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DIFFERENCE IN THE BODE INDEX BETWEEN SMOKER AND NON-SMOKER PATIENTS WITH CHRONIC OBSTRUCTIVE PULMONARY DISEASE (COPD)

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ABSTRACT

Introduction: COPD is increasingly recognized as a multisystem disorder, and multidimensional indices such as the BODE score better reflect functional limitation and systemic involvement than spirometry alone.

Objective: To compare BODE index scores between smokers and non-smokers with COPD and to examine the association between pack-years and multidimensional severity.

Methods: An analytical cross-sectional study was conducted at the Department of Internal Medicine, Isra University, Hyderabad from April 2025 to August 2025. Stable outpatients aged ≥40 years with spirometry-confirmed COPD were recruited consecutively. Smoking status was classified as smoker/non-smoker, and cumulative exposure was quantified as pack-years. BODE index was calculated using BMI, FEV_1% predicted, mMRC dyspnea grade, and six-minute walk distance. Mean BODE scores were compared using an independent samples t-test, and correlation between pack-years and BODE was assessed using Pearson correlation. Pack-years were also compared across BODE severity categories.

Results: A total of 150 patients were analyzed (mean age 60.25 ± 8.91 years; 74% male). Smokers had mean BODE score numerically higher than non-smokers (3.09 ± 1.67 vs 2.70 ± 1.35), but the difference was not statistically significant ($p = 0.223$). Pack-years showed a weak positive correlation with BODE score ($r = 0.268$, $p = 0.001$). Patients with moderate-to-severe disease had higher pack-years than those with mild disease (16.49 ± 11.77 vs 10.64 ± 9.81; $p = 0.003$).

Conclusion: In stable outpatient COPD, smoking status alone did not significantly differentiate BODE severity; however, cumulative exposure (pack-years) showed a modest association with multidimensional disease burden.

Keywords: COPD; BODE index; smoking; pack-years; severity

INTRODUCTION

Recognition of chronic obstructive pulmonary disease (COPD) as a multisystem disorder has shifted severity assessment away from isolated spirometric impairment toward multidimensional constructs that better reflect functional limitation and systemic involvement. Swapna et al.[1] demonstrated that the BODE index correlates strongly with disease severity and extrapulmonary manifestations, supporting its clinical superiority over single physiologic markers.

The prognostic performance of multidimensional indices has been validated across diverse clinical environments. Robles-Hernández et al.[2] confirmed the predictive accuracy of BODE variants for mortality, reinforcing the value of composite severity modeling in long-term risk assessment.

Similarly, Arivumani et al.[3] identified the BODE index as a reliable marker of overall clinical burden, while Reddy et al.[4] reported a significant association between elevated BODE scores and pulmonary hypertension, emphasizing the index's ability to capture cardiopulmonary interaction beyond airflow limitation.

Further strengthening its clinical relevance, Ajay et al.[5] observed that higher BODE scores predicted hospitalization risk. Prospective evaluation by Himavarsh et al.[6] confirmed its utility as a predictor of disease severity, highlighting the transition toward integrated severity frameworks.

Radiologic investigations have also supported multidimensional modeling. Indurkar et al,[7] demonstrated a meaningful correlation between HRCT parameters and BODE-based severity grading. In parallel, patient-reported outcome research by Kaur et al.[8] showed significant agreement between COPD assessment tools and BODE scoring, underscoring the index's ability to reflect symptomatic burden.

More recently, Park et al.[9] validated a modified BODE construct incorporating activity domains, illustrating the adaptability of multidimensional evaluation across clinical contexts.

Smoking remains the principal etiologic driver of COPD and may intensify systemic disease burden through persistent inflammatory activation and oxidative stress. Patel et al.[10] observed that longer disease duration correlated with worsening spirometric impairment and higher BODE scores, suggesting cumulative injury pathways. Biological plausibility is further supported by Hemalatha et al.[11], who linked elevated inflammatory biomarkers with advanced BODE categories. Broader systemic interactions were described by Bonde et al.,[12] who reported associations between vitamin D status and higher BODE scores.

Importantly, exposure–severity relationships have been examined directly. Kalyani et al.[13] demonstrated that increasing smoking index correlated with greater disease severity and comorbidity burden. Meanwhile, Bothara and Holay,[14] described clinically meaningful sex-based differences in COPD expression.

Despite extensive validation of the BODE index, evidence directly comparing multidimensional severity between Smokers and nSmokers remains limited, particularly within stable outpatient cohorts where early risk stratification could influence long-term trajectories.

Given this context, the present study sought to determine whether multidimensional disease burden differs between ever and Non Smokers and to examine the relationship between cumulative smoking exposure and severity among patients with COPD.

METHODS

An analytical cross-sectional design was employed to evaluate the comparison of Bode Index between Smokers and Non-Smokers with Chronic Obstructive Pulmonary Disease. The study was conducted from April 2025 to August 2025, after obtaining ethical approval from the Institutional Review Committee (Approval No: IU/CP. REC(FCS)/2025/487). Participants were recruited from stable outpatient clinics at Isra University Hospital, Hyderabad.

Adults aged ≥40 years with post-bronchodilator spirometry consistent with COPD were eligible. Patients experiencing acute exacerbations, primary bronchial asthma, active pulmonary infections, unstable cardiovascular disease, or conditions precluding functional testing were excluded.

Participants were categorized according to lifetime smoking exposure. Non Smokers were defined as individuals who had smoked fewer than 100 cigarettes in their lifetime. Smokers included participants with documented smoking exposure regardless of current smoking status. Smoking burden was quantified in pack-years, calculated as the number of cigarettes smoked per day divided by 20 and multiplied by years smoked.

The primary outcome was the BODE index, calculated using body mass index, percent predicted FEV_1, modified Medical Research Council dyspnea scale, and six-minute walk distance. Standardized spirometry protocols were followed, and walk testing was conducted under supervision with early termination documented in accordance with prognostic considerations described by Youk et al.[15]

Data were collected on age, sex, socioeconomic status, biomass exposure, COPD duration, exacerbation frequency, and cardiometabolic comorbidities. Body mass index was recorded given its established association with airflow limitation.

Sample size was calculated a priori using G*Power (version 3.1) for comparison of mean BODE scores between ever and nSmokers , assuming α=0.05, 80% power, a moderate effect size (Cohen's d=0.55), and an anticipated outpatient allocation ratio favouring Smokers . The required sample was approximately 150 participants.

Data were analyzed using SPSS version 26. Normality was assessed via Shapiro–Wilk testing. Continuous variables were summarized as mean±SD or median (IQR). Group differences were evaluated using the Mann–Whitney U test. Spearman correlation assessed the relationship between pack-years and BODE score. Smoking exposure was compared with BODE Index by applying independent t test. A p-value <0.05 was considered statistically significant.

RESULTS

A total of 150 patients with spirometry-confirmed stable COPD were included in the final analysis. The mean age of the cohort was 60.25 ± 8.91 years, and males comprised 74% of participants. Biomass exposure was reported in 38% of patients. Current smokers were 78(52%), former and nSmokers were 39(26%) and 33(22%),

respectively. Cardiometabolic comorbidities were common, with hypertension present in 26%, diabetes mellitus in 21.3%, ischemic heart disease in 18%, and heart failure in 10%. Table 1

The mean body mass index was 25.21 ± 4.73 kg/m², and the average duration of COPD was 5.27 ± 3.13 years. The overall mean BODE index was 3.00 ± 1.61, indicating moderate multidimensional disease severity within the study population. Table 1

Smokers (current and former smokers) constituted 78% of the cohort, whereas 22% were nSmokers . The mean BODE score was higher among Smokers compared with nSmokers (3.09 ± 1.67 vs 2.70 ± 1.35); however, this difference did not reach statistical significance (mean difference 0.389; 95% CI −1.016 to 0.239; p = 0.223). Table 2

A statistically significant positive correlation was observed between cumulative smoking exposure and BODE index (r = 0.268, p = 0.001), demonstrating that greater tobacco exposure was associated with increased multidimensional COPD severity. Table 3

When disease severity was dichotomized, patients with moderate-to-severe COPD exhibited significantly higher cumulative smoking exposure compared with those with mild disease (16.49 ± 11.77 vs 10.64 ± 9.81 pack-years). The mean difference of 5.85 pack-years was statistically significant (95% CI 2.06–9.63; p = 0.003), supporting the presence of a clear exposure–severity gradient. Table 4

Table 1: Demographic and Baseline Clinical Characteristics of Study Participants

Variables	**n(%), mean ±SD**
Age (Years)	60.25 ± 8.91
Sex	
Male	**111(74.0)**
Female	**39(26.0)**
Biomass Exposure	
Yes	**57(38.0)**
No	**93(62.0)**
Smoking Status	
Non-Smokers	**33(22.0)**
Former smoker	**39(26.0)**
Current smokers	**78(52.0)**
Smokers (Former + Current smokers)	**117(78.0)**
Educational level	
Primary or less	**48(32.0)**
Secondary	**62(41.3)**
Higher	**40(26.7)**
Comorbidities	
Diabetes mellitus (DM)	**32(21.3)**
Hypertension (HTN)	**39(26)**
Ischemic heart disease	**27(18.0)**
Heart Failure	**15(10.0)**
Body Mass Index (kg/m²)	**25.21 ± 4.73**
Duration of COPD (years)	**5.27 ± 3.13**
Pack-years	
FEV_1 (% predicted)	**63.84 ± 9.68**
FVC (% predicted)	**82.53 ± 7.80**
SpO_2 at rest (%)	**95.68 ± 1.48**
Bode Index	**3.00 ± 1.61**
Bode severity	
Mild	**55(36.7)**
Moderate to Severe	**95(63.3)**

Table 2. Comparison of BODE Index Between Smokers and Non Smokers

Smoking Status	Bode Index (Mean ±SD)	95% CI	P value
Smokers (n=117)	3.09±1.67	-1.016- 0.239	0.223
Non Smokers (n=33)	2.70±1.35		

Table 3. Correlation Between Smoking Exposure and Bode Index

Variables	Correlation Coefficient	P Value
Pack-years vs BODE Index	0.268	0.001

Table 4. Comparison of Pack-Years with severity of COPD

Severity of COPD	Pack-years (Mean ±SD)	95% CI	P value
Mild	10.64±9.81	2.06-9.63	0.003
Moderate to Severe	16.49±11.77		

DISCUSSION

The present study evaluated whether multidimensional COPD severity differs between smokers and non-smokers and further examined the relationship between cumulative tobacco exposure and BODE index in a stable outpatient population. Although smokers demonstrated marginally higher BODE scores, the absence of statistical significance suggests that categorical smoking status alone may inadequately capture the complexity of disease expression. In contrast, the observed positive correlation between pack-years and BODE index indicates that cumulative exposure provides a more discriminative measure of multidimensional impairment. Similar exposure–severity relationships have been described previously, where increasing smoking indices aligned with greater systemic involvement and functional limitation.[13]

The use of the BODE index as the primary severity metric is supported by extensive literature emphasizing its superiority over isolated spirometric markers. Strong correlations between BODE scores and overall disease burden have been demonstrated in stable COPD cohorts,[1,3] while prognostic validation studies confirm its reliability for mortality prediction.[2] Importantly, elevated BODE scores have also been associated with hospitalization risk and adverse clinical trajectories, reinforcing the clinical relevance of multidimensional staging.[5,6]

The lack of a significant smoker versus non-smoker difference in the current study likely reflects the heterogeneous pathobiology of COPD rather than a true absence of exposure effect. Disease duration has been shown to correlate with worsening airflow limitation and higher BODE scores,[10] suggesting that accumulated physiological injury may overshadow simple exposure categorization. Furthermore, systemic inflammatory activation — reflected by elevated biomarkers in advanced BODE categories — supports the concept that COPD severity is partly driven by chronic inflammation.[11] Extrapulmonary contributors also appear relevant; associations between vitamin D deficiency and higher BODE scores indicate that metabolic pathways influence multidimensional decline.[12]

Radiologic and symptomatic correlates further validate the biological framework underlying BODE. Structural abnormalities on high-resolution CT have been shown to parallel severity grading,[7] whereas symptom-based assessment tools demonstrate meaningful concordance with multidimensional indices.[8] The adaptability of this construct is evident in modified activity-integrated models that preserve predictive accuracy across clinical contexts.[9]

Sex-related differences may provide an additional explanatory layer. Variability in COPD expression between males and females has been documented, suggesting that biological susceptibility and hormonal influences can modify disease trajectories independently of smoking exposure.[14] Such effect modification could attenuate group comparisons while still permitting detection of dose–response relationships when exposure is quantified.

Functional limitation remains central to multidimensional severity modeling. Evidence indicates that even early termination of the six-minute walk test may carry prognostic significance comparable to composite indices.[15] Agreement between symptom scales and BODE scoring has also been reported in exacerbation risk assessment, further supporting integrated evaluation strategies.[16]

Beyond individual prognosis, multidimensional indices have important healthcare implications. Economic analyses demonstrate that composite severity tools can predict COPD related medical costs,[17] while broader syntheses emphasize their utility in mortality risk stratification.[18] These findings suggest that incorporating exposure quantification into multidimensional assessment could enhance outpatient risk stratification.

Emerging evidence continues to refine severity modeling. Application of updated airway obstruction thresholds has been shown to alter staging distributions within established COPD cohorts,[19] and physiologic recovery markers such as post-exercise heart rate recovery provide additional prognostic insight when integrated with multidimensional indices.[20] Alternative functional tests, including sit-to-stand substitutions, have demonstrated reliability comparable to traditional BODE components.[21]

Metabolic phenotype is increasingly recognized as a determinant of disease expression. Visceral adiposity has been associated with variation in BODE scores,[22] and regional evidence linking body mass index with airflow limitation further supports the systemic nature of COPD.[23]

Importantly, translating severity assessment into improved outcomes requires structured care pathways. Integrated COPD management implemented within public healthcare settings has demonstrated measurable clinical benefit, underscoring the importance of early identification of high-risk patients through multidimensional evaluation.[24]

Collectively, these findings support a conceptual shift from binary smoking classification toward quantitative exposure assessment. The exposure–severity gradient observed in this study likely reflects cumulative airway injury compounded by systemic inflammation, metabolic dysfunction, and progressive reduction in physiologic reserve. Such an interpretation aligns with contemporary COPD paradigms that emphasize phenotypic heterogeneity and multidimensional disease expression rather than single-factor causation.

In this analytical cross-sectional study of stable outpatient COPD patients, smokers demonstrated slightly higher BODE scores than non-smokers; however, the difference was not statistically significant. In contrast, cumulative smoking exposure measured in pack-years showed a weak but significant positive correlation with multidimensional disease severity, suggesting that exposure burden may better reflect systemic impairment than binary smoking classification. These findings reinforce the clinical value of integrating quantitative exposure history into multidimensional severity assessment rather than relying solely on smoking status. Given the heterogeneous nature of COPD, incorporating pack-year evaluation alongside indices such as BODE may improve early risk stratification in outpatient settings. Larger prospective studies are warranted to clarify causal pathways and determine whether exposure-guided severity assessment translates into improved clinical outcomes.

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