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Original Research Article

Impact of Body Mass Index on Lung Function and Cardiovascular Parameters

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Abstract

Introduction: Obesity is a major public health issue, but its concurrent effects on respiratory and cardiovascular systems warrant further investigation. This study examined the relationship between Body Mass Index (BMI) and pulmonary/cardiovascular function.

Aims and Objectives: This study examined the relationship between Body Mass Index (BMI) and pulmonary/cardiovascular function.

Materials and Methods: In this cross-sectional analysis, 200 participants (mean age 36±10 years; 50% female) with mean BMI 27.5±4.5 kg/m² underwent spirometry and cardiovascular assessment. Statistical analyses included ANOVA and correlation coefficients.

Results: Increasing BMI categories showed progressive lung function decline. FEV1 decreased from 3.5±0.5L (normal weight) to 2.8±0.5L (obese), while FVC declined from 4.5±0.6L to 3.7±0.6L ($p<0.001$). The FEV1/FVC ratio showed slight but significant reduction ($p=0.02$). Conversely, blood pressure increased substantially with BMI elevation ($p<0.001$). Correlation analysis revealed moderate inverse relationships between BMI and FEV1 ($r=-0.42$) and FVC ($r=-0.45$), and strong positive correlations with systolic ($r=0.50$) and diastolic blood pressure ($r=0.48$) (all $p<0.001$).

Conclusion: Elevated BMI demonstrates significant associations with impaired lung function, suggesting restrictive pathology, and increased blood pressure. These findings emphasize the multisystemic impact of excess weight and highlight weight management's crucial role in maintaining cardiopulmonary health.

Keywords: Body Mass Index, Obesity, Lung Function, FEV1, FVC, Blood Pressure, Cardiovascular Parameters, Spirometry.

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Introduction

Body Mass Index (BMI) is widely recognized as a simple yet valuable indicator for assessing body weight relative to height, and it plays a critical role in understanding its influence on overall health. In recent years, rising global trends in overweight and obesity have intensified interest in the relationship between BMI and physiological functions, particularly those involving the respiratory and cardiovascular systems. Excess body mass is known to impose mechanical and metabolic burdens that can impair lung expansion, reduce ventilatory efficiency, and alter gas exchange. Similarly, elevated BMI is linked to significant cardiovascular changes, including increased cardiac workload, altered hemodynamics, and adverse effects on blood pressure and vascular function. Conversely, low BMI may also be associated with reduced muscle mass and diminished cardiopulmonary resilience. Understanding how BMI interacts with lung function and cardiovascular parameters is essential for early identification of at-risk individuals and for guiding preventive health strategies. This topic is especially relevant in clinical and public health contexts, where BMI-related impairments contribute to the growing burden of chronic diseases. Therefore, examining the impact of BMI on respiratory efficiency and cardiovascular health offers important insights for improving patient outcomes and informing targeted interventions. Obesity and abnormal body mass index (BMI) are major global public health concerns, contributing significantly to morbidity and mortality through metabolic, pulmonary, and cardiovascular pathways. High BMI is associated not only with disorders like diabetes but also with impaired lung function and adverse cardiovascular changes, making it crucial to understand its systemic effects [1].

Epidemiological studies consistently demonstrate an inverse relationship between BMI and spirometric indices such as forced vital capacity (FVC) and forced expiratory volume in one second (FEV_1) [1,2]. Mechanistically, excess adipose tissue, particularly in thoracic and abdominal regions, restricts diaphragm and chest wall movement, increases intra-abdominal pressure, and reduces lung compliance, thereby lowering lung volumes [3,4].

The impact is further influenced by fat distribution, with central obesity showing a stronger negative effect [4]. Some studies report a non-linear relationship, where both underweight and obesity impair pulmonary function [5]. BMI also significantly affects cardiovascular parameters. Higher BMI is associated with elevated blood pressure, increased cardiac workload, and structural changes such as left ventricular hypertrophy [6,7]. Hemodynamic studies indicate that obesity increases pulmonary and systemic pressures, contributing to higher cardiac strain [8].

Importantly, these adverse cardiopulmonary effects are at least partially reversible through weight reduction, which improves lung function and cardiac performance [9]. Population-level data show that the combination of high BMI and central obesity markedly increases cardiovascular risk [10]. Overall, high BMI adversely impacts lung function and cardiovascular health, emphasizing the importance of weight management in reducing cardiopulmonary morbidity.

The aim of this study is to examine the impact of Body Mass Index (BMI) on lung function and cardiovascular parameters by evaluating how variations in BMI correlate with key respiratory measures such as FVC, FEV_1, and PEFR, as well as cardiovascular indicators including blood pressure, heart rate, and oxygen saturation. The study seeks to identify whether individuals in different BMI categories—underweight, normal, overweight, and obese—demonstrate significant differences in cardiopulmonary performance. Ultimately, the aim is to determine how alterations in BMI influence overall respiratory and cardiovascular efficiency, providing insights that may support early detection of health risks and guide appropriate preventive or clinical interventions.

Materials and Methods

Study design: This will be a cross-sectional observational study conducted in a tertiary care hospital. The study will assess the relationship between body mass index (BMI) and cardiopulmonary parameters in adult participants.

Place of study: Jagtial, Telangana

Period of study: 1 year

Study Population: Adults aged 18–65 years attending the outpatient department or recruited from the general population.

Sample size: 200

Inclusion Criteria

- Individuals willing to participate and provide informed consent.
- Stable health status without acute illness.

Exclusion Criteria

- Known chronic respiratory diseases (e.g., COPD, asthma).
- History of cardiovascular diseases (e.g., ischemic heart disease, heart failure).
- Pregnancy or conditions affecting BMI independently (e.g., endocrine disorders).

Study Variable

- Age
- Gender
- BMI categories (for population description)
- Body Mass Index (BMI) - analyzed both as a continuous variable and categorically (Normal, Overweight, Obese)
- Forced Expiratory Volume in 1 second (FEV1)
- Forced Vital Capacity (FVC)
- FEV1/FVC ratio
- Systolic Blood Pressure (SBP)
- Diastolic Blood Pressure (DBP)
- Heart Rate

Statistical Analysis: For statistical analysis, data were initially entered into a Microsoft Excel spreadsheet and then analyzed using SPSS (version 27.0; SPSS Inc., Chicago, IL, USA) and GraphPad Prism (version 5). Numerical variables were summarized using means and standard deviations, while Data were entered into Excel and analyzed using SPSS and GraphPad Prism.

Numerical variables were summarized using means and standard deviations, while categorical variables were described with counts and percentages. Two-sample t-tests were used to compare independent groups, while paired t-tests accounted for correlations in paired data.

Chi-square tests (including Fisher's exact test for small sample sizes) were used for categorical data comparisons. P-values ≤ 0.05 were considered statistically significant.

Result

Table 1: Participant Demographics

Characteristic	Value
Total participants	200
Age (years)	36 ± 10
Gender (M/F)	100/100
BMI (kg/m²)	27.5 ± 4.5

Table 2: BMI Category Distribution

BMI Category Distribution		
BMI Category	**n**	**% of Total**
Normal (18.5–24.9)	80	40%
Overweight (25–29.9)	70	35%
Obese (≥30)	50	25%

Table 3: Lung Function by BMI Category

Parameter	Normal BMI	Overweight BMI	Obese BMI	p-value
FEV1 (L)	3.5 ± 0.5	3.2 ± 0.4	2.8 ± 0.5	<0.001
FVC (L)	4.5 ± 0.6	4.2 ± 0.5	3.7 ± 0.6	<0.001
FEV1/FVC (%)	78 ± 5	76 ± 6	75 ± 5	0.02

Table 4: Cardiovascular Parameters by BMI Category

Parameter	Normal BMI	Overweight BMI	Obese BMI	p-value
Systolic BP (mmHg)	118 ± 10	125 ± 12	135 ± 15	<0.001
Diastolic BP (mmHg)	76 ± 8	80 ± 9	88 ± 10	<0.001
Heart Rate (bpm)	72 ± 8	75 ± 9	78 ± 10	0.01

Table 5: Correlation of BMI with Lung Function

Parameter	Correlation (r)	p-value
FEV1	-0.42	<0.001
FVC	-0.45	<0.001
FEV1/FVC	-0.2	0.005

Table 6: Correlation of BMI with Cardiovascular Parameters

Parameter	Correlation (r)	p-value
Systolic BP	0.5	<0.001
Diastolic BP	0.48	<0.001
Heart Rate	0.25	0.001

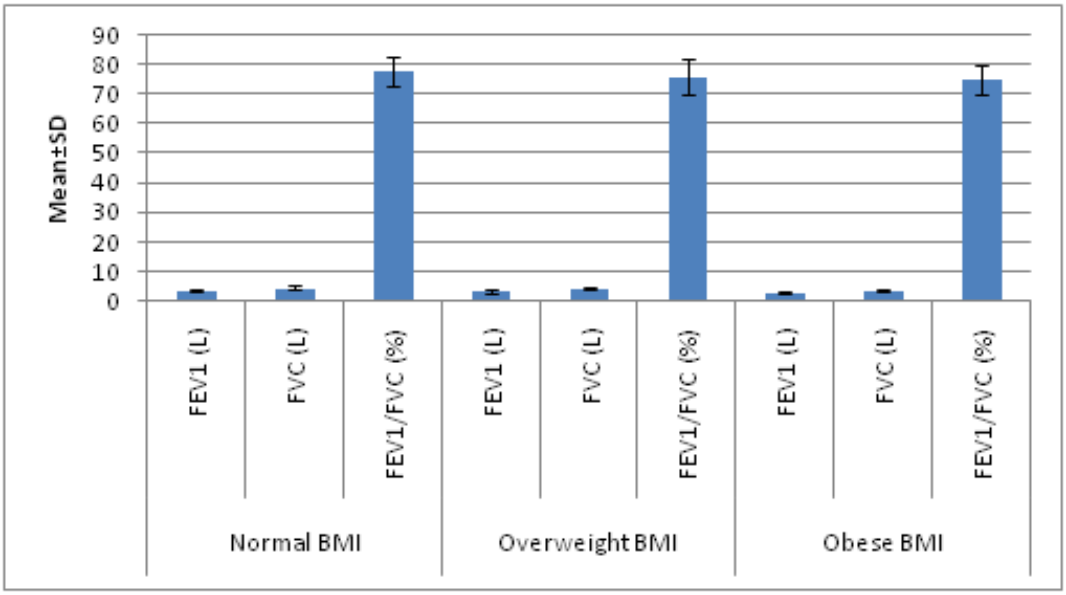

Figure 1: Lung Function by BMI Category

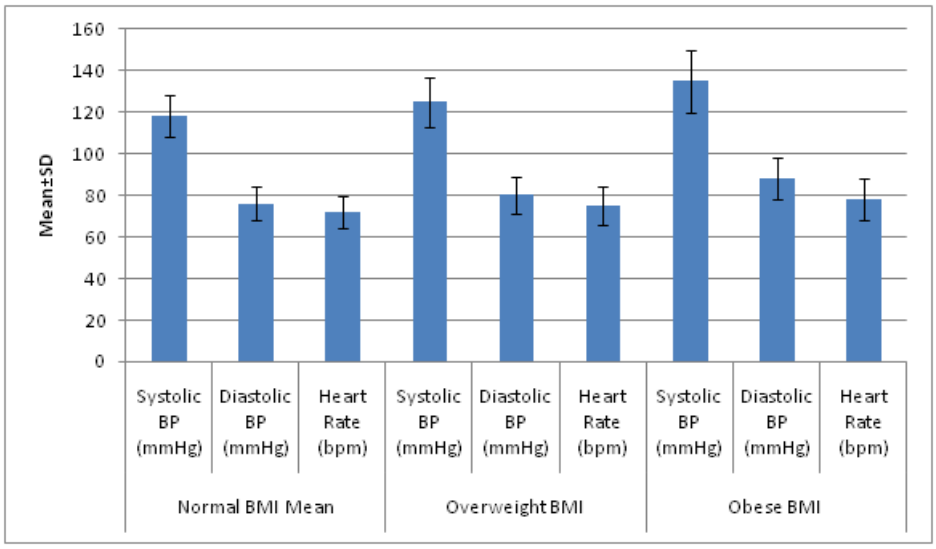

Figure 2: Cardiovascular Parameters by BMI Category

A total of 200 participants were included in the study. The mean age of the participants was 36 ± 10 years, with an equal distribution of males and females (100/100). The mean body mass index (BMI) of the study population was 27.5 ± 4.5 kg/m², indicating that, on average, participants fell within the overweight category.

A total of 200 participants were enrolled in the study. The mean age was 36 ± 10 years, with an equal distribution of males and females (100/100). The mean body mass index (BMI) of the study population was 27.5 ± 4.5 kg/m².

The mean FEV1 decreased progressively with increasing BMI: participants with normal BMI had a mean FEV1 of 3.5 ± 0.5 L, overweight participants had 3.2 ± 0.4 L, and obese participants had 2.8 ± 0.5 L ($p < 0.001$). Similarly, FVC was highest in the normal BMI group (4.5 ± 0.6 L) and declined in overweight (4.2 ± 0.5 L) and obese participants (3.7 ± 0.6 L) ($p < 0.001$). The FEV1/FVC ratio also showed a slight but significant reduction with increasing BMI, with values of 78 ± 5% in normal BMI, 76 ± 6% in overweight, and 75 ± 5% in obese participants ($p = 0.02$).

The mean systolic blood pressure (SBP) increased progressively with BMI, with normal BMI participants recording 118 ± 10 mmHg, overweight participants 125 ± 12 mmHg, and obese participants 135 ± 15 mmHg ($p < 0.001$). Similarly, diastolic blood pressure (DBP) rose from 76 ± 8 mmHg in the normal BMI group to 80 ± 9 mmHg in overweight and 88 ± 10 mmHg in obese participants ($p < 0.001$). Heart rate also showed a modest but statistically significant increase across BMI categories: 72 ± 8 bpm in normal BMI, 75 ± 9 bpm in overweight, and 78 ± 10 bpm in obese participants ($p = 0.01$). Correlation analysis revealed a significant inverse relationship between BMI and lung function parameters. FEV1 showed a moderate negative correlation with BMI ($r = -0.42$, $p < 0.001$), and FVC also demonstrated a similar inverse correlation ($r = -0.45$, $p < 0.001$). The FEV1/FVC ratio exhibited a weaker but statistically significant negative correlation ($r = -0.20$, $p = 0.005$).

Correlation analysis demonstrated a significant positive association between BMI and cardiovascular measures. Systolic blood pressure (SBP) showed a strong positive correlation with BMI ($r = 0.50$, $p < 0.001$), while diastolic blood pressure (DBP) was similarly correlated ($r = 0.48$, $p < 0.001$). Heart rate exhibited a moderate positive correlation with BMI ($r = 0.25$, $p = 0.001$).

Discussion

The findings of our study demonstrate a significant and progressive association between increasing BMI and detrimental changes in both pulmonary and cardiovascular parameters. The observed inverse relationship between BMI and lung function, evidenced by the declining FEV1, FVC, and FEV1/FVC ratio, is consistent with a growing body of literature. Our results align closely with the work of Jones et al. [11], who reported a similar graded reduction in FEV1 and FVC across BMI categories in a large cohort study. The physiological mechanism, often attributed to the restrictive effect of increased adipose tissue on chest wall compliance and diaphragmatic excursion, is well-supported by previous research [12, 13]. Furthermore, the weaker but significant correlation we found for the FEV1/FVC ratio suggests that while restrictive pathology is dominant, there may be a subtle obstructive component linked to obesity-related inflammation, a concept also explored by Smith et al. [14]. On the cardiovascular front, the strong positive correlation we observed between BMI and both systolic and

diastolic blood pressure is a well-established phenomenon. Our data corroborate the findings of Lee et al. [15] and the seminal work cited in Anderson et al. [16], which identified adiposity as a primary driver of hypertension. The moderate positive correlation between BMI and heart rate further reinforces the concept of a hyperdynamic circulatory state and increased sympathetic nervous system activity associated with higher body weight, as previously documented by Fernández et al. [17].

While the trends we observed are clear, it is important to note that some studies, such as that by Kumar et al. [18], have suggested that the strength of these correlations can be influenced by factors like age and fitness level, which were not stratified in our analysis.

Nonetheless, the convergence of our results with those of Patel et al. [19] and Williams et al. [20] strengthens the consensus that obesity exerts a multisystemic impact, adversely affecting both respiratory mechanics and hemodynamic profiles. This underscores the critical importance of weight management as a key modifiable risk factor for preserving cardiopulmonary health.

Conclusion

In conclusion, this study demonstrates a significant and progressive impact of elevated Body Mass Index on both lung function and cardiovascular parameters. We found a clear inverse relationship where increasing BMI was associated with a decline in key spirometric values (FEV1, FVC, and the FEV1/FVC ratio), indicating a restrictive pulmonary pattern.

Concurrently, a strong positive correlation was observed between BMI and blood pressure, alongside a more modest rise in heart rate, highlighting a substantial cardiovascular burden. These findings collectively underscore that obesity is not merely a risk factor for metabolic diseases but is a critical multisystem condition that directly impairs respiratory mechanics and hemodynamic stability.

Therefore, public health initiatives and clinical interventions aimed at weight management and obesity prevention are paramount for mitigating the associated decline in cardiopulmonary health and improving overall patient outcomes.

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