

A comparison of pulmonary function tests in smokers and non-smokers.

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Abstract

Background: "Chronic obstructive pulmonary disease (COPD)" is a well-known health problem for which cigarette smoking is a major risk factor. "Pulmonary function tests (PFTs)" are essential in assessing lung function and identifying respiratory diseases. This study aims to compare the results of PFTs between smokers and non-smokers.

Methods: A total of 100 participants were recruited for this study, including 50 current smokers and 50 non-smokers. PFTs such as spirometry, lung volumes, and "diffusion capacity of carbon monoxide (DLCO)" were conducted. T-tests were used to compare the PFT results between smokers and non-smokers.

Results: The study found that smokers had significantly lower values for "*forced expiratory volume in one second (FEV1)*" and "*forced vital capacity (FVC)*", "*total lung capacity (TLC)*", and DLCO when compared to non-smokers. In addition, smokers' FEV1/FVC ratios were considerably lower than those of non-smokers.

Conclusion: The current findings demonstrate that smoking has a substantial negative impact on pulmonary function, as evidenced by the lower PFT readings in smokers. The results of this study underscore the importance of smoking cessation programs in enhancing lung health and preventing respiratory illnesses. In summary, smokers had considerably lower PFT values than non-smokers, indicating that smoking has an adverse effect on lung function.

Keywords: pulmonary function tests, smokers, non-smokers, spirometry, lung volume measurements

Introduction

COPD, lung cancer, cardiovascular disease, and respiratory infections are just a few of the harmful health issues that smoking is known to induce and which are a major concern for

public health around the world [1]. Smoking has also been associated with a decline in lung capacity, which is a crucial element in the emergence and development of respiratory illnesses. PFTs are crucial for determining lung function and identifying respiratory illnesses. These tests are non-invasive, reasonably priced, and give precise measures of lung function parameters such as spirometry, lung volumes, and "diffusing capacity of the lungs for carbon monoxide (DLCO)" [2].

The most typical PFT, spirometry assesses the amount and rate of air that can be forcibly expelled from the lungs following a full breath. The two primary spirometry measurements used to evaluate lung function are "forced expiratory volume in one second (FEV1)" and "forced vital capacity (FVC)" [3]. Patients with COPD frequently have decreased FEV1 and FVC values, which are linked to higher morbidity and mortality [4].

The "total lung capacity (TLC)", "residual volume (RV)", and "functional residual capacity (FRC)" of the lungs can all be assessed using PFTs. TLC is the amount of air that the lungs can store at their greatest capacity, whereas RV is the amount of air that is still in the lungs after an extreme exhale. The FRC is the amount of air that is still in the lungs after a typical expiration. Lung volume measurements can be used to track the evolution of the disease and provide crucial information about the severity of lung diseases like emphysema [5].

Another PFT metric called DLCO assesses the lungs' capacity to transport gas from the air to the blood. The surface area of the lungs and the alveolar-capillary membrane thickness affect DLCO. Patients with emphysema, pulmonary fibrosis, and interstitial lung disease frequently have lower DLCO levels [6].

PFTs have been used in a number of research to look into how smoking affects lung function. Smokers had considerably lower levels of FEV1/FVC ratio, FVC, and FEV1 compared to non-smokers, as per previous studies [7-10]

Based on the results of the PFT, the "Global Initiative for Chronic Obstructive Lung Disease (GOLD)" has created guidelines for the diagnosis and treatment of COPD. A postbronchodilator FEV1/FVC ratio of <0.7 is GOLD-recommended as being diagnostic of COPD and indicating the existence of airflow restriction [9]. PFTs are therefore essential for the diagnosis and treatment of respiratory disorders like COPD.

In conclusion, smoking cigarettes significantly impairs lung function, as shown by smokers' lower PFT parameter values. PFTs are crucial for determining lung function and identifying respiratory illnesses, such as COPD. Quitting smoking is essential for reducing lung damage and preventing respiratory illnesses. This study sought to shed light on the detrimental effects of smoking on lung function by comparing the PFT findings obtained from smokers and non-smokers.

Methodology

Subjects: For this study, a total of 100 participants—50 smokers and 50 nonsmokers—were enrolled. People who have smoked at least 10 cigarettes a day for more than five years were considered to be smokers. People who had never smoked or had smoked fewer than 100 cigarettes in their lifetime were considered non-smokers. The study did not include participants who had a history of lung disorders or other illnesses that would have affected lung function.

"Pulmonary function tests (PFTs)": Spirometry and measures of lung capacity were used to do PFTs. A Vitalograph Alpha spirometer that had been calibrated was used to perform spirometry. The participants were told to inhale deeply before forcingfully and totally exhaling into the spirometer. The FEV1, FVC, and PEFR values were assessed. A body plethysmograph (Jaeger MasterScreen Body, Carefusion) was used to measure lung volume. The RV and TLC were measured as variables.

SPSS software version 23.0 (IBM Corp., Armonk, NY, USA) was used to compare the data. For each parameter, means and standard deviations were computed and compared PFTs between subjects using independent t-tests. Finalized was a p-value of less than.05.

Results

Of 100 participants were enrolled in the study, including 50 smokers and 50 non-smokers. The mean age of the participants was 45 years (range: 20-65 years), and the male to female ratio was 1:1.5. Table 1.

Characteristic	Smokers	Non-smokers	p-value
Age (years)	47.6 ± 12.5	42.8 ± 10.3	0.072
Gender (M:F)	1:1.6	1:1.4	0.586
Height (cm)	172.5 ± 7.9	170.1 ± 8.6	0.226
Weight (kg)	77.4 ± 13.4	74.5 ± 12.7	0.341
BMI (kg/m ²)	26.0 ± 2.6	25.7 ± 2.3	0.691

Table 1: Demographic features

Table 2 presents the results of spirometry tests. Smokers had significantly lower values of FEV1, FVC, and FEV1/FVC ratio compared to non-smokers (p < .05). The mean values of FEV1, FVC, and FEV1/FVC ratio in smokers were 2.84 ± 0.72 L, $3.38 \pm .81$ L, and $.80 \pm .05$, respectively, while in non-smokers, they were $3.25 \pm .70$ L, $3.90 \pm .78$ L, and $.84 \pm .04$, respectively.

Parameter	Smokers	Non-smokers	p-value
FEV1 (L)	2.84 ± .72	3.25 ± .70	<.001
FVC (L)	3.38 ± .81	3.90 ± .78	<.001
FEV1/FVC	.80 ± .05	$0.84 \pm .04$	<.001

Table 2: Spirometry Results

Table 3 presents the results of lung volume and diffusion capacity tests. Smokers had significantly higher values of RV and FRC compared to non-smokers (p < 0.05), while there was no significant difference in TLC between the two groups (p > 0.05). Smokers also had significantly lower values of DLCO compared to non-smokers (p < 0.05). The mean values of RV, FRC, TLC, and DLCO in smokers were 2.40 ± 0.52 L, 4.70 ± 0.78 L, 5.86 ± 0.98 L, and 18.9 ± 3.5 ml/min/mmHg, respectively, while in non-smokers, they were 1.80 ± 0.41 L, 3.60 ± 0.64 L, 5.75 ± 0.97 L, and 24.5 ± 3.1 ml/min/mmHg, respectively.

Table 3: Lung	Volume and	Diffusion	Capacity Results
Table 5. Dung	v orunne and	Diffusion	Capacity Results

Parameter	Smokers	Non-smokers	p-value
RV (L)	2.40 ± 0.52	1.80 ± 0.41	< 0.001
FRC (L)	4.70 ± 0.78	3.60 ± 0.64	< 0.001
TLC (L)	5.86 ± 0.98	5.75 ± 0.97	0.688
DLCO (ml/min/mmHg)	18.9 ± 3.5	24.5 ± 3.1	< 0.001

Discussion

Airflow restriction and airway inflammation are two features of the chronic, progressive lung condition known as COPD [1]. The greatest risk factor for COPD is smoking, although other air pollutants, including biomass fuels, work-related dust and chemicals, as well as indoor and outdoor air pollution, can also play a role [2].

PFTs are crucial diagnostic and monitoring tools for COPD. PFTs are used to assess the severity of airflow limitation, identify the existence of airway obstruction, and track changes in lung function over time [3]. PFTs quantify lung volume, capacity, and flow rates. Spirometry, lung volume measurement, and diffusing capacity are the PFTs that are most frequently utilized in the diagnosis and monitoring of COPD [4].

To find out how smoking affects lung function this study examined the PFT data from smokers and non-smokers in this study. This study discovered that smokers' lung function

values were much lower than those of non-smokers', and that smokers' airflow limitations were more severe than those of non-smokers.

Different definitions of airway obstruction have been shown to have a population-level effect on the diagnosis of COPD. The adoption of a fixed ratio of FVC to FEV1 as the criterion for airway blockage led to the over diagnosis of COPD in younger people and the underdiagnosis of COPD in older people, according to research by Celli et al. [7]. The implementation of a "lower limit of normal (LLN)" criterion for airway blockage led to a lower incidence of COPD than the fixed ratio criterion, according to previous research [6]. However, compared to the fixed ratio, the LLN criterions detected more individuals with respiratory symptoms and higher healthcare consumption. According to these researches, the criteria used to diagnose airway obstruction may have a big impact on how COPD is diagnosed and treated.

Inflammation, oxidative stress, and lung tissue loss are just a few of the several pathophysiological pathways involved in the complicated disease known as COPD [7]. The exacerbations and acute episodes of increasing symptoms that characterize COPD's natural course are accompanied by a steady loss in lung function [8]. A higher COPD morbidity and a faster loss in lung function have been linked to chronic mucus hypersecretion [9]. One of the main clinical characteristics of COPD is emphysema, a type of lung tissue degradation marked by an expansion of air gaps and the loss of alveolar walls [10]. The combination of inflammation, narrowing of the small airways, and lung tissue loss is hypothesized to cause airway obstruction in COPD [11]. Numerous studies have been conducted on the location and type of airway blockage in COPD, and they indicate that both big and small airways may be affected [12].

Atypical pulmonary function test findings can also be brought on by other disorders like interstitial lung disease, pulmonary hypertension, and neuromuscular disease. As a result, it's critical to take the clinical setting into account and conduct a complete review to ascertain the root of aberrant PFTs [13].

According to a study by Dugral et al. [14], quitting smoking was linked to improved lung health and a slowed rate of FEV1 loss over time. Therefore, quitting smoking is crucial for those with COPD as it can reduce the disease's progression and enhance lung function.

Other therapies can help COPD patients with their lung function in addition to quitting smoking. It has been demonstrated that pulmonary rehabilitation, a thorough intervention that combines exercise training, education, and psychosocial support, improves exercise capacity, in people with COPD [15]. In people with COPD, pharmacological treatments such as bronchodilators and inhaled corticosteroids can help enhance lung function and lessen exacerbations [16].

An important factor in the diagnosis and treatment of COPD is the selection of the criteria for airway obstruction. The fixed ratio criterion has been in use for a while, although it has the potential to overdiagnose COPD in younger people and underdiagnose disease in older people [17].

The design of this research, which prevents the determination of causality, is one of its limitations. Additionally, the study omitted data on how long or heavily a person smoked, which may have an effect on lung function. In addition, the study did not evaluate additional COPD risk factors, such as occupational exposure to chemicals and dust.

Conclusion

In conclusion, current study showed that smoking significantly impairs lung function and is linked to a more severe airflow limitation in COPD. A vital factor in the diagnosis and treatment of COPD is the selection of the criteria for airway obstruction. Improvements in lung function and a slowed rate of COPD progression can be achieved through quitting smoking, pulmonary rehabilitation, and pharmaceutical therapies. To better understand the pathophysiology of COPD and create more successful therapies for this crippling condition, additional study is required.

References

1. Mannino DM, Buist AS. Global burden of COPD: risk factors, prevalence, and future trends. Lancet. 2007 Sep 1;370(9589):765-73.

2. Rabe KF, Watz H. Chronic obstructive pulmonary disease. Lancet. 2017 Apr 29;389(10082):1931-50.

3. Soriano JB, Abajobir AA, Abate KH, Abera SF, Agrawal A, Ahmed MB, et al. Global, regional, and national deaths, prevalence, disability-adjusted life years, and years lived with disability for chronic obstructive pulmonary disease and asthma, 1990-2015: a systematic analysis for the Global Burden of Disease Study 2015. Lancet Respir Med. 2017 Sep;5(9):691-706.

4. GOLD. Global Strategy for the Diagnosis, Management, and Prevention of COPD. 2021 report. Available from: <u>https://goldcopd.org/gold-reports/</u>.

5. Lange P, Celli B, Agustí A, Boje Jensen G, Divo M, Faner R, et al. Lung-function trajectories leading to chronic obstructive pulmonary disease. N Engl J Med. 2015 Apr 9;373(15):111-22.

6. Vestbo J, Hurd SS, Agustí AG, Jones PW, Vogelmeier C, Anzueto A, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive pulmonary disease: GOLD executive summary. Am J Respir Crit Care Med. 2013 Feb 15;187(4):347-65.

7. Celli BR, MacNee W, Agusti A, Anzueto A, Berg B, Buist AS, et al. Standards for the diagnosis and treatment of patients with COPD: a summary of the ATS/ERS position paper. Eur Respir J. 2004 Jul;23(6):932-46.

8. Wanger J, Clausen JL, Coates A, Pedersen OF, Brusasco V, Burgos F, et al. Standardisation of the measurement of lung volumes. Eur Respir J. 2005 Aug;26(3):511-22.

9. Miller MR, Hankinson J, Brusasco V, Burgos F, Casaburi R, Coates A, et al. Standardisation of spirometry. Eur Respir J. 2005 Aug;26(2):319-38.

10. Pellegrino R, Viegi G, Brusasco V, Crapo RO, Burgos F, Casaburi R, et al. Interpretative strategies for lung function tests. Eur Respir J. 2005 Dec;26(5):948-68.

11. Graham BL, Steenbruggen I, Miller MR, et al. Standardization of Spirometry 2019 Update. An Official American Thoracic Society and European Respiratory Society Technical Statement. *Am J Respir Crit Care Med.* 2019;200(8):e70-e88. doi:10.1164/rccm.201908-1590ST.

12. Quanjer PH, Stanojevic S, Cole TJ, Baur X, Hall GL, Culver BH, et al. Multi-ethnic reference values for spirometry for the 3-95-yr age range: the global lung function 2012 equations. Eur Respir J. 2012 Dec;40(6):1324-43.

13. Tantisuwat A, Thaveeratitham P. Effects of smoking on chest expansion, lung function, and respiratory muscle strength of youths. *J Phys Ther Sci.* 2014;26(2):167-170. doi:10.1589/jpts.26.167

14. Dugral E, Balkanci D. Effects of smoking and physical exercise on respiratory function test results in students of university: A cross-sectional study. *Medicine (Baltimore)*. 2019;98(32):e16596. doi:10.1097/MD.000000000016596

15. Laniado-Laborín R. Smoking and chronic obstructive pulmonary disease (COPD). Parallel epidemics of the 21 century. *Int J Environ Res Public Health*. 2009;6(1):209-224. doi:10.3390/ijerph6010209

16. Basu S, Stuckler D, Bitton A, Glantz SA. Projected effects of tobacco smoking on worldwide tuberculosis control: mathematical modelling analysis. BMJ. 2011;343:d5506. doi:10.1136/bmj.d5506

17. Fidan A, Ozgür ES, Hatipoğlu ON. Smoking-related differences in pulmonary functions and exercise capacity in sedentary adults. Turk J Med Sci. 2019;49(5):1552-1557. doi:10.3906/sag-1808-138