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Research Article

Correlation of Peak Expiratory Flow Rate (PEFR) with Waist-to-Hip Ratio (WHR) Among Adult Male Smokers in Rural Western Rajasthan: A Cross-Sectional Study

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ABSTRACT

Background:
Peak expiratory flow rate (PEFR) is an important pulmonary function test (PFT) parameter. Waist-to-hip ratio (WHR) is a reliable index of central obesity. Both the central obesity and smoking are well-recognized independent risk factors causing deterioration of pulmonary functions. Yet, limited studies have evaluated their combined impact, particularly in rural populations.

Objective:
To assess the relationship of PEFR with WHR in male smokers and determine the joint effect of central obesity and smoking on lung function.

Methods:
A cross-sectional study was conducted among 271 adult males (18–47 years) from rural Western Rajasthan. Participants were classified into four groups: Group I—Nonsmoker/Nonobese (control), Group II—Nonsmoker/Obese, Group III—Smoker/Nonobese, and Group IV—Smoker/Obese. WHR was calculated from waist and hip circumferences, and PEFR (L/s) was measured using computerized spirometry. Statistical analysis included ANOVA, Levene's test for homogeneity of variances, Games–Howell post hoc, and Spearman's correlation tests.

Results:
PEFR was lowest in the Smoker/Obese group, followed by Smoker/Nonobese and Nonsmoker/Obese, compared with controls. ANOVA showed a significant difference in PEFR across groups ($p < 0.001$). Spearman's analysis demonstrated a significant inverse correlation of PEFR with both WHR ($p < 0.001$) and smoking status ($p < 0.001$).

Conclusion:
Central obesity and smoking exert additive adverse effects on pulmonary function, with the combined impact being more detrimental than either factor alone. Early detection and integrated preventive strategies are crucial in rural populations with limited healthcare access.

Keywords*: Peak Expiratory Flow Rate, Central Obesity, Waist-to-Hip Ratio, Smoking, Pulmonary Function, Rural Health*

INTRODUCTION:

Peak expiratory flow rate (PEFR) is defined as the maximum rate of airflow (in liters per second) achieved during a forceful expiration following full lung inflation after a deep inspiration (Pocock et al.,1999; DeVrieze et al., 2024)[1,2]. PEFR serves as a sensitive indicator of large airway function; as it reflects the forced expiratory phase of the breathing maneuver and. As a volitional measure, PEFR is influenced by both respiratory muscle strength and the subject's maximal effort.

PEFR is widely recognized for its utility in both acute and chronic respiratory care. It offers clinicians a quick, objective, and cost-effective means to monitor lung function, assess the severity of airway obstruction, and evaluate therapeutic

outcomes (Neuspiel et al., 2020; Quanjer et al., 1997)[3,4]. The British Medical Journal (1959) endorsed PEFR as a practical bedside test, citing its simplicity and reproducibility[5]. First introduced by Hadorn in 1942, PEFR became a standardized component of pulmonary function testing by 1949 (Ferretti et al., 2001)[6]. The use of peak flow meters has since gained popularity for their affordability and ease of use in both clinical and field settings (Dhungel et al., 2008)[7].

Obesity, particularly central obesity or abdominal adiposity, is a well-established risk factor for many chronic diseases, like cardiovascular disorders (CVDs), non-alcoholic fatty liver disease and type 2 diabetes (Kasper et al., 2005; McClean et al., 2008)[8,9]. It is also known to adversely affect pulmonary functions by impairing the respiratory mechanics, increasing work of breathing, and altering gas exchange dynamics (Flegal et al., 2002)[10]. The accumulation of abdominal fat restricts diaphragmatic movement and chest wall compliance; which compromises the lung volumes and peak expiratory flow.

Although, body mass index (BMI) is being widely used in epidemiological studies to assess general obesity, but it has notable limitations in predicting pulmonary function, as it can't differentiate fat from lean mass or indicate fat distribution. In contrast, other more reliable indicators of central obesity, like waist-to-hip ratio (WHR) and waist circumference (WC) have shown to better correlate with impaired lung functions (Mamtani et al., 2005; Carey et al., 1999; Chen et al.,1999; Pouliot et al., 1994)[11–14]. Adiposity (central obesity) exerts inflammatory and mechanical influences on the respiratory system, reducing lung compliance, limiting chest wall expansion, and increasing airway resistance (King et al., 2005; Biring et al., 1999; Luce et al., 1980)[15–17].

Multiple studies have highlighted a significant inverse relationship between central adiposity markers (such as WC and WHR) and PFT parameters, including PEFR (Chen et al., 1993; Collins et al., 1995)[18,19]. Reports from the World Health Organization (2000, 2011) have also reinforced the importance of WC and WHR as key anthropometric markers of central obesity and related health risks. [20,21]. In particular, Saxena et al. (2011) concluded that waist to hip ratio (WHR) is a far more accurate index of central obesity than BMI or body weight; which can be used as predictor of expiratory flow. [22].

Reduced PEFR in obese individuals reflects a restrictive ventilatory defect. Excess adipose tissue imposes mechanical constraints on thoracic and diaphragmatic movement, ultimately limiting effective lung expansion (Raison et al., 2001; Benjaponpitak et al., 1999) [23,24]. However, the extent to which various adiposity indices predict PEFR impairment, may vary across different populations and contexts. There has been a significant implication of adipokine imbalance in the pathogenesis of COPDs, asthma and airway dysfunction in obese individuals (Alipour et al., 2015)[25].

Cigarette smoking is another well-documented contributor to cardio-vascular and pulmonary deterioration. It induces structural and inflammatory changes in the airways, including epithelial cell damage, mucus hypersecretion, squamous metaplasia, and fibrosis (Ambrose et al., 2004; Bohadana et al., 2004)[26,27]. All of these important changes lead to bronchial wall thickening, narrowing of the airways, and impaired airflow. Smoking is the primary cause of chronic obstructive pulmonary disease (COPD), which is characterized by chronic inflammation of airways and airflow limitation. The chronic exposure to tobacco smoke results in irreversible damage to lung architecture and function (U.S. Surgeon General's Report, 2010)[28]. In clinical settings, PEFR is routinely used to monitor disease severity, guide treatment, and assess response to interventions in evaluating obstructive pulmonary diseases such as COPD and asthma. (Enright et al., 2014)[29].

Previously, there had been many studies showing direct adverse effects of smoking on pulmonary health (Medabala et al., 2013; Wannamethee et al.,1995)[30,31]. Similarly, many studies showing impact obesity (abdominal obesity) alone on pulmonary functions (Lazarus et al., 1997; Chen et al., 2007; Costa et al., 2008; Gupta et al., 2010; Chinn et al., 1996)[32-36]. However, very limited research literature is available about the combined effect of central adiposity (abdominal obesity) and smoking on PEFR, especially among adult male populations.

Study Rationale-

Given the individual impact of central obesity and smoking on pulmonary function, it is plausible that their coexistence may have additive or synergistic effects, resulting in a more pronounced decline in PEFR. However, this interaction has not been extensively studied. The present study aims to fill this gap by evaluating the combined effects of abdominal obesity and cigarette smoking on PEFR among adult males. It is hypothesized that the co-occurrence of these risk factors will result in significantly reduced PEFR compared to individuals with either risk factor along.

AIMS AND OBJECTIVES:

Aims-

The primary aim of our study was to examine the impact of central obesity on peak expiratory flow rate (PEFR) among adult smoker males, and to explore the correlation of PEFR with waist to hip ratio (WHR) as anthropometric marker of central obesity.

Objectives-

1-To compare the PEFR of obese adult males with non-obese counterparts (based on WHR) within the age group of 18–47 years.

2-To evaluate the impact of tobacco smoking on PEFR, and to further compare their PEFR values with nonsmokers.
3-To assess the combined influence of central obesity (WHR) and smoking on PEFR by comparing values between obese and non-obese smokers

MATERIALS AND METHODS:

Study Design

This study was an observational, cross-sectional, analytical study; approved by the Ethical Committee of Dr. S. N. Medical College, Jodhpur; for research involving human participants in health sciences (Approval No. SNMC/IEC/2022/Plan/540; dated 24.03.2022).

Study Participants

A total of 271 adult male participants of rural areas, of age group 18 to 47 years, were recruited through random sampling from the general population and local health facilities in rural Western Rajasthan. A written informed consent (IC) was taken from all subjects prior to data collection.

Grouping of Participants

To evaluate the individual and combined effects of central obesity (WHR) and smoking on PEFR, subjects were categorized into four groups based on their waist-hip ratio (WHR) and smoking status:

1. Group I – Nonsmoker, Nonobese (Control group)
2. Group II – Nonsmoker, Obese
3. Group III – Smoker, Nonobese
4. Group IV – Smoker, Obese

This classification allowed for comparison of PEFR across both isolated and combined conditions of obesity and smoking.

Inclusion Criteria

- ***Obesity classification:***
  - Nonobese: WHR < 0.90
  - Obese (central obesity): WHR ≥ 0.90
- ***Smoking classification:***

* Current smokers: Actively smoking daily at the time of study.
* Subjects must have smoked continuously for at least 1 year, with smoking experience of at least 100 or more beedis/cigarettes during his lifetime.
* Ex-smokers: who used to smoke in the past, but he has quit smoking at least 1 year before recording his spirometry and not smoking currently.
* To qualify as Smoker-Obese, participants had to meet both the obesity and smoking criteria.

- ***Nonsmokers included:***

Individuals who had either never smoked at all during their lifetime, or they had smoked fewer than 100 beedis/cigarettes up to the date of spirometry testing.

Exclusion Criteria

Subjects were excluded from our study if they had any of the following:

- Cardiovascular diseases (CVDs)
- Previously diagnosed respiratory disorders i.e. Respiratory infections, COPD or bronchial asthma
- Musculoskeletal disorders
- Were on medications affecting cardio-respiratory function

Methodology

Waist-to-Hip Ratio (WHR) Measurements

- Measurements of waist circumference (WC) and hip circumference (HC) were taken using a non-stretchable, flexible measuring tape and recorded in meters.
- WC was measured at the end-expiration point of normal breathing, putting the tape at the midpoint between the lower rib margin and iliac crest; with participants standing erect, feet together, and arms at their sides.
- HC was measured by positioning the tape at the widest part of the buttocks.
- Waist-to-Hip Ratio (WHR) was then calculated by dividing values of WC by HC.

Peak expiratory flow rate (PEFR) Measurement

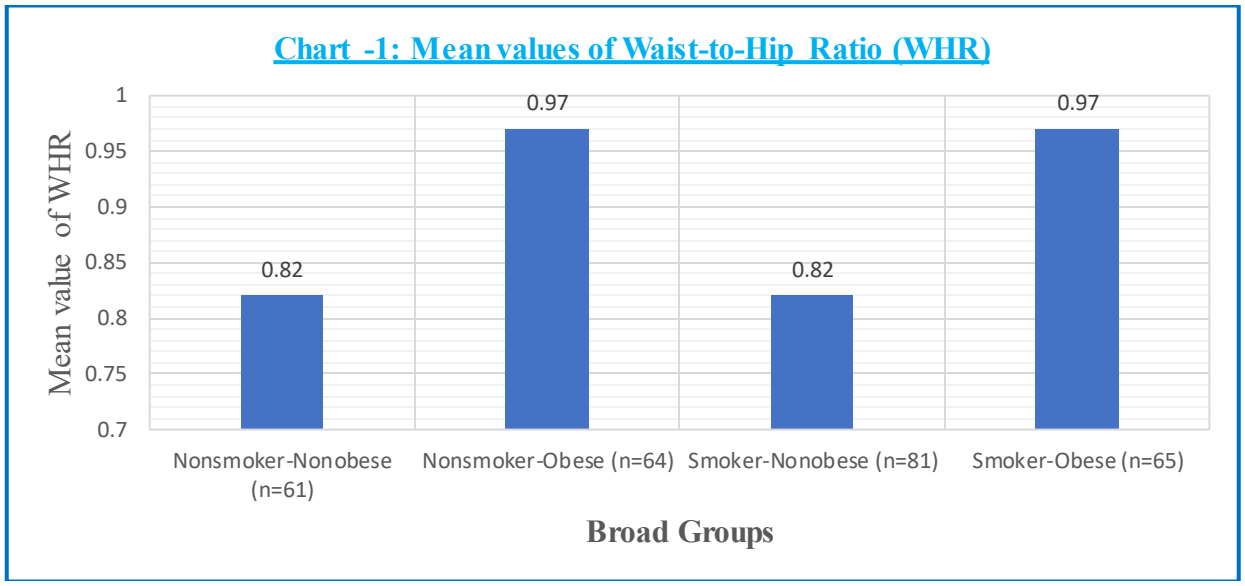
PEFR (in liters/second) measurements were done using a **computerized (electronic) spirometer** (Spiro Excel, Medicaid Systems; PC/Laptop based), following the standardized protocols of the **European Respiratory Society (ERS)** and **American Thoracic Society (ATS)**.

- The spirometer was **calibrated prior to each session**.
- Participants were given 15 minutes of rest and were thoroughly instructed on the manoeuvre.

- After attaching a nose clip and mouthpiece, subjects performed a forceful and rapid expiration following a maximum inspiration.
- A minimum of three acceptable and reproducible recordings were taken, and the highest PEFR value was selected for final analysis.

OBSERVATION AND RESULTS:

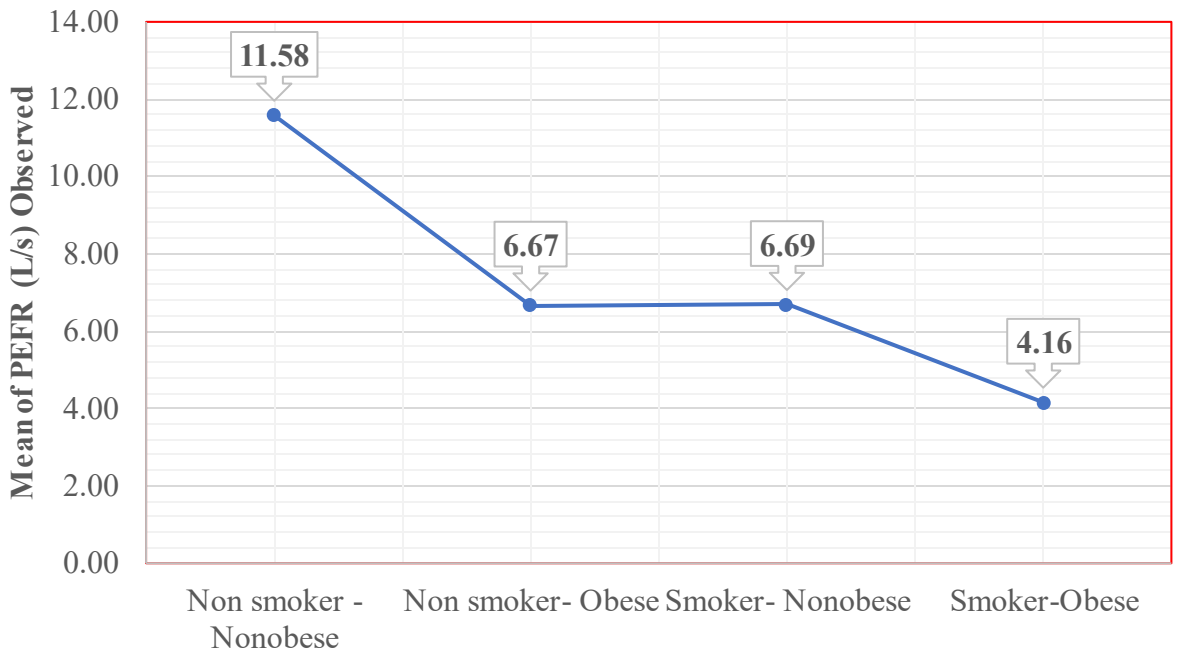
Table-1: - Mean ± SD of Waist-to-Hip Ratio (WHR) in all 4 different broad groups-				
Central Obesity parameter	**Nonsmoker-Nonobese (n=61)**	**Nonsmoker-Obese (n=64)**	**Smoker-Nonobese (n=81)**	**Smoker-Obese (n=65)**
WHR	0.82±0.02	0.97±0.02	0.82±0.03	0.97±0.02



- As shown in above table no.1 and chart no. 1 in our current study we found that Obesity is clearly associated with significantly (and positively) higher WHR.
- Smoking status doesn't notably affect the WHR measures; obesity remains the main contributor.

Table-2:- Mean ± SD PEFR (liters/second) and PEFR (as % of control) in all 4 different broad groups-					
S.No.	**Parameters**	**Nonsmoker-Nonobese (n=61)**	**Nonsmoker-Obese (n=64)**	**Smoker-Nonobese (n=81)**	**Smoker-Obese (n=65)**
1.	Mean **PEFR** (L/s)	11.58±0.89	6.67±2.42	6.69±2.92	4.16±2.77
2.	**PEFR** (as % of control)	100%	57.60%	57.77%	35.92%

Chart-2: Mean of PEFR (L/S) observed in braod groups



- As depicted above both smoking and obesity independently reduce PEFR to about 57% of control. while Combined smoking and obesity further reduce PEFR dramatically to just 36% of control. (Table 2 & chart 2)
- This suggests **additive or possibly synergistic detrimental effects** of smoking and obesity on lung function.

Table -3: Statistical analysis of PEFR (L/s) Observed by ANOVA (Analysis of Variance)

		Sum of Squares	df	Mean Square	F	Sig.
PEFR (L/s) Observed	Between Groups	1809.33	3	603.11	101.23	0.00
	Within Groups	1590.77	267	5.96		
	Total	3400.10	270			

- As shown in above table no. 3 the very high F-value and $p < 0.001$ indicate **statistically highly significant differences** in PEFR between all the four groups included in our current study.

Table 4: Statistical analysis of PEFR (L/s) Observed by Levene's Test (Homogeneity of Variances)

		Levene Statistic	df1	df2	Sig.
PEFR (L/s) Observed	Based on Mean	27.31	3.00	267.00	0.00
	Based on Median	18.34	3.00	267.00	0.00
	Based on Median and with adjusted df	18.34	3.00	220.53	0.00
	Based on trimmed mean	26.04	3.00	267.00	0.00

- Levene's statistics (table no. 4) shows clearly that all p-values are statistically significant (**0.00**) meaning the variances between groups **are not equal,** this justifies for using a **Games-Howell** post-hoc test for further statistical analysis in multiple group comparison.

Table 5: Games-Howell Post Hoc Test for Multiple group Comparison of PEFR (L/s) Observed between the groups

Games-Howell Dependent Variable		Mean Difference (I-J)	Std. Error	Sig.	95% CI	
					LB	UB
Nonsmoker- Nonobese	Nonsmoker- Obese	4.91*	0.32	0.00	4.06	5.75

	Smoker- Nonobese	4.90*	0.34	0.00	3.99	5.79
	Smoker- Obese	7.42*	0.36	0.00	6.47	8.37
Nonsmoker- Obese	Smoker- Nonobese	-0.01	0.44	1.00	-1.17	1.14
	Smoker -Obese	2.51*	0.46	0.00	1.32	3.70
Smoker- Nonobese	Smoker- Obese	2.52*	0.47	0.00	1.30	3.76

***. The mean difference is significant (p-value) at the 0.05 level.**
(Sig.=Significance, CI=confidence interval, LB=Lower Bound, UB=Upper Bound)

Key findings of Games-Howell Post Hoc Test: (table-5):

- A highly significant PEFR differences were obtained between Nonsmoker-Nonobese and all other groups ($p < 0.001$), similar type of statistically significant PEFR differences was observed between Nonsmoker-obese & Smoker -Obese and between Smoker-Nonobese & Smoker- Obese ($p < 0.001$).
- The difference between Smoker-Nonobese and Nonsmoker-Obese is not significant ($p = 1.00$).
- PEFR is lowest in Smoker-Obese, significantly worse than all others.

Interpretation:

- Independently both smoking and obesity impair lung function significantly and reduce PEFR but combined effect (Smoker–Obese) causes the greatest decline.
- The lack of difference between smoking and obesity alone (when not combined) implies **similar individual impacts** on PEFR as shown in below table no. 6.

Table 6- Interpretation of Post Hoc Test for Multiple group Comparison

Comparison	Mean Difference	p-value	Significant?
Nonsmoker - Nonobese **vs** Others	~4.9 to 7.4	0.00	✅ Yes
Nonsmoker - Obese **vs** Smoker- Obese	2.51	0.00	✅ Yes
Smoker - Nonobese **vs** Smoker- Obese	2.52	0.00	✅ Yes
Nonsmoker - Obese **vs** Smoker - Nonobese	0.01	1.00	❌ No

Table 7: Spearman's rho correlation of PEFR (L/s) with WHR & Smoking status

SPEARMAN'S RHO CORRELATION		PEFR L/S (Observed)	Strength
WHR	Correlation Coefficient	-.476**	Moderate negative
	Sig. (2-tailed)	0.000	
	N	271	
SMOKING STATUS	Correlation Coefficient	-.527**	Moderate negative
	Sig. (2-tailed)	0.000	
	N	271	
SMOKING FREQUENCY	Correlation Coefficient	-.647**	Fairly strong negative
	Sig. (2-tailed)	0.000	
	N	271	
SMOKING DURATION	Correlation Coefficient	-.402**	Moderate negative
	Sig. (2-tailed)	0.000	
	N	271	

****. Correlation is significant at the 0.01 level (2-tailed).**

- Spearman's Rho correlation test was performed to assess the direction and strength of monotonic relationships of PEFR with WHR, Smoking status, Smoking frequency and Smoking duration. All these correlations are statistically significant ($p < 0.001$). (Table -7)

Interpretation:

Table no. 08- Summary of Results of Spearman's Rho Correlation

Variable	Correlation (ρ)	p-value	Interpretation
WHR	-0.476	0.000	Moderate Negative
Smoking Status	-0.527	0.000	Moderate Negative
Smoking Frequency	-0.647	0.000	**Strong Negative**
Smoking Duration	-0.402	0.000	Moderate Negative

All correlations are **negative and significant**, suggesting that:

- As WHR, smoking status, frequency, and duration increase, PEFR decreases.
- Smoking frequency has the strongest negative correlation with PEFR, even more than duration or WHR.

DISCUSSION:

The present study was designed to investigate the impact of central obesity and smoking—independently and in combination, on pulmonary function. In our current study we used Waist Hip ratio (WHR) to assess central obesity and peak expiratory flow rate (PEFR) to measure lung function, among adult male subjects.

Total 271 subjects were included in our study after random selection. The study population was than categorized into four groups based on waist-hip ratio (WHR) and smoking status: Nonsmoker–Nonobese (control), Nonsmoker–Obese, Smoker–Nonobese, and Smoker–Obese.

On statistical analysis of mean values of WHR and PEFR findings, a significant decline in PEFR associated with both central obesity and smoking was observed. PEFR values were significantly reduced in all groups with either smoking or obesity, when compared with the Nonsmoker–Nonobese (control group). Specifically:

- The **Nonsmoker–Obese** group demonstrated a substantial decline in PEFR (~42.4% reduction). The possible underlying mechanism responsible for this significant reduction in PEFR by central obesity can be multipronged. Increased visceral adiposity restricts diaphragmatic excursion and expansion of chest wall, lowering total lung capacity (TLC) and vital capacity. Reduced thoracic compliance and external compression of small airways increase resistance and predispose to airway closure during forced expiration. Additionally, adipose tissue–derived cytokines (e.g., IL-6, TNF-α) contribute to low-grade systemic inflammation and airway hyperresponsiveness, further impairing expiratory flow.
- The **Smoker–Nonobese** group showed a nearly identical reduction (~42.2%) in PEFR; which could be because of the fact that tobacco smoking independently reduces PEFR via chronic airway inflammation, mucosal remodeling, and mucus hypersecretion. Destruction of alveolar walls diminishes elastic recoil, while oxidative stress damages airway epithelium, compounding airflow limitation.
- The **Smoker–Obese** group exhibited the **lowest PEFR**, reflecting a maximum (**~64%) reduction** in PEFR relative to the control group. This is because central obesity and smoking, when present combined, they exert additive and potentially synergistic effects. Mechanical restriction from obesity and loss of recoil from smoking markedly reduce expiratory force, while amplified inflammation, oxidative stress, and impaired immune defense accelerate decline in PEFR.

These findings suggest that both smoking and central obesity exert independent adverse effects on pulmonary function. Importantly, their **combined effect appears additive or possibly synergistic**, resulting in a greater than expected reduction in expiratory flow.

A one-way **ANOVA** test showed a statistically very significant difference in PEFR across the four groups ($F = 101.23$, $p < 0.001$), indicating that group classification had a significant impact on expiratory flow. This confirms that PEFR is influenced by both central adiposity and smoking status. Multiple group comparisons by **Games–Howell Post Hoc Test** revealed **highly significant differences ($p < 0.001$)** in PEFR between the control group and all other groups. A highly significant difference in mean value of PEFR of the **Smoker–Obese** group with all other groups, confirming the **compounded negative impact** of concurrent smoking and central obesity.

Levene's test showed a significant result ($p < 0.001$), confirming the violation of the assumption of homogeneity of variances in this study. Since, the variances across groups are unequal, so this justified appropriately the use of the **Games–Howell post hoc test for multiple comparisons**.

Correlation Analysis (Spearman's rho) of PEFR with **Waist–Hip Ratio (WHR):** $r = –0.476$, $p < 0.001$; with **Smoking status:** $r = –0.527$, $p < 0.001$; with **Smoking frequency:** $r = –0.647$, $p < 0.001$; with **Smoking duration:** $r = –0.402$, $p < 0.001$ was also assessed and our results showed a moderate to strong inverse relationship of PEFR with both the **degree of central adiposity** and **smoking exposure**. Notably, **smoking frequency** demonstrated the strongest negative correlation with PEFR, indicating that habitual tobacco use is a critical determinant of reduced lung function.

Results of our study are consistent with the findings of many previous researches. Across diverse populations (Chinese, European, Indian, Thai), evidence consistently shows that **central obesity and smoking are independent risk factors for PEFR decline, but their coexistence produces the greatest impairment**. Multiple studies (Costa, Salam, Canoy, Inthachai) confirm an **additive/synergistic interaction**, aligning with our finding that smoker–obese males had the lowest PEFR values as compared to the Smoker-Nonobese or Nonsmoker-Obese group.

Similar to ours, several studies have previously examined the interaction of central obesity and smoking in relation to pulmonary function decline, particularly peak expiratory flow rate (PEFR). A research study in Chinese adults reported that centrally obese smokers exhibited significantly lower PEFR compared to non-obese smokers, with above-normal

values of waist circumference (WC) and waist–hip ratio (WHR), exerting a stronger influence than general adiposity (Chen et al., 2014)[37]. Similar to it, in a large European cohort study of older men, demonstrated a strong inverse correlation between WC/WHR and PEFR, with both smoking and abdominal fat acting as independent yet additive determinants of reduced lung function (Wannamethee et al., 2005)[38].

Findings of a study by Costa et al. further supported this association, reporting a very significant inverse relationship between PEFR and WC in male smokers, in whom the central obesity exacerbating smoking-induced pulmonary impairment (Costa et al. (2008)[39]. Comparable findings were observed in an Indian population, where it was found that PEFR decline was most pronounced in centrally obese smokers, particularly in those with WC >90 cm and high WHR, with smoking frequency (>10 cigarettes/day) compounding the effect (Salam et al., 2013)[40].

Additional evidence in support of our study, reinforces the synergistic burden of these risk factors. Studies reported that obese smokers exhibited the steepest reductions in lung function parameters compared with either exposure alone (Canoy et al., 2004)[41]. Likewise, another study published in European journal highlighted an accelerated PEFR decline among overweight middle-aged male smokers, underlining the additive risk of obesity and smoking combined (Wannamethee et al., 2005)[42]. More recently, a Thai cross-sectional study by confirmed this interaction, showing that young adult men (20-40 years age group) with both abdominal obesity and smoking exposure had the greatest deterioration in PEFR and respiratory muscle strength compared with controls or single-risk groups (Inthachai et al., 2020)[43].

Collectively, these studies provide consistent evidence across diverse populations that central obesity and smoking impair pulmonary function not only independently but also act synergistically, producing the steepest PEFR decline when both exposures coexist. When coexistent, they act in an amplifying manner, leading to disproportionately greater deterioration of lung function This aligns with the current study's findings that smoker–obese males exhibited the lowest PEFR values, highlighting the additive burden of these two modifiable risk factors.

Table no. 9 - Summary of Statistical Significance

Test	Indicates	Result
Descriptive Statistics	Central tendencies & variation	PEFR lowest in smoker-obese
One-Way ANOVA	Differences between groups	$p < 0.001$ (highly significant)
Levene's Test	Homogeneity of variances	$p < 0.001 \rightarrow$ violated
Games-Howell	Pairwise group differences	Most significant
Spearman's Correlation	Relationship with PEFR	All negative & significant

CONCLUSION: Implications for Practice and Research

Central obesity and smoking act as independent yet synergistic risk factors for impaired pulmonary function, underscoring the need for early screening of PEFR in at-risk populations. Targeted public health interventions promoting smoking cessation and obesity reduction should be prioritized to preserve respiratory capacity and prevent premature decline in lung health.

At the research level, longitudinal and large-scale population studies focusing specifically on PEFR are warranted to strengthen causal inferences and quantify long-term effects. In addition, mechanistic studies exploring the interplay of mechanical, inflammatory, and oxidative pathways may provide deeper insights to guide more effective prevention and treatment strategies.

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CONFLICT OF INTEREST: - None was reported by any author.
ETHICAL APPROVAL: Was taken from the Institutional Ethics Committee.

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