on lung function from tobacco is unclear. We found that although both substances were associated with increased lung volumes, there was little evidence of airflow obstruction or reduced gas transfer with cannabis use. It is possible that the participants had simply not smoked enough cannabis for it to have a measurable effect on lung function, but this seems unlikely in view of the evidence for increased lung volumes and R_{aw}. Apart from the active ingredients of cannabinoids and nicotine, the inhaled combustion products in cannabis and tobacco smoke are qualitatively similar [7], although cannabis smokers may inhale more tar per cigarette/joint [8–10]. One possibility is that delta-9-tetrahydrocannibiol, which is known to act as a short-term bronchodilator [5], also has long-term biological effects. Another possibility is that differences are due to the technique of

TABLE 4 Longitudinal analyses of cannabis and tobacco exposure with spirometric lung function

	Subjects n	Cannabis			Tobacco		
		Coefficient	95% CI	p-value	Coefficient	95% CI	p-value
FEV_1 mL	779	4.4	-1.4–10.3	0.137	-4.3	-8.2– -0.4	0.031
FVC mL	779	10.7	3.9–17.5	0.002	0.2	-4.4–4.8	0.928
FEV_1/FVC %	779	-0.07	-0.14–0.01	0.069	-0.09	-0.14– -0.04	0.001

FEV_1: forced expiratory volume in 1 s; FVC: forced vital capacity. Linear regression analyses of lung function at age 32 yrs using both cannabis and tobacco smoking as predictors, adjusted for lung function at age 15 yrs. Analyses also adjusted for sex, height, change in height between age 15 and 32 yrs, and current asthma at age 32 yrs. Coefficients represent the difference in lung function at age 32 yrs associated with each joint-year of cannabis or pack-year of tobacco up to age 32 yrs, adjusted for the use of the other substance.

smoking cannabis. Cannabis smokers tend to inhale more deeply and hold their breath for longer than tobacco smokers [8]. It is plausible that this alters the distribution of smoke throughout the lungs and thereby alters the associated physiological effects on lung function. Alternatively, it is possible that some of the findings are due to the repeated deep inhalation and breath-holding techniques themselves.

This study has a number of limitations. Cannabis use was reported for the previous year at the four assessments, rather than for all of the intervening years. Our joint-years variable assumes that the consumption of cannabis was similar for the intervening years. We do not know how much cannabis was used on each occasion or whether the cannabis joints were smoked directly or through a device such as a bong/water pipe. By comparison, tobacco smoking histories were taken for all years of the assessment period, cigarettes tend to vary less in tobacco content [15], and the practice of smoking tobacco through devices such as a bong is very unusual. Hence, the measure of cannabis exposure may be less accurate than that of tobacco, although this measurement error is unlikely to have biased the findings with respect to lung function. Errors in cannabis and tobacco consumption will also have occurred because if data were missing for an assessment, we used the amount reported at the next assessment for the calculation of joint- and pack-years. However, repeating the analysis after excluding those with missing data provided very similar findings. It is also possible that study members were less honest in reporting cannabis use than tobacco use, because it is an illegal substance. However, self-reports of cannabis use correlate well with biological markers of use [26], and our well-established record of confidentiality and nonintervention over 30 yrs of the lives of the study members tends to encourage frank reporting of these behaviours. We cannot rule out the possibility that some smokers mixed cannabis with tobacco in the same joint. Although this is not a common practice in New Zealand [15], any mixing of the two substances is most likely to have obscured the differences in the pattern of lung function changes between the two and is unlikely to explain our findings.

The study also has a number of strengths. Both cannabis and tobacco smoking were assessed on a number of occasions throughout early adult life in a population-based cohort with minimal loss to follow-up. We have a comprehensive assessment of lung function at age 32 yrs and, although plethysmography was only performed at the most recent assessment, we have measurements of spirometry pre-dating the exposure to cannabis and tobacco to investigate whether baseline lung function influenced the propensity to smoke (*e.g.* a ''healthy smoker'' effect). This analysis provided no evidence for an association between spirometry at age 15 yrs and subsequent use of tobacco or cannabis.

In conclusion, cannabis and tobacco smoking are each associated with a distinct pattern of lung function changes in young adults. Cannabis was associated with evidence of hyperinflation and increased large airway resistance, with little evidence of airflow obstruction or impairment of gas transfer, whereas tobacco was associated with airflow obstruction, gas trapping and lower transfer factors. These findings suggest that smoking cannabis and tobacco have different physiological consequences for the lungs.

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STATEMENT OF INTEREST

None declared.

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