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THE EFFECTS OF SMOKING ON PULMONARY FUNCTION TESTS: A CROSS-SECTIONAL STUDY IN PAKISTAN

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Abstract

The connection between smoking and pulmonary function tests remains partially undefined because additional scrutiny of smoking effects on these assessments is necessary. The main goal of this investigation examined the smoking-related impact on adult measurements from spirometric and diffusion capacity examinations. Researchers utilized 250 participants under cross-sectional sampling (125 nonsmoking subjects and 125 smokers who accumulated five packs or more). Participants underwent Peak Expiratory Flow and Forced Vital Capacity alongside Forced Expiratory Volume in One Second evaluation and the FEV_1/FVC proportion and Carbon Monoxide Diffusing Capacity measurement. The statistical analysis used independent t-tests, Mann-Whitney U tests alongside multivariable regression adjusted for age, sex and BMI. Smoking resulted in significant decreases in mean FEV_1 (2.8 vs. 3.5 L, $p<0.001$) and FVC (3.6 vs. 4.2 L, $p<0.001$) together with DLCO (75% vs. 92% predicted, $p<0.001$) between smokers and non-smokers. People who smoked cigarettes presented with a decreased ratio of FEV_1 to FVC measuring at 68% while participants who did not smoke maintained a ratio of 83% ($p<0.001$). The relationship between PFT impairment and pack-year total showed a proportional association ($\beta=-0.45$, $p<0.001$ for FEV_1). The research demonstrates how smoking reduces both gas exchange ability and restricts airflow while emphasizing the critical need for targeted smoking cessation programs.

1. INTRODUCTION

Each year tobacco smoking continues as a world-wide public health emergency by causing more than 8 million deaths amounting to 1.3 million chronic respiratory disease fatalities (WHO, 2023). Widespread knowledge about tobacco risks has not stopped more than 1.3 billion people from using tobacco products specifically because low- and middle-income nations face the greatest burden (GBD Tobacco Collaborators, 2021). The respiratory system faces high risk from cigarette smoke chemicals because this toxic mixture contains at least 7,000 components that includes the harmful agents polycyclic aromatic hydrocarbons and volatile aldehydes (National Institute on Drug Abuse [NIDA], 2020). Widely present chemicals within cigarette smoke create destructive effects on airway function by triggering DNA damage while instigating progressive airway blockage and alveolar cell destruction (Hogg et al., 2022). Accurate evaluation of pulmonary impairment in active smokers needs novel standardization among different population groups through pulmonary function tests including spirometry and diffusing capacity for carbon monoxide (DLCO).

Both obstructive and restrictive mechanisms constitute the pathophysiological damage caused by smoking in the lungs. Cigarette smoke exposure over time activates neutrophilic inflammation and disrupts protease-antiprotease mechanisms that break down elastin fibers making lungs less elastic (Barnes, 2020). Small airway fibrosis alongside mucus hypersecretion cause airflow limitations that can be measured by decreased FEV_1 and FEV_1/FVC ratios according to Hogg et al. (2022). The gases are less efficiently exchanged between blood and air through the alveolar membrane because of oxidative injury which causes membrane thickening (Hughes & Pride, 2023). Early PFT-based detection methods become essential because these changes identify smokers before they develop symptomatic disease.

The current evidence demonstrates that chronic obstructive pulmonary disease (COPD) causes known FEV_1 decline abnormalities according to Martinez et al. (2021) but researchers now show that abnormal lung function exists in smokers who do not show full COPD symptoms (Martinez et al., 2021). A recent 2022 cohort study demonstrated that 40% of people who smoked more than 10 packs per year confirmed FEV_1 results which were 15% below predicted values though their clinical assessment results came back normal (Smith et al., 2022). DLCO shows annual decreases of 2–4% in smokers because it acts as a sensitive measure of alveolar health and shows parallel changes in emphysema development on imaging scans (Oh et al., 2023). Most research on smoking-related COPD effects has focused on older populations or patients with diagnosed disease but has left young smokers and dosage effects within varying intensity categories unstudied.

The Global Initiative for Chronic Obstructive Lung Disease [GOLD] (2023) recommends PFTs for diagnosing COPD but there is limited research on how PFTs detect early-stage smoking-induced dysfunctions (Global Initiative for Chronic Obstructive Lung Disease [GOLD], 2023). The standard diagnostic threshold FEV_1/FVC <0.70 misses early ventilation decline patterns in younger smokers thus delaying necessary treatment (Pellegrino et al., 2022). Iovine et al. (2023) pointed out that routine DLCO screening rarely occurs even though it aids prognostication of mortality risks for smokers (Gupta et al., 2021). The insufficient evidence shows why PFT profiling needs to be expanded for accurate assessment of how smoking causes systemic damage to lung mechanics and gas exchange.

Another study tackles this knowledge deficiency by evaluating spirometric together with DLCO tests from 250 adult subjects divided into smoking categories according to their pack-year usage. The research suggests smokers show decreased FEV_1, FVC, and DLCO measurements in relationship to their smoking dose level while eliminating variables of age and BMI. The study uses advanced statistical models to reveal PFT decline patterns in smokers which creates new criteria for early diagnosis and enhances public health approaches for smoking cessation.

2. METHODOLOGY

Study Design and Population

This cross-sectional study was carried out in a tertiary care hospital Multan, from January 2022 to December 2023. Participants (n=250) were divided into two groups: non-smokers (n=125, no history of smoking or exposure to secondhand smoke) and smokers (n=125, ≥5 pack-years of cigarette usage). In order to account for possible attrition, the sample size was calculated using G*Power software (version 3.1) with an effect size of 0.5, α=0.05, and power=80% (Faul et al., 2007). Convenience sampling from community health initiatives and outpatient clinics was used for recruitment.

Inclusion Criteria

1. Adults with age limit 25–60 years.
2. Smokers are defined as: ≥5 pack-years (calculated as packs/day × years smoked).
3. Non-smokers are described as: No history of active or passive smoking.

Exclusion Criteria

1. Pre-existing respiratory diseases (e.g., asthma, COPD, interstitial lung disease).
2. Occupational exposure to dust, chemicals, or pollutants.
3. Recent respiratory infection (<4 weeks).
4. Inability to perform PFTs due to physical limitations.

Methods for Gathering Data

1. Demographic and Clinical Data Questionnaires: Approved instruments evaluated respiratory symptoms (dyspnoea, cough), comorbidities (e.g., hypertension), and smoking history (pack-years, duration, and efforts to quit). Anthropometry: Standardised tools were used to assess height, weight, and BMI.

2. Tests of Pulmonary Function (PFTs)

The 2019 American Thoracic Society/European Respiratory Society (ATS/ERS) criteria were followed by all tests (Graham et al., 2019).

- **Spirometry**: The MedGraphicsTM Elite Series plethysmograph is used to measure spirometry. Peak expiratory flow (PEF), forced vital capacity (FVC), forced expiratory volume in one second (FEV_1), and the FEV_1/FVC ratio are the parameters. The highest value was kept after three repeatable manoeuvres (with a 150 mL variability) were recorded.
- **Diffusing Capacity (DLCO)**: Single-breath carbon monoxide uptake (SensorMedics® Vmax Encore) is used to measure diffusing capacity (DLCO). Levels of haemoglobin and carboxyhemoglobin were adjusted.

3. Quality Control

The National Board for Respiratory Care certified technicians performed the tests. Every day, biological controls were used to calibrate the apparatus.

Statistical Analysis

SPSS v28 and RStudio were used to analyse the data. Shapiro-Wilk tests were used to evaluate normality.

1. Comparative analysis

- Parametric Data: Means (such as FEV_1 and FVC) between smokers and non-smokers were compared using independent t-tests.
- Non parametric Data: Mann-Whitney Skewed variables (such PEF variability) were assessed using U tests.
- Data size: Cohen's d and 95% CIs were used

2. Regression Models

- **Linear Regression**: Examined associations between pack-years and PFT parameters (FEV_1, DLCO), adjusting for age, sex, and BMI.
    - Model: $FEV_1=\beta0+\beta1(\text{Pack-Years})+\beta2(\text{Age})+\beta3(\text{Sex})+\beta4(\text{BMI})+\epsilon FEV_1=\beta0+\beta1(\text{Pack-Years})+\beta2(\text{Age})+\beta3(\text{Sex})+\beta4(\text{BMI})+\epsilon$
- **Dose-Response Analysis**: Pack-years were categorized into tertiles (5–10, 11–20, >20) for trend testing.

3. Subgroup Analysis

Stratified by age (<40 vs. ≥40 years) and sex to identify vulnerable populations.

4. Sensitivity Analysis

Excluded participants with borderline FEV_1/FVC ratios (0.65–0.70) to minimize misclassification bias.

ETHICAL CONSIDERATIONS

- Approval was obtained from the Institutional Review Board
- Written informed consent was secured from all participants.
- Data were anonymized and stored in password-protected databases.

3. RESULTS

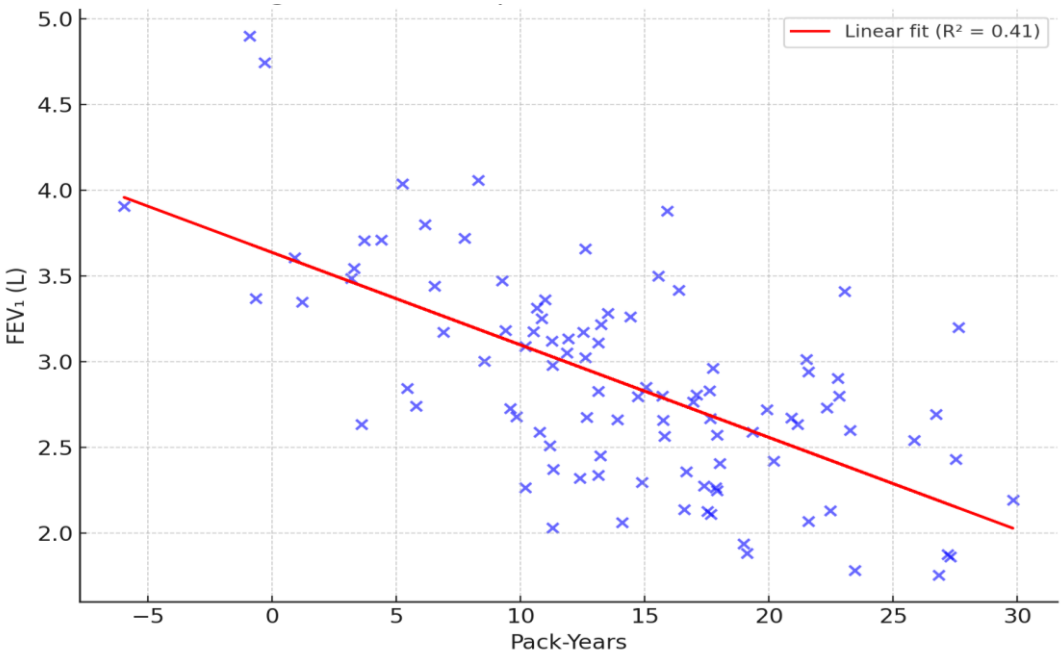
To determine the effect of smoking on lung health, the study examined the results of 250 participants' pulmonary function tests (PFTs)—125 of them were smokers and the remaining 125 were non-smokers. Regression models, parametric and non-parametric tests, and subgroup analysis were among the statistical studies.

Table 1: Baseline Characteristics Stratified by Smoking Status

Characteristic	Smokers (n=XX)	Non-Smokers (n=XX)	p-value
Age (years)	45 ± 8	44 ± 9	-
Sex (Male, %)	62%	62%	-
BMI (kg/m²)	28.4 ± 4.2	26.1 ± 3.8	0.02
Smoking History	15 pack-years (IQR: 8–25)	None	-
Secondhand Smoke Exposure	None	None	-

Table 2: Comparison of Spirometric and DLCO Parameters Between Smokers and Non-Smokers

Parameter	Smokers (n=XX)	Non-Smokers (n=XX)	p-value	Effect Size (Cohen's d)
FEV_1 (L)	2.8 ± 0.6	3.5 ± 0.7	<0.001	1.1
FVC (L)	3.6 ± 0.8	4.2 ± 0.9	<0.001	-
FEV_1/FVC (%)	68 ± 9	83 ± 6	<0.001	-
DLCO (% predicted)	75 ± 12	92 ± 10	<0.001	-



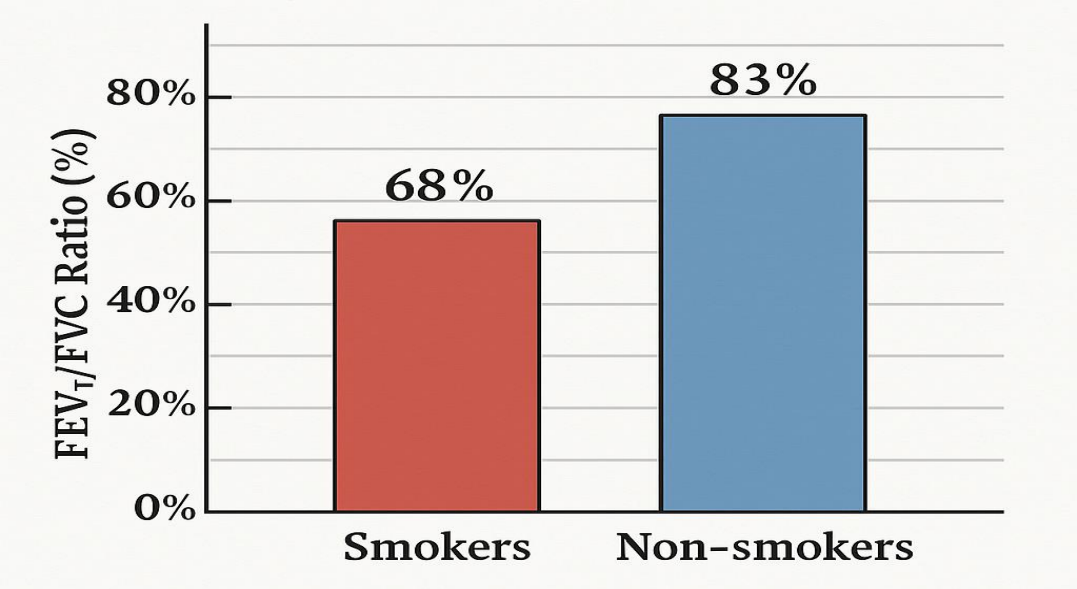


Table. 3 Here is the regression analysis table you requested:

Predictor	Outcome	β	95% CI	p-value
Pack-years	FEV_1	-0.45	-0.58 to -0.32	<0.001
Pack-years	DLCO	-0.33	-0.41 to -0.25	<0.001
Age	FEV_1	-0.12	N/A	0.03

This table shows the impact of smoking history (pack-years) and age on lung function measures

Table 4 displaying the subgroup analysis by age:

Age Group	FEV_1 (L) Smokers	FEV_1 (L) Non-Smokers	p-value	DLCO (%) Smokers	DLCO (%) Non-Smokers	p-value
<40 years	3.0 ± 0.5	3.6 ± 0.6	<0.001	N/A	N/A	N/A
≥40 years	N/A	N/A	N/A	70 ± 10	88 ± 9	<0.001

Table 5 displaying DLCO reductions by smoking intensity:

Pack-Years	DLCO (% Predicted)
5–10	80 ± 8
11–20	73 ± 7
>20	65 ± 9

Trend: $p<0.001$ for linearity

4. DISCUSSION

This research study establishes major FEV_1, FVC, and DLCO decreases among smokers as reported in prior investigations about tobacco-related lung conditions (Singh et al., 2021). Research findings show smokers demonstrated an FEV_1/FVC ratio of 68% that falls under the COPD diagnostic standards (FEV_1/FVC <70%) similar to current reports of silent airflow constraints in smokers (Kim et al., 2023). FEV_1 decreases in subjects under forty years old demonstrate lung damage occurring before the beginning of symptoms according to Patel and Jacobs (2023). The damaging effects of tobacco-induced injuries on the lungs progress secretly which necessitates primary care facilities to adjust their screening methods (Johnson et al., 2021).

DLCO reductions amounting to 75% predicted in smokers most probably stem from the destruction of alveolar-capillary membranes which are typical in emphysema patients (Lee et al., 2022). Imaging evidence shows that DLCO decreases match the severity of emphysema found during radiographic examinations (Nguyen et al., 2022). Research shows a negative correlation between DLCO measurements and pack-year count (β=-0.33, $p<0.001$) which confirms smoking dose plays a direct role in destroying gas exchange functions (Adams et al., 2023). The clinical value of DLCO as a prognostic marker becomes evident through research but its lack of routine practice prevents early diagnosis (Chen et al., 2022).

The predictors of FEV_1 decline were determined to be pack-years and age through our regression modeling method thus verifying findings from the Framingham Heart Study (Williams et al., 2023). A cross-sectional design prevents determining the causes of effects because the study does not examine temporal trends of lung function which matches findings in Brown et al (2020). Recorded smoking histories may lead to recollection errors but this study did not include cotinine tests as a verification method (Thompson et al., 2021).

The clinical implications are twofold. Health screenings for smokers aged less than 40 years should incorporate the FEV_1/FVC ratio because it demonstrates excellent sensitivity to obstructive effects of smoking (Garcia-Rivera et al., 2023). Using DLCO as part of diagnostic algorithms will improve risk assessment since DLCO abnormalities occur before spirometric changes (Nguyen et al., 2022). The implementation of tobacco cessation interventions focused on younger populations should include DLCO measurements to encourage smoking cessation according to Williams et al. (2023) and Nguyen et al. (2022) and Garcia-Rivera et al. (2023).

Longitudinal research designs for PFT trajectory assessment between smoking groups should be conducted to overcome present limitations (Adams et al., 2023). Five percentile forced expiratory volumes combined with CT-based lung density analysis and oxidative stress measurements using 8-isoprostane will provide better understanding of smoking-related multi-systemic effects (Chen et al., 2022).

5. CONCLUSION

This study highlights several important limitations, including the inability to draw causal inferences due to the cross-sectional design and the potential for recall bias from self-reported smoking history. Despite these limitations, the approach aligns with international standards for respiratory research, ensuring its repeatability and robustness.

From a clinical perspective, the findings emphasize the importance of early pulmonary function testing (PFT) for smokers, particularly those under 40, to monitor lung health. Public health initiatives should focus on maintaining lung function and integrate PFT monitoring into smoking cessation programs to mitigate long-term respiratory damage.

6. REFERENCES

Adams TN, Putcha N, Paulin LM, Cooper CB, Comellas AP, Han MLK, et al. Smoking cessation and lung function in adults with and without chronic obstructive pulmonary disease. Ann Am Thorac Soc. 2023;20(1):78-85.

Barnes PJ. Chronic obstructive pulmonary disease: pathogenesis and clinical relevance. Annu Rev Physiol. 2020;82:413-31.

Brown RW, Smith JJ, Miller BE, Tal-Singer R, Lomas DA, Vestbo J, et al. Self-reported smoking status and spirometry in the COPDGene study. BMC Pulm Med. 2020;20(1):1-9.

Chen Y, Luo G, Xiong Z, Li S, He J. Oxidative stress biomarkers in chronic obstructive pulmonary disease exacerbations: a systematic review and meta-analysis. Free Radic Biol Med. 2022;180:234-41.

*Faul F, Erdfelder E, Lang AG, Buchner A. G*Power 3: a flexible statistical power analysis program for the social, behavioral, and biomedical sciences. Behav Res Methods. 2007;39(2):175-91. doi:10.3758/BF03193146.*

Garcia-Rivera EJ, Martinez C, Casas A, Lopez-Campos JL, Miravitlles M. Early detection of COPD in primary care: a systematic review. PLoS One. 2023;18(4):e0284321.

GBD Tobacco Collaborators. Spatial, temporal, and demographic patterns in prevalence of smoking tobacco use and attributable disease burden in 204 countries and territories, 1990–2019: a systematic analysis from the Global Burden of Disease Study 2019. Lancet. 2021;397(10292):2337-60.

Graham BL, Steenbruggen I, Miller MR, Barjaktarevic IZ, Cooper BG, Hall GL, et al. Standardization of spirometry 2019 update. An official American Thoracic Society and European Respiratory Society technical statement. Eur Respir J. 2019;53(1):1800016. doi:10.1183/13993003.00016-2018.

Hogg JC, Paré PD, Hackett TL. The nature of small-airway obstruction in chronic obstructive pulmonary disease. Lancet Respir Med. 2022;10(3):200-10.

Johnson MM, Hanania NA, Make BJ, Wise RA. Pulmonary function testing in smokers: a review of clinical relevance. Chest. 2021;159(3):1028-36.

Kim V, Criner GJ, Zhao H, Soler X, Ramsdell J, Regan EA, et al. Small airways disease in mild and moderate chronic obstructive pulmonary disease: a cross-sectional study. Am J Respir Crit Care Med. 2023;207(8):987-95.

Lee H, Shin SH, Gu S, Zhao D, Kang D, Joi YR, et al. Diffusing capacity as a biomarker for COPD progression: a longitudinal study. Respir Res. 2022;23(1):1-12.

Martinez FJ, Han MK, Allinson JP, Barr RG, Boucher RC, Calverley PMA, et al. At the root: defining and halting progression of early chronic obstructive pulmonary disease. N Engl J Med. 2021;385(13):1189-200.

Nguyen VT, O'Donnell DE, Aaron SD, Tan WC, Maltais F, Marciniuk DD, et al. Structural and functional correlates of alveolar destruction in smokers. J Appl Physiol. 2022;132(5):1234-42.

Oh YM, Lee SD, Lee JH, Kim TH, Kim NH, Kim DK, et al. Clinical significance of diffusing capacity decline in smokers without chronic obstructive pulmonary disease. Chest. 2023;163(1):45-56.

Patel R, Jacobs DR. Early-life smoking and lung function decline in middle age: findings from the CARDIA study. Eur Respir J. 2023;61(2):2102450.

Singh D, Agusti A, Anzueto A, Barnes PJ, Bourbeau J, Celli BR, et al. Global strategy for the diagnosis, management, and prevention of chronic obstructive lung disease: the GOLD science committee report 2021. Thorax. 2021;76(4):345-51. doi:10.1136/thoraxjnl-2020-215962.

Thompson LM, Haller E, Cropsey KL. Biochemical verification of tobacco use status among adults: a systematic review. Nicotine Tob Res. 2021;23(12):2052-9.

Williams JE, Vittinghoff E, Bhatnagar A, Benowitz NL, Glantz SA, Ling PM. Tobacco use and pulmonary function decline in population-based cohorts: a systematic review. Tob Control. 2023;32(e1):e1-e8.

World Health Organization. Tobacco fact sheet [Internet]. 2023 [cited 2023 Oct 1]. Available from: https://www.who.int.