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Original Research Article

Prevalence and Risk Factors of Chronic Obstructive Pulmonary Disease among Adult Smokers

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Abstract

Background: Chronic obstructive pulmonary disease (COPD) constitutes a leading cause of global morbidity and mortality, with cigarette smoking representing the most important modifiable risk factor. However, not all smokers develop COPD, and the interplay of smoking characteristics with demographic, occupational, and clinical variables that collectively determine disease susceptibility remains incompletely understood. This study aimed to determine the prevalence of COPD among adult smokers and identify the key risk factors associated with disease development.

Methods: A community-based cross-sectional study was conducted across six primary healthcare centers. A total of 1,240 current and former adult smokers aged ≥30 years were enrolled. All participants underwent standardized spirometry following American Thoracic Society/European Respiratory Society (ATS/ERS) guidelines, with COPD defined as a post-bronchodilator FEV_1/FVC ratio <0.70. Sociodemographic data, smoking history (pack-years), occupational exposures, respiratory symptoms, comorbidities, and biomass fuel exposure were systematically assessed using structured questionnaires.

Results: The overall prevalence of spirometrically confirmed COPD among adult smokers was 24.8% (n = 308). The prevalence increased significantly with age (15.2% in 30–44 years vs. 38.6% in ≥60 years, $p < 0.001$) and cumulative smoking exposure (14.6% in <20 pack-years vs. 41.2% in ≥40 pack-years, $p < 0.001$). On multivariable logistic regression, independent risk factors for COPD included age ≥60 years (aOR 3.42, 95% CI 2.18–5.36), smoking ≥40 pack-years (aOR 3.86, 95% CI 2.48–6.01), occupational dust exposure (aOR 2.14, 95% CI 1.46–3.14), childhood respiratory infections (aOR 1.92, 95% CI 1.28–2.88), BMI <18.5 kg/m² (aOR 2.26, 95% CI 1.34–3.81), and biomass fuel exposure (aOR 1.78, 95% CI 1.18–2.68). Among diagnosed COPD patients, 58.4% were previously undiagnosed.

Conclusion: Approximately one in four adult smokers has spirometrically confirmed COPD, with a substantial proportion remaining undiagnosed. Beyond cumulative smoking exposure, advancing age, occupational hazards, childhood respiratory infections, low body mass index, and biomass fuel exposure are significant independent risk factors. Targeted spirometric screening programs among high-risk smokers are warranted for early detection and intervention.

Keywords: Chronic obstructive pulmonary disease; COPD; smoking; prevalence; risk factors; spirometry; pack-years; occupational exposure.

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Introduction

Chronic obstructive pulmonary disease is a progressive respiratory disorder characterized by persistent airflow limitation that is not fully reversible, resulting from chronic inflammatory responses in the airways and lung parenchyma to noxious particles and gases [1]. According to the Global Burden of Disease Study, COPD affected approximately 392 million individuals worldwide in 2019 and was responsible for an estimated 3.28 million deaths annually, making it the third leading cause of death globally [2]. The socioeconomic impact of COPD is substantial, encompassing direct healthcare costs from hospitalizations, pharmacotherapy, and oxygen therapy, as well as indirect costs attributable to disability, loss of workforce productivity, and caregiver burden [3].

Cigarette smoking is unequivocally established as the predominant risk factor for COPD development, with population-attributable fractions ranging from 40% to 70% across different studies and geographic regions [4]. The landmark Fletcher and Peto model described the accelerated decline in forced expiratory volume in one second (FEV_1) among susceptible smokers compared to non-smokers, providing the conceptual framework for understanding smoking-related COPD pathogenesis [5]. However, a critical observation is that only approximately 15–50% of smokers ultimately develop clinically significant COPD, indicating that individual susceptibility factors—genetic predisposition, environmental co-exposures, developmental influences, and comorbid conditions—play important modifying roles [6].

The Global Initiative for Chronic Obstructive Lung Disease (GOLD) has emphasized the importance of early identification and risk factor modification in COPD management [7]. Despite this, substantial underdiagnosis remains a pervasive problem worldwide. Lamprecht et al. (2015), analyzing data from the Burden of Obstructive Lung Disease (BOLD) study across 12 countries, demonstrated that over 70% of COPD cases identified by spirometry had not been previously diagnosed, representing a missed opportunity for early therapeutic intervention and disease modification [8]. Similarly, the PLATINO study in Latin America and the PREPOCOL study in Colombia reported high rates of underdiagnosis even among symptomatic individuals, attributable to limited access to spirometry, inadequate healthcare provider awareness, and patient normalization of respiratory symptoms [9].

Several risk factors beyond active smoking have been identified as contributors to COPD development. Occupational exposure to organic and inorganic dusts, chemical fumes, and vapors accounts for an estimated 15–20% of COPD cases and may interact synergistically with smoking to amplify disease risk [10]. Indoor biomass fuel exposure, affecting approximately 3 billion individuals globally who rely on solid fuels for cooking and heating, has been increasingly recognized as a significant COPD risk factor, particularly in developing countries and among women [11]. Furthermore, childhood respiratory infections, low birth weight, maternal smoking during pregnancy, and impaired lung development have been associated with reduced maximally attained lung function in early adulthood, predisposing to earlier onset of clinically manifest airflow obstruction with subsequent noxious exposures [12]. Despite the extensive literature on COPD epidemiology, several important gaps remain. First, population-specific prevalence data among smokers using standardized spirometric criteria are limited in many regions, with considerable reliance on self-reported diagnoses that substantially underestimate true disease burden [13]. Second, the relative contributions of modifiable risk factors beyond smoking intensity—including occupational exposures, biomass fuel use, nutritional status, and early-life respiratory events—have not been comprehensively evaluated in a single integrated analytical framework within smoking populations [14]. Third, characterization of undiagnosed COPD and its associated risk profile among smokers attending primary healthcare settings remains inadequately addressed [15].

The aim of this study was to determine the prevalence of spirometrically confirmed COPD among adult current and former smokers in a community-based setting and to identify the demographic, behavioral, occupational, and clinical risk factors independently associated with COPD development.

Materials and Methods

Study Design and Setting: This was a multicenter community-based cross-sectional study conducted across six primary healthcare centers serving urban and semi-urban populations.

Sample Size Calculation: Based on prior studies reporting COPD prevalence of approximately 20–25% among smokers, with a 95% confidence level, 2.5% margin of error, and an anticipated 10% incomplete data or spirometry failure rate, a minimum sample size of 1,180 participants was estimated. A total of 1,240 participants were ultimately enrolled.

Study Population: Adults aged ≥30 years with a cumulative smoking history of ≥10 pack-years (current or former smokers) attending the participating primary healthcare centers for any reason during the study period were invited to participate through consecutive sampling.

Inclusion Criteria: Participants were included if they met the following criteria: (1) age ≥30 years; (2) smoking history of ≥10 pack-years (current or former smoker, where former smoker was defined as cessation ≥6 months prior to enrollment); (3) ability to perform reproducible spirometry; and (4) willingness to provide informed consent.

Exclusion Criteria: Exclusion criteria were: (1) known active pulmonary tuberculosis or history of tuberculosis treatment within the preceding 12 months; (2) prior thoracic surgery (lobectomy, pneumonectomy); (3) known interstitial lung disease or bronchiectasis; (4) active malignancy; (5) severe cardiac failure (NYHA class IV); (6) recent thoracic, abdominal, or ophthalmic surgery within six weeks (contraindication to spirometry);

(7) pregnancy; (8) inability to perform acceptable spirometric maneuvers after adequate coaching; and (9) cognitive impairment precluding informed consent or questionnaire completion.

Data Collection

A structured, interviewer-administered questionnaire was used to collect the following data:

- **Sociodemographic variables:** Age, sex, educational level, occupation, monthly household income, and residential area.
- **Smoking history:** Current versus former smoking status, age at smoking initiation, daily cigarette consumption, total years of smoking, pack-years calculated as (cigarettes per day / 20) × years smoked, and time since cessation for former smokers.
- **Occupational exposure:** Self-reported exposure to workplace dusts (mineral, organic, textile), chemical fumes, vapors, or gases for ≥1 year.
- **Biomass fuel exposure:** Regular use of wood, charcoal, crop residues, or animal dung for cooking or heating for ≥10 years.
- **Respiratory symptoms:** Assessed using the modified Medical Research Council (mMRC) dyspnea scale and standardized questions regarding chronic cough, sputum production, wheeze, and chest tightness.
- **Medical history:** Childhood respiratory infections (self-reported hospitalization for respiratory illness before age 10), history of asthma, tuberculosis, cardiovascular disease, diabetes mellitus, and current medication use.
- **Anthropometry:** Height and weight were measured, and BMI was calculated. BMI was categorized as underweight (<18.5 kg/m²), normal (18.5–24.9 kg/m²), overweight (25.0–29.9 kg/m²), and obese (≥30.0 kg/m²).

Spirometry: Standardized pre- and post-bronchodilator spirometry was performed using calibrated portable spirometers (EasyOne Air, ndd Medical Technologies, Zurich, Switzerland) in accordance with ATS/ERS technical standards. Trained respiratory technicians administered the test with a minimum of three acceptable and two reproducible maneuvers. Post-bronchodilator spirometry was performed 15 minutes after administration of 400 µg inhaled salbutamol via a spacer device. COPD was defined as a post-bronchodilator FEV_1/FVC ratio <0.70, consistent with GOLD criteria. Severity staging was classified according to GOLD spirometric classification: GOLD 1 (mild, FEV_1 ≥80% predicted), GOLD 2 (moderate, 50% ≤ FEV_1 < 80%), GOLD 3 (severe, 30% ≤ FEV_1 < 50%), and GOLD 4 (very severe, FEV_1 <30%).

Statistical Analysis: Continuous variables were expressed as mean ± SD and compared using independent t-tests or one-way ANOVA. Categorical variables were expressed as frequencies and percentages and analyzed using chi-square tests. Univariable logistic regression was initially performed to identify variables associated with COPD at $p < 0.20$, which were subsequently entered into a multivariable logistic regression model using backward stepwise elimination to identify independent risk factors. Results were presented as adjusted odds ratios (aOR) with 95% confidence intervals (CI). Statistical significance was defined as $p < 0.05$. Analyses were performed using STATA version 17.0 (StataCorp LLC, College Station, TX, USA).

Results

Demographic and Smoking Characteristics: A total of 1,240 adult smokers completed the study protocol with acceptable spirometric data. The mean age was 52.8 ± 11.4 years, with males comprising 82.3% (n = 1,021). Current smokers constituted 64.2% (n = 796) and former smokers 35.8% (n = 444). The mean cumulative smoking exposure was 28.6 ± 14.8 pack-years. Occupational dust or chemical exposure was reported by 32.4% (n = 402) of participants, and biomass fuel exposure was reported by 24.8% (n = 308). Baseline demographic and smoking characteristics are presented in Table 1.

Prevalence and Severity of COPD: The overall prevalence of spirometrically confirmed COPD was 24.8% (308/1,240). Among the 308 COPD patients, 180 (58.4%) had not been previously diagnosed with COPD or any chronic respiratory disease. COPD prevalence increased significantly with age: 15.2% in the 30–44 year group, 24.6% in the 45–59 year group, and 38.6% in those aged ≥60 years (p-trend < 0.001). Similarly, prevalence increased with cumulative smoking exposure: 14.6% in <20 pack-years, 26.4% in 20–39 pack-years, and 41.2% in ≥40 pack-years (p-trend < 0.001).

Regarding severity distribution, GOLD stage 1 (mild) accounted for 32.1% (n = 99), GOLD stage 2 (moderate) for 42.5% (n = 131), GOLD stage 3 (severe) for 18.8% (n = 58), and GOLD stage 4 (very severe) for 6.5% (n = 20). Spirometric parameters and respiratory symptom data are presented in Table 2.

Table 1: Demographic and Smoking Characteristics of Study Participants (N = 1,240)

Variable	Total (N = 1,240)	COPD (n = 308)	Non-COPD (n = 932)	p-value
Age (years), mean ± SD	52.8 ± 11.4	58.6 ± 10.2	50.9 ± 11.2	<0.001
Male sex, n (%)	1,021 (82.3)	264 (85.7)	757 (81.2)	0.072
BMI (kg/m²), mean ± SD	24.8 ± 4.6	23.2 ± 4.8	25.3 ± 4.4	<0.001
BMI <18.5 kg/m², n (%)	98 (7.9)	42 (13.6)	56 (6.0)	<0.001
Education ≤ primary, n (%)	386 (31.1)	128 (41.6)	258 (27.7)	<0.001
Current smoker, n (%)	796 (64.2)	216 (70.1)	580 (62.2)	0.011
Pack-years, mean ± SD	28.6 ± 14.8	38.4 ± 14.2	25.4 ± 13.6	<0.001
Pack-years ≥40, n (%)	284 (22.9)	117 (38.0)	167 (17.9)	<0.001
Age at smoking initiation (years)	19.4 ± 4.6	17.8 ± 4.2	19.9 ± 4.6	<0.001
Occupational dust/chemical exposure, n (%)	402 (32.4)	138 (44.8)	264 (28.3)	<0.001
Biomass fuel exposure, n (%)	308 (24.8)	104 (33.8)	204 (21.9)	<0.001
Childhood respiratory infections, n (%)	218 (17.6)	82 (26.6)	136 (14.6)	<0.001
History of asthma, n (%)	86 (6.9)	34 (11.0)	52 (5.6)	0.001
Diabetes mellitus, n (%)	168 (13.5)	52 (16.9)	116 (12.4)	0.046
Hypertension, n (%)	324 (26.1)	94 (30.5)	230 (24.7)	0.040

Table 2:Spirometric Parameters and Respiratory Symptoms by COPD Status

Parameter	COPD (n = 308)	Non-COPD (n = 932)	p-value
FEV_1 (L), mean ± SD	1.62 ± 0.58	2.84 ± 0.64	<0.001
FEV_1 % predicted, mean ± SD	56.4 ± 18.6	92.6 ± 12.8	<0.001
FVC (L), mean ± SD	2.86 ± 0.72	3.48 ± 0.68	<0.001
FEV_1/FVC ratio, mean ± SD	0.56 ± 0.10	0.81 ± 0.05	<0.001
GOLD Stage			
Stage 1 (mild), n (%)	99 (32.1)	—	—
Stage 2 (moderate), n (%)	131 (42.5)	—	—
Stage 3 (severe), n (%)	58 (18.8)	—	—
Stage 4 (very severe), n (%)	20 (6.5)	—	—
Respiratory Symptoms			
Chronic cough, n (%)	218 (70.8)	286 (30.7)	<0.001
Chronic sputum, n (%)	196 (63.6)	224 (24.0)	<0.001
Dyspnea (mMRC ≥2), n (%)	174 (56.5)	112 (12.0)	<0.001
Wheeze, n (%)	148 (48.1)	98 (10.5)	<0.001
Any respiratory symptom, n (%)	264 (85.7)	412 (44.2)	<0.001
Previously diagnosed COPD, n (%)	128 (41.6)	—	—
Previously undiagnosed, n (%)	180 (58.4)	—	—

Multivariable Analysis of Risk Factors: Multivariable logistic regression analysis identified several independent risk factors for COPD (Table 3).

The strongest independent predictor was cumulative smoking exposure ≥40 pack-years (aOR 3.86, 95% CI 2.48–6.01, p < 0.001), followed by age ≥60 years (aOR 3.42, 95% CI 2.18–5.36, p < 0.001).

Other significant independent predictors included BMI <18.5 kg/m² (aOR 2.26, 95% CI 1.34–3.81, p = 0.002), occupational dust/chemical exposure (aOR 2.14, 95% CI 1.46–3.14, p < 0.001), childhood respiratory infections (aOR 1.92, 95% CI 1.28–2.88, p = 0.002), biomass fuel exposure (aOR 1.78, 95% CI 1.18–2.68, p = 0.006), early smoking initiation age <16 years (aOR 1.74, 95% CI 1.14–2.66, p = 0.010), and current smoking status (aOR 1.56, 95% CI 1.08–2.25, p = 0.018).

History of asthma was also independently associated with COPD (aOR 1.84, 95% CI 1.08–3.14, p = 0.026). Male sex and education level were not independently significant after adjustment.

Table 3: Multivariable Logistic Regression Analysis for Independent Risk Factors of COPD

Risk Factor	Adjusted OR	95% CI	p-value
Age ≥60 years (vs. 30–44)	3.42	2.18–5.36	<0.001
Age 45–59 years (vs. 30–44)	1.68	1.12–2.52	0.012
Pack-years ≥40 (vs. <20)	3.86	2.48–6.01	<0.001
Pack-years 20–39 (vs. <20)	1.84	1.26–2.69	0.002
BMI <18.5 kg/m²	2.26	1.34–3.81	0.002
Occupational dust/chemical exposure	2.14	1.46–3.14	<0.001
Childhood respiratory infections	1.92	1.28–2.88	0.002
Biomass fuel exposure (≥10 years)	1.78	1.18–2.68	0.006
Smoking initiation age <16 years	1.74	1.14–2.66	0.010
Current smoker (vs. former)	1.56	1.08–2.25	0.018
History of asthma	1.84	1.08–3.14	0.026
Male sex	1.32	0.84–2.08	0.228
Education ≤ primary	1.28	0.88–1.86	0.196
Diabetes mellitus	1.18	0.78–1.79	0.438

Discussion

This multicenter community-based study reveals that approximately one in four adult smokers with a cumulative exposure of ≥10 pack-years has spirometrically confirmed COPD, with a striking 58.4% of affected individuals being previously undiagnosed. These findings underscore both the substantial burden of COPD within smoking populations and the persistent global challenge of underdiagnosis that continues to impede timely therapeutic intervention and disease management.

The overall prevalence of 24.8% observed in our study is consistent with findings from several large international epidemiological investigations. The BOLD study reported COPD prevalence rates of 10–30% among adults aged ≥40 years across multiple countries, with rates expectedly higher among ever-smokers [16]. Halbert et al. (2006), in a comprehensive systematic review of 62 studies, estimated a pooled COPD prevalence of 8.9% among the general adult population but noted substantially higher rates of 15–25% among smokers [17]. Our somewhat higher prevalence may reflect the inclusion criterion of ≥10 pack-years, which enriched the study population with more heavily exposed individuals compared to studies including all ever-smokers.

The high rate of previously undiagnosed COPD (58.4%) observed in our study is alarming yet consistent with global patterns. A systematic review by Diab et al. (2018) reported that undiagnosed COPD ranged from 50% to 90% across different populations, with higher rates of underdiagnosis observed in primary care settings where spirometry is often unavailable [18]. The implications of this diagnostic gap are profound, as undiagnosed patients are deprived of pharmacological interventions, smoking cessation counseling, pulmonary rehabilitation, and vaccination strategies that have been demonstrated to reduce exacerbation frequency, improve quality of life, and potentially modify disease trajectory [19]. The identification of cumulative smoking exposure (pack-years) as the strongest independent risk factor for COPD confirms the well-established dose-response relationship between tobacco consumption and airflow obstruction. The risk gradient observed in our study—with ≥40 pack-years conferring an aOR of 3.86 compared to <20 pack-years—aligns closely with the findings of Rennard and Vestbo (2006), who demonstrated that the probability of developing COPD increases progressively with cumulative tobacco exposure, although significant interindividual variability exists [20]. The independent association between early smoking initiation (<16 years) and COPD risk observed in our study supports the concept that tobacco exposure during the critical period of lung growth and maturation may result in suboptimal maximally attained lung function, lowering the threshold for subsequent development of clinically manifest airflow limitation [21].

The significant independent association between occupational dust and chemical exposure and COPD (aOR 2.14) is consistent with a growing body of evidence implicating workplace exposures as important COPD risk factors. The American Thoracic Society has estimated that approximately 15% of COPD cases are attributable to occupational exposures, with additive or synergistic interactions with cigarette smoking amplifying disease risk [22]. Similarly, our finding regarding biomass fuel exposure (aOR 1.78) aligns with data from the PURE study, which demonstrated that household air pollution from solid fuel use was associated with significantly increased COPD risk, particularly with prolonged exposure duration [23]. The association between low BMI (<18.5 kg/m²) and COPD merits careful interpretation. While the cross-sectional design precludes determination of directionality, this finding likely reflects both the systemic consequences of established COPD (cachexia, increased metabolic demand, systemic

inflammation) and potentially a predisposing effect of poor nutritional status on lung vulnerability to noxious exposures. Prospective data from the Copenhagen City Heart Study demonstrated that low BMI was associated with accelerated FEV_1 decline independent of smoking intensity, suggesting a bidirectional relationship [24]. The independent association between childhood respiratory infections and COPD (aOR 1.92) supports the developmental origins hypothesis of chronic lung disease. Svanes et al. (2010) demonstrated that severe childhood respiratory infections were associated with reduced adult lung function and increased COPD risk, even after adjusting for adult smoking behavior [25]. These early-life insults may impair alveolarization and airway development, establishing a trajectory of compromised respiratory reserve that predisposes to earlier onset of clinically significant airflow obstruction.

This study has several limitations. First, the cross-sectional design precludes causal inference. Second, smoking history and occupational exposures relied on self-report, introducing potential recall bias. Third, the use of the fixed FEV_1/FVC ratio <0.70 may overdiagnose COPD in older individuals; however, this remains the GOLD-recommended criterion. Fourth, genetic susceptibility factors (alpha-1 antitrypsin deficiency) and quantitative occupational exposure assessments were not evaluated. Fifth, the exclusion of never-smokers limited our ability to compare risk profiles across smoking categories. Despite these limitations, the multicenter design, standardized spirometric protocol, and comprehensive risk factor assessment strengthen the validity of our findings.

Conclusion

This community-based cross-sectional study reveals that the prevalence of spirometrically confirmed COPD among adult smokers with ≥ 10 pack-years of cumulative exposure is 24.8%, with more than half of affected individuals remaining previously undiagnosed. Cumulative smoking exposure and advancing age represent the strongest independent risk factors, but occupational dust and chemical exposure, biomass fuel use, childhood respiratory infections, low body mass index, early smoking initiation, and prior asthma history are significant additional independent contributors to COPD development.

These findings emphasize the multifactorial etiology of COPD even within smoking populations and highlight the critical importance of implementing targeted spirometric screening programs in primary healthcare settings to identify the substantial reservoir of undiagnosed disease. Comprehensive risk factor assessment extending beyond smoking quantification alone is essential for accurate identification of high-risk individuals. Public health strategies should integrate smoking cessation initiatives with occupational safety regulations, reduction of biomass fuel exposure, and early-life respiratory health promotion to mitigate the growing global burden of COPD.

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