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Comparison of FEV_3 and FEV_3/FVC Ratio Between Smokers and Non-Smokers Who Engage in Regular Exercise

Ida Rizqa Nuraini[1*], Nurul Widyawati[2], Nita Damayanti[3], Hartati Tuna[4], Ronald Aprianto Parubak[5], Galuh Dharanindya Ica Manohara[6], Dita Puspita Damayanti[7], Alisya Nuril Firdausi Nuzula[8], Ketut Suhendro[9]

[1-9]Faculty of Medicine, Bhakti Wiyata Institute of Health Sciences, Kediri, Indonesia

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Corresponding author:
Ida Rizqa Nuraini

E-mail address:
ida.rizqa@iik.ac.id

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ABSTRACT

Introduction. Smoking is a major risk factor for chronic obstructive pulmonary disease (COPD). In the early stages of disease, these pathological processes predominantly affects the small airways, known as Small Airway Disease (SAD), which is often clinically silent. FEV_3 and FEV_3/FVC ratio have been proposed as potential indicators of SAD. Regular exercise has been suggested to attenuate smoking-related pulmonary decline. However, evidence regarding its effect on small airway function in smokers in human studies, particularly among physically active individuals, remains limited. This study aimed to compare FEV3 and FEV3/FVC ratio between smokers and non-smokers who regularly engage in physical exercise. **Methods.** 30 adult male with regular exercise (18 smokers and 12 non-smokers) were included in this cross-sectional comparative study. Spirometric parameters, including percentage predicted values of Forced Expiratory Volume in three seconds (FEV_3), and FEV_3/FVC were measured and compared between groups using the independent samples t-test. Welch's t-test was applied as a more robust method. Statistical analysis was computerized using Jamovi 2.3.28 software. **Results.** Non-smokers showed higher mean percentage predicted FEV_3 and FVC compared with smokers. However, no significant differences in FEV_3 (p= 0.608; effect sizes=-0.1870; 95% CI -0.911 to 0.555) and FEV_3/FVC ratio (p= 0.100; effect sizes=0; 95% CI -0.487 to 0.984) between groups. **Conclusion.** This study found no significant differences in FEV_3 or FEV_3/FVC ratio between smokers and non-smokers with regular exercise. Further studies are needed to clarify the relationship between smoking, exercise, and early small airway impairment.

1. Introduction

Smoking is a major risk factor for pulmonary function impairment. Approximately 20% of smokers develop Chronic Obstructive Pulmonary Disease (COPD), the third most common cause of mortality globally.[1,2] Chronic exposure to cigarette smoke triggers increased oxidative stress, persistent airway inflammation, mucus hypersecretion resulting from goblet cell hyperplasia, ciliary dysfunction of the respiratory tract, airway hypersensitivity to exogenous stimuli, and bronchospasm.[3,4] These pathological changes lead to airflow limitation in COPD, as indicated by a reduced FEV_1/FVC ratio on spirometry. In the early stages of disease, these pathological processes predominantly affects the small airways (terminal bronchioles), known as Small Airway Disease (SAD).[5]

Small Airway Disease is often clinically silent, early detection of SAD remains challenging.[5] At this stage, smokers may appear healthy based on physical examination, spirometry, and radiographic findings despite the presence of subclinical airway abnormalities. This condition is commonly referred to as "healthy smoker".[6] Conventional spirometric parameters often remain within normal ranges because FEV_1 primarily reflects large airway function, whereas SAD mainly involves the small airways. Consequently, FEV_1/FVC ratio may remain within normal limits despite the presence of underlying pathological changes, contributing to under-diagnosis.[7] According to global studies, the prevalence of under-diagnosis is estimated at approximately 60%–86% among individuals with COPD.[8] To improve early detection, recent studies[9,10] have highlighted the potential role of the FEV_3/FVC ratio in the early assessment of COPD by detecting SAD. Physiologically, the FEV_3/FVC ratio reflects airflow from both early and mid-to-late expiratory phases. Since SAD causes air trapping and delayed emptying, the FEV_3/FVC ratio is thought to be a sensitive indicator of SAD. A lower FEV3/FVC ratio is linked to a higher chance of lung function declining

quickly and developing COPD, even in individuals with normal spirometric results.[10]

Recently, factors influencing the progression of smoking-related pulmonary decline have gained increasing attention. Numerous previous studies[11,12] have suggested that regular exercise may attenuate smoking-related pathological changes and delay pulmonary function decline. However, it does not fully prevent smoking-related airway impairment. These beneficial effects are mediated by several physiological mechanism, including enhanced respiratory muscle strength, lung capacity, and natural bronchodilation.[4,13,14]

In small airways, regular exercise may attenuate cigarette smoke–induced injury through upregulation of endogenous antioxidant systems (such as GPx and SOD), reduction of reactive oxygen species (ROS), and modulation of inflammatory mediators in lung tissue, thereby helping to preserve airway integrity and reduce structural damage such as alveolar destruction and loss of lung elasticity.[15] However, these findings are primarily derived from experimental animal studies, while evidence regarding the effects of regular exercise on small airway function in humans remains limited. This highlights the challenge in assessing these effects in human studies.

On the other hand, FEV_3 has been proposed as a potential indicator of early expiratory flow limitation and small airway obstruction. However, evidence regarding FEV_3 and the FEV_3/FVC ratio is still evolving and has not been widely explored in smoker populations, particularly with regular exercise. Most previous research has focused on conventional spirometric parameters such as FEV1, which primarily reflects large airway function and may not adequately detect early small airway impairment. This highlights a gap in evidence regarding the use of FEV_3 and FEV_3/FVC as an early marker in populations with smoking exposure, particularly among individuals who regularly engage in physical activity. The novelty of this study lies in its focus on a physically active smoking population and the use of FEV3/FVC as a potentially more sensitive marker of early small airway changes that may not be captured by conventional spirometric parameters.

Therefore, this study aimed to compare FEV_3 and the FEV_3/FVC ratio between smokers and non-smokers who regularly exercised. We hypothesized that there would be no significant differences in FEV3 and FEV3/FVC ratio between smokers and non-smokers who regularly exercised.

2. Methods

The design of this study was cross-sectional comparative design. Adult male participants aged over 18 years and those who engaged in regular exercise for at least the past six month were included in this study. Regular exercise in this study was defined as aerobic exercise (e.g., jogging, cycling, brist walking, or swimming) performed for 150-300 minutes per week. Exercise data were obtained using a structured questionnaire and reported by participants based on frequency and duration. No objective assessment of exercise intensity was performed. Participants with a history of active smoking within the past year were classified into the smoker group, while the remaining participants were categorized as non-smokers. Smoking exposure was quantified using pack-years, calculated using the following formula: pack-years = (number of cigarettes smoked per day/20) x number of years smoked.[16] The number of pack-years was classified as light (<10 pack-years), moderate (10-20 pack-years), and heavy (>20 pack-years) smokers.[17]

A total of 30 subjects were included using a convenience sampling technique, consisting of 18 smokers and 12 non-smokers. Individuals who have previously had a respiratory disease (such as asthma, COPD, and tuberculosis) or who consumed bronchodilators were excluded from this study. Written informed consent was obtained from all eligible participants prior to the study.

Data collection was performed in March 2026 at the Bhakti Wiyata Institute of Health Sciences, Kediri, Indonesia. A structured questionnaire was utilized to obtain information on participants' demographic profile, history of respiratory disease, current respiratory symptoms, comorbidities, and exercise behaviors (including type, frequency, and intensity). Chest auscultation was conducted on all participants by general practitioners to detect any abnormal respiratory sounds. Pulmonary function parameters were measured using CONTEC SPM-D Spirometer (Contec Medical Systems Co., Ltd., Qinhuangdao, Hebei, China), which displays measurement values and produces computerized reports when integrated with the manufacturer's software. Spirometry testing was performed following standardized procedures based on European Respiratory Society (ERS) recommendations.[18] Participants were asked to perform spirometry maneuver three times in an upright seated and relaxed position. The best percentage predicted values of forced vital capacity (FVC), forced expiratory volume in three seconds (FEV3), and FEV3/FVC were selected for analysis.

Data were presented using descriptive statistics and presented as mean ± standard deviation (SD) if normally distributed, otherwise the data were presented as median and interquartile range (IQR). The number of patients and percentage were used as categorical variables. Analysis of distribution were performed using Shapiro-Wilk. If normally distributed, comparisons between smoker and non-smoker groups were performed using the independent samples t-test. Due to unequal sample sizes between groups, Welch's t-test was applied as a more robust method. The Mann-Whitney U test was used to assess data that was not normally distributed. Effect size and 95% confidence interval (CI) were also

calculated to assess the magnitude and precision of differences between groups. Cohen's d was used for parametric analyses, while rank-biserial correlation was applied for non-parametric analyses. The homogeneity of variance was evaluated using Levene's test. Statistical analysis was computerized using Jamovi 2.3.28 software.[19] Statistical significance was defined as a P value of less than 0.05. Graphs were made using PRISM 11.0.0 software (Graph Pad Software, La Jolla, CA, USA).

3. Results

Baseline participants characteristics are presented in Table 1. BMI was normally distributed in both groups (p = 0.063 in the smoker group and p = 0.215 in the non-smoker group). Homogeneity of variance was confirmed (p = 0.874), and BMI did not significantly differ between the groups (p = 0.867). Most participants in both groups had a normal BMI (72.2% in the smoker group and 58.3% in the non-smoker group), while the remainder were categorized as overweight.

Age distribution was non-normal in both groups (p = 0.013 for smokers and p = 0.016 for non-smokers). Nevertheless, homogeneity of variance was confirmed (p = 0.844), and age differences between the groups were not statistically significant (p = 0.251).

The degree of smoking exposure was not normally distributed (p Shapiro-wilk = 0.012), with a median of 4.4 (IQR = 1.9 – 9.68). Most participants (83.3%) were classified as light smokers, while 16.7% were moderate smokers.

As shown in Table 2 and Figure 1, the non-smoker group demonstrated higher mean percent predicted values of FVC (96.2 ± 13.8) and FEV_3 (92.9 ± 11.75) compared to the smoker group (FVC: 92.5 ± 16.9; FEV_3: 90.2 ± 16.49). However, no significant differences were observed (p = 0.510 for FVC; p = 0.608 for FEV_3). Both parameters demonstrated small effect size (FEV_3 = -0.1870; FVC = -0.2440).

Similarly, Table 3 showed no significant difference was found in the percent predicted ratio of FEV_3/FVC between the two groups (p = 0.100) and the effect size was negligible (rank-biserial correlation = 0.000).

Table 1. Baseline participants characteristics

Characteristics	Smoker Group (n=18)	Non-Smoker Group (n=12)	*P* value
Age (Median (Q1-Q3))	28.5 (21.8 – 43)	25 (20 – 32.5)	0.251
Body Mass Index, Mean ± SD	24.3 ± 2.83	24.5 ± 3.10	0.867
Normal, n (%)	13 (72.2%)	7 (58.3%)	
Overweight, n (%)	5 (27.7%)	5 (41.6%)	
Pack-years, (Median (Q1-Q3))	4.4 (1.9-9.68)	0	<0.001
Light smoker, n (%)	15 (83.3%)		
Moderate smoker, n(%)	3 (16.7%)		
Heavy smoker, n (%)	0		

Table 2. Comparison of FEV3 and FVC between smoker and non-smoker group with regular exercise

Parameters	Mean ± SD		Mean Difference	Effect Size	95% CI	P value
	Smoker Group	Non-Smoker Group				
FEV_3	90.2 ± 16.49	92.9 ± 11.75	-2.678	-0.1870	-0.911 to 0.555	0.608
FVC	92.5 ± 16.9	96.2 ± 13.8	-3.767	-0.2440	-0.970 to 0.499	0.510

FEV3 = Forced Expiratory Volume in three seconds; FVC = Forced Vital Capacity;
SD = Standart Deviation; CI = Confidence Interval.
Independent sample t-test was used. Effect size is reported as Cohen's d with 95% CI.

Table 3. Comparison of FEV3/FVC ratio between smoker and non smoker group with regular exercise

Parameters	Median (IQR)		Effect Size	95% CI	P value
	Smoker Group	Non-Smoker Group			
FEV_3/FVC	98.1(97.9 – 98.1)	98.1 (95.4 – 98.4)	0.000	-0.487 to 0.984	0.100

FEV3 = Forced Expiratory Volume in three seconds; FVC = Forced Vital Capacity; CI = Confidence Interval.
The Mann-Whitney U test was used. Effect size is reported as rank-biserial correlation with 95% CI.

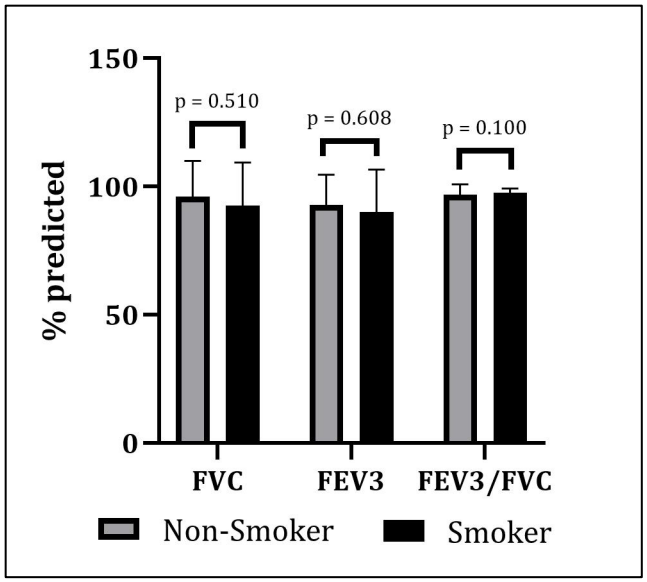

Figure 1. Percent predicted values of FVC, FEV3, and FEV3/FVC parameters

4. Discussion

This study compared FEV_3 and FEV_3/FVC ratio between smokers and non-smokers among individuals who regularly engage in exercise. Although the non-smoker group demonstrated higher mean percent predicted values of FEV_3 and FVC, no statistically significant differences were observed between groups. Furthermore, all measured parameters were classified as having small effect sizes, indicating minimal clinical relevance. These findings suggest that the measured FEV_3 and FEV_3/FVC ratio in this study were relatively similar between smokers and non-smokers with regular exercise.

Several factors may explain the absence of significant differences observed in this study. First, the participants were relatively young adults, which may have resulted in relatively limited smoking exposure. This was supported by the finding that most smokers (83.3%) were classified as light smokers. The degree of smoking exposure is thought to contribute to the development of SAD. Previous study[20] has demonstrated a dose-response relationship between smoking exposure and SAD, in which higher pack-year exposure was associated with greater impairment in SAD. In addition, Pisi *et al*[21] have reported significantly lower FEV_3/FVC ratio in heavy smokers compared to mild smokers, further supporting the association between the degree of smoking exposure and SAD.

Second, the absence of significant differences may be influenced by healthy smoker bias. Healthy smokers are individuals who smoke but remain asymptomatic and demonstrate normal clinical findings (e.g. physical examination, spirometry, and radiographic imaging), despite continued smoking exposure. Nevertheless, healthy smokers are not truly healthy.[6] Previous study has reported[21] no significant differences in spirometric parameters between healthy/asymptomatic smokers and never-smokers. These findings are consistent with the results of the present study, in which no significant differences were observed between the smoker and non-smoker group. The possibility of healthy smoker bias in this study may be related to the characteristics of the smoker group, all of whom routinely engaged in regular exercise. This is supported by previous studies reporting that smokers with regular physical activity tended to demonstrate better spirometric parameters compared to physically inactive smokers.[22] These positive effects of regular exercise are mediated by several physiological mechanisms. Previous studies[13] have shown that exercise reduces vagal tone in the airways, thereby decreasing airway resistance and facilitating airflow. Therefore, exercise is considered as natural bronchodilator. Lung capacity also increases as an adaptive response to regular exercise[14]. In addition, endurance exercise enhances respiratory muscle strength, which in turn contributes to greater respiratory muscle endurance.[4]

In addition, the relatively small sample size may have influenced the study findings by limiting the statistical power to detect differences between groups. Consequently, small differences in spirometry parameters have not reached statistical significance, increasing the possibility of Type II error.

This study has several limitations. Exercise characteristics were obtained through self-reported questionnaires without objective assessment of exercise intensity, such as Metabolic Equivalents (METs) or VO_2max measurements. In addition, potential confounding factors such as age, BMI, and smoking exposure were not adjusted using multivariable analysis due to the limited sample size. The causal relationship between smoking, regular exercise, and spirometry parameters (FEV_3, FVC, and FEV_3/FVC ratio) cannot be established due to the cross-sectional methodology.

5. Conclusion

This study found no significant differences in FEV_3 or FEV_3/FVC ratio between smokers and non-smokers with regular exercise. These findings should be interpreted cautiously considering the relatively low smoking exposure, possible healthy smoker bias, and limited sample size. Further studies with larger sample sizes and more comprehensive exposure assessments are needed to clarify the potential role of regular exercise in preserving lung function and attenuating early smoking-related functional decline.

6. Author Contribution

All authors contributed to writing the paper or critically reviewed the manuscript. Ida designed the study experiments, collected and analyzed data, and performed statistical analysis. Nurul and Ronald contributed to collected data. Nita, Hartati, and Dita supervised data collection. Galuh, Alisya, and Ketut recruited participants.

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