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NO₂ exposure and lung function decline in a cohort of adults in Mysore, India

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Abstract

Increasing air pollution in low- and middle-income countries (LMICs) may be contributing to worsening respiratory health, yet to date most relevant studies have been conducted in industrialized nations. Particularly, there are few studies for India, the world’s second most populated country, and on this country’s poorest populations, who may be at the highest risk. We investigated the influence of long-term nitrogen dioxide (NO₂) exposure at residential location on lung function decline over a 5-year period in a cohort of low-income adults in Mysore, Karnataka, India. In 2012–2014 and in 2017–2018, we conducted standardized interviews and performed in-home field spirometry before and after bronchodilation. We estimated annual average NO₂ in 2017 based on interpolation of seasonal air pollution sampling and used linear mixed effects models with a person-specific random effect to estimate NO₂ versus lung function cross-sectionally at baseline and longitudinally, adjusting for potential confounders (age at baseline, sex, smoking status, and long-term seasonality). Among healthy participants (with no COPD or asthma based on lung function tests), NO₂ levels were associated with a decline in lung function pre- and post-bronchodilation (−21.7 ml [95% CI: −42.1, −1.3] for FEV1 and −22.2 ml [95% CI: −46.8, 2.3] for FVC pre-bronchodilation, −25.2 ml [95% CI: −48.4, −4.1] for FEV1 and −26.6 ml [95% CI: −51.1, −2.2] for FVC post-bronchodilation) per interquartile range (10 ppb) increase in NO₂. Longitudinal impacts of air pollution on lung function were not statistically significant. Results suggest that air pollution exposure is associated with worse lung function among apparently healthy individuals among urban poor communities in India. Future studies should further characterize time-varying air pollution exposures and collect further longitudinal health data in these understudied communities.

Introduction

According to the 2019 Global Burden of Disease (GBD) Study, chronic respiratory diseases are among the leading causes of mortality and morbidity worldwide. With chronic obstructive pulmonary disease (COPD) and asthma leading to 5.8% and 0.82% of global deaths annually [1]. COPD and asthma are particularly problematic...
in low- and middle-income (LMIC) country settings such as India, where respiratory morbidity more often leads to mortality [2].

Ambient air pollution is an established risk factor for both the development of incident respiratory diseases [3–6] as well as acute exacerbations among those with pre-existing health conditions [7–10]. Large cities in India have some of the highest levels of air pollution in the world [11]. Thus, increasingly high ambient air pollution levels may be contributing to worsening health outcomes in India. According to a discussion of the Indian portion of the GBD study, air quality in Indian cities continues to worsen [12]. Analysis of regulatory monitoring data that are publicly available from the Central Pollution Control Board (CPCB) of India indicates that concentrations of air pollution exceed national ambient air quality standards at over half of the country’s monitoring stations [13, 14]. However, we lack local evidence of direct associations with respiratory disease. The contribution of traffic and point sources of pollution to elevated ambient pollution levels is an important concern for long-term respiratory health, particularly in rapidly developing countries that are experiencing increasing levels of air pollution due to growth in pollution sources [15]. Nitrogen dioxide (NO$_2$), which is emitted by combustion of fossil fuels and can be considered a proxy for traffic pollution [16], is one of the criteria pollutants currently monitored by the CPCB under the National Air Quality Monitoring Program (NAMP) [13]. Currently, there are 573 operating air pollution monitoring stations within the NAMP. With an estimated population of 1.37 billion [17], this equates to approximately 1 air pollution monitor per 2,400,000 persons. The existing air pollution monitoring network heavily favors urban areas of India [17].

Previous studies of pulmonary health effects from NO$_2$ air pollution focused on levels currently observed in urban centers in high income countries in Europe and North America; this literature indicated that elevated ambient NO$_2$ exposure is associated with decreased lung function and increased risk of pulmonary disease outcomes [18–21]. While there is evidence of a relationship between NO$_2$ exposure and decreased respiratory health [22], the chronic impact of elevated levels of air pollutants on lung function in adults is not well understood in LMICs [23], especially among people living in relatively low income communities in urban centers of LMICs [24]. More importantly, there is an urgent need for estimation of exposure-response functions in LMIC’s such as India, to understand the source, threshold concentrations of specific air pollutants, population susceptibility and health disparities.

In this study, we evaluated the relationship between air pollution and long-term changes in lung function in a cohort of adults age $\geq 35$ y, as part of the Burden of Obstructive Lung Diseases (BOLD) cohort study in Mysore, Karnataka, India. We measured associations between NO$_2$ and forced expiratory volume in 1 s (FEV$_1$) and forced vital capacity (FVC), objectively measured estimates of respiratory health that are utilized as early indicators of pulmonary and systemic inflammation [25, 26].

**Methods**

**Study design and participants**

The study area covers the urban center of Mysore, Karnataka State, India (figure 1) [27, 28]. The population of Mysore and surrounding peri-urban areas was 920,550 in 2011 [29].

The BOLD study had three primary objectives. First, the study seeks to estimate the global prevalence of COPD and risk factors associated with COPD globally. Second, the study aims to quantify the impact of COPD on quality of life. Finally, the BOLD study aims to project the future global burden of disease attributable to COPD [30]. As a participant site in the BOLD study, we collected lung function data in a cohort of adults age $\geq 35$ y in 2012 (sampling design is shown in figure 2). At baseline, households were selected into the cohort using a multi-stage random sampling procedure. In the first stage, all 17 wards of the municipal constituency were selected for sampling. In the second stage, houses within each ward were sampled in clusters of 10. In the third stage, all individuals age $\geq 35$ y were invited to participate. Original lung function data were collected from a total of 725 participants between July 2012 and July 2014.

We conducted a subsequent round of lung function data collection between August 2017 and February 2018 among all members of the original cohort who had not moved from their 2012 home location, died, or refused to participate in the second round. Within the cohort from 2017, 128 participants with measurements could not be appropriately linked to data from the same participants in 2012, likely due to issues with deidentification of data in the field. Finally, 38 persons sampled in 2012 were not resampled in 2017 due to loss to follow up. A total of 449 participants provided lung function samples at both 2012 and 2017, from a total $n_{2012} = 573$ and total $n_{2017} = 593$. Additional sampling details are illustrated in figure 2.

**Lung function data collection and outcome ascertainment**

At each time point, participants were administered at least three trials of the spirometry test using the procedure for spirometry recommended by the American Thoracic Society [31], and results for usable spirometry data
Figure 1. Map of Mysore, India Urban Area.

Figure 2. Study sampling design.
were recorded. Participants were then administered salbutamol for bronchodilation and the procedure was repeated. From this cohort, anyone without sufficient replicability of the lung function testing (3 consistent and valid tests pre- and post-bronchodilation) or missing data on covariates of interest (smoking status, date of birth, etc) was excluded. During spirometry data collection, the population prevalence of asthma and COPD was assessed utilizing pre- and post-bronchodilation FEV₁ and FVC data. COPD was diagnosed in participants with post bronchodilator FEV₁/FVC % < 0.7. Asthma was diagnosed in patients with FEV₁ reversibility (comparing pre- and post-bronchodilation FEV₁) of more than 12% and 200 ml.

In 2012–2014, spirometry data were collected using the ndd EasyOne spirometer, within participants’ homes. Subsequently, participants were asked a series of questions comprising the BOLD questionnaire by a survey administrator, including questions regarding smoking status, gender, date of birth/age, indicators of socioeconomic status (SES) such as income (measured as monthly household income), education of self and parents, and primary cooking fuel (liquefied petroleum gas (LPG), biomass fuel, or other liquid fuel such as kerosene), using hard copy surveys to collect qualitative data. Responses were recorded on paper.

During follow-up (2017–2018), we followed the same health data collection procedure from 2012–2014. Participants receiving a diagnosis of asthma or COPD at the original baseline sample, or any participant recalling such treatment at the follow-up visit, were excluded from the second round of data due to the potential for meaningful improvement in lung function attributable to treatment. In the second round of data collection, participants were asked a series of qualitative questions by the same survey administrators who conducted the first round of data collection; responses were recorded using the Qualtrics online survey on wireless tablets. These questions included the BOLD standard questionnaire, described above, as well as questions regarding perception of pollution exposure. Biometric data on height, weight, and blood pressure were also recorded at both time points. Validity of each trial during both rounds of data collection was assessed using the BOLD study quality control measure, which is more rigorous than the spirometer’s own measure of quality control. The standardized procedure for health and qualitative data collection for the BOLD cohort has been described in previous studies [31].

**Air pollution exposure assessment**

Air pollution exposure estimates were based on data collected in 2016–2017. The existing regulatory monitoring network (figure 3) provided insufficient data on the spatial distribution of NO₂ levels in the city, although it does suggest that long-term air pollution levels have been increasing. The region does not have good geolocation information, particularly in low-income neighborhoods. Therefore, we assessed the precise home location for each participant using Global Positioning System (GPS) tagging. We assigned to each study participant the annual average ambient pollution exposure of NO₂ at their geotagged home location. This exposure estimate was based on a spatial model developed using universal kriging, as previously described [32]. Briefly, we developed a geostatistical model of annual levels of NO₂ using data from NO₂ monitoring at 150 locations throughout Mysore, as well as available land use characteristics, pollution point sources, and population demographics. The model performed well in comparison with previous studies in LMIC settings (model

![Figure 3. Map of predicted NO₂ concentrations (parts per billion, ppb), and approximate location of the homes of all included study participants, and 2 operational regulatory monitors in Mysore.](image-url)
The recommended annual maximum NO₂ exposure according to the WHO is 21.3 ppb. Figure 3 shows approximate home locations and modeled pollution exposure, illustrating that air pollution exposures in parts of the city exceed the WHO’s health protective standards.

Daily movement of residents through different neighborhoods (i.e., time-activity patterns) within an urban area is difficult to determine in low-income urban communities in India. We asked study participants about their specific home and work neighborhoods, as a proxy for movement around the city. Over 97.9% of participants indicated that their work and home locations were within the same neighborhood, indicating that home location air pollution exposure is a good estimate of overall exposure to air pollution for study participants. Additionally, a majority of the participants do not have access to a car, and would move around their neighborhood in open rickshaws, by bicycle, or on foot, making their air pollution exposures during transit similar to background levels. Neighborhood sizes varied from < 1 km² to approximately 10 km².

Due to a lack of accessible information regarding long-term air pollution levels within the city of Mysore based on only 2 existing CPCB monitors located at industrial point sources of pollution, and a lack of spatially resolved NO₂ exposure data in 2012, we assumed that the same level of ambient NO₂ exposure was experienced at the home location by each participant at baseline and follow up. This design assumes that members of the cohort living in high pollution areas within the city relative to their neighbors in 2017, have been living in relatively higher pollution zones since their recruitment into the study.

**Standard protocol approvals and informed consent**

The study was approved by the Yale University Human Subjects Committee in the US (HSC#1607018137), and the JSS University Institutional Ethical Committee in Mysore, India (JSSMC/IEC/08/1991/2017–18). Participants provided written consent for their inclusion in the study or documented verbal consent in the case of those who are not able to read/write.

**Statistical analysis**

The sampling procedure described previously generated an unbalanced longitudinal cohort design. Distributions of sociodemographic characteristics were calculated as mean (±SD) for continuous variables and proportions (%) for categorical variables. Estimates of pollutant exposure are presented as mean (SD).

We used linear mixed models for repeated measures to study the relation of residential air pollution to both baseline lung function as well as longitudinal lung function decline. All models adjusted for pollutant, visit, visit by pollutant interaction, and a series of pre-defined sociodemographic characteristics considering previous evidence, including month of data collection as a categorical variable to account for time trends in the data; age as a continuous variable at time of enrollment; smoking: ever smoker versus never smoker; and gender: men versus women. The models fitted participants as a random effect and used a compound symmetry covariance matrix. We analyzed pollutant exposures as both continuous measures and by quartile. The dependent variable of interest was lung function in person i at time j of the study. All study participants indicated either steady lung function or decline over time, which is consistent with past literature and clinical evidence regarding longitudinal measures of lung function among adults.

In the primary analysis, we excluded participants meeting the criteria for a diagnosis of COPD or asthma (based on the criteria described previously) at the time that they meet that criteria, and at all subsequent rounds of analysis (we assumed that all participants diagnosed with asthma or COPD in the first round would have
received treatment, in accordance with BOLD study objectives. We also present the same model with those participants included at the time of their diagnosis of COPD or asthma and excluded from subsequent rounds of the study.

In sensitivity analyses, we considered additional potential effect modifiers such as Body Mass Index (BMI), calculated from survey data, and SES, measured as self-reported household level income in 2017, in the models. Because the majority of households (99%) reported LPG use for cooking fuel, we did not control for indoor air pollution in the main or sensitivity analyses.

Statistical analyses were conducted using R 3.5.2 GUI 1.70, El Capitan Build (The R Foundation for Statistical Computing).

Results

The average age at enrollment was 46 years (table 1); most participant characteristics remained similar between baseline and follow-up, with the exception of a higher proportion of smokers in the follow-up compared with baseline (primarily participants who took up smoking in the interim). As expected, a large majority of the cohort (>99% of participants) used LPG as residential cooking fuel due to the Pradhan Mantri Ujjwala Yojana, the Indian government scheme to distribute LPG connections to women of Below Poverty Line families [38], reducing indoor exposure to biomass as a potential cause of respiratory disease. Over 75% of the cohort reported a household level income less than $565/month or $6780/year, which is lower than the annual average individual income for urban areas of India according to the World Bank, indicating poverty [39]. While table 1 indicates that the members of our cohort live in relatively lower air pollution exposure areas compared with health protective standards, figure 3 indicates that some members of the cohort live in high pollution zones.

Over time, among participants with data collected at both time points, we observed annual decreases in lung function that were comparable with previous studies (table 2) [40, 41].
Cross-sectional results show a statistically significant association with lower pre- and post-bronchodilation measures of lung function (table 2). Adjustment for confounders substantially attenuated the observed associations, suggesting the potential for residual confounding. In all participants (including those with COPD and asthma at baseline and removing those participants from subsequent rounds of data collection assuming they have received treatment), in adjusted models, higher levels of residential air pollution exposures are not statistically significantly associated with lower pre- and post-bronchodilation measures of lung function (table 3). Association between air pollution and lung function

In healthy participants (excluding those with COPD or asthma at baseline), in models adjusted for age at baseline, sex, month of data collection, and smoking status, higher levels of residential air pollution exposures remained statistically significantly associated with lower pre- and post-bronchodilation measures of lung function (table 3). Adjustment for confounders substantially attenuated the observed associations, suggesting the potential for residual confounding. In all participants (including those with COPD and asthma at baseline and removing those participants from subsequent rounds of data collection assuming they have received treatment), in adjusted models, higher levels of residential air pollution exposures are not statistically significantly associated with lower pre- and post-bronchodilation measures of lung function (table 4).

In longitudinal analysis, higher levels of air pollution were associated with a faster decline in lung function, although results were not statistically significant. This was consistent across unadjusted and adjusted models, and across all healthy participants as well as the model including participants with COPD or asthma at time of diagnosis (tables 1 and 4). Figure 4 illustrates the comparison between cross-sectional and longitudinal impacts of air pollution exposure on lung function metrics, as well as the comparison between healthy individuals and all members of the cohort.

In sensitivity analyses, we additionally adjusted the models of healthy participants for BMI and SES indicators. These analyses showed either no or weak evidence for potential confounding of the air pollution lung function associations (Supplemental Materials, Tables S1 & S2 (available online at stacks.iop.org/ERC/3/055001/mmedia)).

Table 2. Initial lung function and average annual decline in lung function; Forced Expiratory Volume (FEV₁) and Forced Vital Capacity (FVC).

|                | Pre-Bronchodilation | Post-Bronchodilation |
|----------------|---------------------|-----------------------|
|                | FEV₁               | FVC                   | FEV₁               | FVC                   |
| Initial (L)    | 2.11               | 2.65                  | 2.12               | 2.63                  |
| Change per Year (L./yr) | −0.027          | −0.042                | −0.027             | −0.042                |

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Discussion and conclusions

This study contributes evidence of an association between long-term exposure to ambient pollution and decreased lung function in developing country settings, specifically among a cohort of low-income adults. Cross-sectional results show a statistically significant relationship between NO₂ exposure and pre- and post-bronchodilation lung function in healthy adults, with decreasing lung function associated with increasing pollution levels. Although we did see elevated annual decline in lung function over time as compared with previous work, the longitudinal analysis did not show a differential effect of air pollution on lung function over time. Thus, while we observed associations between NO₂ and lower lung function at baseline, we did not observe an increase in the rate of decline in lung function associated with higher air pollution exposure. It is possible that the time period covered by the study (6 years between enrollment and follow-up) is not sufficient to disentangle the effect of time (or natural FEV decreases with age) from the effect of NO₂: future rounds of lung function data collection are ongoing, and may yield an improved understanding of the relationship between air pollution and longitudinal decline in lung function.

Since we removed the individuals with COPD or asthma from the cohort after their lung function tests indicate illness (assuming that they would receive treatment, consistent with the aims of the BOLD study), it is difficult to make conclusions about the ‘decline’ in lung function comparing those with COPD or asthma at baseline to those without. One interpretation of our results comparing healthy individuals with the whole cohort may be that, among individuals with existing health conditions, air pollution exposure is a less important contributor to ill health compared to healthy individuals.

NO₂ attributable to automobile emissions has been linked to premature deaths across the world [42, 43]. Results from this study are consistent with previous studies, which found an adverse effect of elevated NO₂ exposure on lung function in children [44] and healthy adults [6, 22]. These previous studies, which primarily focused on populations in high-income countries, indicate consistent associations between NO₂ and lower FEV₁ and FVC, among other indicators of respiratory health. Relatively few studies have been conducted on NO₂ and respiratory health in LMICs. One study in Iran estimated 0.38% increased risk of COPD hospitalization associated with 10 μg m⁻³ increase in NO₂ [45]. These studies highlight a major challenge of conducting research in low-income populations in LMICs, the lack of long-term cohort data from these difficult-to-access communities.

| Change per Year (L./yr) | −0.027          | −0.042                | −0.027             | −0.042                |
|------------------------|----------------|-----------------------|-------------------|----------------------|
| Initial (L)            | 2.11           | 2.65                  | 2.12              | 2.63                 |

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Table 3. Association of NO$_2$ with Forced Expiratory Volume (FEV$_1$) and Forced Vital Capacity (FVC) decline (ml), before and after adjustment$^a$ for potential confounders.

| NO$_2$ (10 ppb) (Cross-sectional) | Pre-Bronchodilation | Post-Bronchodilation |
|----------------------------------|--------------------|----------------------|
| NO$_2$ (10 ppb) (Cross-sectional) |                   |                      |
| NO$_2$ x Time (10 ppb) (Longitudinal) |                   |                      |
| Unadjusted model | Adjusted model | Unadjusted model | Adjusted model | Unadjusted model | Adjusted model | Unadjusted model | Adjusted model |
| FEV$_1$ (95% CI) |          | FEV$_1$ (95% CI) |          | FEV$_1$ (95% CI) |          | FEV$_1$ (95% CI) |          |
| -40.5 (-59.2, -21.8)$^{***}$ | -21.7 (-42.1, -1.31)$^*$ | -44.8 (-67.6, -22.0)$^{***}$ | -22.2 (-46.8, 2.30) | -38.4 (-58.2, -18.6)$^{***}$ | -25.2 (-46.4, -4.07)$^*$ | -44.9 (-68.3, -21.6)$^{***}$ | -26.6 (-51.1, -2.19)$^*$ |
| PVC (95% CI) |          | PVC (95% CI) |          | PVC (95% CI) |          | PVC (95% CI) |          |
| -13.6 (-39.3, 12.1) |          | -13.9 (-36.8, 9.01) |          | -16.2 (-47.2, 14.8) |          | -5.7 (-23.4, 14.8) |          |
| FEV$_1$ (95% CI) |          | FEV$_1$ (95% CI) |          | FEV$_1$ (95% CI) |          | FEV$_1$ (95% CI) |          |
| -30.3 (-59.2, -2.13) |          | -34.2 (-56.1, -12.3) |          | -21.7 (-47.2, 4.14) |          | -28.4 (-56.3, -10.6) |          |
| PVC (95% CI) |          | PVC (95% CI) |          | PVC (95% CI) |          | PVC (95% CI) |          |
| -16.2 (-47.2, 14.8) |          | -16.2 (-47.2, 14.8) |          | -16.2 (-47.2, 14.8) |          | -16.2 (-47.2, 14.8) |          |

$^a$ Models adjusted for age, sex, month of spirometry data collection, and smoking status.

$^{***}$Significant at $p = 0.0001$ $^*$Significant at $p = 0.05$ Significant at $p = 0.1$. 
Table 4. Association of NO\textsubscript{2} with Forced Expiratory Volume (FEV\textsubscript{1}) and Forced Vital Capacity (FVC) decline (mL), before and after adjustment\textsuperscript{a} for potential confounders; including participants with COPD or Asthma, at time of diagnosis.

| NO\textsubscript{2} (10 ppb) (Cross-sectional) | Pre-Bronchodilation | Post-Bronchodilation |
|-----------------------------------------------|---------------------|----------------------|
| Unadjusted Model                              | Adjusted Model      | Unadjusted Model     | Adjusted Model      | Unadjusted Model | Adjusted Model |
| (95% CI)                                       | (95% CI)            | (95% CI)             | (95% CI)            | (95% CI)         | (95% CI)        |
| NO\textsubscript{2} x Time (10 ppb) (Longitudinal) |                     |                      |                     |                   |                   |
| (95% CI)                                       |                     |                      |                     |                   |                   |

\textsuperscript{a} Models adjusted for age, sex, month of spirometry data collection, and smoking status.

\textsuperscript{**}Significant at \( p = 0.0001 \).
Some previous studies assessed air pollution exposure utilizing proximity to nearby roadways or other pollution sources [19, 26], or relied on availability of nearby regulatory monitoring data, which are sparse in LMICs. A study of the respiratory health of women in slum areas of New Delhi, India did not identify a link between ambient air pollution assessed using regulatory monitors and reduced lung function, although the authors hypothesized an effect of air pollution on health [24]. In Mysore, data are available from only 2 regulatory monitors (figure 3). The spatial distribution of air pollution in Indian urban settings exhibits high heterogeneity, indicating the potential for exposure misclassification in studies using only regulatory monitoring. In this study, the home estimate of NO₂ is a good measure of overall ambient exposure at the individual level, due to a low level of personal movement throughout the day. The operational network of regulatory monitors in Mysore would not suitably captured pollutant concentrations at the household and would likely introduce exposure misclassification. In addition to limited spatial resolution of regulatory monitors, data available from the CPCB lack high temporal resolution as such data are available as monthly averages [14]. A major strength of our study is the estimate of annual average ambient air pollution exposure at the individual level based on geotagged home location, providing a spatially heterogeneous estimate of exposures. Future work may aim to similarly improve temporal exposure assessment, through ongoing air pollution monitoring campaigns coincident with continued follow-up in the BOLD cohort.

Several methodological issues related to outcome and exposure assessment may have impacted our results. First, we only had measurements from two spirometry time points that were not matched on season, which may be important due to higher potential for respiratory illness during colder winter months, or allergic outcomes during monsoon season when bioallergens may become more prevalent. This may have decreased precision in estimating lung function decline. We aimed to address this issue by adjusting for month of data collection to address potential temporal confounding. Second, data from a single annual average air pollution exposure model showing exposure to NO₂ in 2017 was utilized to assess residential air pollution exposure for both health data collection time points for each participant. This may lead to issues with exposure misclassification; however, given the limitations of the existing regulatory sampling at only 2 locations throughout the city, we believe our estimation of air pollution exposure represents an improvement in the existing capacity to assess pollution exposures in the city. Our assumption is that patterns of air pollution exposure will remain spatially consistent across time; however, if variability in spatial trends exist, exposure misclassification may be introduced. The sampling design and recruitment procedure to meet standards of the BOLD study yielded a cohort in neighborhoods with lower air pollution than in Mysore overall (figure 3), which may further attenuate results. We excluded participants with the worst health outcomes from analysis in 2017; we treat this as noninformative censoring, but some studies suggest a potential ‘healthy survivor effect’ in longitudinal studies, particularly with elderly populations and health outcomes associated with aging [46]. If cohort participants who were treated for respiratory disease were included in follow-up analysis, we may have observed artificial improvements in lung function over time due to treatment. However, by removing them from analysis, we estimate that the longitudinal results are biased towards the null.

Spirometry devices were updated with new software within each round of lung function data collection, and a different spirometer was used during follow-up than at baseline. Within-round assessments of spirometry standardization were completed as recommended by the American Thoracic Society [47]. However, calibration was not possible across sampling rounds, since the original spirometer was unavailable to perform comparison tests with the follow-up spirometer. Such changes can be an inherent source of differences in measured lung function, especially the long-term temporal change. We attempted to account for this by controlling for the time of data collection.

The current study, which includes repeat measures from a low-income cohort living in high pollution areas in urban India with highly localized exposure assessment at the home locations, provides evidence of an adverse association between ambient air pollution and lung function in adults. These results have policy relevance to minimize the public health burden of ambient pollution, such as support for zoning of buildings near high-traffic corridors or the anti-idling policies that have become prevalent in Indian cities. While results were not statistically significant longitudinally, future studies should aim to utilize temporally as well as spatially heterogeneous estimates of pollution exposure to further characterize the longitudinal relationship between air pollution and lung function.

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Data availability statement

The data that support the findings of this study are available upon reasonable request from the authors.

Summary conflict of interest statements

Dr. Wellenius has served as a paid member of multiple expert panels for the Health Effects institute (Boston, MA) providing expertise on the health effects of air pollution. Dr. Wellenius currently serves as a paid visiting scientist at Google Research. The remaining authors declare no competing interests in the publication of this manuscript.

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Credit author statement

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