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The correlation between smoking cumulative dose based on Brinkman Index with peak expiratory flow rate

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Abstract

Background: Indonesia is the country with the highest prevalence of smokers above 15 years old according to WHO, with a percentage of 76.2%. Smoking-induced lung damage is characterized by inflammation, leading to the destruction of lung parenchyma and airway obstruction, ultimately worsening lung function parameters. This study aims to find correlation between cumulative dose of smoking based on Brinkman index (BI) with Peak Expiratory Flow Rate (PEFR).

Methods: This is a cross-sectional correlative study conducted on January–March 2020. Data were collected through history taking and PEFR measured with a peak flowmeter by taking the average of three peak flowmeter measurements, each separated by a 2-min interval. Inclusion criteria include male active smokers, aged 30 to 40 years, who have had a smoking history for at least 1 year. Exclusion criteria are as follows: uses e-cigarettes for smoking, has a history of chronic lung diseases such as tuberculosis, pneumonia, post-COVID-19 syndrome, asthma, and has not smoked in the last 28 days.

Results: A total of 48 male smokers are included in this study. The mean age of participants was 35.91 years, with a history of smoking of 18.12 years, and 12.5 cigarettes smoked daily. Patients included in this study had BI classified as mild (47.91%), moderate (47.91%), and severe (4.16%). PEFR in patients was classified into green (10.41%), yellow (83.33%), and red (6.25%). Analysis showed significant negative correlation between BI and PEFR ($r = -0.721$; $p < 0.001$) suggesting that increasing Brinkman Index may lead to a decrease in PEFR.

Conclusion: Higher BI correlates with a decrease in PEFR. PEFR may prove to be useful in early detection of reduced pulmonary function. Further trials conducted on larger sample sizes and evaluating other lung function parameters are recommended.

KEYWORDS

Brinkman Index, Cigarette, PEFR, Smoking

1 | INTRODUCTION

Cigarettes are the leading cause of death among adults in developing countries. An analysis by health experts in Europe states that in developing countries, smoking causes 24% of deaths in men and 7% of deaths in women. Indonesia has a high smoking prevalence, with a percentage of 76.2% among adults. According to data from the 2011 Global Adult Tobacco Survey Indonesia Report (GATS), 59.8 million adults in Indonesia smoke.[1] Men are more likely to smoke than women, and the prevalence of smoking in men is also higher than in women.[2] In men, the highest percentage of daily smokers was found in the age ranges of 25 to 44 years (63.8%) and 45 to 64 years (62.8%).[1]

Cancer, cardiovascular disease, and chronic obstructive pulmonary disease (COPD) are major health problems that are closely related to smoking.[2] Smoking carries an estimated coronary heart disease risk of 57% for coronary heart disease and 31% for stroke.[3] The increase in smoking duration is in line with the cumulative dose of a smoker. The cumulative dose of smoking can be measured by the Brinkman index (BI). The BI is an assessment carried out by calculating the number of cigarettes smoked per day multiplied by the number of years of smoking.[4] High cumulative doses can increase the risk of developing various lung diseases, including lung cancer. Smoking can also cause damage to the lungs, which is characterized by inflammation, airway obstruction, and destruction of the lung parenchyma.[5] Chronic exposure to cigarette smoke increases the production of matrix metalloproteinase (MMP) by macrophages, which causes alveolar wall destruction.[6]

Peak expiratory flow (PEF) is defined as the maximum expiratory flow rate after maximum inspiration using a device called a peak flowmeter. A study in Andhra Pradesh, India, found a negative correlation between smoking and PEF (peak expiratory flow).[7] Another study in Hyderabad, India, also found that PEF in smokers was significantly lower than in people who did not smoke. As supported by research conducted by Sawant (2019), the prevalence of abnormal PEF was found in 84% of smokers.[8]

Another parameter that can be used to see a decrease in lung function due to smoking is forced expiratory volume in 1 second (FEV1). A study conducted in Switzerland reported a decrease in FEV1 of 10.4 mL in men and 13.8 mL in women for each pack of cigarettes used per day.[9] A separate study in the United States also found that the decrease in FEV1 among smokers is directly proportional to the number of cigarettes consumed daily.[10] Based on the explanation of the problem above, it can be inferred that the cumulative dose of smoking is correlated with a decline in lung function. This study aims to determine the significant relationship between the BI score and peak expiratory flow rate (PEFR) and to determine whether an increase in the BI score is accompanied by a decrease in the PEFR value.

2 | MATERIALS AND METHODS

Verbal and written informed consent was obtained for all participants included in this study. Ethical clearance has been approved by the Institutional Review Board (approval number: 017/K-LKJ/ETIK/I/2020). This study is a cross-sectional study conducted in February–April 2022 in Jakarta, Indonesia. Participants were recruited from the general population with the following inclusion criteria include male active smokers, aged 30 to 40 years, who have a smoking history for at least 1 year. Exclusion criteria are as follows: (1) uses e-cigarettes for smoking, (2) has a history of chronic lung diseases such as tuberculosis, pneumonia, post-COVID-19 syndrome, and asthma, and (3) has not smoked in the last 28 days. Data was collected by history taking which encompasses information on smoking history, height, and age to calculate the BI score. PEF was measured by taking the average of three peak flowmeter measurements, each separated by a 2-min interval conducted by a researcher. The PEF examination results obtained with the peak flowmeter will be compared with the predicted PEF values to calculate the PEFR. The Pneumobile Project formula as follows is used to calculate the predicted PEF value:

$$\text{Predicted PEF in male} = -10.86040 + 0.12766 \times (\text{age in years}) + 0.11169 \times (\text{height in cm}) - 0.0000319344 \times (\text{age in years})^3 \pm 1.70935$$

$$\text{Predicted PEF in female} = -5.12502 + 0.06980 \times (\text{age in years}) + (\text{height in cm}) - 0.00145669 \times (\text{age in years}) \pm 1.77692.$$

Collected research data is entered into Microsoft Excel 2019. The data is analyzed using SPSS (Statistical Package for the Social Sciences) software version 25.0, employing the Spearman correlation test analysis method to determine the correlation between the BI score variables and Peak Expiratory Flow Rate (PEFR).

3 | RESULTS

Forty-eight men who have given their informed consent and fulfilled inclusion and exclusion criteria are included in this study. Table 1 shows the characteristics of participants included in this study. The mean age of participants was 35.91 years ±3.40, with a history of smoking of 18.12 ± 6.54 years, and 12.5 ± 4.97 cigarettes smoked daily. Patients included in this study had BI classified as mild (47.91%), moderate (47.91%), and severe (4.16%). PEFR in patients where classified into green (10.41%), yellow (83.33%), and red (6.25%). The average PEFR value for the sample is 64.11 ± 11.68%, which means the average PEFR for the sample population is in the yellow zone. The lowest PEFR in the sample population was 43.04%, and the highest was 92%.

Based on the Kolmogorov–Smirnov data normality test, BI had significant test results ($p < 0.05$). This showed that data was not normally distributed. Kolmogorov–Smirnov data normality test on the PEFR variable were not significant ($p > 0.05$). This shows the normal distribution of data.

A correlation test was then conducted to determine the strength and direction of the relationship between the independent variable (BI) and the dependent variable (PEFR) and its statistical significance. The correlation test used was the Spearman correlation test because the assumption of normally distributed data was not met, thus non-parametric analysis was used. The results obtained are as follows:

Table 2 shows the correlation test conducted between BI and PEFR using the Spearman correlation test. This test showed a correlation value of $r = -0.721$ and p-value < 0.001. According to Colton,

TABLE 1 Characteristics of participants.

Characteristic	N	Prevalence	Mean
Age (years)			
30–35	20	50%	
36–40	20	50%	
			35.91 ± 3.40
History of smoking (years)			
1–5	2	4.16	
6–10	7	14.58	
11–15	7	14.58	
16–20	14	29.16	
21–25	13	27.08	
26–30	5	10.41	
			18.12 ± 6.54
Amount of cigarretes smoked daily			
<5	1	2.08	
5–9	11	22.91	
10–14	20	41.67	
15–24	15	31.25	
≥25	1	2.08	
			12.5 ± 4.97
Smoking intensity (Brinkman Index)			
Mild (<200)	23	47.91	
Moderate (200–600)	23	47.91	
Severe (>600)	2	4.16	
			228.5 ± 140.29
PEFR zone			
Green (>80%)	5	10.41	
Yellow (50%–80%)	40	83.33	
Red (<50%)	3	6.25	
			64.11 ± 11.68%

TABLE 2 Spearman correlation test results.

		BI	PEFR
BI	Correlation coefficient	1	−0.721
	Sig. (1-tailed)		0.000
	N	48	48
PEFR	Correlation coefficient	−0.721	1
	Sig. (1-tailed)	0.000	
	N	48	48

the strength of the relationship between two variables can be qualitatively divided into four levels as shown in Table 3.[8]:

Based on the results obtained in Tables 2 and 3, it can be concluded that the relationship between the BI score and PEFR shows a strong inverse relationship ($r = -0.721$). These results also indicate a significant relationship between the two variables ($p < 0.01$). A scatter plot regarding BI and PEFR showed a strong correlation with a negative linear pattern. R^2 value was 0.427 indicating BI affects 42.7% of PEFR value (Figure 1).

TABLE 3 Strength of correlation according to Colton.[8]

R-value	Interpretation of correlation strength
0.00–0.25	Weak or no linear relationship
0.26–0.50	A fair degree of linear relationship
0.51–0.75	Moderately strong linear relationship
0.76–1.00	Very strong linear relationship/Perfect linear

4 | DISCUSSION

The results of data analysis related to peak flowmetry examination are present in Table 1, indicating that the sample population has an average PEFR value of 64.11 ± 11.68%. This shows that the average PEFR for the sample population falls within the yellow zone. The abnormal peak flowmetry results in this smoking population are consistent with the findings of a study conducted by Sawant et al., which reported a 3.33 times higher risk for smokers to have an abnormal PEFR ($p < 0.05$).[11] Abnormal peak flowmetry results in the sample population who smoke can be attributed to inflammation, which is a consistent pathological factor in smokers. Airflow limitation in the respiratory tract of smokers is induced by inflammatory mediators resulting from cigarette smoke, leading to airway remodeling characterized by decreased cilia motility in the mucosa, an increased number of goblet cells, and mucus hypersecretion.[12] Gurung[13] also stated that the lungs of active smokers undergo histological changes and experience a decline in lung function, leading to abnormal peak flowmetry results.

The correlation test between the BI and PEFR yielded significant negative results, as shown in Table 2 ($r = -0.721$, $p < 0.001$), indicating that a higher BI corresponds to a lower PEFR value. Thus, the hypothesis can be established that an increase in the BI score will result in a decrease in the PEFR value. These findings align with a study conducted by Rao et al., which demonstrated a strong negative correlation between cigarette years and the PEFR value ($r = -0.83$, $p < 0.01$).[7] Supported by the results of Pearson correlation analysis, a strong negative correlation between smoking intensity and PEF was observed, indicating that higher smoking intensity is associated with lower PEF values.[14]

The cause of the decrease in PEFR as the BI increases can be caused by chronic inflammation that continues to occur in the walls of the respiratory tract. Cigarette smoke modulates inflammation by directly activating epithelial cells in the respiratory tract and inducing the release of chemokines and inflammatory mediators such as TNFa, GM-CSF, IL-8, MCP-1, and TSLP. These mediators recruit and promote the survival of neutrophils, macrophages, T cells, and dendritic cells, resulting in a chronic inflammatory process.[15] This leads to the narrowing of the respiratory tract, ultimately causing obstruction and resulting in decreased lung function parameter measurements. Cigarettes have been shown to have a significant impact on the lungs and their effects damage tissue and impact lung function. Smokers experience significant airway inflammation, and airway inflammation worsens

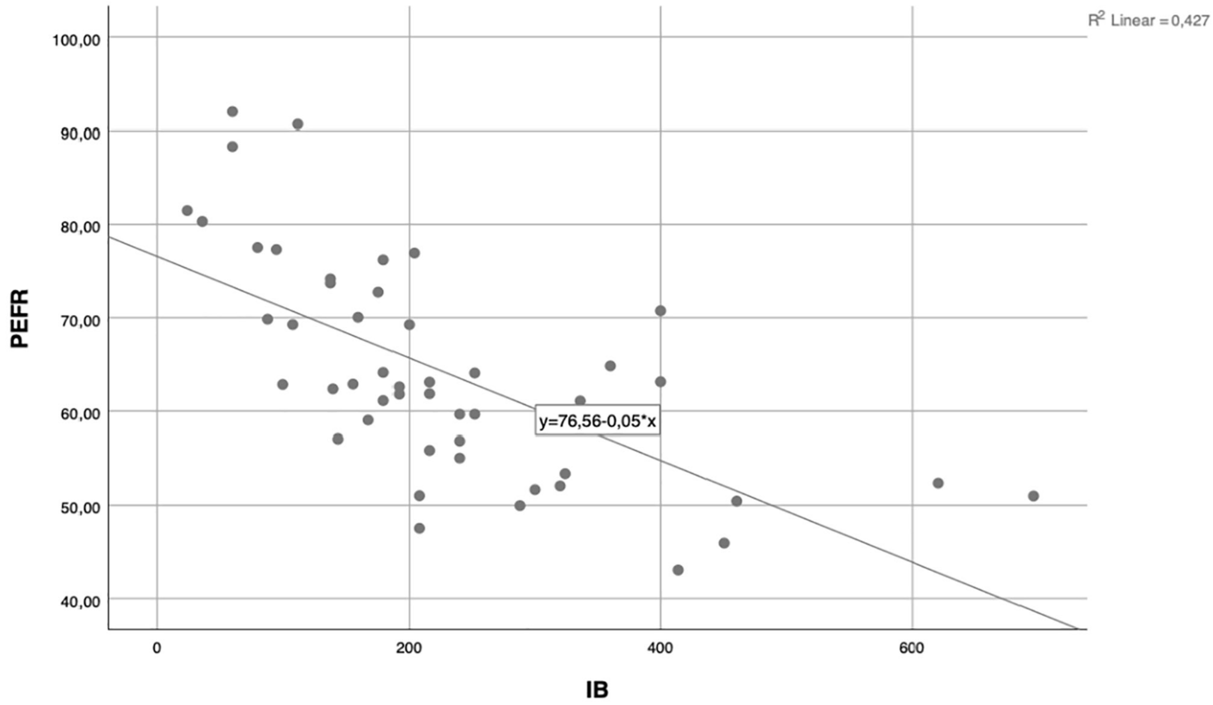

FIGURE 1 Scatter plot BI and PEFR.

remodeling due to repeated epithelial damage.[16] The involvement of inflammatory cells CD8+ T lymphocytes and eosinophils, contributes to the structural changes leading to airway obstruction.[17]

PEFR is an effective parameter for measuring lung function due to its relatively easy and straightforward procedure, affordable equipment, and absence of contraindications for the examination. PEFR is generally considered a primary indicator of changes in elastic retrograde pressure and/or inflammatory changes in the bronchiolar wall. The diagnosis of COPD in smokers can be established through a spirometry test to assess obstructions in the respiratory tract affecting PEFR.[18] Consistent with research conducted by Metha et al., which demonstrates a statistically significant reduction in PEFR levels between smokers and non-smokers. The PEFR score can decrease as the number of cigarettes smoked per day increases.[19] In smokers, PEFR values were found to be significantly lower than in non-smokers.[20]

This research uses PEFR as the indicator for lung function parameters. Thus, height, age, and ethnicity, which may influence PEF value are put into consideration. This research is limited by its sample size and sample distribution, wherein this research was only conducted in one city. Other lung function parameters such as FEV1 and FEV1/FVC using a spirometer were also not conducted in this research. Further research conducted on a larger sample size with a more representative population which also evaluates other lung function parameters is recommended.

5 | CONCLUSIONS

Higher Brinkman Index correlates with a decrease in peak expiratory flow rate. The simplicity of measuring peak expiratory flow rate may prove to be useful in detecting early decrease in pulmonary function in smokers and encourage smoking habit cessation in younger male smokers. Due to the limitations of this study, gold standard measurement should still be considered before making any presumptive diagnoses.

CONFLICT OF INTEREST STATEMENT

Authors declare no conflict of interests for this article.

DATA AVAILABILITY STATEMENT

Data is available online in the Pelita Harapan University Repository. Data may be accessed by contacting and acceptance by the institution.

ETHICS APPROVAL STATEMENT

Ethical approval have been obtained by the Institution Review Board of Pelita Harapan (Approval Code: 071/K-LKJ/ETIK/I/2020) on January 6, 2020.

PATIENT CONSENT STATEMENT

Verbal and written informed consent for all participants in this study have been obtained prior to initiating the study.

CLINICAL TRIAL REGISTRATION

Clinical trial registration is not required for this study as no intervention is conducted. Informed consent has been obtained.

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