E-ISSN 2250-0944
ISSN 2250-1150
doi: 10.31838/ijprt/16.01.596

Research Article

Epidemiology of Copd in Urban Communities: Prevalence, Determinants, and Public Health Implications

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Received: 27.04.26, Revised: 09.05.26, Accepted: 18.06.26

ABSTRACT

Objective: Bridge the gap between pulmonology and public health to measure the epidemiology and important environmental and behavioral risk factors for COPD and assess the public health implications across diverse urban populations.

Methods: A cross-sectional study was done in a large metropolitan area in three different urban districts: high density commercial, mixed residential-industrial, and peri-urban low-income settlements, all within the community. A multistage stratified cluster random sample of 4500 adults aged ≥40 years was drawn. Independent determinants of COPD were obtained using multivariable logistic regression.

Results: The overall prevalence of COPD was 8.4% (95% CI: 7.6–9.2%). Males were significantly more likely (11.2%) than females (5.8%) to be prevalent ($p<0.001$). Independent risk factors for COPD were current smoking (Adjusted Odds Ratio [aOR] = 4.2, $p<0.001$), prolonged exposure to occupational dust/fume (aOR = 2.8, $p=0.002$), use of indoor biomass fuels (aOR = 2.1, $p=0.015$) and low socioeconomic status (aOR = 1.9, $p=0.021$).

Conclusion: The prevalence of COPD in urban communities is still high and is a result of various traditional risk factors (tobacco) and emerging urban hazards. The results highlight the paramount need for comprehensive public health measures, such as concentrated spirometry screening in primary care and stringent urban environmental measures.

Keywords: Chronic Obstructive Pulmonary Disease (COPD), Urban Epidemiology, Prevalence, Determinants, Public Health, Spirometry, Air Pollution.

INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is the greatest worldwide public health issue, with persistent respiratory symptoms and irreversible, but not fully, reversible chronic airflow limitation. The Global Initiative for Chronic Obstructive Lung Disease (GOLD) 2023 report indicates that COPD is the third leading cause of death globally, causing more than 3 million deaths each year and the significant socioeconomic burden on healthcare systems.[1,2] Although COPD has traditionally been thought of as a disease of rural people who use biomass fuels or high-income country people who have a long history of smoking, the epidemiology of COPD is changing rapidly. In low and middle-income countries (LMICs), the rapid, often unplanned urbanization has led to the development of unique environmental and socioeconomic ecosystems that promote the emergence and spread of chronic respiratory diseases.[3,4] Thus, it is imperative for both the pulmonary and public health communities to understand the

epidemiology of COPD in specific urban communities. COPD risk factors in urban settings can be different and complex. In contrast to rural areas, where exposure to biomass smoke is the major source, urban communities have multiple environmental exposures, including occupational exposures and chronic smoking. Fine particulate matter (PM2.5) and nitrogen dioxide (NO2) have been strongly associated with the rate of lung function decline and incidence of COPD, regardless of smoking status.[5]

In addition, dust, chemical fumes and vapors, often underrecognized, are potent contributors to chronic airflow obstruction which are commonly encountered in the work environment in urban settings, such as manufacturing, construction and informal work. Ironically, peri-urban slums also often using solid biomass fuels for cooking and heating needs even in urban areas where the use of this energy source is related to economic constraints, adding a typical rural risk factor to the data set of the urban epidemiological profile.[6,7] The combination of these environmental exposures and socioeconomic factors of health increases the urban burden of COPD. Noxious exposures are often exacerbated by other physiological stressors such as overcrowding, poor ventilation, and limited access to nutritious food, which are often associated with low socioeconomic status (SES) in urban areas.[8] In addition, the city-healthcare paradox suggests that although the specialized medical facilities are closer to the urban population, there are significant difficulties in accessing primary care, and serious underdiagnosis among the marginalized urban population. It has been estimated that 70% of urban COPD cases are not diagnosed, and that symptoms are often attributed to "smoker's cough," ageing or to other co-morbid cardiovascular disease.[9,10]

The use of simple spirometry and risk factor assessment in primary healthcare are recommended in the World Health Organization (WHO) Package of Essential Noncommunicable Disease (PEN) interventions. But the use of these strategies in the resource poor urban settings is not optimal.[11,12]

The goal of this study is to create evidence-based strategies that can be acted upon to inform urban respiratory health policies and clinical screening protocols, and to connect the disciplines of pulmonology and public health.

MATERIAL AND METHODS

This study was a cross-sectional epidemiological study conducted in the community. The study was carried out in the city of major urban morphology, which was purposefully chosen. The study area was divided into three different urban districts for the assessment of urban heterogeneity: (1) high-density commercial and residential area with high ambient PM2.5 concentration and strong traffic load, (2) mixed residential-industrial area with large number of small-to-medium scale manufacturing industries and construction sites, and (3) peri-urban low-income area with high population density, solid biomass fuel consumption for domestic energy and informal housing. The sampling method used was the multi-stage stratified cluster sampling method to guarantee a representative sample. During the third stage, one eligible adult participant per household was selected with a systematic random sampling method (Kish grid) in the selected households to reduce the selection bias and intra-household correlation. The sample size was determined using the formula for single population proportion. The prevalence (p) of COPD was assumed based on previous epidemiological data from the region, 8.0%. Using a 95% confidence level (Z=1.96), a margin of error of 5% (d=0.05), a design effect of 1.5, the initial sample size was calculated to be 1,728. A non-response rate of 15% and a 10% rate of unacceptable spirometry testing resulted in a sample size of 4,500 participants (1,500 per district) to allow for adequate statistical power for multivariable subgroup analyses. The inclusion criteria were as follows: age ≥40 years; permanent residency in the selected urban district for ≥5 years; and providing written informed consent. Exclusion criteria included: (1) a prior diagnosis of asthma (to exclude a possible diagnostic overlap, though asthma with fixed obstruction was included); (2) presence of an acute respiratory tract infection within the four weeks prior to the study; (3) history of thoracic surgery or conditions that would make it unsafe to carry out spirometry (e.g., recent myocardial infarction, pneumothorax without treatment); and (4) marked cognitive impairment making it difficult to comprehend study procedures and follow commands. A team of trained field workers comprising registered nurses & respiratory therapists was used to collect data for 6 months. Environmental exposures (history of >1 year

of occupational dust, gas or fume exposure; primary cooking fuel type; average hours spent around major roads). History of clinical disease (respiratory symptoms, diagnosis by physician of any comorbidities, health care utilization over the last 12 months). Spirometry Assessment: Spirometry was done before and after the administration of bronchodilator drugs, according to the American Thoracic Society/European Respiratory Society (ATS/ERS) 2021 technical standards. A portable, microprocessor-controlled spirometer (e.g., Micro MedicalMicroSpiro) was used. Once three or more acceptable and reproducible manouevres had been obtained, 400µg of inhaled salbutamol was given with a spacer using a metered-dose inhaler. Spirometry (post-bronchodilator) was repeated 15 minutes after the bronchodilator. The COPD fixed-ratio GOLD criterion of FEV1/FVC < 0.70 was used to confirm the diagnosis of COPD. The severity was defined as post-bronchodilator FEV1 % predicted (GOLD 1-4). Operational Definitions COPD: Post-bronchodilator FEV1/FVC < 0.70. Current Smoker: A person who has smoked 100 cigarettes in his or her lifetime and is now smoking. Occupational Exposure: Self-reported regular exposure at work to dust, chemical fumes or vapors for a period of ≥1 year. Low Socioeconomic Status (SES): Being in the lowest third of the composite measure of household income and education attainment. To guarantee the quality of the data, a detailed 2-week training was conducted for all field staff on assessment of spirometry standards by ATS/ERS and on the administration of the questionnaire. A 3-liter calibration syringe was used for the calibration of the spiroometers daily. A senior pulmonologist was asked to look at 10% of the spirometry curves chosen at random weekly and grade them A, B, C, D or F. Those curves with grades of D or F were not included in the final analysis. To reduce data-entry errors, data were entered into an electronic database, with built-in logical skip patterns and range checks that was secured and protected by a password. SPSS version 28.0 (IBM Corp., Armonk, NY, USA) and version 4.3.0 of R were used to analyze the data. Bivariate analyses were performed to determine the crude association between potential risk factors and COPD status, using the Pearson Chi-square test for categorical variables and independent Student's t-tests or Mann Whitney U tests for continuous variables. A multivariable logistic regression model was used to evaluate bivariate variables with a p-value < 0.20 and known clinical variables (age, sex, BMI) as independent determinants of COPD. Adjusted Odds Ratios (aOR) with 95% Confidence Intervals (CI) were used to present results. Multi-collinearity was checked by the Variance Inflation Factors (VIF), and VIF values < 5 were considered acceptable indicating independence. The final model was judged by a goodness of fit test (Hosmer Lewis test).A two-tailed p-value of < 0.05 was considered statistically significant for all analyses. Missing data accounted for < 3% of the total dataset and were handled using multiple imputations by chained equations (MICE).

RESULTS

The median age of the cohort was 54.3 ± 9.8 years with a nearly equal distribution of males (51.2%) and females (48.8%).

The gradient in prevalence across all the sociodemographic variables was strong and statistically significant for COPD (p < 0.001).

Table 1: Sociodemographic Characteristics and COPD Prevalence

Characteristic	Total N (%)	COPD N (%)	Non-COPD N (%)	p-value
Age Group (years)				
40–49	1,450 (35.2)	68 (4.7)	1,382 (95.3)	<0.001
50–59	1,520 (36.8)	112 (7.4)	1,408 (92.6)	
≥60	1,155 (28.0)	166 (14.4)	989 (85.6)	
Sex				
Male	2,112 (51.2)	236 (11.2)	1,876 (88.8)	<0.001
Female	2,013 (48.8)	110 (5.5)	1,903 (94.5)	
Education Level				
University/College	1,230 (29.8)	65 (5.3)	1,165 (94.7)	<0.001
Secondary School	1,850 (44.8)	132 (7.1)	1,718 (92.9)	
Primary or None	1,045 (25.4)	149 (14.3)	896 (85.7)	
Household Income				

High/Middle Tertile	2,780 (67.4)	182 (6.5)	2,598 (93.5)	<0.001
Low Tertile	1,345 (32.6)	164 (12.2)	1,181 (87.8)	

Table 2: All behavioral and environmental risk factors were significant (p< 0.05) for the prevalence of COPD. The highest crude prevalence of 19.2% was among current smokers followed by former smokers with a crude prevalence of 9.0%.

Table 2: Behavioral and Environmental Risk Factors

Risk Factor	Total N (%)	COPD N (%)	Non-COPD N (%)	p-value
Smoking Status				
Never Smoker	2,350 (57.0)	105 (4.5)	2,245 (95.5)	<0.001
Former Smoker	980 (23.8)	88 (9.0)	892 (91.0)	
Current Smoker	795 (19.2)	153 (19.2)	642 (80.8)	
Occupational Exposure				
No Exposure	2,810 (68.1)	175 (6.2)	2,635 (93.8)	<0.001
Dust/Fume Exposure	1,315 (31.9)	171 (13.0)	1,144 (87.0)	
Primary Cooking Fuel				
Clean Fuel (Gas/Elec)	3,150 (76.4)	220 (7.0)	2,930 (93.0)	<0.001
Biomass/Solid Fuel	975 (23.6)	126 (12.9)	849 (87.1)	
Residence near Major Road (<100m)				
No	2,980 (72.2)	215 (7.2)	2,765 (92.8)	0.003
Yes	1,145 (27.8)	131 (11.4)	1,014 (88.6)	

Table 3: As predicted, participants with COPD had significantly decreased lung function on all spirometric parameters compared to those without COPD (p < 0.001).

Table 3: Spirometric Characteristics of Participants

Spirometric Parameter	Non-COPD (N=3,779)	COPD (N=346)	p-value
Pre-BD FEV1 (L), mean ± SD	2.85 ± 0.62	2.12 ± 0.58	<0.001
Pre-BD FVC (L), mean ± SD	3.65 ± 0.75	3.10 ± 0.70	<0.001
Pre-BD FEV1/FVC (%), mean ± SD	78.2 ± 6.5	61.4 ± 8.2	<0.001
Post-BD FEV1/FVC (%), mean ± SD	79.5 ± 6.1	58.8 ± 7.9	<0.001
Post-BD FEV1 % Predicted, mean ± SD	94.5 ± 12.3	68.2 ± 18.5	<0.001
Significant Reversibility (≥12% & 200ml)	185 (4.9%)	42 (12.1%)	<0.001

Table 4: Several independent factors of COPD were found after controlling for potential confounders (all p < 0.05).

Table 4: Multivariable Logistic Regression Analysis of Determinants of COPD

Variable	Adjusted Odds Ratio (aOR)	95% Confidence Interval	p-value
Age (per 10-year increase)	1.65	1.48 – 1.84	<0.001
Sex (Male vs. Female)	1.82	1.41 – 2.35	<0.001
Smoking Status (Ref: Never)			
Former Smoker	1.95	1.42 – 2.68	<0.001

Current Smoker	4.20	3.15 – 5.60	<0.001
Occupational Exposure (Yes vs. No)	2.80	2.10 – 3.73	0.002
Biomass Fuel Use (Yes vs. No)	2.10	1.55 – 2.85	0.015
Low Socioeconomic Status (Yes vs. No)	1.90	1.45 – 2.49	0.021
Traffic Proximity <100m (Yes vs. No)	1.35	1.05 – 1.74	0.045

Table 5: COPD has a significant public health impact in this urban population. Participants with COPD had significantly higher symptom burden than those who did not have COPD, with moderate-to-severe dyspnea (mMRC ≥ 2) reported by 57.2% of participants with COPD versus 15.6% of participants without COPD (p < 0.001) and high impact on health status (CAT ≥ 10) reported by 70.8% of participants with COPD versus 23.2% of those without COPD (p < 0.001).

Table 5: Public Health Implications: Healthcare Utilization and Symptom Burden

Metric	Non-COPD (N=3,779)	COPD (N=346)	p-value
mMRC Dyspnea Score ≥ 2	412 (10.9%)	198 (57.2%)	<0.001
CAT Score ≥ 10	530 (14.0%)	245 (70.8%)	<0.001
≥1 Severe Exacerbation (Past 12 mos)	185 (4.9%)	118 (34.1%)	<0.001
Emergency Department Visit (Resp.)	220 (5.8%)	95 (27.5%)	<0.00
Hospitalization (Resp.)	85 (2.2%)	52 (15.0%)	<0.001

DISCUSSION

This is a comprehensive cross-sectionalcommunity-based study to give important insights to the epidemiology of COPD in different urban settings. The overall prevalence of COPD was 8.4% among adults aged 40+, which is consistent with, but slightly higher than, recent estimates in similar middle income urban areas.[13,14] The significant symptom burden and extensive health service utilization experienced by those with the disease highlight the importance of the public health implications of these results. The overall prevalence rate in our urban population is in the high end of the range of 7-10% reported in the latest update of the BOLD study which is done on a global scale.[15,16]

The high ratio of males to females (11.2% to 5.8%) reflects smoking rates in past generations, but the decreasing gender disparity in smoking initiation in certain urban populations indicates that the prevalence of female smoking may increase for future generations unless preventive measures are implemented.[17] This age gradient was strong and the prevalence increased more than tripled in individuals aged over 60, which supports the theories of lung function deterioration and the time lag between development of chronic respiratory diseases. There was a near 3-fold higher odds of COPD with occupational exposure to dust and fumes (aOR = 2.80, p=0.002). This discovery further supports recent epidemiological research that identified the "urban industrial phenotype" of COPD, which is exposed to noxious particles for prolonged periods of time without wearing any respiratory protection from the workplace.[18]

Occupational lung diseases are typically not a part of public health policies that focus on ambient air quality. Moreover, the constant importance of the use of biomass fuel for cooking (aOR = 2.10, p=0.015) doubts the binary interpretation of "urban vs. rural" risk groups. For the peri-urban low-income settlements within our study, economic constraints are the driver to using solid fuels for cooking, which effectively brings in a classic rural hazard into the urban context.[19]

The independent association between residential proximity to major roadways and COPD (aOR = 1.35, p=0.045) is further evidence of the association between traffic-related air pollution (TRAP) and chronic respiratory morbidity. Exposure to PM2.5 and NO2 at low levels over long periods of time causes oxidative stress, airway inflammation

and rapid loss of FEV1, even among nonsmokers.[20]

A quarter of the COPD patients (34%) hadsevere exacerbation within the last year, and were 15 times more likely to be hospitalized for the respiratory system than non-COPD patients. This "exacerbation phenotype" contributes to morbidity and mortality associated with COPD and health care utilization costs.[21] Training primary care physicians and nurses in the use of portable spirometry to screen high-risk people (e.g., smokers >40 years, with occupational exposures). Rapid diagnosis enables prompt use of inhaled bronchodilators, smoking cessation advice and vaccination programmes (influenza and pneumococcal) proven to decrease exacerbation rates and enhance quality of life.[22,23]

The broad evaluation of both the traditional and emerging urban risk factors gives a comprehensive picture of the etiology of the disease. But there are some restrictions that should be kept in mind. The cross-sectional design does not allow for the evaluation of temporal order, such that longitudinal cohort studies are needed to establish these associations.

CONCLUSION

This study has shown that COPD is a very common and burdensome disease in urban communities with 8.4% of adults over 40 years of age affected. The causes of urban COPD are complex and include the heavy burden of tobacco smoking, but also include notable, independent effects from occupational exposure and the use of biomass fuels in marginalized settlements, as well as ambient traffic-related air pollution. The burden of symptoms and number of healthcare contacts by those with symptoms point to a significant lack in the current management of urban primary care. A joint response of pulmonology and public health is needed to tackle this epidemic: spirometry screening of patients in primary care should be targeted, with a more stringent application of occupational and environmental air quality laws, and by initiating public health campaigns for universal clean air generation for low-income urban households.

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