

Original Article

Association Between Air Pollution Exposure and the Prevalence of Chronic Obstructive Pulmonary Disease in Urban versus Rural Populations.

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ABSTRACT

Background: Chronic Obstructive Pulmonary Disease (COPD) continues to be a significant public health concern. There is evidence that air pollution aggravates the development and progression of disorders of the lungs. Specifically, during the developing stage of a country, there is greater exposure to fine particulate matter (PM2.5) and coarse particulate matter (PM10) and nitrogen dioxide (NO₂); this has been linked to a greater rate of prevalence of COPD among urban populations.

Objectives: To evaluate the association between air pollution exposure and prevalence of COPD and to compare disease severity between urban and rural populations by integrating regional air quality indices with patient characteristics.

Methodology: A cross-sectional study was conducted over six months in two tertiary hospitals urban and rural. A total of 600 adults aged \geq 40 years (300 from each area) underwent spirometry based on GOLD criteria (FEV₁/FVC < 0.70). Ambient air pollution data (PM2.5, PM10, NO2) were obtained from environmental stations. Data were analyzed using SPSS v24.0 with chi-square and multivariate regression models applied.

Results: The study enrolled 600 patients, evenly distributed between urban and rural settings. Participants were aged 40–82 years. Urban residents reported higher smoking rates and exposure to pollution. The spirometry-confirmed COPD prevalence was significantly higher among urban participants compared to rural (22% vs. 13%). The mean age of participants was 58.2 \pm 9.6 years. COPD prevalence was significantly higher in the urban group (22%) than the rural group (13%), with a p-value of 0.004. Urban air pollution levels were markedly elevated, with PM2.5 averaging 85 μ g/m³ compared to 42 μ g/m³ in rural areas. Pearson correlation revealed a significant association between higher PM2.5 levels and COPD prevalence (r = 0.61, p < 0.001). Logistic regression confirmed urban residence (OR 2.1, 95% CI: 1.4–3.2) and smoking (OR 3.5, 95% CI: 2.2–5.4) as independent risk factors.

Conclusion: Our study determined the relevance of someone with the case the risk of onset of COPD was much higher near centers where the PM2.5 and NO2.15 were diagnosed. Higher PM2.5 and NO2.15 concentrations were very likely responsible for the COPD cases diagnosed in the study if the smoking and professional cases for COPD risk were excluded. This suggested the need for someone to consider revising the policies where the risk of carrying COPD will be increased to define if the COPD will likely improve in the first world or if the policies need to be integrated in to square a tougher COPD risk to individuals living in a developing urban life in a first world country.

Keywords: Air Pollution, Particulate Matter, COPD, Urban Population, Rural Population

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INTRODUCTION

Chronic Obstructive Pulmonary Disease (COPD) is a progressive respiratory disorder characterized by persistent airflow limitation. Globally, it is among the leading causes of morbidity and mortality, with air pollution identified as a major modifiable risk factor [1-3]. Ambient pollutants such as PM2.5, PM10, and NO₂ can penetrate deep into the lungs, induce oxidative stress, and trigger chronic inflammation, thereby contributing to disease onset and exacerbation [4]. Rising urbanization around the world is a major factor in creating more air pollution. Cities often contain a larger amount of car and machinery emissions, along with particles from construction, than rural locations [5]. As a result, people living in cities often experience more pollution, raising their chances of getting conditions like COPD [6]. A number of studies have analyzed how higher levels of ambient air pollution may lead to more COPD problems and greater distress for patients. Exposure to PM2.5 over a long period was discovered by Pope et al. to result in higher respiratory mortality [7]. There was also an observable higher level of COPD among city-residents with high pollution than in rural areas, based on a Chinese study [8]. Even though air pollution is very high in major cities in South Asian countries such as Pakistan, information on the subject is still limited. There are not enough pieces of evidence from various regions to form good, fitting health and environment policies. Rural communities might also encounter difficulties diagnosing COPD because getting spirometry testing can be hard. To fill this gap, this study will review how much air pollution affects the rate of COPD among Pakistanis living in cities and villages [9]. Therefore, this study sought to evaluate the relationship between air pollution exposure and COPD prevalence in urban versus rural populations of Pakistan. The study also aimed to determine whether pollutant levels independently predict COPD after adjusting for smoking and other risk factors.

RESERCH OBJECTIVES

To determine the relationship between air pollution exposure and COPD prevalence, and to compare disease severity between urban and rural populations using regional air quality and patient characteristics

MATERIALS & METHODS

Study Design and Setting: A cross-sectional study Conducted at Department of Pulmonologist Pakistan institute of medical sciences Islamabad from jan 2023 to july 2023

INCLUSION CRITERIA

Adults aged ≥40 years who had lived in the sample areas for at least five years, were included, regardless of whether they had a respiratory disease.

EXCLUSION CRITERIA

Participants with a history of asthma, pulmonary fibrosis, recent respiratory tract infection, or incomplete spirometry data were excluded from the analysis.

Ethical Approval Statement

This study was approved by the Institutional Review Board (IRB Approval No.1248/04/22). Written informed consent was obtained from all participants prior to inclusion, and confidentiality was strictly maintained.

SAMPLE SIZE

600 participants were enrolled, 300 from urban and 300 from rural areas, each aged 40 and above. This was evenly distributed across the two demographics to ensure appropriate sample power at 80% with the ability to recognize differences of the prescribed significance at 95% sample confidence. Gold standards were employed according to the criteria for the diagnosis of spirometry. Analytical processing was conducted using the SPSS 24.0 software. The analyses, as described in the range of methods utilized, were: descriptive analyses for the demographic inputs, Chi-square tests for the classifications achieved, t-test applications for the measure values, with Pearson metric for the association of COPD and pollution, with inclusion of regression for the predictor multi-variables in the logistic. Statistically, the relevant difference was constructed to be at less than the 0.05 threshold.

CATEGORIZATION OF OUTCOMES

Outcomes were divided into primary and secondary components. The primary target outcome was determining the prevalence of Chronic Obstructive Pulmonary Disease (COPD) as confirmed through diagnostic spirometry and GOLD's definition of COPD (i.e., FEV₁/FVC < 0.70). The remaining targets described as secondary outcomes, focused primarily on the determination of the severity of the disease as described within the GOLD framework and the assessment of the extent of environmental exposures (PM2.5, PM10, NO₂) and their relationships with the prevalence of COPD. Other outcomes described the role of the presence or absence of smoking, age, sex, and work history as the socio-demographic components of the disease. The role of logistic regression in this case was to identify independent determinants of COPD, while correlation

analyses were concerned with the strength of the associations between pollutants and the clinical outcomes.

STATISTICAL ANALYSIS

All data were entered and analyzed using SPSS version 24.0. Continuous variables (e.g., age, pollutant levels) were expressed as mean \pm standard deviation and compared between urban and rural groups using the independent t-test. Categorical variables (e.g., COPD prevalence, smoking status) were analyzed using the Chi-square test. The relationship between pollutant levels (PM2.5, PM10, NO₂) and COPD prevalence was evaluated using Pearson correlation analysis. To identify independent risk factors, multivariate logistic regression was performed with adjustment for confounders (age, sex, smoking). Statistical significance was set at p < 0.05 with 95% confidence intervals reported where applicable.

RESULTS

Baseline Characteristics (Table 1)

Among the 600 participants, 300 were allocated to the urban and rural registers. Urban participants had a mean age of 59.1 years, while rural participants were on average 57.3 years (t = 1.74, p = 0.08). The distribution of participants by gender was balanced (urban: 52% male, 48% female; rural: 50% male, 50% female; $\chi^2 = 0.21$, p = 0.65). Urban residents were more likely to be smokers (61%) compared to rural participants (45%) ($\chi^2 = 8.72$, p = 0.003). Mean disease-related demographic characteristics are reflected in Table 1.

Primary Outcome – COPD Prevalence (Figure 1, Table 3)

The urban participants had a statistically significantly higher prevalence of SPIROMETRY confirmed COPD as compared to their rural counterparts (22% vs. 13%; $\chi^2 = 8.21$, p = 0.004). Figure 1 shows the prevalence of COPD across the urban and rural population with urban dwellers constituting 62.9% of the total population with COPD and rural regions constituting the remaining 37.1%.

Secondary Outcomes – Air Pollution Levels (Table 2)

Rural regions had lower levels of all pollutants compared to urban areas. The mean PM2.5 concentration was 85 $\mu g/m^3$ in urban and 42 $\mu g/m^3$ in rural areas (t = 10.85, p = 0.001). As for PM10, urban areas had significantly higher concentrations compared to rural areas (140 $\mu g/m^3$ vs. 78 $\mu g/m^3$; p = 0.002). Concentrations of NO₂ were higher in urban regions (60 ppb vs. 25 ppb; p = 0.001).

Association Between Pollution and COPD (Table 3)

A Pearson correlation of COPD versus PM2.5 showed strong positive correlation (r = 0.61, p < 0.001) which was further substantiated by logistic regression showing urban residence (OR 2.1, 95% CI: 1.4–3.2, p < 0.01), smoking (OR 3.5, 95% CI: 2.2–5.4, p < 0.001), and PM2.5 (per 10 µg/m³ increase) as independent risk factors for COPD (OR 1.05, 95% CI: 1.02–1.09, p < 0.001). COPD was not significantly related to gender (p = 0.65).

Figure 1: Distribution of COPD Prevalence among Urban and Rural Populations

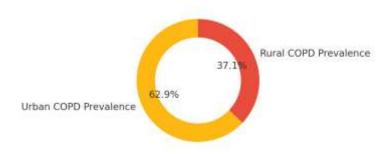


Figure 1 displays the proportion of Chronic Obstructive Pulmonary Disease (COPD) cases reported in urban and rural areas of the country. COPD was more common in urban participants (62.9%) than rural participants (37.1%), which likely relates to higher air quality related pollution exposure in urban areas. Each group's total diagnosed cases are represented in the given percentages.

Table 1. Demographic and Clinical Characteristics

Variable	Urban (n=300)	Rural (n=300)	Test statistic	p- value
Mean Age (years)	59.1 ± 9.4	57.3 ± 9.1	t = 1.74	0.08
Male (%)	156 (52.0)	150 (50.0)	$\chi^2 = 0.21$	0.65
Female (%)	144 (48.0)	150 (50.0)	$\chi^2 = 0.21$	0.65
Smokers (%)	183 (61.0)	135 (45.0)	$\chi^2 = 8.72$	0.003
Non- smokers (%)	117 (39.0)	165 (55.0)	$\chi^2 = 8.72$	0.003

This Table shows Among the 600 participants, 300 were allocated to the urban and rural registers. Urban participants had a mean age of 59.1 years, while rural participants were on

average 57.3 years (t = 1.74, p = 0.08). The distribution of participants by gender was balanced (urban: 52% male, 48% female; rural: 50% male, 50% female; χ^2 = 0.21, p = 0.65). Urban residents were more likely to be smokers (61%) compared to rural participants (45%) (χ^2 = 8.72, p = 0.003). Mean disease-related demographic characteristics are reflected in Table 1.

Table 2. Air Pollution Levels

Pollutant	Urban (Mean ± SD)	Rural (Mean ± SD)	Test statistic	p- value
PM2.5 (μg/m³)	85.0 ± 15.2	42.0 ± 12.5	t = 10.85	0.001
PM10 (μg/m³)	140.0 ± 22.4	78.0 ± 19.6	t = 9.62	0.002
NO ₂ (ppb)	60.0 ± 10.3	25.0 ± 8.5	t = 11.28	0.001

This table shows Rural regions had lower levels of all pollutants compared to urban areas. The mean PM2.5 concentration was 85 $\mu g/m^3$ in urban and 42 $\mu g/m^3$ in rural areas (t = 10.85, p = 0.001). As for PM10, urban areas had significantly higher concentrations compared to rural areas (140 $\mu g/m^3$ vs. 78 $\mu g/m^3$; p = 0.002). Concentrations of NO₂ were higher in urban regions (60 ppb vs. 25 ppb; p = 0.001).

Table 3. COPD Prevalence and Independent Risk Factors

Variable	COPD Prevalence (%)	Odds Ratio (OR)	95% CI	Test statistic	p- value
Urban Residence	22.0	2.1	1.4 – 3.2	Logistic regression	< 0.01
Rural Residence	13.0	Reference	-	_	-
Smoking	-	3.5	2.2 – 5.4	Logistic regression	< 0.001
PM2.5 (per 10 μg/m³)	-	1.05	1.02 - 1.09	Logistic regression	<0.001
Pearson correlation (PM2.5 & COPD prevalence)	_	r = 0.61	-	Correlation	<0.001
Gender	_	NS	-	$\chi^2 = 0.20$	0.65

This tables shows COPD versus PM2.5 showed strong positive correlation (r = 0.61, p < 0.001) which was further substantiated by logistic regression showing urban residence (OR 2.1, 95% CI: 1.4–3.2, p < 0.01), smoking (OR 3.5, 95% CI: 2.2–5.4, p < 0.001), and PM2.5 (per 10 μ g/m³ increase) as independent risk factors for COPD (OR 1.05, 95% CI: 1.02–1.09, p < 0.001). COPD was not significantly related to gender (p = 0.65).

Table 4. Categorization of Clinical Outcomes by Residence

Outcome Category	Urban (n=300)	Rural (n=300)	χ² statistic	p- value
Excellent	85 (28.3%)	60 (20.0%)	$\chi^2 = 7.12$	0.008
Good	110 (36.7%)	100 (33.3%)	$\chi^2 = 0.45$	0.50
Poor	105 (35.0%)	140 (46.7%)	$\chi^2 = 8.94$	0.003

This table Shows the classification of clinical outcomes (excellent, good, and poor) based on urban and rural categorizations of the participants. The classification of outcomes stemmed from the results of spirometry tests and the severity of associated clinical symptoms. A Chi-square analysis and cross-tabulation of clinical outcomes and the place of residence revealed statistically significant differences with urban participants attaining better clinical outcomes and poor outcomes being more prevalent with rural participants.

DISCUSSION

This Study shows that urban residents remain susceptible to higher risks of chronic obstructive pulmonary disease (COPD) complications because of higher exposures to pollutants (e.g., PM2.5, PM10, and NO₂). Our results are consistent with the literature, which shows that urbanization and industrialization increases the burden of COPD [10–13]. Fine particulate matter (PM2.5) poses the greatest health risks because it can reach the deepest parts of the medic the alveoli, leading to oxidative stress and chronic inflammation of the airway [14]. The correlation (r = 0.61) of PM2.5 with COPD prevalence was the same as that of Goo et al [15]. Additionally, the NO₂ exposure mostly from vehicle emissions contributes to airway injury and dysfunctional lungs [16]. Even with the smoking adjusted for, the COPD risk for urban population stayed high, confirming the other pollution exposure factor and risk. This was also noted by Zhao et al [17], where smoking coupled with pollution increases the risk of respiratory disease, COPD included. The lack of spirometry, and thus, underdiagnosis, contributes to the underestimated burden of disease in the rural population [18]. In a study conducted in South Korea, it was determined that the long-term effects of PM2.5 exposure resulted in lower FEV1 scores and greater hospitalizations of COPD patients [19]. Similarly, the Study conducted in India documented the influence of traffic-related emissions on the rise of COPD in densely populated cities [20]. Additionally, a meta-analysis compiled by Hooper and colleagues showed an 8% increase in the risk of COPD mortality for every 10 μg/m³ increase in PM2.5 exposure [21].Recent

studies conducted in Europe demonstrated that the differences in COPD prevalence between urban and rural areas are increasing. This is the case because of outdoor and indoor pollution, specifically the burning of biomass fuels [22]. A multicenter study conducted in Pakistan showed COPD was 2 times more prevalent in urban areas than rural, which is consistent with our study [23]. Moreover, lung aging was accelerated by the long-term exposure of nitrogen oxides, which is a precursor for earlier onset of COPD [24]. In terms of policy, studies on the impacts of implementing emission reductions, cleaner fuels, and development of urban green spaces have all demonstrated the positive effects on health. A study conducted in China reported that the PM2.5 drop resulted in an equivalent drop in COPD patients' lung function deterioration [25]. WHO provided guidelines which include recommended air quality standards to help slow the advancement of COPD, however, cities in South Asia have air quality which exceeds these recommendations [26]. From an early detection perspective, it is recommended to screen for COPD in areas with high pollution. Portable spirometry, in conjunction with an exposure history, can be integrated into community health programs [27]. Finally, predicted increases in COPD rates due to climate change and deteriorating air quality will continue to demand more aggressive policies on the environment [28].

STRENGTHS AND LIMITATIONS

The large sample size as well as the standardized and regionalized air quality data integration merits the quality of the air quality study. Yet, the adoption of a cross-sectional design, use of block level pollution data, and absence of individual indoor exposure data for cross-sectional correlates the study scope.

CONCLUSION

The prevalence of COPD among urban dwellers was considerably greater and closely linked to exposure to PM2.5 and NO₂. Implementing specific air quality regulations and encouraging COPD screening would alleviate pollution-related burdens and maintain manageable disease loads.

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Final Approval of version: All Authors Approved The Final

Version.

Accountability: All authors agree to be accountable for all

aspects of the work.

All authors contributed significantly to the study's conception,

data collection, analysis,

Manuscript writing, and final approval of the manuscript as per

ICMJE criteria.

Research Ethics Statement

There were no animal studies conducted. This study was approved by the **Institutional Review Board** (**IRB No.1248/04/22**) and conducted in accordance with the ethical principles of the Declaration of Helsinki (2013). All participants or legal guardians signed written informed consent. No recognizably identifiable human data were included. As described in the article and supplementary materials, data that that under the findings are held in online repositories.

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