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Correlation of Waist Circumference (WC) with Lung Age in Adult Male Smokers of Rural Western Rajasthan: A Cross-Sectional Study

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ABSTRACT

Background: Independently, central obesity (abdominal adiposity) and tobacco smoking are major risk factors for pulmonary decline; but in their combined state, their effect on lung age - a marker of respiratory aging - remains underexplored in rural India. So, our study targeted to examine the joint impact of these two risk factors on lung age in adult males residing in rural Western Rajasthan.
Aims and Objectives: To evaluate the correlation between Lung age and central obesity (waist circumference) among adult male smokers and to assess the individual and combined impact of central obesity and smoking on lung age.
Materials & Methods: In this cross-sectional study, 271 adult males (18–47 years) from rural Western Rajasthan were classified into four groups by smoking status and obesity (i.e. Nonsmoker-Nonobese, Nonsmoker-Obese, Smoker-Nonobese & Smoker-Obese group). Waist circumference (WC) was calculated using standard anthropometry, and lung age was estimated from FEV_1 and height via the Morris and Temple equation. Data were analyzed using one-way ANOVA and Spearman's correlation.
Results: Mean lung age rose progressively from controls to the smoker–obese group, with significant intergroup differences in ANOVA [$F(3,267) = 92.32$, $p < 0.001$]. As per Spearman's rho correlation, Lung-age was correlated positively with WC ($rs = +0.433$) and smoking status ($rs = 0.587$) ($p \leq 0.001$ for both).
Conclusion: Central obesity and smoking independently accelerate lung aging, with their combination exerting the greatest effect. Dual-targeted interventions are essential to preserve pulmonary health in rural populations.
Keywords: Lung age, Smoking, Central obesity, Waist circumference, Rural population.

INTRODUCTION

Chronic diseases such as lung cancer, airway cancers, chronic obstructive pulmonary disease (COPD) and asthma, ultimately lead to a progressive deterioration of lung functions, resulting in premature aging of the lungs. One of the most significant contributing factors to this

decline is cigarette smoking, which releases a mixture of thousands of lethal chemicals—great many of them exert deleterious effects at cellular level. Due to this, cigarette smoking is widely recognized as one of the most important risk factors causing lung cancers and COPDs.

Obstructions of airways caused by smoking significantly affect the parameters of pulmonary function test (PFT), such as forced expiratory volume in the first second (FEV1), forced vital capacity (FVC), and peak expiratory flow rate (PEFR). [1]

Several PFT-based studies on asymptomatic smokers have shown notable impairments in lung function, particularly in tests related to airway obstruction, when compared with nonsmokers. [2]

Lung age is an emerging and informative concept in respiratory evaluation. It correlates the subject's current pulmonary functions with the age at which such values would be considered normal. Thus, a higher lung age as compared to the individual's chronological age is indicative of poorer lung function and suggests premature aging of the lungs. [3]

Research studies have demonstrated that presenting lung age to smokers in primary care settings improves smoking cessation rates at one-year follow-up. However, these studies did not explain any negative psychological effects of communicating a normal lung age to smokers. [4,5]

Smoking is a widespread community health issue. Disturbingly, it has become ingrained in modern lifestyles, particularly among youth. To combat this epidemic, effective community-level interventions must aim not only at reducing smoking but at its complete eradication.

Newbury et al. (2012) assessed the accuracy of various predictive equations for estimating lung age in smokers and demonstrated that newer equations provided superior reliability compared with traditional models. Given that lung age is frequently employed as a motivational tool in smoking cessation programs, accurate estimation is essential; underestimation may diminish the perceived risks of smoking, whereas overestimation may artificially amplify them. The retrospective analysis of a randomly selected cohort enhances the robustness and generalizability of these findings. [6]

Morris and Temple (1985), in the *Preventive Medicine*, first introduced the concept of "lung age" and demonstrated its utility as a potentially powerful motivational tool is the estimation of lung age in smokers, and comparing it with predicted values for healthy nonsmokers. Highlighting the premature aging of lungs in smokers could serve as a psychological incentive to encourage smoking cessation. [7]

Similarly, the Framingham Offspring Study (Becklake MR & Kauffmann F, 1999) provided evidence of a dose–response relationship, showing that even light smoking contributes to accelerated pulmonary decline when compared with nonsmokers. [8]

Furthermore, central obesity (adiposity) influences pulmonary functions in adults (both middle-aged and elderly). Many mechanisms explain the correlation between central obesity (i.e. waist circumference) and lung functions. The central obesity itself is a more direct restrictive factor, often associated with reduced lung volumes. Additionally, physical activity appears to have an independent association with lung function, not fully explained by either general or central obesity.

There are two principal mechanisms (i.e. mechanical and inflammatory factors) explaining the impact of adiposity (i.e. thoracic and abdominal obesity) on lung function: The excess adipose tissue in the abdominal and thoracic regions applies external pressure (i.e. mechanical factors) on the lungs, limiting their expansion during inspiration. This reduces vital capacity and consequently, the expiratory flow is also diminished. [9]

Similarly, the adipose tissue also acts as a source of inflammatory mediators. Chronic inflammation due to weight gain can damage lung tissue and narrow airway

diameters, contributing to reduced pulmonary function. [10-12]

Central (abdominal) obesity is thus linked to a restrictive pattern of lung disease, characterized by reduced lung volumes with minimal airflow obstruction. The studies by Zammit et al. (2010) supported this hypothesis, which found that abdominal obesity correlates more with restrictive, rather than obstructive, ventilatory patterns. [13]

Many other research studies have independently examined the relationship of central obesity (WC) and lung age. [14-17]

Similarly, many previous studies showed correlation between smoking and lung age. [18-21]

Study rationale:

Since, only very limited evidence exists on whether central obesity in young adult male smokers is associated with lung age deterioration. Therefore, the objective of this study was to examine the combined deleterious effect of both risk factors on lung health (i.e. lung- aging) when present in same individual simultaneously.

Aims & Objectives:

- To evaluate the effect of central obesity (WC) on lung age in adult male smokers.
- To compare these findings with those of obese only (i.e. Nonsmoker-Obese group), smoker only (i.e. Smoker-Nonobese group) and controls (i.e. Nonsmoker-Nonobese group).

MATERIALS & METHODS

Study Design

This was an analytical, observational, and cross-sectional study.

Study Participants

Total 271 adult male subjects, aged 18 to 47 years, were selected by random sampling from the general population and health facilities in the Western rural Rajasthan. Prior to data collection, we obtained a written informed consent (IC) from each and every participant.

Group Categorization

To examine the separate and joint effects of central obesity (WC) and tobacco smoking on lung age, we divided all subjects into four broad groups: Control group (i.e. Nonsmoker -Nonobese), Nonsmoker-Obese group, Smoker-Nonobese group and Smoker-Obese group. This classification helped in comparative analysis of lung age across various combinations of smoking and obesity statuses.

Inclusion Criteria

- **Obesity:** Participants were classified as per criteria for central obesity in Asian populations. They were declared non-obese if their waist circumference was less than 0.90 meter; and obese if waist circumference was either equal or more than 0.90 meter; (i.e. Nonobese=WC $<$ 0.90 m and Obese=WC $\geq$ 0.90 m).
- **Smoking:** Participants were declared as smokers those who had smoked continuously for duration of minimum one year or had smoked at least 100 units of cigarettes/beedis during their lifetime till date of spirometry.
  - Smokers were further divided into current smokers and ex-smokers.
  - Current smokers are those who are smoking even today (i.e. on date of spirometry).
  - Ex-smokers are those who used to smoke in the past but have quit at least 1 year before the date of their spirometry.
  - Nonsmokers were individuals who either never smoked at all or had smoked fewer than 100 units of cigarettes/beedis in their whole life until the spirometry date.
- **Smoker-Obese** group included subjects meeting the criteria of both the obesity and smoking.

Exclusion criteria-

Those subjects were excluded from our study who were suffering from- Respiratory

ailments, COPDs, asthma, cardiovascular diseases (CVDs), musculoskeletal disorders, and those under-treatment with medicines influencing functions of cardio-respiratory systems.

Time Period of study-
Data collection took 1.5 years (July-2022 to December-2024).

METHODOLOGY

Measurements of anthropometric parameters:

- **Height measurements:** Height of subjects was measured in inches using measuring tape. During recording, subjects were standing barefoot upright on flat surface against wall (back/buttocks/heels touching the wall) and with their feet together.
- **Waist circumference (WC) Measurements-** It was measured in metres, by placing measuring tape in horizontal plane at midpoint between the lowest rib and the iliac crest, feet together & subject looking straight forward. The average of WC readings taken after inspiration and after expiration, was made.

Spirometry measurements for FEV1:
Subject's FEV1 was measured using a computerized (electronic) spirometer of Medicaid Systems) following ERS/ATS guidelines. [22]

- Calibration was performed before each test. Subjects were given 15 minutes rest before the measurement and were instructed on the procedure.
- Measurements were performed using a nose clip and mouthpiece. Subjects were asked to perform deep inspiration, and then a forced-rapid expiration.
- Among at least three acceptable readings, the highest value was selected.

Lung Age Estimation-
Lung age was estimated using Morris and Temple's predictive equation, based on measured height (in inches) and FEV1 (in liters):[7]

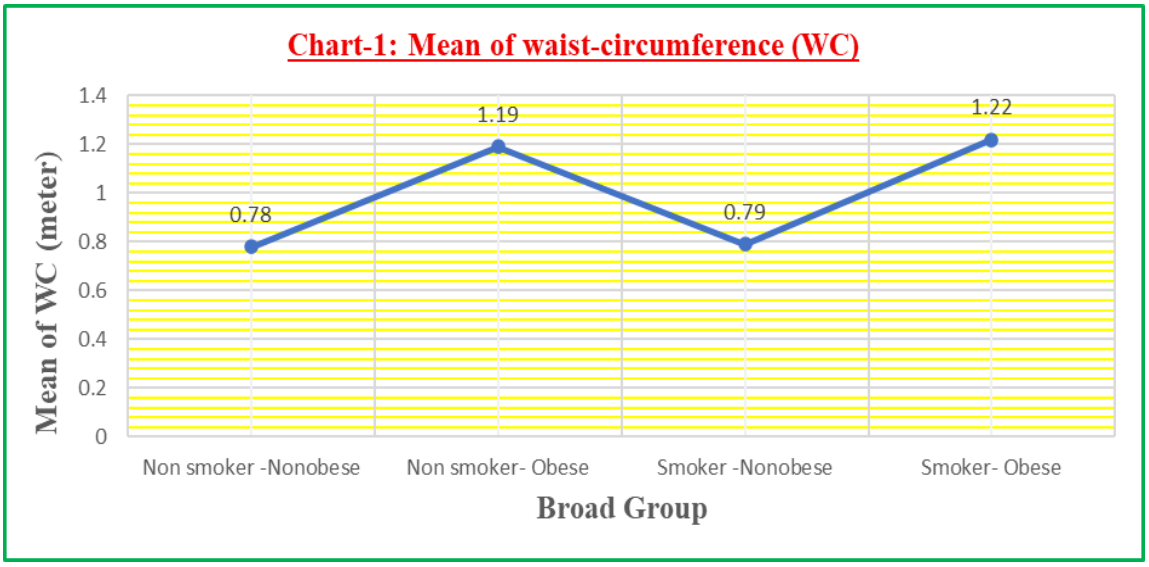
Morris and Temple's predictive equation for Lung age in males:
$Men_{Lung\text{-}Age} = [2.87 \times height\ (inches)] - [31.25 \times observed\ FEV_1\ (lit)] - 39.375$

- Lung age was then compared to the participant's chronological age.

OBSERVATIONS AND RESULTS

Table-1: Mean ± SD of various anthropometric parameters (Chronological age, height and waist-circumference) in all 4 different broad groups-

Anthropometric Parameter	Nonsmoker-Nonobese (n=61)	Nonsmoker-Obese (n=64)	Smoker-Nonobese (n=81)	Smoker-Obese (n=65)
Chronological Age (years)	35.35±8.24	38.18±6.5	28.9±6.5	26.77±6.9
Height (inches)	67.37±1.99	66.53±2.47	67.37±2.33	67.19±3.22
WC (m)	0.78±0.03	1.19±0.12	0.79±0.03	1.22±0.12

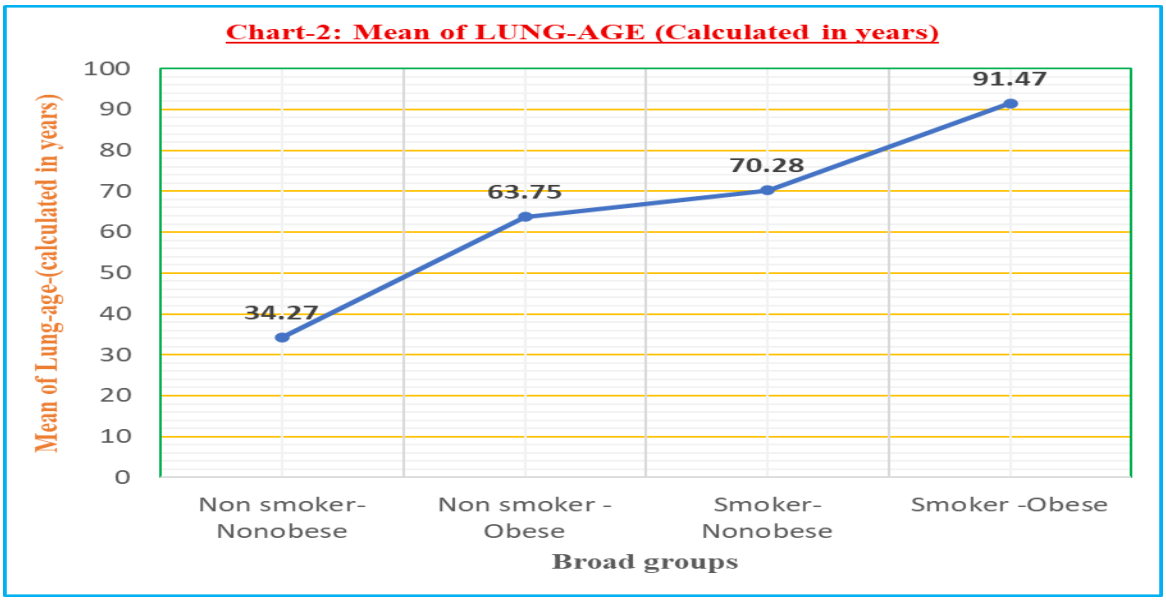


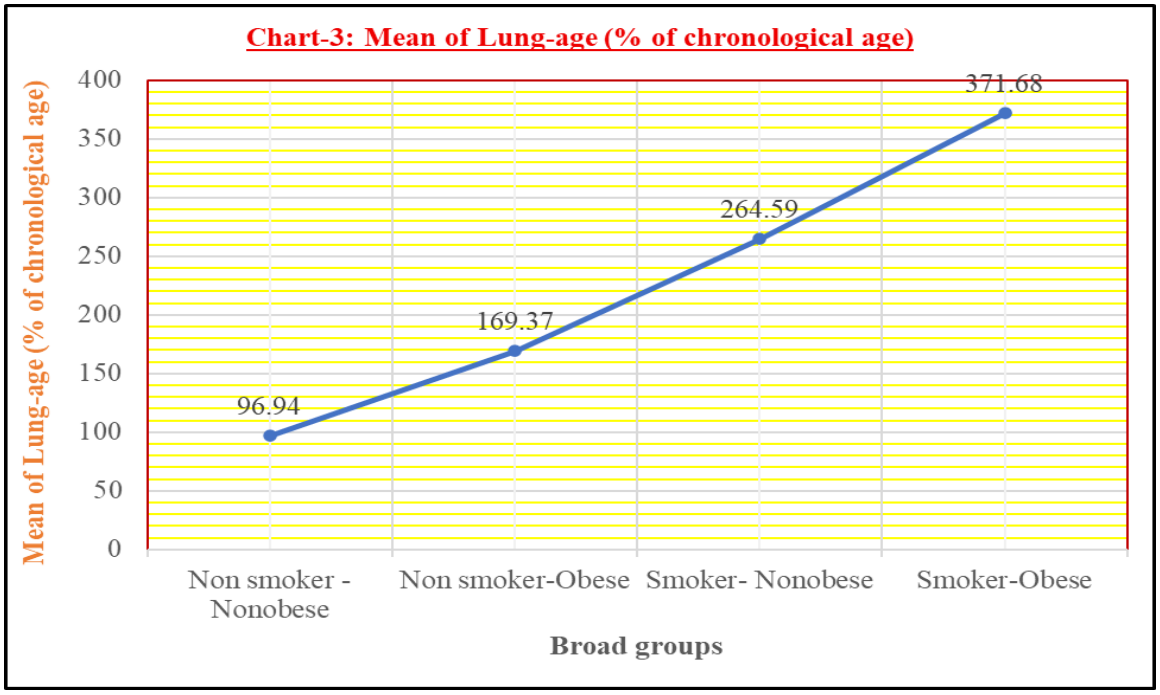
Comparative descriptive analysis of Anthropometric Parameters-

- Nonsmoker–Obese participants were the oldest on average (38.18 years), whereas Smoker–Obese were the youngest (26.77 years).
- Mean height was almost equal (i.e. 67 inches approx.) in 3 groups (Nonsmoker–Nonobese, Smoker–Nonobese & Smoker–Obese). While it was 66.5 inches in Nonsmoker–Obese group.
- Waist circumference was significantly greater in obese groups (~1.22 m) compared with non-obese (~0.79 m), consistent with the expected anthropometric profile. (Table -1 & chart-1).

Table 2: Mean±SD of Respiratory parameters and lung age in all 4 different broad groups-

S. No.	Respiratory Parameters	Nonsmoker-Nonobese (n=61)	Nonsmoker-Obese (n=64)	Smoker-Nonobese (n=81)	Smoker-Obese (n=65)
1.	FEV1 (Liters)	3.83±0.27	2.81±0.52	2.68±0.74	1.98±0.93
2.	Lung age (calculated in years)	34.27±8.06	63.75±15.80	70.28±21.75	91.47±25.82
3.	Lung age (% of chronological age)	96.94±2.59	169.37±40.97	264.59±131.61	371.68±156.46





Intergroup Comparison of Respiratory parameters-

- Lung functions, assessed by FEV_1, was the highest among Nonsmoker–Nonobese individuals (3.83 L) and the lowest in Smoker–Obese individuals (1.98 L). (Table-2).
- Calculated lung age (years) closely matched chronological age (years) in Nonsmoker–Nonobese participants (34.27 years), whereas Smoker–Obese individuals exhibited markedly elevated lung age (91.47 years). (Table-2 & Chart-2).
- When expressed as a percentage of chronological age, premature lung aging was minimal in Nonsmoker–Nonobese (~97%) but pronounced in Smoker–Obese (~372%), highlighting the severe synergistic impact of smoking and central obesity on respiratory health. (Table-2 & Chart-3).
- There was a progressive increase in lung age from healthy to unhealthy categories. The highest lung aging in Smoker-Obese, followed by Smoker-Nonobese, then Nonsmoker-Obese. This demonstrates the additive detrimental effect of smoking and obesity. (Chart-2 & Chart-3).

Table-3: ANOVA (analysis of variances) of Lung-age (calculated in years)

		Sum of Squares	df	Mean Square	F	Sig.
Lung Age (Calculated in years)	Between Groups	105394.00	3.00	35131.40	92.32	0.00
	Within Groups	101600.00	267.00	380.53		
	Total	206995.00	270.00			

- ANOVA test shows highly significant difference in lung age among all 4 groups, with F-value = 92.32, p = 0.000. (Table -3):

Table-4: Levene's test of Homogeneity of variances based on mean of Lung-age (years)

		F-value	df1	df2	Sig.
Lung Age (Calculated)	Based on Mean	28.12	3.00	267.00	0.00

Further, the Levene's test for mean Lung age (years), shows that the variances are unequal across the groups. [F (3, 276) =28.12, ($p < 0.05$)]. (Table-4)

Table-5: Games-Howell post-hoc test for multiple comparison of Lung-age (calculated in years) between the groups

	Games-Howell Dependent Variable		Mean Difference (I-J)	Std. Error	Sig.
Lung Age (calculated in years)	Nonsmoker- Nonobese	Nonsmoker- Obese	-29.49*	2.25	0.00
		Smoker- Nonobese	-36.02*	2.65	0.00
		Smoker-Obese	-57.21*	3.39	0.00
	Nonsmoker- Obese	Smoker-Nonobese	-6.53	3.14	0.17
		Smoker- Obese	-27.72*	3.79	0.00
	Smoker- Nonobese	Smoker-Obese	-21.20*	4.04	0.00

*. The mean difference is significant at the alpha level of p= 0.05 level.

Unequal variances proved by Levene's statistics validates the use of Games-Howell post-hoc test for multiple comparisons between the groups.

- The multiple group comparisons showed significant differences in mean lung age between almost all groups (p = 0.00).
- The largest difference in mean lung age (i.e. 57.21 years) was found between Smoker-Obese and Nonsmoker-Nonobese groups (p = 0.00).
- The minimum difference in mean lung age (i.e. 6.53 years) was found between Nonsmoker-Obese and Smoker-Nonobese groups, that is quite insignificant (p = 0.17).
- It suggests that obesity and smoking may exert comparable but independent adverse effects on lung functions, but their compounded joint effect (i.e. Smoker-Obese group) is far more deleterious. (Table-5)

Table-6: Spearman's rho correlation of lung-age (calculated) with WC and Smoking status and Smoking frequency)

SPEARMAN'S RHO CORRELATION		LUNG AGE (years) Calculated	Strength
Waist circumference (WC)	Correlation Coefficient	.433**	Moderate positive
	Sig. (2-tailed)	0.000	
	N	271	
SMOKING STATUS	Correlation Coefficient	.587**	Moderate positive
	Sig. (2-tailed)	0.000	
	N	271	

**. Significance level of correlation is at the level of p=0.01 (2-tailed).

As per Spearman's rho correlation test, Lung Age is moderately (& positively) correlated with waist circumference: r = 0.433 (p < 0.01). This means the lung age increases with increase in degree of central obesity (WC). Similarly, Spearman's rho correlation of Lung Age with Smoking Status was also found as moderate (& positive) correlation: r = 0.587 (p < 0.01). It shows that the smokers have higher lung age as compared to their nonsmoker counterparts. (Table-6)

DISCUSSION

The present analysis evaluates the combined and independent effects of smoking and central obesity (waist circumference) on lung function and estimated lung age. The data from 271 participants grouped into four distinct categories—Nonsmoker-Nonobese, Nonsmoker-Obese, Smoker-Nonobese, and Smoker-Obese—reveals compelling evidence of lung function deterioration, expressed as reduced FEV1, which is further translated into an accelerated lung aging.

Impact of Smoking and Obesity on Lung Function (individually and in combination)-

In Nonsmoker-Obese group, we observed that mean FEV1 was 2.81 L and calculated lung age was found 63.75 years, far exceeding their mean chronological age (38.18 years). Possible underlying mechanism for this deterioration of lung

functions is that central obesity (adiposity) exerts restrictive effects on pulmonary mechanics. Excess thoracoabdominal fat reduces diaphragmatic excursion and chest wall compliance, lowering lung volumes such as ERV and FRC. A state of low-grade systemic inflammation, characterised by elevated cytokines (e.g., TNF-α, IL-6), contributes to airway hyperresponsiveness and fibrosis. Obesity also disrupts ventilation–perfusion balance and increases the respiratory load of breathing, accelerating fatigue of respiratory mechanics and decline in effective lung capacity.

While in Smoker-Nonobese group, we observed mean FEV1 (2.68 L) and calculated lung age (70.28 years), significantly exceeding to their mean chronological age (28.9 years). Probable explanation of this diminished lung age by tobacco smoking is that it accelerates pulmonary aging through multiple mechanisms. Reactive oxygen species (i.e. free radicles) in cigarette smoke induce oxidative stress, DNA damage, and telomere shortening; leading to premature cellular senescence. Chronic airway inflammation mediated by neutrophils and macrophages, promotes structural remodelling, mucus hypersecretion, and progressive airflow limitation; that is reflected in reduced FEV1. Additionally, destruction of alveolar walls impairs elastic recoil and causes air trapping characteristic of emphysema, while ciliary dysfunction compromises mucociliary clearance, increasing susceptibility to infection and further functional decline.

But the most striking outcome is observed in the Smoker-Obese group, which exhibits the lowest mean FEV1 (1.98 L) and highest calculated lung age (91.47 years), far exceeding their mean chronological age (26.77 years). This suggests a severe compromise in pulmonary function, with the lungs resembling those of individuals many decades older. This is because the combined exposure to smoking and obesity produces synergistic harm. Inflammatory responses from both conditions amplify the tissue damage, while oxidative stress from cigarette smoke and adipose metabolism increases the burden of cellular injury. Mechanical restriction from obesity and alveolar destruction from smoking further compromise ventilatory capacity. Together, these factors impair the repair mechanisms, hasten physiological lung aging, and accentuate decline in pulmonary function.

In contrast, the Nonsmoker-Nonobese group maintained near-normal lung age (34.27 years), closely mirroring their actual age (35.35 years), thereby validating the protective effect of avoiding both risk factors.

Statistical Validation-

The statistical analyses strongly reinforce these findings. ANOVA results demonstrate highly significant differences in lung age across all 4 broad groups (with $p < 0.001$). The multiple comparisons by Games-Howell post-hoc analysis (appropriate for unequal variances), confirms significant pairwise differences between most groups, especially between the extremes (Smoker-Obese vs Nonsmoker-Nonobese, $p < 0.001$). Notably, the difference in mean lung age between Smoker-Nonobese and Nonsmoker-Obese groups was statistically insignificant ($p = 0.17$), indicating a similar detrimental influence of smoking and obesity on lung aging when acting alone.

Correlations and Predictors-

The *Spearman rho correlation analysis* further clarifies the predictive value of the studied variables. A moderate positive correlation of lung age was found with smoking status ($r = 0.587$), and WC ($r = 0.433$). These values suggest that the smoking status alone or obesity, has clinically significant independent toxic effect of on lung tissues.

Previous Research Studies in Support of Our Findings-

In line of our current study, a growing body of evidences indicates that smoking and

central obesity interact to accelerate pulmonary function decline and increase calculated lung age, particularly in men. Large-scale epidemiological investigations have consistently demonstrated this dual burden.

Similar to our study findings, Peters *et al.* (2018) showed in their study that concurrent presence of smoking and obesity exerted additive deleterious effects on pulmonary function, evidenced by accelerated declines in FEV_1 values and increased frequency of chronic respiratory symptoms. [23]

In alignment to our study, Canoy *et al.* (2004) reported that smoking and obesity contributed both independently and synergistically to lung function impairment and elevated respiratory morbidity among middle-aged adults. [24]

Supporting our findings, analyses from the NHANES III survey by researchers Chen et al., (2007) revealed that obese smokers exhibited a disproportionately higher prevalence of impaired lung function and chronic bronchitis compared with individuals who were either non-obese or non-smokers. [25]

Targeted studies in male cohorts provide more detailed insights. Ali et al. (2015) conducted a cross-sectional study of Saudi university men, demonstrating that WC was significantly (& positively) correlated with lung age and was negatively associated with PFT parameters like FEV_1, FVC, and FEF25-75%. So, WC emerging as a superior predictor compared to BMI. [26]

A larger Korean cohort conducted by Oh et al. (2014) showed that the lung-age minus chronological-age difference increased with both smoking exposure (pack-years) and adiposity indices. Multivariate modelling revealed that each 3.7 cm increase in WC corresponded to approximately one additional year of lung aging. [27]

Longitudinal data from the Takahata Study by Sato et al. (2013) in Japanese male smokers further demonstrated that greater abdominal circumference was independently associated with a faster rate of annual FEV_1 decline, a surrogate of accelerated lung aging. [28]

Comparable to our results, Mitsumune et al. (2009), in a study of over 3,000 Japanese men, confirmed that both obesity and smoking were associated with accelerated lung aging. Smokers consistently showed significantly higher lung age than non-smokers. Obese smokers had the worst lung age profiles, supporting the hypothesis of an additive or synergistic detrimental effect. This study was among the first in Asia to confirm that central obesity and smoking do not just impair FEV_1, but also contribute to premature lung aging. It highlights lung age as a simple, communicable marker to motivate for modification in lifestyle (e.g., smoking cessation, weight reduction etc.). [29]

Beyond BMI, several studies underscore the value of central adiposity indices in capturing respiratory risk. For example, a Mediterranean cohort study reported that WC and WHtR were more strongly associated with impaired lung function than BMI, with WHtR values ≥ 0.55 linked to higher lung age.[30]

Mechanistic insights from these studies suggest that central adiposity and smoking impair respiratory health through both mechanical and biological pathways. Abdominal fat accumulation restricts diaphragmatic excursion and chest wall compliance, while systemic inflammation and oxidative stress from obesity and smoking act synergistically to accelerate airway narrowing and parenchymal injury.

Taken together, all the above evidences (in coherence to our study findings), demonstrate a consistent pattern:

- ✓ Male smokers with greater central adiposity exhibit significantly worse spirometric indices and higher lung age, both in cross-sectional and longitudinal analyses.
- ✓ Central obesity measures, particularly WC and WHtR, outperform BMI in predicting respiratory aging, highlighting the need to incorporate

these markers into clinical risk stratification.

- ✓ The dose–response relationship between smoking intensity, abdominal obesity, and lung age underscores the potential value of early lifestyle interventions targeting both exposures to mitigate premature respiratory decline in men.

CONCLUSION: with Clinical and Public Health Implications-

- ➢ This study demonstrates that both adiposity (central obesity) and smoking independently contribute to lung aging, as evidenced by findings of our study by the intermediate lung age values observed in the Nonsmoker-Obese (63.75 years or 169.37% of chronological age) and Smoker-Nonobese (70.28 years or 264.59% of chronological age) groups. Although both show considerable deviation from normal lung age, their impact appears additive rather than synergistic, indicating that even in isolation, each factor exerts substantial physiological stress on the respiratory system.
- ➢ The highest lung age was observed in smoker–obese individuals (i.e. 91.47 years or 371.68% of chronological age), underscoring the compounded risk.
- ➢ Our study showed that waist circumference, as central adiposity markers, was stronger predictor of premature lung aging than BMI, highlighting its value in clinical risk stratification. These findings establish waist circumference (WC) as central adiposity marker and lung age as a clinically valuable practical and sensitive marker for assessing premature respiratory decline, offering both diagnostic utility and motivational potential in patient counselling.
- ➢ From a preventive public health perspective, these results of our study reinforce the importance of integrated preventive strategies that combine the smoking cessation and obesity management (weight reduction) programs. Measuring lung age may also serve as an effective motivational tool, particularly for young smokers who underestimate their immediate health risks. Early interventions targeting these modifiable risk factors may substantially mitigate premature respiratory decline and preserve long-term lung health in adult men.

Declaration by Authors

Ethical Approval: Approved (*Approval No. SNMC/IEC/2022/Plan/540; dated 24.03.2022*).

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Conflict of Interest: The authors declare no conflict of interest.

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