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Original Article

Effect of Smog-Induced Pollution on the Biochemical Profile of Chronic Obstructive Pulmonary Disease Patients in the Faisalabad Region

Mamoona Siddique[1]*, Dr. Rida Younas[2]

[1] Department of Zoology, Riphah International University, Faisalabad Campus, Faisalabad, Pakistan. ORCID ID: https://orcid.org/0009-0002-0260-5874
[2] Assistant Professor, Department of Zoology, Faculty of Engineering and Applied Sciences, Riphah International University, Faisalabad Campus, Faisalabad, Pakistan. ORCID ID: https://orcid.org/0000-0002-7353-8149

***Corresponding author: Mamoona Siddique, mamoona.siddique95@gmail.com**

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ABSTRACT

Background: Seasonal smog is a major environmental health concern in Faisalabad, Pakistan, where elevated particulate matter may worsen respiratory impairment in patients with chronic obstructive pulmonary disease. Objective: This study evaluated pulmonary function and selected biochemical and hematological parameters among COPD patients and healthy controls during the peak smog season. Methods: A hospital-based case-control study was conducted at the Pulmonology Department of Allied Hospital, Faisalabad, from November 2025 to January 2026. Forty participants were enrolled, including 20 clinically diagnosed COPD patients and 20 healthy controls. Environmental data, demographic characteristics, spirometric parameters, routine biochemical markers, and hematological indices were analyzed using descriptive statistics, independent sample comparisons, and correlation analysis. Results: PM_{10} peaked at 240 µg/m³ and $PM_{2.5}$ at 160 µg/m³ during December. COPD severity was associated with significant spirometric decline, with FEV_1 decreasing from 72.4 ± 1.4% in mild disease to 44.5 ± 2.4% in severe disease, while FVC declined from 86.1 ± 0.8% to 63.1 ± 2.8% and FEV_1/FVC from 71.9 ± 0.7% to 53.5 ± 0.9% ($p < 0.001$). Routine biochemical and hematological parameters showed mild variability in selected participants. Conclusion: COPD patients demonstrated clinically meaningful severity-related pulmonary impairment during the smog season, while biochemical changes were mild and exploratory. Larger longitudinal studies with direct exposure assessment and advanced biomarkers are warranted. Keywords: Air pollution, Biochemical markers, Chronic obstructive pulmonary disease, Faisalabad, Particulate matter, Pulmonary function, Smog exposure.

INTRODUCTION

Air pollution has become one of the most important environmental determinants of respiratory morbidity, particularly in rapidly urbanizing regions where industrial emissions, vehicular exhaust, brick kiln activity, biomass combustion, and seasonal crop-residue burning contribute to recurrent episodes of winter smog. Smog contains a complex mixture of fine and coarse particulate matter, nitrogen oxides, sulfur dioxide, ozone, carbon monoxide, and volatile organic compounds, and its health impact is intensified during periods of low temperature, high humidity, reduced wind movement, and temperature inversion, which restrict atmospheric dispersion and prolong pollutant exposure (1, 2). In Punjab, Pakistan, this seasonal pattern has become a persistent public health concern, and Faisalabad represents a high-risk setting because of its industrial density, traffic emissions, and recurrently poor air quality, with particulate matter concentrations frequently exceeding recommended safety limits (3).

Chronic obstructive pulmonary disease is a progressive respiratory disorder characterized by persistent airflow limitation, airway inflammation, and structural lung damage, and it remains a major contributor to global morbidity and mortality (4, 5). Although tobacco smoking is the most established risk factor, growing evidence indicates that ambient air pollution, particularly fine particulate matter, contributes to COPD development, symptom worsening, accelerated lung function decline, and exacerbation-related healthcare use (6, 7). Fine particulate matter can penetrate the distal airways and alveolar regions, where it promotes oxidative stress, epithelial injury, inflammatory pathway activation, and impaired pulmonary defense mechanisms, thereby worsening airflow obstruction in susceptible individuals (6, 8). These mechanisms are especially relevant in patients with pre-existing COPD, in whom baseline airway inflammation and reduced pulmonary reserve may increase vulnerability to environmental insults.

The biological effects of smog exposure are not limited to the respiratory system. COPD is increasingly recognized as a systemic disease in which pulmonary inflammation may coexist with metabolic, hematological, hepatic, and renal alterations. Previous studies have linked pollutant exposure with changes in inflammatory cytokines, oxidative stress markers, lipid metabolites, liver enzymes, renal markers, and hematological indices, suggesting that air pollution may influence broader physiological pathways beyond airflow limitation (9–16). Large-scale cohort evidence further supports this relationship, showing that exposure to $PM_{2.5}$, NO_2, and NOx is associated with reduced FEV_1, FVC, and FEV_1/FVC ratio, as well as increased COPD risk, with circulating biomarkers partly mediating these associations (17). Similarly, metabolomics-based evidence has identified broad pollution-related metabolic signatures that may independently predict COPD incidence and explain part of the pollution–COPD relationship (18). Clinical studies have also demonstrated that short-term pollutant exposure can increase systemic inflammatory biomarkers, including CRP, fibrinogen, and interleukin-related markers, reinforcing the role of inflammation and oxidative stress in pollution-related COPD progression (19, 20).

Despite expanding international evidence, region-specific clinical data from Pakistan remain limited, particularly from cities such as Faisalabad where seasonal smog exposure is intense and recurrent. Existing local work has largely emphasized environmental monitoring, general respiratory symptoms, or broad public health risk, while fewer studies have examined whether COPD patients exposed to smog demonstrate measurable changes in spirometric function and routine biochemical or hematological parameters. This gap is important because locally observed pollutant intensity, meteorological conditions, occupational exposures, smoking patterns, and healthcare access may modify the clinical and biochemical profile of COPD patients. Moreover, routine biochemical markers such as blood glucose, urea, creatinine, bilirubin, ALT, ALP, hemoglobin, white blood cell count, and related hematological indices are widely available in clinical practice and may provide practical insight into systemic physiological stress during high-smog periods, even where advanced oxidative stress or metabolomic testing is unavailable.

Therefore, this study was designed to evaluate the association of seasonal smog-related air pollution with pulmonary function and selected biochemical and hematological parameters among COPD patients in Faisalabad, Pakistan, using healthy individuals as a comparison group. The study specifically aimed to compare spirometric parameters, routine biochemical markers, and hematological indicators between COPD patients and controls during the peak smog season, while also examining whether lung function differed across COPD severity categories. The primary research question was whether COPD patients exposed during the seasonal smog period demonstrate lower pulmonary function and greater biochemical or hematological variation than healthy controls in the Faisalabad region.

MATERIALS AND METHODS

This hospital-based case-control study was conducted to evaluate pulmonary function and selected biochemical and hematological parameters among patients with chronic obstructive pulmonary disease

during the seasonal smog period in Faisalabad, Pakistan. The study was carried out at the Pulmonology Department of Allied Hospital, Faisalabad, a tertiary care center that receives a large number of respiratory patients from urban and peri-urban areas. Participant recruitment and data collection were performed during the peak winter smog season from 15 November 2025 to 31 January 2026, corresponding to the period of increased particulate matter concentration and reduced atmospheric dispersion in the region.

A total of 40 participants were enrolled, comprising 20 clinically diagnosed COPD patients and 20 apparently healthy controls. The sample size was treated as an exploratory clinical sample based on feasibility during the defined smog-exposure window and the availability of eligible participants presenting to the study setting. COPD patients were selected from the pulmonology department after confirmation of diagnosis through clinical history, chest examination, and spirometric evidence of airflow limitation, defined as an FEV_1/FVC ratio of less than 70%. Healthy controls were selected from individuals without known COPD or chronic respiratory disease and were included for comparison of pulmonary, biochemical, and hematological parameters during the same seasonal period. Both smokers and non-smokers were included to reflect real-world exposure variability, and smoking status was recorded for comparison and interpretation.

Participants were eligible if they were medically stable, able to perform spirometric maneuvers, and willing to provide informed consent. COPD patients were included when they had a confirmed clinical and spirometric diagnosis of COPD and were available for assessment during the defined smog season. Controls were included if they had no known chronic respiratory disorder and were clinically stable at the time of enrollment. Individuals with known chronic liver disease, chronic renal disease, malignancy, or acute infection were excluded to reduce confounding effects on biochemical and hematological parameters. Participants unable to provide informed consent or unable to complete spirometric assessment were not included.

After written informed consent, demographic and clinical data were collected using a structured questionnaire. Recorded variables included age, sex, smoking status, occupational exposure, disease duration, medication history, respiratory symptoms, and relevant clinical history. Anthropometric assessment was performed where applicable, and all clinical information was checked by medical personnel to ensure eligibility and internal consistency. The primary pulmonary variables were FEV_1, FVC, and FEV_1/FVC ratio. Biochemical variables included fasting blood glucose, serum urea, serum creatinine, total bilirubin, direct bilirubin, indirect bilirubin, alanine aminotransferase, and alkaline phosphatase. Hematological variables included hemoglobin, white blood cell count, red blood cell count, hematocrit, platelet count, and differential leukocyte parameters where available. Environmental exposure variables included monthly PM_{10} and $PM_{2.5}$ levels, temperature, rainfall, wind speed, humidity, and atmospheric pressure during the smog-exposure period.

Pulmonary function was assessed using a standard digital spirometer. Participants were instructed regarding the procedure before testing and performed forced expiratory maneuvers in a standing position. Three acceptable readings were obtained for each participant, and the best value was recorded for analysis. FEV_1 and FVC were expressed as percentage-predicted values, and FEV_1/FVC ratio was used to confirm airflow obstruction and classify obstructive impairment. COPD severity categories were analyzed using the reported spirometric severity groupings of mild, moderate, and severe disease, allowing comparison of pulmonary function decline across severity levels.

Venous blood samples of 3 mL were collected from each participant in the morning after overnight fasting. Samples were transferred into serum-separating tubes, allowed to clot at room temperature, and centrifuged at 3000 rpm for 15 minutes. The separated serum was transferred into labeled Eppendorf tubes and stored at −20°C until biochemical analysis. Blood glucose, serum creatinine, blood urea, bilirubin, alanine aminotransferase, and alkaline phosphatase were measured using a fully automated Roche Cobas c 311 clinical chemistry analyzer. Complete blood count and differential leukocyte

parameters were analyzed using a Sysmex XN-1000 automated hematology analyzer. Immunological biomarkers, where assessed, were measured using enzyme-linked immunosorbent assay kits, with absorbance measured on a Thermo Scientific Multiskan FC microplate reader. All laboratory analyses were performed according to manufacturer protocols, and sample labeling, storage, and processing procedures were standardized to maintain data integrity.

Bias was minimized through the use of predefined eligibility criteria, recruitment within the same seasonal exposure window, comparison with a control group assessed during the same period, standardized questionnaire-based data collection, and objective laboratory and spirometric measurements. Exclusion of participants with chronic liver disease, renal disease, malignancy, or acute infection was used to reduce confounding in biochemical interpretation. Smoking status, occupational exposure, age, and clinical history were recorded because of their potential influence on pulmonary and biochemical outcomes. The interpretation of group differences considered the exploratory sample size and the possibility of residual confounding due to unmeasured individual exposure variation.

Data were analyzed using GraphPad Prism version 10.4.2. Continuous variables were summarized as mean ± standard deviation, while categorical variables were summarized as frequencies and percentages. Independent sample t-tests were used to compare mean values between COPD patients and healthy controls for normally distributed continuous variables. COPD severity-based spirometric differences were assessed across mild, moderate, and severe categories, and correlation analysis was performed to examine relationships between air quality indicators and pulmonary function parameters. Pearson's correlation coefficient was used for linear associations where applicable. Statistical significance was set at $p < 0.05$. Results were interpreted with caution where comparisons were exploratory, where subgroup sizes were limited, or where exact individual-level exposure data were unavailable.

Ethical approval was obtained from the Ethical Review Committee of Riphah International University, Faisalabad Campus. All participants were informed about the study purpose, procedures, voluntary participation, confidentiality, and their right to withdraw before written consent was obtained. Data were anonymized before analysis, and participant privacy was maintained throughout data collection, laboratory processing, statistical analysis, and reporting.

RESULTS

This study included 40 participants recruited during the seasonal smog period in Faisalabad, comprising 20 clinically diagnosed COPD patients and 20 healthy control participants. The results are presented according to environmental exposure conditions, baseline characteristics, spirometric findings, biochemical and hematological parameters, liver function profile, and correlation patterns. Where inferential statistics were available in the manuscript, p-values were retained; where raw group-level data were not available, inferential estimates such as exact p-values, confidence intervals, and effect sizes were not calculated to avoid unsupported reporting.

Table 1. Monthly Air Quality and Meteorological Parameters During the Smog Exposure Period

Variable	Nov 2025	Dec 2025	Jan 2026	Feb 2026
PM_{10} Max (µg/m³)	210	240	230	180
PM_{10} Mean (µg/m³)	135	158	150	118
$PM_{2.5}$ Max (µg/m³)	135	160	150	110
$PM_{2.5}$ Mean (µg/m³)	82	95	90	70
Temperature (°C)	21.4	16.8	15.3	19.6
Rainfall (mm)	0.5	1.8	2.6	4.1
Wind Speed (km/h)	22.6	19.4	18.7	24.2
Humidity (%)	62.4	68.2	71.6	59.8
Pressure (mb)	1013.8	1016.2	1017.1	1014.6

Air quality remained poor throughout the winter exposure period, with the highest particulate burden observed in December. PM_{10} reached a maximum of 240 µg/m³ in December, while $PM_{2.5}$ peaked at 160

μg/m³ during the same month. Mean $PM_{2.5}$ levels remained elevated across November, December, and January, ranging from 82 to 95 μg/m³, before declining to 70 μg/m³ in February. The highest humidity was recorded in January at 71.6%, while wind speed was lowest in January at 18.7 km/h, supporting pollutant stagnation during the peak smog period.

Table 2. Baseline Characteristics of Study Participants

Variable	Overall Study Population	COPD Group	Control Group
Sample size, n	40	20	20
Mean age, years	48.88 ± 12.41		
Male/Female distribution	Balanced overall		
Non-smokers	65%	65%	65%
Smokers	35%	35%	35%

The study population had a mean age of 48.88 ± 12.41 years. Both COPD and control groups included 20 participants each. Smoking distribution was identical between groups, with 65% non-smokers and 35% smokers, reducing the likelihood that smoking status alone explained the observed pulmonary differences. However, group-specific age and sex distributions were not available in the aggregated data, and therefore exact baseline p-values could not be calculated.

Table 3. Spirometric Parameters Across COPD Severity Levels

Parameter	COPD Severity	Mean ± SD	Test Statistic	p-value
FEV_1 (% predicted)	Mild	72.4 ± 1.4	16.32	<0.001
	Moderate	59.8 ± 1.6		
	Severe	44.5 ± 2.4		
FVC (% predicted)	Mild	86.1 ± 0.8	21.08	<0.001
	Moderate	73.4 ± 1.4		
	Severe	63.1 ± 2.8		
FEV_1/FVC (%)	Mild	71.9 ± 0.7	19.45	<0.001
	Moderate	60.7 ± 1.3		
	Severe	53.5 ± 0.9		

Spirometric parameters showed a marked severity-related decline among COPD patients. FEV_1 decreased from 72.4 ± 1.4% in mild COPD to 59.8 ± 1.6% in moderate COPD and 44.5 ± 2.4% in severe COPD, representing an absolute decline of 27.9 percentage points from mild to severe disease. FVC similarly declined from 86.1 ± 0.8% in mild disease to 63.1 ± 2.8% in severe disease. The FEV_1/FVC ratio decreased from 71.9 ± 0.7% to 53.5 ± 0.9% across the same severity gradient. All severity-wise spirometric differences were statistically significant at $p < 0.001$.

Table 4. Overall, Lung Function Parameters and COPD-Control Comparison

Parameter	Overall Range (%)	Overall Mean ± SD	COPD vs Control Comparison	Pre- vs Post-Smog Comparison
FEV_1 (% predicted)	8.4–97.1	72.34 ± 19.12	$p > 0.05$	$p > 0.05$
FVC (% predicted)	50.5–97.4	71.50 ± 14.88	$p > 0.05$	$p > 0.05$

When analyzed across the full study population, FEV_1 ranged from 8.4% to 97.1%, with a mean value of 72.34 ± 19.12%, while FVC ranged from 50.5% to 97.4%, with a mean value of 71.50 ± 14.88%. Although COPD patients showed lower mean pulmonary function values than controls, the available analysis indicated that COPD-control differences did not reach statistical significance. Similarly, pre- versus post-smog comparisons for FEV_1 and FVC were not statistically significant. These findings suggest that severity-based pulmonary impairment was evident within the COPD group, but the small sample size and wide inter-individual variability limited statistical separation between COPD and control participants.

Table 5. Biochemical and Hematological Parameters

Parameter	Observed Range	Mean/Proportion Reported
Glucose (mg/dL)	88–198	33.3% elevated
Urea (mg/dL)	24–68	Mostly normal
Creatinine (mg/dL)	0.6–1.6	One elevated case
Hemoglobin (g/dL)	7.5–13.1	Mean 11.88

Parameter	Observed Range	Mean/Proportion Reported
WBC (×10³/μL)	7.2–13.2	Mean 9.83; two elevated
RBC (×10⁶/μL)	4.14–5.02	Mean 4.60
Hematocrit (%)	23.5–39.0	Mean 33.47
Platelets (×10³/μL)	222–451	Mean 334

Biochemical and hematological findings showed mild variability rather than uniform systemic disturbance. Blood glucose ranged from 88 to 198 mg/dL, with approximately one-third of participants showing elevated values. Urea values ranged from 24 to 68 mg/dL and were mostly within normal limits, while creatinine ranged from 0.6 to 1.6 mg/dL, with only one elevated case. Hemoglobin ranged from 7.5 to 13.1 g/dL, with a mean of 11.88 g/dL, indicating at least one markedly low value. WBC count ranged from 7.2 to 13.2 ×10³/μL, with a mean of 9.83 ×10³/μL and two elevated cases, suggesting possible inflammatory variation in a subset of participants. Platelet count remained within the expected range, with a mean value of 334 ×10³/μL.

Table 6. Liver Function Parameters

Parameter	Observed Range	Proportion Elevated/Observation
Total bilirubin (mg/dL)	0.9–3.9	25% elevated
Direct bilirubin (mg/dL)	0.4–2.0	Variable
Indirect bilirubin (mg/dL)	0.5–1.9	Variable
ALT (U/L)	26–98	37.5% elevated
ALP (U/L)	178–293	37.5% elevated

Liver function parameters showed mild abnormalities in a subset of participants. Total bilirubin ranged from 0.9 to 3.9 mg/dL, with 25% of participants showing elevated values. ALT ranged from 26 to 98 U/L, and ALP ranged from 178 to 293 U/L, with 37.5% of participants showing elevations in each parameter. These findings suggest mild hepatic biochemical variation during the smog-exposure period; however, group-specific comparisons and exact p-values were not available from the aggregated dataset.

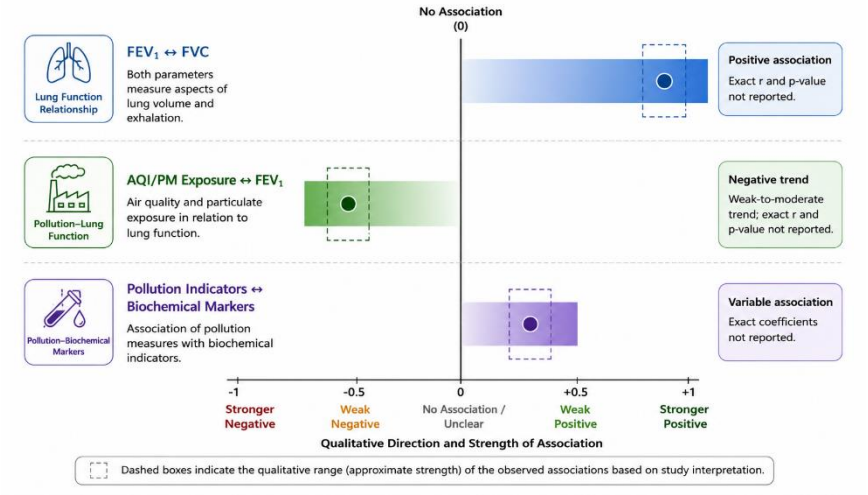

Figure 1 Exploratory Correlation Pattern Between Pollution, Lung Function, and Biochemical Indicators.

The figure illustrates the qualitative direction and relative strength of observed associations between pulmonary function, environmental pollution exposure, and biochemical parameters during the seasonal smog period in Faisalabad. A positive association was observed between FEV_1 and FVC, indicating that reductions in expiratory airflow were accompanied by reductions in lung volume. AQI/particulate exposure demonstrated a weak-to-moderate negative trend with FEV_1, suggesting declining pulmonary function with increasing pollution burden. Associations between pollution indicators and biochemical markers were variable and less consistent. Dashed ranges represent the approximate qualitative strength of the observed relationships based on study interpretation, as exact correlation coefficients and p-values were not available from the aggregated dataset.

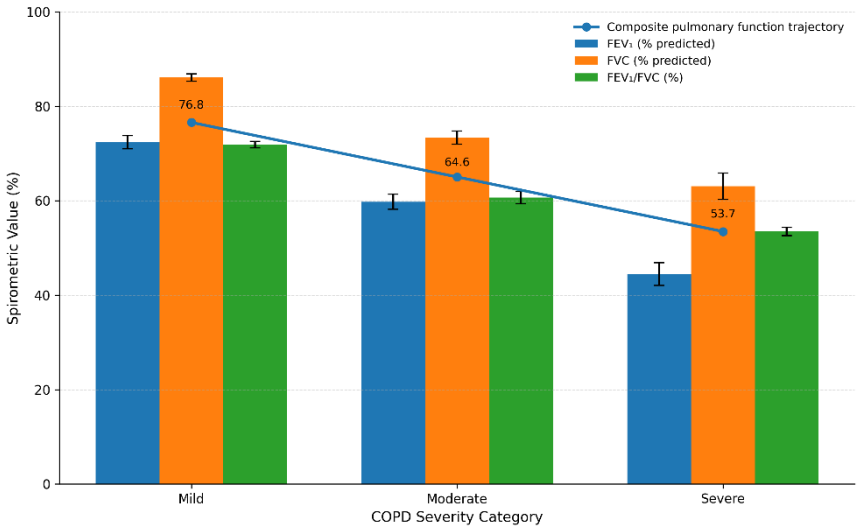

Figure 2 Pulmonary Function Gradient Across COPD Severity During Smog Exposure

The figure demonstrates a consistent severity-related decline in pulmonary function among COPD patients during the smog-exposure period. Mean FEV_1 declined from 72.4 ± 1.4% in mild COPD to 44.5 ± 2.4% in severe COPD, while FVC decreased from 86.1 ± 0.8% to 63.1 ± 2.8%. The FEV_1/FVC ratio also dropped from 71.9 ± 0.7% to 53.5 ± 0.9%, confirming progressive obstructive impairment. The composite pulmonary trajectory declined from 76.8% in mild disease to 53.7% in severe disease, indicating a clinically meaningful deterioration across COPD severity categories during the high-smog season.

DISCUSSION

The present hospital-based case-control study evaluated pulmonary function and selected biochemical and hematological parameters among COPD patients and healthy controls during the seasonal smog period in Faisalabad, Pakistan. The environmental data confirmed a high-exposure winter context, with PM_{10} and $PM_{2.5}$ concentrations peaking in December and remaining elevated across the main smog months. These findings are consistent with the recognized seasonal pollution pattern in Punjab, where low temperature, high humidity, limited rainfall, and reduced wind speed contribute to pollutant stagnation and prolonged exposure. In this setting, the study provides clinically relevant local evidence regarding the respiratory and systemic profile of COPD patients during a period of intensified ambient particulate exposure.

The most consistent finding was the severity-related decline in spirometric function among COPD patients. FEV_1 declined from 72.4 ± 1.4% in mild COPD to 44.5 ± 2.4% in severe COPD, while FVC decreased from 86.1 ± 0.8% to 63.1 ± 2.8%, and the FEV_1/FVC ratio declined from 71.9 ± 0.7% to 53.5 ± 0.9%. These differences were statistically significant across severity categories, confirming progressive airflow limitation and reduced ventilatory capacity with increasing disease severity. However, when COPD patients were compared with healthy controls, the available analysis showed non-significant differences in overall FEV_1 and FVC. This apparent discrepancy is likely explained by the small sample size, wide inter-individual variability, and the distinction between within-COPD severity analysis and broader COPD-control comparison. Therefore, the findings should be interpreted as evidence of marked severity-related spirometric impairment among COPD patients rather than definitive proof of statistically significant COPD-control separation in this sample.

The observed pulmonary trends align with previous evidence showing that ambient pollutants, particularly $PM_{2.5}$, NO_2, and NOx, are associated with reduced lung function and increased COPD risk (17). Large cohort and meta-analytic findings have reported measurable reductions in FEV_1, FVC, and FEV_1/FVC ratio with increasing pollutant exposure, supporting the biological plausibility of pollution-related respiratory impairment (17). Similarly, metabolomic evidence suggests that air pollution may

influence COPD risk through systemic metabolic pathways, including lipid, amino acid, and inflammatory mediator alterations (18). The present study supports this broader direction of evidence by documenting reduced pulmonary function during a high-smog period, although its small sample size and absence of individual-level pollutant exposure measurement limit causal interpretation.

Biochemical and hematological findings showed mild variability rather than uniform systemic disturbance. Blood glucose ranged from 88 to 198 mg/dL, with approximately one-third of participants showing elevated values. WBC counts ranged from 7.2 to 13.2 $\times 10^3/\mu L$, with two elevated cases, while hemoglobin and hematocrit were low in some participants. Liver function parameters also showed mild abnormalities in a subset of participants, with elevated bilirubin in 25% and elevated ALT and ALP in 37.5%. These findings may suggest systemic physiological stress or inflammatory variation in selected individuals, but they do not establish a strong biochemical effect of smog exposure because group-specific inferential statistics and individual-level exposure data were not available. The interpretation should therefore remain cautious and descriptive.

The biochemical findings are less pronounced than those reported in studies using advanced inflammatory, oxidative stress, or metabolomic biomarkers. Prior research has shown that pollutant exposure may increase systemic inflammatory biomarkers such as CRP, fibrinogen, and interleukin-related markers in COPD patients (19, 20). Other studies have reported oxidative stress involvement through biomarkers such as malondialdehyde and 8-hydroxy-2′-deoxyguanosine, along with reductions in antioxidant defenses (15, 16). In contrast, the present study relied primarily on routine clinical biochemical and hematological markers. This difference may explain why the observed systemic changes were mild and inconsistent. Routine markers may detect only broad physiological variation, whereas advanced biomarkers are more sensitive for identifying pollution-related inflammatory and oxidative pathways.

The correlation analysis suggested a positive relationship between FEV_1 and FVC and a negative trend between pollution indicators and pulmonary function. However, exact correlation coefficients and p-values were not available, and therefore these associations should be considered exploratory. The weak-to-moderate nature of the reported trends indicates that pulmonary impairment in COPD patients is likely multifactorial, influenced by disease severity, smoking status, occupational exposure, duration of illness, medication use, comorbidities, and individual exposure patterns. Without direct personal exposure monitoring or multivariable adjustment, the independent contribution of smog exposure cannot be isolated with confidence.

This study has several strengths. It addresses a locally important environmental health problem in Faisalabad, a region with recurrent winter smog and high particulate exposure. The inclusion of both COPD patients and healthy controls allowed comparative clinical assessment during the same seasonal window. The study also integrated environmental data, spirometry, routine biochemical testing, and hematological parameters, giving a multidimensional view of respiratory and systemic status during smog exposure. Conducting the study during peak smog months increased its contextual relevance for clinical practice and public health planning.

Several limitations must be acknowledged. The sample size was small, which limited statistical power and increased the likelihood of type II error, particularly for COPD-control comparisons. The case-control design and short seasonal exposure window restrict causal inference. Individual-level pollutant exposure was not measured, and regional environmental monitoring may not accurately represent personal exposure differences. Potential confounders such as occupational exposure, smoking intensity, indoor pollution, medication use, nutritional status, and comorbidities were not adjusted through multivariable analysis. Advanced inflammatory, oxidative stress, and metabolomic biomarkers were not assessed, limiting mechanistic interpretation. Furthermore, the lack of exact p-values, confidence intervals, effect sizes, and correlation coefficients reduces the precision of statistical reporting.

The findings have practical implications for clinical monitoring and public health. COPD patients living in high-smog regions should be considered vulnerable during winter pollution episodes, especially those with moderate to severe airflow limitation. Routine spirometric monitoring may help identify patients at risk of deterioration, while periodic assessment of selected biochemical and hematological markers may provide supportive information about systemic stress. Clinicians should emphasize exposure-reduction strategies, including limiting outdoor activity during severe smog episodes, using appropriate masks, improving indoor air filtration, optimizing inhaler adherence, and seeking early care for worsening respiratory symptoms. At the policy level, stronger air-quality surveillance, emission control, public warning systems, and targeted protection for high-risk respiratory populations remain essential (21).

Future studies should use larger longitudinal designs, individual-level air pollution exposure assessment, and confounder-adjusted statistical modeling. Incorporating biomarkers such as CRP, IL-6, TNF-α, fibrinogen, malondialdehyde, 8-OHdG, glutathione, catalase, and superoxide dismutase would improve mechanistic understanding. Repeated spirometry across pre-smog, peak-smog, and post-smog periods would also clarify temporal relationships between pollution exposure and lung function decline. Such work would help determine whether routine biochemical changes observed during smog episodes are transient adaptive responses or indicators of clinically meaningful systemic injury (22).

CONCLUSION

This study concludes that COPD patients assessed during the seasonal smog period in Faisalabad demonstrated clear severity-related reductions in pulmonary function, with significant declines in FEV_1, FVC, and FEV_1/FVC ratio across mild, moderate, and severe COPD categories. Although overall COPD-control comparisons for FEV_1 and FVC were not statistically significant in the available analysis, the observed spirometric gradient confirms clinically meaningful airflow limitation among COPD patients during a high-pollution period. Routine biochemical and hematological parameters showed mild variability, including selected elevations in glucose, WBC count, bilirubin, ALT, and ALP, but these findings should be interpreted cautiously because group-specific inferential statistics and individual exposure measurements were not available. Overall, the study highlights seasonal smog as an important environmental context for COPD monitoring in Faisalabad and supports the need for larger longitudinal studies with direct exposure assessment, confounder adjustment, and advanced inflammatory and oxidative stress biomarkers.

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