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Original Research Article

Study of Pulmonary Function in Smoker's Vs Non-Smokers

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Abstract:

Background: Smoking is a major global public health concern, responsible for millions of deaths annually. Tobacco smoke contains harmful substances that damage the respiratory system and contribute to diseases such as chronic bronchitis, emphysema, and COPD.

Aim: To evaluate the effect of smoking on pulmonary function.

Methodology: A cross-sectional study was conducted on 100 males aged 20–40 years, including 50 smokers and 50 non-smokers, for one year. Smokers with a history of at least 10 cigarettes per day for five years were included. Pulmonary function was assessed using spirometry, and the smoking index was calculated.

Results: Smokers showed a significant reduction in pulmonary function compared to non-smokers. Parameters such as FVC, FEV_1, FEV_1/FVC ratio, PEFR, and FEF 25–75 were significantly decreased ($p < 0.05$). A dose–response relationship was observed with increasing smoking intensity and duration.

Conclusion: Smoking significantly impairs pulmonary function, particularly airway parameters. Early cessation may improve lung function and reduce disease progression, emphasizing the need for preventive strategies and awareness programs.

Keywords: Smoking, Pulmonary Function Tests, Spirometry, FVC, FEV_1, COPD.

Introduction

Annually, cigarettes are estimated to cause the deaths of approximately five million individuals worldwide. The World Health Organization (WHO) indicated that tobacco smoking resulted in the deaths of 100 million individuals globally in the 20th century and cautioned that it may cause one billion fatalities in the 21st century.

The act of consuming tobacco is a contagious and dangerous issue. All possible measures are being implemented at the governmental level to eradicate smoking. It has been designated as a criminal offense and a penalized act. An ordinance imposing penalties on cigarette smokers has been enacted; nonetheless, cigarette smoking continues to thrive [1]. Deaths attributable to tobacco would rise to almost 10 million annually. Since 1975, tobacco smoking rates have diminished in industrialized nations; nevertheless, low-income countries have experienced a concomitant 50% rise in smoking rates.

Cigarette smoking significantly impacts respiratory function and is unequivocally associated with the development of several respiratory disorders, notably chronic bronchitis, emphysema, and bronchial cancer [2]. In the process of burning tobacco in cigarettes, over 4000 identifiable compounds are produced, including those that are present in the tobacco itself or novel compounds that are generated as a result of pyrolysis, prosynthesis, distillation, sublimination, hydrogenation, oxidation, decarboxylation, and dehydration. They comprise smoke dust particles that disrupt respiratory airway function, tears that irritate bronchial epithelium (tar is the residual matter after removing nicotine and moisture), and nicotine, which accelerates heart rate and raises systolic blood pressure [3].

Inhalation of tobacco smoke results in an instantaneous increase in airway resistance that lasts for a minimum of one hour. This results from vagally mediated smooth muscle contraction, likely through the stimulation of submucosal irritant receptors. Experimental investigations indicate that extended cigarette smoking disrupts ciliary movements, while the restriction of alveolar macrophage activity results in hypertrophy and hyperplasia of mucus-secreting glands [4]. It is likely that smoke inhibits antiproteases and prompts polymorphonuclear leukocytes to abruptly release proteolytic enzymes.

Cigarette smoking is the predominant risk factor for COPD, with tobacco significantly contributing to this risk. Chronic obstructive pulmonary disease (COPD) is defined by the gradual blockage of airflow and the deterioration of lung tissue, resulting from the prolonged exposure of genetically predisposed individuals to environmental causes. Tobacco smoking has been linked to the risk of COPD since the 1950s; it was identified as a causal risk factor in a 38-year prospective analysis including 792 men, and these findings were corroborated by the larger and more extensive Framingham cohort offspring study [5]. Subsequently, subsequent research has concentrated on smoking as the primary risk factor for COPD; several prevalence studies have been conducted only among smokers, and most clinical trials for COPD include only smokers with a minimum of 20 pack-years of cigarette exposure [6].

In India, smoking is a prevalent habit that is present in both urban and rural areas, regardless of the method of smoking, such as cigarettes, pipes, cigars, or hookahs. Cigarette smoke is a heterogeneous aerosol generated by the incomplete burning of tobacco leaves. Tobacco consumption in India predominantly occurs in the form of bidis (54%), followed by smokeless tobacco (27%) and cigarettes (9%) [7]. Cigarette smoking significantly impacts respiratory function and is unequivocally associated with the development of several respiratory disorders, notably chronic bronchitis, emphysema, and bronchial cancer.

Pulmonary function tests that measure the diameter of airways, such as forced expiratory flow in one second (FEV), experience a precipitous decline as a result of smoking. Even in adolescents who have smoked for only a few years, maximum expiratory flow volume curves exhibit a reduction in flow rate at low lung capacities, indicating airway obstruction. Lung function tests are a potent instrument in evaluating respiratory conditions [8].

Diagnosing an obstructive airway illness without assessing airflow is likely as improper as diagnosing hypertension without evaluating blood pressure. The lung function test can assist in the diagnosis, as well as in the objective evaluation of severity and the monitoring of the response to treatment [9]. Smoke may be injurious because it contains an unrefined form of tobacco as compared to cigarettes which was proved experimentally. Smoking is a leading avoidable cause of morbidity and death globally and is a recognized risk factor for chronic respiratory illnesses. Cigarette smoke comprises many toxic agents that compromise airway function, diminish lung elasticity, and hasten the deterioration of pulmonary function, frequently resulting in chronic obstructive pulmonary disease (COPD). Pulmonary function tests (PFTs) provide an objective and dependable means of evaluating respiratory health and identifying early airflow restrictions.

This cross-sectional study aimed to analyze and compare pulmonary function parameters between adult male smokers and non-smokers aged 20–40 years, and to determine the impact of smoking amount and duration on lung function. This study seeks to elucidate the dose-dependent effects of cigarette smoking on respiratory physiology and underscore smoking as a modifiable risk factor for respiratory dysfunction.

Methodology

Study Design: This study was designed as a cross-sectional observational study to assess and compare pulmonary function parameters between smokers and non-smokers. Adult male participants aged 20–40 years were evaluated at a single point in time. The design enabled assessment of the association between smoking exposure and lung function without follow-up.

Study Area: This study was carried at the Department of Physiology, Lord Buddha Koshi Medical College and Hospital, Saharsa, Bihar, India.

Study Duration: The study was conducted over a period of one year.

Study Size: All 100 male individuals aged 20 to 40 years were recruited and categorized into two groups: 50 smokers and 50 non-smokers. The sample size comprised all eligible cases presenting during the study period and fulfilling the inclusion criteria.

Study Population: The population was chosen from the Department of Physiology at Lord Buddha Koshi Medical College and Hospital in Saharsa, Bihar, India, ensuring that both smokers and non-smokers were age-matched for cross-sectional analysis.

Data Collection: Data were collected by categorizing participants into smokers and non-smokers according to World Health Organization criteria. Pulmonary function tests were performed using a Schiller/Spandan spirometer to measure parameters such as FVC, FEV_1, FEV_1/FVC ratio, PEFR, and FEF 25–75. For smokers, the smoking index was calculated based on the number of cigarettes smoked per day and the duration of smoking to assess dose–response relationships.

Inclusion Criteria:

- Males of 20-40 years of age.
- Minimum of 10 cigarettes/day for 5 years.
- Lord Buddha Koshi Medical College and Hospital, Saharsa, Bihar, India
- Schiller/Spandan Spirometer with laptop.
- Control groups are non-smokers of the same age.

Exclusion Criteria:

- Females
- Athletes.
- Males having diseases of lung/any chronic medication.
- Guthka or pan chewing or any other types of nicotine intake 15. Males < 10 cigarettes/day

Study Procedure: The research was performed in the Department of Physiology at Lord Buddha Koshi Medical College and Hospital in Saharsa, Bihar, India, spanning one year from November 2014 to October 2015. All 100 male individuals aged 20 to 40 years were recruited and categorized into two groups: 50 smokers and 50 non-smokers.

Smokers were classified according to WHO guidelines, necessitating a minimum consumption of 10 cigarettes daily for a duration of at least 5 years. The smoking index was computed for each smoker to evaluate the dose-duration response, utilizing the formula:

Smoking Index
= Average number of cigarettes per day
× Duration in years

Women, athletes, individuals with chronic medication or respiratory diseases, and those who consumed alternative nicotine products (gutkha, pan, etc.) were excluded from the study. Pulmonary function testing was conducted with a Schiller/Spandan Spirometer with laptop assistance. The assessed metrics were Forced Vital Capacity (FVC), Forced Expiratory Volume in one second (FEV1), FEV1/FVC ratio, Peak Expiratory Flow Rate (PEFR), and Forced Expiratory Flow (FEF).

The gathered data underwent statistical analysis to compare lung function in smoker's vs non-smokers and to assess the relationship between smoking quantity/duration and pulmonary function degradation.

Statistical Analysis: The investigation demonstrated a statistically significant reduction in all principal lung function metrics among smoker's vs non-smokers. The impairment exhibited a dose-response relationship, with more significant decreases noted in those who smoked a higher number of cigarettes daily and for extended periods.

The results affirm that smoking is significantly linked to airway blockage and diminished pulmonary function, underscoring its status as a principal risk factor for chronic respiratory illnesses like COPD. The study indicates that lung function declines more swiftly among smokers, with the deterioration correlating with both the severity and duration of smoking."

Result

In table 1 the data indicates that, of the 50 smokers examined, 58% were categorized as light smokers, 28% as moderate smokers, and 14% as heavy smokers. The data indicates that most participants were classified as light smokers, with a decreasing number in the moderate and heavy smoking categories, mirroring the general distribution of smoking intensity among the study population.

Table:1: Grading of Smokers

Grade of Smokers	Number of Smokers	Percentage (%)
Light smokers	29	58
Moderate smokers	14	28
Heavy smokers	7	14
Total	**50**	**100**

The evaluation of lung function tests in Table 2 indicated that smokers had markedly lower results than non-smokers. The mean values of FVC, FEF 25–75, PEFR, FEV_1, and the FEV_1/FVC ratio were decreased in smokers, with statistically significant differences ($p < 0.05$). This unequivocally demonstrates that smoking significantly deteriorates lung function, notably influencing airway flow and capacity.

Table 2: Comparison of Various Pulmonary Function Tests between Smokers and Non-Smokers

Sr. No	Parameter	Non-Smokers (Mean ± SD)	Smokers (Mean ± SD)	P Value
1	FVC (L)	2.85 ± 0.57	1.71 ± 0.60	< 0.05
2	FEF 25–75 (L/s)	2.77 ± 1.05	1.27 ± 0.52	< 0.05
3	PEFR (L/s)	5.93 ± 1.92	3.07 ± 1.68	< 0.05
4	FEV_1 (L)	2.40 ± 0.51	1.20 ± 0.39	< 0.05
5	FEV_1 / FVC (%)	84.64 ± 9.35	72.15 ± 14.28	< 0.05

In Table 3 Pulmonary function deteriorated substantially with daily cigarette use. Individuals smoking 10–20 cigarettes per day had comparatively superior values; however, those consuming 21–30 cigarettes per day showed additional declines, with the most diminished values observed in smok-

ers of 31 or more cigarettes per day. All indices, including FVC, FEF 25–75, PEFR, and FEV_1, exhibited substantial reductions ($p < 0.05$), substantiating that increased daily cigarette use results in larger deterioration of pulmonary function.

Table 3: Effect of No. of Cigarette Smoked Per Day on Pulmonary Function Tests				
Parameter	**10–20 Cig/day (n ≈ 20)**	**21–30 Cig/day (n ≈ 18)**	**≥31 Cig/day (n ≈ 12)**	**P Value**
FVC (L)	1.90 ± 0.63	1.75 ± 0.56	1.50 ± 0.33	< 0.05
FEF 25–75 (L/s)	1.27 ± 0.60	0.87 ± 0.39	0.80 ± 0.41	< 0.05
PEFR (L/s)	3.16 ± 1.30	3.10 ± 2.26	2.79 ± 1.02	< 0.05
FEV_1 (L)	1.40 ± 0.41	1.09 ± 0.32	0.99 ± 0.31	< 0.05
FEV_1/FVC (%)	73.57 ± 17.14	69.76 ± 13.15	73.94 ± 9.69	< 0.05

In **Table 4,** Pulmonary function tests deteriorated progressively with increased smoking duration. Individuals who smoked for 5–10 years exhibited elevated levels; however, the decreases were more pronounced in the 11–15-year cohort, and minimal in the 16–20-year group. All measures, including FVC, FEF 25–75, PEFR, and FEV_1, exhibited substantial reductions ($p < 0.05$), substantiating that continued smoking induces progressive lung function deterioration.

Table 4: Effect of Duration since Smoking on Pulmonary Function Test				
Parameter	**5–10 years (n ≈ 18)**	**11–15 years (n ≈ 17)**	**16–20 years (n ≈ 15)**	**P Value**
FVC (L)	1.91 ± 0.40	1.75 ± 0.71	1.50 ± 0.53	< 0.05
FEF 25–75 (L/s)	1.19 ± 0.45	1.01 ± 0.44	0.93 ± 0.65	< 0.05
PEFR (L/s)	4.05 ± 2.00	2.74 ± 1.46	2.87 ± 1.58	< 0.05
FEV_1 (L)	1.39 ± 0.26	1.23 ± 0.42	1.05 ± 0.38	< 0.05
FEV_1/FVC (%)	74.48 ± 15.14	72.16 ± 12.76	70.70 ± 16.05	< 0.05

In **table 5** pulmonary function testing indicated substantial declines in smokers from both age demographics relative to non-smokers. In the 21–30-year cohort, smokers exhibited significantly reduced values for FVC, FEF 25–75, PEFR, FEV_1, and FEV_1/FVC ratio compared to non-smokers, with analogous reductions noted in the 31–40-year cohort. All differences were statistically significant ($p < 0.05$), indicating that smoking adversely affects pulmonary function irrespective of age.

Table 5: Comparison of Various Pulmonary Function Tests among Smokers and Non-Smokers in Relation to Different Age Groups				
Parameter	**Age Group (Years)**	**Non-Smokers (Mean ± SD)**	**Smokers (Mean ± SD)**	**P Value**
FVC (L)	21–30	2.87 ± 0.47	1.00 ± 0.46	< 0.05
	31–40	2.86 ± 0.43	1.66 ± 0.43	< 0.05
FEF 25–75 (L/s)	21–30	2.80 ± 0.89	1.04 ± 0.43	< 0.05
	31–40	2.75 ± 0.86	1.03 ± 0.42	< 0.05
PEFR (L/s)	21–30	5.97 ± 1.59	3.05 ± 1.22	< 0.05
	31–40	5.90 ± 1.52	3.04 ± 1.20	< 0.05
FEV_1 (L)	21–30	2.42 ± 0.38	1.20 ± 0.33	< 0.05
	31–40	2.41 ± 0.36	1.18 ± 0.33	< 0.05
FEV_1/FVC (%)	21–30	84.78 ± 8.08	72.31 ± 11.76	< 0.05
	31–40	84.46 ± 8.03	72.73 ± 11.39	< 0.05

Discussion

The dangers of smoking are widely recognized; nonetheless, the continuation of this practice has prompted this study, which aims to compare the lung function tests of smokers with those of non-smokers. This research contrasted 40 male smokers with 40 healthy nonsmokers. The smokers group comprised individuals who have smoked a minimum of one pack of cigarettes daily for at least five years. The quantification of tobacco smoking was achieved by determining the smoking index for smokers. 42.5% of smokers belonged to the 20-30 age group, while 57.5% were in the 31-40 age group."

Light smokers comprised 57.5% of the smokers; moderate smokers comprised 27.5%, and severe smokers comprised 15%, as indicated by the smoking criteria. Spirometry was conducted on both smokers and non-smokers to assess lung function testing. FVC was considerably reduced in smoker's vs to non-smokers. The pulmonary function test parameters of smokers were compared with those of non-smokers, serving as the control group. The results indicated a statistically significant reduction

in FVC, FEV, FEV/FVC ratio, PEFR, and FEF in smokers relative to non-smokers. This study assessed the impact of smoking quantity and duration on several pulmonary function tests (PFTs), revealing a significant association between pulmonary function impairment and both the length of smoking and the number of cigarettes smoked daily [10].

A study of gold miners revealed that all participants who succumbed to COPD were smokers; an interaction between smoking history and dust exposure influencing COPD mortality was identified [11]. A recent study conducted among smokers and non-smokers concluded that the actual values of FVC, FEV1, the FEV1/FVC ratio, FEF25-75%, and PEFR are diminished in smokers relative to non-smokers, with all values further declining with prolonged smoking duration and an increased number of cigarettes smoked daily [12].

The current investigation revealed no significant difference in FVC between the two groups. Gupta et al. and Mahajan et al. reported no alterations in FVC among smokers [13]. In the current study, the female population in the nonsmoker group exceeded that of males. In females, the parameters are lower than in men, perhaps due to the differences in lung size between the sexes [14]. Males often possess bigger lung capacity relative to their height [15]. The lack of significance in metrics such as FVC and the FEV1/FVC ratio between the two groups may be attributed to this explanation.

This demonstrated a dose-response connection. Their investigation found that pulmonary function tests among smokers and non-smokers of the same age group and BMI were not substantially correlated with most spirometric parameters. In our study, decreased FEV1 in smokers signifies obstructive lung disease. The study's limitations were the lack of matching in age and height among individuals in both groups. Furthermore, the non-smoker group had a higher proportion of females.

This provides evidence that lung function in smokers, collectively, deteriorates more swiftly than that of non-smokers. The impact of cigarette smoking, as a controllable variable, has been thoroughly examined, revealing that lung function improvement occurs subsequent to a reduction in smoking. Additional research is necessary to ascertain the threshold at which a rapid decrease can be mitigated and to identify the associated traits.

Conclusion

This cross-sectional study clearly indicates that cigarette smoking significantly impairs lung function. Smokers had a significant decline in all principal spirometry metrics, FVC, FEV_1, FEV_1/FVC ratio, PEFR, and FEF 25–75, relative to age-matched non-smokers, signifying primary airway obstruction. Lung function deterioration was determined to be dose-dependent, with more significant impairment noted in persons who smoked a greater quantity of cigarettes daily and for an extended period.

The decline in pulmonary function was apparent across all examined age demographics, affirming that smoking negatively impacts respiratory health regardless of age. These findings demonstrate that lung function in smokers declines more swiftly than in non-smokers and underscores smoking as a significant modifiable risk factor for the onset of chronic respiratory illnesses, including COPD. Prompt detection of pulmonary function deterioration with spirometry and prompt cessation of smoking can markedly enhance respiratory outcomes and decelerate disease development. Consequently, robust public health initiatives, educational programs, and counseling focused on smoking prevention and cessation are critically required to alleviate the burden of smoking-related respiratory morbidity.

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