

Original Article



Effect of Ambient Air Pollutants on Acute changes in Pulmonary Variables and Inflammatory Markers in Healthy Adults: A Pilot Study

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Abstract

OBJECTIVE: Air pollution is associated with adverse health effects, particularly on respiratory and cardiovascular systems. Smog, prevalent in Northern India, contains particulate matter (PM₁₀, PM_{2.5}) and gaseous pollutants that can impair pulmonary function. Ambient air pollution can be quantified by the air quality index (AQI). Understanding the acute effects of air quality on respiratory physiology and inflammation is essential.

MATERIAL AND METHODS: A pilot prospective longitudinal observational study evaluated healthy volunteers during high (HAQI, AQI >350) and low pollution (LAQI, AQI <150) phases. Spirometry and impulse oscillometry (IOS) assessed lung function; and biomarkers (interleukin-6, tumor necrosis factor alpha) were measured from exhaled breath condensate (EBC) and serum. Paired t-tests or Wilcoxon signed-rank tests were used for analysis.

RESULTS: A total of 21 participants (mean age 25.4±7.4 years, female 33.3%, body mass index 23.9±3.5 kg/m²) completed measurement at both HAQI (395.7±43.8) and LAQI (85.4±9.7). Spirometry revealed significantly lower slow vital capacity (SVC%, 81.6±11.2 vs. 87.7±7.7%, *P* = 0.008) and forced vital capacity (FVC%, 86.2±9.8 vs. 90.9±9.4%, *P* = 0.0005) under HAQI compared to LAQI. Forced expiratory volume at 1 second (FEV₁) was also reduced (*P* < 0.0001), while FEV₁/FVC remained unchanged. IOS showed higher airway resistance (R_s, R₂₀) during HAQI (*P* < 0.0001). Inflammatory biomarkers in serum and EBC showed no statistical differences between HAQI and LAQI. Despite measurable differences, spirometry and IOS parameters remained within normal limits.

CONCLUSION: Acute air pollution exposure impairs lung function and increases airway resistance in healthy adults. These findings underscore the need for larger longitudinal studies to clarify the mechanisms linking acute air pollution exposure to chronic health outcomes.

KEYWORDS: Air pollution, spirometry, impulse oscillometry, particulate matter, inflammatory markers

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INTRODUCTION

Air pollution has been associated with detrimental health effects and reduced life expectancy.¹ The composition of air pollutants exhibits marked regional and temporal variation with differential health effects. Smog is a particularly detrimental type of air pollution, prevalent in Northern India due to a combination of emissions from vehicles, industries, and crop residue burning, which undergo photochemical reactions in sunlight. During winter, temperature inversion in this part of the country traps these pollutants near the surface, leading to dense, persistent smog.² Smog contains high concentrations of small particulate matter, i.e., PM_{2.5} (≤2.5 µm diameter) and PM₁₀ (≤10 µm diameter). PM_{2.5} and PM₁₀ are known to have significant negative effects on human health.³ These particles, although small, have a relatively large surface area that facilitates the attachment of toxic substances. Due to their small size, they can evade

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nasal filtration, penetrate deep into the respiratory system, and reach the alveoli. Once deposited in the lungs, $PM_{2.5}$ and PM_{10} can induce local inflammation and may even enter the bloodstream, causing systemic adverse effects.⁴ Apart from particulate matter, ozone (O_3), sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), and lead are the main contaminants of outdoor air.

The Central Pollution Control Board's website (Ministry of Environment, Forestry and Climate Change, India) provides real-time access to the air quality index (AQI).⁵ The AQI ranges from 0–500 and is calculated from the concentration of 7 pollutants: PM_{10} , $PM_{2.5}$, NO_2 , O_3 , CO, SO_2 , ammonia (NH_3). There are 6 categories of AQI: good, satisfactory, moderately polluted, poor, very poor, and severe. AQI is the generally accepted method for monitoring air pollution in India.

Acute exposure to air pollutants might result in respiratory symptoms, cardiovascular problems, hospital admissions, and even death.⁶ Further, lung cancer, atherosclerosis, chronic bronchitis, and increased mortality have all been connected to prolonged exposure to air pollution.⁷ Understanding the acute effects of air pollution on the respiratory system physiology is also important for comprehending the pathophysiological mechanisms of chronic detrimental effects.

In this study, we assessed the acute effects of changes in ambient air quality (measured by AQI) on lung volumes, capacities, airway mechanics and inflammatory biomarkers in North India.

MATERIAL AND METHODS

This was a prospective longitudinal observational study aimed at generating pilot data. Healthy volunteers aged 18–40 years who were never smokers, with no history of chronic lung diseases, thoracic surgery, cardiovascular and systemic diseases, and with no acute upper respiratory diathesis (runny nose, cough, sore throat, etc.) or fever, were recruited through convenience sampling at a tertiary care center in New Delhi, India. Given the exploratory nature of the study, a formal sample size and power calculation was not performed. However, we aimed to maximize enrollment to achieve the highest possible power within real-world resource limitations.

The AQI is the air quality metric determined by the Central Pollution Control Board of India, calculated the highest indexed value among seven ambient pollutants: PM_{10} , $PM_{2.5}$, NO_2 , O_3 , CO, SO_2 , and NH_3 .⁵ Their official AQI calculator and its

recommended interpretation are available in the Supplementary Material 1. Briefly, AQI values of 0–50 are classified as “good” (minimal impact), 51–100 as “satisfactory” (minor breathing discomfort), 101–200 as “moderate” (discomfort for sensitive individuals), 201–300 as “poor” (discomfort with prolonged exposure), 301–400 as “very poor” (potential respiratory illness with prolonged exposure), and values above 400 as “severe” (respiratory effects even in healthy individuals).

For each volunteer, comprehensive evaluations of respiratory parameters were made in two phases: an initial high pollution phase (HAQI) and a later low pollution phase (LAQI). Phase definitions were determined based on typical local AQI trends in New Delhi in 2018, the year preceding the study, to ensure adequate contrast between high and low AQI exposures while maintaining sufficient sampling windows. In 2018, the mean AQI during winter months (November–January) was 341 ± 59 , during the rainy season (July–September) it was 109 ± 42 , and during the remaining months it was 231 ± 70 . Therefore, HAQI was defined as an average daily AQI of >350 for at least 7 consecutive days, and LAQI as an average daily AQI of <150 for at least 7 consecutive days. Written informed consent was obtained from all participants at the time of enrollment.

Spirometry was used to measure lung volumes and capacities, and impulse oscillometry (IOS) was used to estimate respiratory impedance at two time points, i.e., HAQI and LAQI. Exhaled breath condensate (EBC) and blood were collected to measure airway and systemic inflammation by estimating biomarkers, including tumor necrosis factor alpha (TNF- α) and interleukin-6 (IL-6), at both time points.

The study was approved by the All-India Institute of Medical Sciences, New Delhi's Ethics Committee for human subjects (ref. no.: IEC-622/6.09.2019, RP-15/2019).

Assessment of Lung Volumes and Capacities by Spirometry (Spiro'air Pulmonary Function Test System-Medisoft)

Spirometry was performed as per the guidelines of the American Thoracic Society and the European Respiratory Society (ERS).⁸ Of the three acceptable and repeatable slow vital capacity (SVC) and forced vital capacity (FVC) maneuvers, the highest values were considered. The parameters recorded were SVC, FVC, forced expiratory volume in the first second (FEV_1), FEV_1 /FVC ratio, and peak expiratory flow (PEF).

Assessment of Respiratory Impedance by Impulse Oscillometry (MS-IOS Digital JAEGER System)

IOS, is a simple, non-invasive technique that mainly uses forced oscillations, to evaluate the mechanics of the lungs or airways. The participant's effort is minimal when this method is compared to spirometry, to evaluate lung function. Sound waves of different frequencies ranging between 5Hz and 35Hz are superimposed over normal tidal breathing, and impedance is measured by the ratio of pressure signal to flow signal. Sound waves of smaller frequencies (<15 Hz) can reach the alveoli, but higher frequencies lose their strength in the central airways. The procedure was explained to the participants, and while the participants were in a sitting position, the sound waves were

Main Points

- Acute exposure to air pollution can cause measurable worsening of respiratory function and airway parameters in healthy subjects.
- Higher air quality index (AQI) (worse air pollution) is associated with lower slow vital capacity and forced expiratory volume at 1 second.
- Higher AQI is associated with increased respiratory impedance (R_5 and R_{20}) as measured on the impulse oscillometry.

superimposed onto normal tidal breathing for 90 seconds. A tight seal between lips and mouthpiece was ensured. The cheeks were held firmly by the participant with their hands. The parameters recorded were airway impedance (Z), airway resistance at 5Hz and 20Hz (R_5 , R_{20}), and airway reactance at 5Hz and 20Hz (X_5 , X_{20}). The other oscillometry indices taken into consideration were peripheral airway resistance (R_5 - R_{20}), resonant frequency, and an area under reactance.⁹

Assessment of inflammatory biomarkers using EBC and enzyme-linked immunosorbent assay (ELISA). EBC was performed according to ERS guidelines.¹⁰ EBC was collected using an R-tube (Respiratory Research, Inc., USA). R-tube is a disposable collection system that consists of a large Tee section made of polypropylene, which separates saliva from the exhaled breath, a one-way valve (made of silicone rubber), and a polypropylene collection tube, which is cooled by a cooling aluminium sleeve placed around it. Subjects were instructed to breathe tidally by inhaling through the nose and exhaling through the mouthpiece connected to the R-tube for 10 minutes. Approximately 1.5 mL of the condensate was collected and immediately stored at -20 °C. Separated serum from peripheral blood samples was also stored at -20 °C. Human ELISA kits of Boster Biotech, USA (Cat No: EK0525, EK0410) were used to quantify serum levels of TNF- α and IL-6, respectively. According to the manufacturer's instructions, ELISA was performed, and a microplate reader (BioTek, EpochTM 2 microplate reader) was used to read the colour that developed in the 96-well plates. The detection range was 15.6-1000 pg/mL for TNF- α (sensitivity 1 pg/mL) and 4.69-300 pg/mL for IL-6 (sensitivity 0.3 pg/mL).

Statistical Analysis

GraphPad Prism 9.0.1 for Windows (GraphPad Software, Inc., USA) was used for analysis. Each parameter was tested for distribution of the data based on standard normality tests (the D'Agostino-Pearson normality test, the Anderson-Darling test, the Shapiro-Wilk test). Based on the normality of the data, group comparisons were carried out using the paired t-test or

the Wilcoxon signed-rank test along with the appropriate post-hoc comparison tests. The level of statistical significance was set at $P < 0.05$. Bonferroni corrections were applied within each test domain (spirometry, IOS, and biomarkers) to adjust the significance thresholds for multiple comparisons.

RESULTS

A total of 21 participants (mean age 25.4 ± 7.4 years, female 33.3%, body mass index 23.9 ± 3.5 kg/m²) who completed recordings at both time points was considered for analysis. The mean AQI was 395.7 ± 43.8 during HAQI and 85.4 ± 9.7 during LAQI phases ($P = 0.0001$). Detailed distribution of pollutants during the two phases is available in Table 1.

Spirometry Parameters

SVC_(%pred) was lower in HAQI (81.6 ± 11.2) compared to LAQI (87.7 ± 7.7 , $P = 0.0008$). Similarly, FVC_(%pred) was lower in HAQI (86.2 ± 9.8 vs. 90.9 ± 9.4 , $P = 0.0005$). While FEV_{1(%pred)} was lower during HAQI [83 (75-89) vs. 89 (86-94), $P < 0.0001$], FEV₁/FVC was similar between both groups (95.0 ± 11.2 vs. 96.4 ± 10.0 , $P = 0.6$). Results of spirometry are summarized in Table 2.

IOS Parameters

The airway resistance was higher in HAQI compared to LAQI, as measured by R_5 (%pred) [104.0 (94.2-123.2) vs. 75.0 (68.4-92.8), $P < 0.0001$] and R_{20} (%pred) [108.7 (100.0-122.6) vs. 77.3 (69.5-89.6), $P < 0.0001$]. The reactance [X_5 (%pred) and X_{20} (%pred)] was similar in both groups ($P = 0.3$ and $P = 0.4$), although absolute values of X_5 were more negative in HAQI [-0.11 (-0.16-(-0.09)) vs. -0.07 (-0.09-(-0.05)), $P = 0.001$]. The results of the IOS parameters are summarized in Table 3.

Inflammatory Biomarkers

There was no difference in the serum and EBC levels of TNF- α ($P = 0.1$ and $P = 0.4$) and IL-6 ($P = 0.3$ and $P = 0.5$) between the two groups. These results are summarized in Table 4.

Table 1. Air pollution parameters at two different time-points

Parameters	High Pollution (AQI >350 for 7 consecutive days)	Low pollution (AQI <150 for 7 consecutive days)	P value
AQI	395.7 ± 43.8	85.4 ± 9.7	<0.0001
PM _{2.5}	394.6 ± 43.9	54.0 ± 17.0	<0.0001
PM ₁₀	369.6 ± 55.7	64.7 ± 19.4	<0.0001
NO ₂	51 (49-58)	39 (26-41)	0.02
NH ₃	6.6 ± 1.3	23.1 ± 0.7	<0.0001
SO ₂	14.0 ± 1.2	16.4 ± 2.7	0.07
CO	55.4 ± 10.0	64.6 ± 34.8	0.5
O ₃	19.6 ± 1.7	35.1 ± 9.9	0.01

Values presented are mean $\pm$ standard deviation or median (minimum-maximum), analyzed by Paired t-test (two-tailed) or Wilcoxon signed-rank test (two-tailed), respectively

AQI: air quality index, PM_{2.5}: particulate matter 2.5, PM₁₀: particulate matter 10, NO₂: nitrogen dioxide, NH₃: ammonia, SO₂: sulfur dioxide, CO: carbon monoxide, O₃: ozone

Table 2. Spirometry parameters at two different time-points

Spirometry parameters	High pollution (AQI >350 for 7 consecutive days)	Low pollution (AQI <150 for 7 consecutive days)	P value*
SVC (%predicted)	81.6±11.2	87.7±7.7	0.0008
FVC (%predicted)	86.2±9.8	90.9±9.4	0.0005
FEV ₁ (%predicted)	83 (75-89)	89 (86-94)	<0.0001
FEV ₁ /FVC	95.0±11.2	96.4±10.0	0.6
MMEF (%predicted)	75.5±23.1	88.0±18.5	0.003
PEF (%predicted)	83.2±24.1	95.5±17.8	0.003

*The Bonferroni-corrected significance threshold was set at $P < 0.0083$ (6 comparisons)

Values presented are mean ± standard deviation or median (minimum-maximum), analyzed by Paired t-test (two-tailed) or Wilcoxon signed-rank test (two-tailed) respectively

AQI: air quality index, SVC: slow vital capacity, FVC: forced vital capacity, FEV₁: forced expiratory volume at 1st second, MMEF: maximum mid expiratory flow, PEF: peak expiratory flow

Table 3. Impulse oscillometry parameters at two different time-points

Impulse oscillometry parameters	High pollution (AQI >350 for 7 consecutive days)	Low pollution (AQI <150 for 7 consecutive days)	P value*
R ₅ [kPa/(L/s)]	0.27 [0.25-0.39]	0.22 [0.18-0.29]	<0.0001
R ₅ (%predicted)	104.0 [94.2-123.2]	75.0 [68.4-92.8]	<0.0001
R ₂₀ [kPa/(L/s)]	0.25 [0.23-0.34]	0.18 [0.15-0.23]	<0.0001
R ₂₀ (%predicted)	108.7 [100.0-122.6]	77.3 [69.5-89.6]	<0.0001
R ₅ -R ₂₀ [kPa/(L/s)]	0.04 [0.02-0.05]	0.03 [0.02-0.05]	0.09
X ₅ [kPa/(L/s)]	-0.11 [-0.16-(-0.09)]	-0.07 [-0.09-(-0.05)]	0.001
X ₅ (%predicted)	-333.3 [-491.7- 412.5]	-200.0 [-308.3-395.8]	0.2
X ₂₀ [kPa/(L/s)]	.06 [-0.01- 0.09]	0.04 [0.01-0.07]	0.2
X ₂₀ (%predicted)	66.7 [-11.7-100.0]	57.1 [18.3-88.9]	0.4
Ax [kPa/L]	0.30 [0.21-0.90]	0.28 [0.17-0.56]	0.03
Fres [1/sec]	13.8 [10.7-18.7]	15.1 [11.3-18.4]	0.8

*The Bonferroni-corrected significance threshold was set at $P < 0.0045$ (11 comparisons)

Values presented are mean ± standard deviation or median (minimum-maximum), analyzed by Paired t-test (two-tailed) or Wilcoxon signed-rank test (two-tailed) respectively

AQI: air quality index, R₅: resistance at 5Hz, R₂₀: resistance at 20 Hz, R₅-R₂₀: peripheral airway resistance, X₅: reactance at 5Hz, X₂₀: reactance at 20Hz, Ax: area under reactance, Fres: resonant frequency

Table 4. Inflammatory biomarker parameters at two different time-points

Inflammatory biomarkers	High pollution (AQI >350 for 7 consecutive days)	Low pollution (AQI <150 for 7 consecutive days)	P value*
TNF-α in EBC (pg/mL)	6.4±1.7	7.2±0.6	0.1
TNF-α in serum (pg/mL)	4.6 (0.8-7.5)	4.71 (0.0-6.5)	0.4
IL-6 in EBC (pg/mL)	0.3 (0.1-0.7)	0.3 (0.2-0.5)	0.3
IL-6 in serum (pg/mL)	0.5 (0.2-1.1)	0.4 (0.0-1.6)	0.5

*The Bonferroni-corrected significance threshold was set at $P < 0.0125$ (4 comparisons)

Values presented are mean ± standard deviation or median (minimum-maximum), analyzed by Paired t-test (two-tailed) or Wilcoxon signed-rank test (two-tailed), respectively

AQI: air quality index, TNF-α: tumor necrosis factor-alpha, IL-6: interleukin 6, EBC: exhaled breath condensate

DISCUSSION

To the best of our knowledge, this is the first study to prospectively evaluate comprehensive respiratory parameters using spirometry, IOS, and inflammatory markers in healthy volunteers regarding short-term changes in air pollution. Despite a small sample size for a pilot study, we found statistically significant differences associated with higher air pollution, including lower lung capacities, lower expiratory volume, and higher airway resistance. It is important to note, however, that these values remained within the normal prescribed limits. There was no statistically significant difference in the inflammatory markers noted in our study. Overall, higher ambient air pollution was associated with deleterious effects on clinically measurable pulmonary and airway parameters even over relatively short exposure periods in healthy individuals.

Our spirometry results are supported by conclusions from previous studies. In a 2019 systematic review, Edington et al. reported that short-term exposure to higher $PM_{2.5}$ level was associated with a statistically significant decrease in FEV_1 .¹¹ In a retrospective cohort study in Belgium, Int Panis et al.¹² reported that FVC, FEV_1 , and PEF were negatively correlated with PM_{10} concentrations on the day of examination. In a study on asthmatic patients in Thailand, Chujit et al.¹³ found that PEF was most affected by NO_2 levels 2 days before and PM_{10} levels 6 days before spirometry, proposing that this lag is required for these pollutants to affect the airway.

While SVC and FVC are generally expected to be similar in healthy individuals, a reduction in FVC and FEV_1 with a preserved SVC may serve as a sensitive marker of small airway limitation. Therefore, both parameters were measured and reported. In our study, however, the mean SVC% was lower than the mean FVC%, likely reflecting a measurement artifact. Given the more relaxed nature of the SVC manoeuvre, suboptimal respiratory effort may occur despite extensive participant coaching, particularly when compared to the more forceful and dynamic effort required during the FVC manoeuvre.

Although there is less literature exploring the acute impact of air pollution on IOS parameters, previous studies provide a theoretical framework for understanding the increased resistance observed during HAQI. In a cross-sectional study, De¹⁴, observed that long-term exposure to high levels of ambient air pollution during early life is associated with increased respiratory impedance in children, compared to those with lower pollution levels. This finding shows that ambient air pollution affects small airway resistance and elastic characteristics but not proximal airway resistance. Additionally, children who live in more polluted areas had a greater absolute magnitude of X_5 (more negative), suggesting that exposure to ambient air pollution may alter the respiratory system's elastic properties or increase ventilation heterogeneity.¹⁴ In a retrospective study of chronic obstructive pulmonary disease (COPD) patients, Zhu et al.¹⁵ found that short-term exposure to higher $PM_{2.5}$ was associated with worsening of IOS parameters, including respiratory impedance and reactance.

Friedman et al.¹⁶ measured spirometry and IOS parameters among area workers and residents, who had breathed dust and fumes from the World Trade Centre tragedy in 2001. Among

these participants, cases, i.e., those who developed lower respiratory symptoms, were more likely to have abnormal spirometry and increased respiratory impedance compared to controls, which persisted 7-8 years after exposure. They concluded that these abnormalities were primarily associated with dysfunction in the peripheral airways.

We observed that HAQI and LAQI had comparable levels of serum IL-6 and TNF- α in both EBC and serum. This is likely an artifact arising from our small sample size. Robust longitudinal measurements by Sabeti et al.¹⁷ in Iran show that ambient $PM_{2.5}$ and metal concentrations in places with high pollution levels is positively associated with increased TNF- α but negatively correlated with IL-6. A study on healthy Swiss adults found that serum levels of IL-6 and TNF- α were positively associated with short-term exposure to PM_{10} . These findings provide strong support for the notion that short-term exposure to PM_{10} is probably sufficient to induce systemic inflammation. Nevertheless, it is plausible that the very short duration of exposure to high pollution in our study (7 days), within a setting characterized by year-round elevated pollution levels, may have limited our ability to detect appreciable changes in inflammatory markers. Furthermore, unmeasured factors such as occult infections and other environmental exposures could have influenced baseline inflammatory status, potentially obscuring subtle differences.

Acute exposure to air pollutants can significantly impair respiratory function through several pathophysiological mechanisms. Inhaled pollutants, including particulate matter ($PM_{2.5}$ and PM_{10}), O_3 , NO_2 , and heavy metals, can irritate the respiratory mucosa, triggering oxidative stress and inflammation.¹⁸⁻²⁰ This leads to increased production of inflammatory cytokines, recruitment of immune cells, and disruption of epithelial barriers in the airways and gaseous exchange surfaces.^{21,22} Further, this can cause bronchoconstriction, airway edema, and reduced mucociliary clearance, as evidenced by exacerbations of conditions like asthma and COPD.²³ Our study demonstrates, even in apparently healthy individuals, these processes can cause small yet measurable adverse effects on airway mechanics and pulmonary function. Further research is needed to elucidate the exact mechanisms by which this acute inflammatory reaction to air pollutants translates to long-term respiratory complications and other detrimental health effects.

While our results demonstrate a correlation between higher pollution levels and worse respiratory parameters, it is important to note that we did not adjust for other environmental confounders, including ambient temperature and humidity. Since the HAQI phase was predominantly recorded during winter months, cold temperatures may have influenced the spirometry and IOS measurements. Although cold dry air is generally known to induce bronchoconstriction, the effect of the winter season on spirometry parameters is complex. Recent studies suggest a paradoxical linear increase in FEV_1 and FVC with colder temperatures,²⁴ likely driven by factors such as reduced outdoor allergen exposure during winter months. The impact of seasonal variation in temperature and humidity on IOS parameters remains unclear. Future analyses should adjust for these covariates to better isolate the independent effect of ambient air pollution.

Interestingly, ambient levels of NH_3 , SO_2 , CO , and O_3 during the HAQI phase were similar to or paradoxically lower than those during the LAQI phase compared to the LAQI phase, despite substantially higher $\text{PM}_{2.5}$, PM_{10} , and AQI. This trend may reflect chemical conversion of these gases into particulate matter or other compounds, leading to artificially low readings during the HAQI period.²⁵ Temperature, humidity, and local wind patterns may also contribute. Nevertheless, it is important to note that despite lower gas levels, the elevated particulate matter and smog, during HAQI periods, result in worse overall air quality.

Study Limitations

This study should be interpreted in the context of several limitations. The use of a small convenience sample introduces potential selection bias and increases the risk of type II errors. Airway measurements may have been influenced by environmental confounders, beyond pollution, including ambient temperature, humidity, air conditioning, and circulating seasonal viruses. Participant-level factors such as time since last meal, recent exercise, stress, sleep, and caffeine intake were also not accounted for. Additionally, we did not record information regarding time spent indoors versus outdoors and recent travel history, which could impact cumulative exposure to air pollution. We have also not used any instrument to measure the volunteers' personal exposure to air pollution. Given the relatively young age of our study population, these findings may not be generalizable to older individuals. Finally, we were unable to evaluate a dose-response relationship as only two measurements were made for each participant.

CONCLUSION

Acute increase in air pollution adversely affects spirometry parameters and respiratory impedance in healthy adults. Larger longitudinal studies are needed to elucidate the effect of air pollution on local and systemic inflammation, as well as their link to chronic health outcomes.

Ethics

Ethics Committee Approval: The study was approved by the All-India Institute of Medical Sciences, New Delhi's Ethics Committee for human subjects (ref. no.: IEC-622/6.09.2019, RP-15/2019).

Informed Consent: Written informed consent was obtained from all participants at the time of enrollment.

Footnotes

Authorship Contributions

Surgical and Medical Practices: Surgical and Medical Practices: G.B., A.T., Concept: G.B., A.T., Design: G.B., A.G., A.T., Data Collection or Processing: A.G., L.M., S.G., K.S., Analysis or Interpretation: K.S., K.N.J., G.B., A.T., Literature Search: K.S., A.G., K.N.J., L.M., S.G., G.B., A.T., Writing: A.G., K.S., G.B.

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