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Prevalence and Predictors of COPD in Indonesian Smokers Attending Posbindu Community Health Centers: A Cross-Sectional Analysis

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ABSTRACT

Introduction: Chronic Obstructive Pulmonary Disease (COPD) is a major global health burden, particularly in low- and middle-income countries. In Indonesia, the high prevalence of smoking, coupled with underdiagnosis and limited healthcare access, contributes substantially to COPD cases. Identifying key risk factors is critical to strengthen prevention and management strategies. This study aimed to determine the prevalence of COPD among smokers attending POSBINDU community health center in Indonesia and to identify significant predictors associated with the disease.

Methods: A population-based cross-sectional study was conducted in March 2023 across Integrated Care Posts (Posbindu) in Yogyakarta and East Java. A total of 548 smokers aged ≥40 years were recruited using convenience sampling, which may limit generalizability. Only 17 COPD cases were identified (3.1% prevalence), constraining statistical power for multivariate analyses. Data collection involved structured questionnaires and spirometry testing following American Thoracic Society guidelines. Descriptive statistics, bivariate analysis, and multivariate logistic regression were used to identify independent predictors of COPD.

Results: The prevalence of COPD was 3.1% (17/548). Most participants were male (98.2%) and aged 40-49 years (44.2%). Bivariate analysis showed significant associations between COPD and sputum production (OR = 4.79; P = 0.001), shortness of breath (OR = 3.11; P = 0.017), asthma (OR = 23.03; P <0.001), cardiovascular disease (OR = 11.67; P = 0.005), and abnormal spirometry (OR = 12.59; P <0.001). In multivariate analysis, sputum production (AOR = 3.99; P = 0.019) and asthma (AOR = 8.64; P = 0.020) remained strong independent predictors. Conversely, a Brinkman Index ≥20 pack-years showed a paradoxical protective effect (AOR = 0.16; P = 0.023).

Conclusion: This study highlights the importance of early screening and intervention among high-risk smoker populations. Community-based platforms such as Posbindu can play a critical role in facilitating prevention, early diagnosis, and improved management of COPD.

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INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD), characterized by persistent airflow limitation and chronic airway inflammation, ranks as the third leading cause of death globally (1). The burden is disproportionately high in low- and middle-income countries (LMICs) including Indonesia, where the interplay of high smoking prevalence, increasing air pollution, and aging populations accelerates the epidemiological transition (2). In Indonesia, the prevalence of COPD is projected to rise significantly, yet the disease remains largely underdiagnosed and undertreated due to limited access to spirometry in primary care settings (3,4).

While cigarette smoking is the most established risk factor for COPD, there is significant heterogeneity in disease susceptibility, not all smokers develop airflow obstruction (5). This variability suggests that specific clinical phenotypes and modifiable risk factors interact with tobacco exposure to drive disease progression. Therefore, identifying these predictors among smokers is critical for early detection. The selection of predictors in this study is grounded in the pathophysiology of airway inflammation and systemic oxidative stress.

Respiratory symptoms, particularly chronic sputum production, were selected as key predictors because they serve as clinical markers for the "chronic bronchitis" phenotype. This phenotype is associated with accelerated lung function decline and higher exacerbation risks in smokers, independent of baseline airflow limitation (6). Sputum production, a hallmark of chronic bronchitis phenotype, reflects mucus hypersecretion from goblet cell hyperplasia and submucosal gland hypertrophy, exacerbating airflow obstruction via airway inflammation (7). Furthermore, a history of asthma was included due to the growing recognition of Asthma-COPD Overlap (ACO), where asthmatic smokers exhibit more severe persistent airflow limitation and systemic inflammation compared to those with COPD alone (8). Cardiovascular comorbidities contribute via systemic inflammation and hypoxemia-induced pulmonary vascular remodeling (9). Brinkman Index measures cumulative smoke exposure, driving oxidative stress central to COPD (7).

Beyond clinical history, lifestyle factors such as diet and physical activity provide theoretical insight into the systemic defense mechanisms against tobacco smoke. Fruit and vegetable intake was assessed as a proxy for antioxidant capacity. The imbalance between oxidative stress from cigarette smoke and antioxidant defense is a core mechanism in COPD pathogenesis; thus, low intake of dietary antioxidants is theoretically linked to greater lung tissue damage (10). Similarly, physical inactivity is both a consequence and a predictor of COPD, driven by systemic inflammation and skeletal muscle dysfunction (11).

Despite the importance of these factors, data integrating clinical phenotypes (sputum, asthma history) with lifestyle determinants (diet, activity) among the smoking population in Indonesia remains very limited. The use of questionnaires and spirometry in this study is intended to enhance measurement reliability. This approach helps clarify how lifestyle interacts with respiratory risk, there by supporting more targeted prevention efforts. In the context of the escalating COPD epidemic, strategies such as spirometry-based screening and active case-finding are becoming increasingly crucial (12,13). Most previous studies have relied on hospital-based cohorts, which are less able to capture early-stage cases common at the community level. Therefore, this study aims to determine the prevalence of COPD among smokers aged ≥40 years in Indonesia and to assess the theoretical relevance of these various predictors within a community context by Posbindu.

METHOD

Study Design & Setting

This study employed a population-based, cross-sectional design to assess COPD prevalence and risk factors among smokers in Yogyakarta and East Java provinces, Indonesia. Data was collected in March 2023 at Integrated Care Posts (Posbindu), community health centers that provide services for non-communicable diseases. These provinces were selected for their diverse demographics and varying COPD prevalence, enabling a comprehensive assessment of risk factors across different settings.

Population and Sample

The target population comprised smokers aged 40 years and older residing in Yogyakarta and East Java. A total of 548 participants was recruited through convenience sampling at Posbindu centres. Eligibility criteria included: 1) being a current or former smoker, 2) being aged ≥40 years, 3) residing in Yogyakarta or East Java, and (4) providing

informed consent. Exclusion criteria were: 1) current use of bronchodilators, 2) medical conditions preventing spirometry (e.g., recent abdominal, lung, or chest surgery within three months), or 3) testing positive for COVID-19. This age group was chosen because COPD risk increases with age, and individuals are more likely to have accumulated sufficient exposure to smoking and other risk factors.

The minimum sample size was determined using the single population proportion formula to estimate the prevalence of COPD. Based on previous national health data indicating a COPD prevalence of approximately 3.7% in Indonesia, with a 95% confidence level ($Z\alpha/2=1.96$) and a desired precision (d) of 5%, the minimum required sample size was calculated to be 220 participants. The final recruitment of 548 smokers exceeded this requirement, providing sufficient statistical power to estimate the prevalence of COPD within the target population with a high degree of precision.

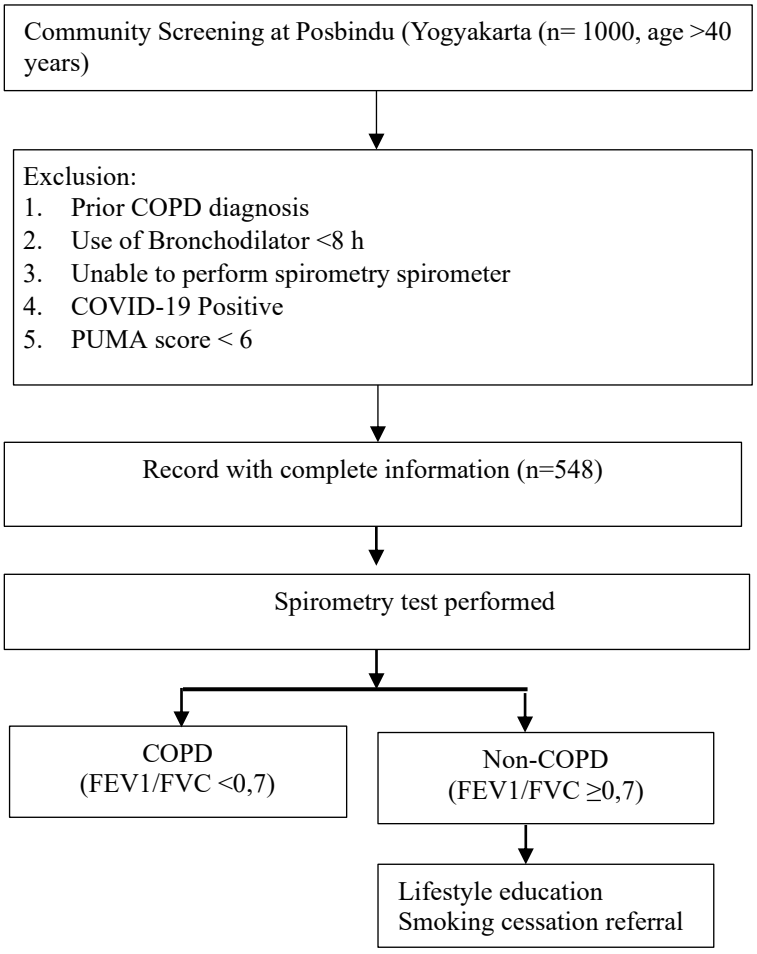

Figure 1. Flow Diagram of Participant Selection

Data Sources and Data Collection

Data were collected using a structured questionnaire adapted from the PUMA study (14), covering medical history (including comorbidities such as diabetes, hypertension, asthma, and cardiovascular disease) and socioeconomic information. Spirometry was conducted on all participants using CHEST spirometers (CHEST or CHEST HI-801) by pulmonologists or trained healthcare professionals following standardized procedures. Participants with a pre-bronchodilator FEV1/FVC ratio <0.7 underwent a 400mcg salbutamol challenge, followed by repeat spirometry after 15–20 minutes. A post-bronchodilator FEV1/FVC ratio <0.7 was used to confirm COPD, in

accordance with American Thoracic Society (ATS) guidelines (1). Questionnaires were administered confidentially in private rooms, and informed consent was obtained before participation. Quality control measures were applied to ensure accuracy and reliability. A history of hypertension, asthma, diabetes and cardiovascular disease can be obtained by asking about a patient's previous medical history.

COPD was defined according to ATS guidelines (1). Brinkman Index was used to define the degree of smoking that grouped into low risk (<20 packs year) and high risk (>20 packs year) (15). Central obesity was defined using WHO criteria (16). Fruit and vegetable intake frequency was defined as low (less than two meal a day) and normal (more than two meal a day) (17). Physical activity was defined as low (less than twice per week), and normal (2-3 times per week) (17). Cardiovascular diseases include heart conditions and disorders of the blood vessels, such as coronary heart disease, stroke, heart failure, heart rhythm problems (arrhythmias), and peripheral artery disease (18).

Data Analysis Procedure

Prior to statistical modeling, the dataset was inspected for completeness. A complete-case analysis approach was adopted, where participants with missing values for the primary outcome (spirometry results) or key predictor variables (demographics, PUMA questionnaire responses) were excluded during the data cleaning phase. Consequently, no statistical imputation methods were applied. The data collected were analyzed using SPSS Ver. 25 (IBM Corp., NY). Descriptive statistics summarized demographic characteristics and COPD prevalence. Bivariate analysis (chi square or Fisher test) explored associations between risk factors and COPD, while multivariate logistic regression identified independent predictors, adjusting for confounders. Association among variables were shown in odds ratio (OR) and adjusted odds ratio (AOR). Statistical significance was set at $P<0.05$.

Ethical Approval and Consent to Participate

This study was approved by the Directorate General of Disease Prevention and Control Ministry of Health. Approval No. TM.04.01/C. II/446/2023. All participants were informed about the objectives and procedures of the study and provided written informed consent before participation.

RESULTS

This study included 548 smokers aged 40 years and older, with an overall COPD prevalence of 3.1% (17 subjects) (Figure 1). Participants were predominantly male (98.2%) and middle-aged, with nearly half falling into the 40–49 age bracket. A significant proportion (38.3%) worked in high-risk occupations. regarding smoking habits, the majority had a Brinkman Index below 20 pack-years, and conventional cigarettes were the primary tobacco product used. Hypertension and central obesity were the most prevalent comorbidities, affecting approximately one-third of the study population. In terms of lifestyle, a substantial number of participants reported unhealthy behaviours, including low fruit and vegetable intake and physical inactivity. General characteristics of study participants are shown in Table 1.

Table 1. Characteristics of Study Participants

Category	Frequency	Percentage	P-value*
Age group (years)			
40-49	242	44.2	reff
50-59	183	33.4	0.773
>60	123	22.4	0.403
Gender			
Female	10	1.8	reff
Male	538	98.2	0.568
Occupation			
Not at risk	338	61.7	reff
At risk	210	38.3	0.202

Category	Frequency	Percentage	P-value*
Brinkman Index			
<20 packs year	412	75.18	reff
≥20 packs year	136	24.82	0.901
Cigarette types			
E-cigarettes	16	2.9	reff
Conventional cigarettes	532	97.1	0.832
Previous spirometry			
Normal	526	96.0	reff
Abnormal	22	4.0	0.003
Shortness of breath			
No	422	77.0	reff
Yes	126	23.0	0.000
Cough			
No	419	76.5	reff
Yes	129	23.5	0.125
Sputum			
No	438	79.9	reff
Yes	110	20.1	0.014
Comorbids			
Hypertension	153	27.9	0.082
Asthma	11	2.0	0.000
Diabetes	26	4.7	0.071
Cardiovascular diseases[a]	8	1.5	0.003
Central obesity	163	29.7	0.077
Low fruit and vegetable intake			
No	219	40	reff
Yes	329	60	0.329
Lack of physical activity			
No	294	53.6	reff
Yes	254	46.4	0.184
Alcohol consumption			
No	534	97.4	reff
Yes	14	2.6	0.649

COPD: chronic obstructive pulmonary disease
[a] coronary heart disease, stroke, heart failure, heart rhythm problems (arrhythmias), and peripheral artery disease
*Calculated using Chi square

Bivariate analysis identified five significant predictors of COPD (Table 2). Asthma showed the strongest association (OR=23.03, 95% CI: 5.99-88.50, P<0.001), with 36.4% of asthmatic smokers having COPD versus 2.4% without asthma. Sputum production increased COPD risk nearly 5-fold (OR=4.79, 95% CI: 1.80-12.72, P=0.001), reflecting chronic bronchitis phenotype. Previous abnormal spirometry (OR=12.59, P<0.001) and cardiovascular disease (OR=11.67, P=0.005) also showed strong associations. Shortness of breath was significant (OR=3.11, P=0.017), while age ≥50 years (OR=2.64, P=0.082) and cough (OR=2.34, P=0.082) showed borderline significance. Smoking intensity, gender, occupation, and lifestyle factors showed no significant associations (Table 2).

Table 2. Factors Associated with COPD

Variables	Subject group[b]		OR	95% CI	P-value*
	COPD	Non-COPD			
Age group (years)					
40-49	4 (1.7)	238 (98.3)	2.64	0.850-8.202	0.082
≥50	13 (4.2)	293 (95.8)			
Gender					
Female	0	10 (100)	1.03	1.017-1.049	0.568
Male	17 (3.2)	521 (96.8)			
Occupation					
Not at risk	13 (3.8)	325 (96.2)	0.48	0.156-1.509	0.203
At risk	4 (1.9)	206 (98.1)			
Brinkman Index					
<20 packs year	13 (3.2)	399 (96.8)	0.93	0.298-2.902	0.901
≥20 packs year	4 (2.9)	132 (97.1)			
Cigarette types					
E-cigarettes	0	16 (100)	1.03	1.017-1.049	0.468
Conventional cigarettes	17 (3.2)	515 (96.8)			
Previous spirometry					
Normal	12 (2.3)	514 (97.7)	12.59	3.990-39.776	<0.001
Abnormal	5 (22.7)	17 (77.3)			
Shortness of breath					
No	9 (2.1)	413 (97,9)	3.11	1.175-8.241	0.017
Yes	8 (6.3)	118 (93,)			
Cough					
No	10 (2.4)	409 (97.6)	2.34	0.875-6.295	0.082
Yes	7 (5.4)	122 (94.6)			
Sputum					
No	8 (1.8)	430 (98.2)	4.79	1.804-12.720	0.001
Yes	9 (8.2)	101 (91.8)			
Hypertension					
No	10 (2.5)	385 (97.5)	1.84	0.690-4.940	0.216
Yes	7 (4.6)	146 (95.4)			
Asthma					
No	13 (2.4)	524 (97.6)	23.03	5.994-88.502	<0.001
Yes	4 (36.4)	7 (63.6)			
Diabetes					
No	15 (2.9)	507 (97.1)	2.81	0.609-13.023	0.167
Yes	2 (7.7)	24 (92.3)			
Cardiovascular diseases[a]					
No	15 (2.8)	525 (97.2)	11.67	2.173-62.635	<0.001
Yes	2 (25.0)	6 (75.0)			
Central obesity					
No	13 (3.4)	372 (96.6)	0.720	0.231-2.242	0.569
Yes	4 (2.5)	159 (97.5)			
Low fruit and vegetable intake					
No	5 (2.3)	214 (97.7)	1.62	0.563-4.665	0.456
Yes	12 (3.6)	317 (96.4)			

Variables	Subject group[b]		OR	95% CI	P-value*
	COPD	Non-COPD			
Lack of physical activity					
No	12 (4.1)	282 (95.9)	0.47	0.164-1.358	0.155
Yes	5 (2.0)	249 (98.0)			
Alcohol consumption					
No	17 (3.2)	517 (96.8)	0.96	0.953-0.983	0.498
Yes	0	14 (100)			

COPD: chronic obstructive pulmonary disease; OR: odds ratio; CI: confidence interval
[a] coronary heart disease, stroke, heart failure, heart rhythm problems (arrhythmias), and peripheral artery disease
[b] Presented in frequency (%)
*Calculated using Chi square

Multivariate logistic regression identified several independent predictors of COPD (Table 3). A Brinkman Index of ≥20 pack-years was unexpectedly associated with a lower likelihood of COPD (AOR = 0.16; 95% CI: 0.032–0.777; P = 0.023). In contrast, the presence of sputum predicted the risk of COPD (AOR = 3.99; 95% CI: 1.262–12.651; P = 0.019). Asthma as a comorbid condition also remained a strong independent predictor, with an 8.6-fold higher risk of COPD (AOR = 8.64; 95% CI: 1.395–53.557; P = 0.020). Abnormal spirometry showed a nearly four-fold increased risk (AOR = 3.96), though this did not reach statistical significance (P = 0.072). Occupation classified as low risk demonstrated a protective effect (AOR = 0.29; P = 0.074), but the association was borderline and not statistically significant.

Table 3. Independent Variables Associated with COPD in Multivariate Analysis

Variables	B	AOR	95% CI		P-value
			Lower	Upper	
Brinkman Index ≥20 packs year	-1.852	0.16	0.032	0.777	0.023
Presence of sputum	1.358	3.99	1.262	12.651	0.019
High-risk occupation	-1.229	0.29	0.079	1.122	0.074
Asthma as comorbid	2.157	8.64	1.395	53.557	0.020
Abnormal spirometry	1.378	3.96	0.883	17.806	0.072

AOR: adjusted odds ratio; CI: confidence interval

DISCUSSION

The overall prevalence of COPD in this study population was 3.1%, consistent with previous Indonesian studies, although variations are noted due to differences in populations and diagnostic criteria (19). Sputum production emerged as a significant predictor of COPD, reflecting its role as a marker of chronic airway inflammation and damage that contributes to airflow limitation and disease progression (20,21). The inverse association between the Brinkman Index and COPD in the multivariate model requires further investigation; genetic predisposition or environmental exposures may influence the relationship between smoking and COPD (22). One possible explanation is survivor bias, whereby individuals with high smoking exposure who are susceptible to COPD may have already died or been diagnosed, leaving a cohort of more resilient smokers in the analysis. While the association between smoking and COPD is well established, this anomaly highlights the complexity of smoking-related risk. In contrast, the strong association between asthma and COPD observed in this study is consistent with previous research, reinforcing evidence of overlap between the two conditions (13,23,24).

These findings underscore the need for targeted interventions to reduce COPD among smokers in Indonesia. The prevalence aligns with other epidemiological studies using similar diagnostic criteria (25,26). Tobacco smoke remains the primary risk factor for COPD worldwide (23,27). Public health measures should prioritise smoking cessation, reduction of air pollution exposure, and improved access to early diagnosis and treatment (28,29). Further

research is required to clarify the complex interplay of smoking history, genetic susceptibility, and COPD development, with longitudinal studies needed to track disease progression and identify predictors of severity and outcomes.

Comorbidities also play a critical role. COPD patients are more likely to experience major adverse cardiovascular events (MACEs) following exacerbations, highlighting the importance of integrated management addressing both respiratory and cardiovascular health (30). Asthma, conceptually a risk factor, showed a strong association with COPD in this study, consistent with evidence of asthma COPD overlap (21).

Regarding dietary habits, although theoretical models suggest that low fruit and vegetable intake exacerbates oxidative stress and COPD progression (34), our study did not observe a statistically significant association between low intake and COPD risk (OR = 1.62; P = 0.456). This discrepancy with broader literature may be attributed to the cross-sectional nature of the data or recall bias inherent in self-reported dietary assessments. However, given the high prevalence of poor nutrition in this cohort (60%), promoting antioxidant-rich diets should remain a general public health priority for smokers, even if the direct statistical link was not established in this specific sample.

Regarding occupational exposures, our analysis did not demonstrate a statistically significant association between high-risk occupations (e.g., laborers, farmers) and COPD (AOR = 0.29; P = 0.074). This finding contrasts with global literature identifying occupational dust as a major risk factor. The lack of association and the trend towards a protective effect observed in our model likely reflects the 'healthy worker effect'. This phenomenon occurs when workers with respiratory symptoms or declining lung function leave physically demanding jobs, leaving a survivor population of healthier individuals in high-risk sectors (31). Therefore, interpretations regarding occupational risks in this cross-sectional cohort must be made with caution, as the data does not support occupation as a primary driver of COPD prevalence in this specific sample.

Age was another important determinant. COPD prevalence increased significantly among participants 50 years and older, consistent with regional and international studies showing age as a major driver of COPD (32). Another study demonstrated a notable difference in risk between age groups, with individuals aged ≥50 years having a 2.6-fold higher risk of developing COPD compared to those aged 40–49 years, and smoking were identified as the most important determinants of COPD (33). In China, the incidence of COPD has been observed to rise sharply with age, reaching its peak among individuals aged 95 years and older (34). Similarly, studies in Sweden have shown that smoking and occupational exposure, in addition to advanced age, are significant risk factors for COPD (35).

The inverse association observed between a higher Brinkman Index (≥20 pack-years) and COPD prevalence (AOR = 0.16) is paradoxically consistent with the 'healthy smoker effect' described in epidemiological literature. This phenomenon suggests a selection bias wherein smokers susceptible to rapid lung function decline develop symptoms early and subsequently cease smoking, thereby limiting their cumulative exposure (lower Brinkman Index). Consequently, the remaining cohort of heavy smokers likely represents a 'survivor phenotype' with innate physiological or genetic resistance to tobacco induced airway remodeling (36,37). This aligns with the 'susceptible smoker' hypothesis, which posits that only a fraction of smokers develops significant airflow obstruction due to variations in genetic detoxification pathways and inflammatory responses.

Effective COPD management must therefore integrate lifestyle modifications, comorbidity control, and preventive strategies. Strengthening community-based services such as Posbindu can support smoking cessation, dietary improvement, regular spirometry, and comorbidity management. Public health campaigns should raise awareness of COPD symptoms and the benefits of early medical care (38). The morbidity and economic burden of COPD in Indonesia can be mitigated by strengthening public awareness of prevention, particularly regarding lifestyle and respiratory health, expanding screening programmes, and promoting healthier behaviours (39). Primary healthcare workers play a pivotal role by addressing individual risk factors, especially among smokers, through smoking cessation counselling, dietary guidance, and regular follow-ups such as spirometry testing. Their responsibilities also include referring patients to specialists every three months, monitoring blood pressure and cholesterol, educating high-risk workers on the use of protective equipment, providing respiratory muscle training, managing comorbidities, controlling asthma, and supporting the establishment of COPD self-help groups. The Posbindu platform offers an effective entry point for community-based COPD prevention and management initiatives.

Limitations and Cautions

This study has several limitations. Its cross-sectional design precludes causal inference, and convenience sampling may have introduced selection bias. Reliance on self-reported data could lead to recall bias, while restriction to two provinces limits generalisability. The relatively small sample size may have reduced statistical power, and genetic factors such as alpha-1 antitrypsin deficiency were not considered. Despite these limitations, the study provides valuable insights into COPD risk factors among Indonesian smokers and highlights priorities for prevention and management.

Recommendations for Future Research

Future study should incorporate longitudinal cohort designs to better understand the causal pathways between smoking exposure, respiratory symptoms, and COPD development, particularly to clarify the paradoxical protective effect observed with higher Brinkman Index scores. Larger and more diverse populations, including women, younger smokers, and individuals with varied occupational exposures are needed to improve generalisability and to explore potential genetic or environmental modifiers. Future studies should also investigate asthma, COPD overlap using objective biomarkers and advanced lung function tests, as well as evaluate the role of lifestyle factors such as diet and physical activity in COPD progression. Additionally, research exploring the effectiveness of community-based screening platforms like Posbindu in early COPD detection and long-term management would provide valuable evidence to strengthen public health strategies in Indonesia.

CONCLUSION

The prevalence of COPD among Indonesian smokers aged ≥40 years in this community cohort was 3.1%. This study provides empirical evidence that specific clinical phenotypes, specifically the chronic bronchitis phenotype (sputum production) and Asthma COPD overlap are potent, identifiable predictors in primary care settings. The findings validate the Posbindu screening model not merely as a data collection point, but as a critical strategic node for active case finding. By shifting focus from general smoking cessation alone to targeted screening of these high-risk phenotypes, Indonesia can optimize limited healthcare resources to intercept COPD progression earlier.

AUTHOR'S CONTRIBUTION STATEMENT

All authors contributed substantially to the conception and design of the study, data collection, analysis, and interpretation. Dian Meutia Sari led the data analysis, manuscript drafting, and overall coordination of the research. Besral contributed to the study design, data validation, and critical revision of the manuscript for important intellectual content. Ratna Djuwita and Triya supervised the study implementation, provided methodological guidance, and reviewed the final version of the manuscript. Farida Murtiani reviewed the final version of the manuscript. All authors have read and approved the final manuscript and agree to be accountable for all aspects of the work.

CONFLICTS OF INTEREST

The authors declare no conflicts of interest related to this study. The research was conducted independently, without any commercial or financial relationships that could be construed as a potential conflict of interest.

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

During the preparation of this manuscript, the authors used generative artificial intelligence (AI) and AI-assisted tools, including ChatGPT for language refinement, structure of the text, and improvement of clarity, as well as Grammarly for grammar and style checking. The authors reviewed and edited all generated content to ensure accuracy, originality, and compliance with the study's scientific context. The final version of the manuscript reflects the authors' own analysis, interpretation, and intellectual contributions.

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