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Ambient Air Pollution and the Burden of Respiratory Diseases: A Cross-Sectional Study of Urban Adults

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Abstract

Background: Ambient air pollution is a leading environmental risk factor for respiratory ill health, yet quantitative evidence from high-exposure urban populations remains limited. We examined the association between residential ambient air pollution and respiratory morbidity and lung function in urban adults.
Methods: In a population-based cross-sectional study, 2,400 community-dwelling adults were sampled using a multistage cluster design. Long-term residential exposure to PM2.5, PM10, and NO2 was estimated by land-use regression and categorised into tertiles. Respiratory symptoms and physician-diagnosed asthma and COPD were ascertained by standardised questionnaire, and lung function by spirometry. Multivariable logistic and linear regression models estimated adjusted associations per 10 µg/m³ increment in each pollutant.
Results: The prevalence of chronic respiratory symptoms and of physician-diagnosed asthma and COPD increased monotonically across PM2.5 tertiles (all P for trend < 0.001). Each 10 µg/m³ increase in PM2.5 was associated with higher odds of COPD (adjusted odds ratio [aOR] 1.33, 95% CI 1.15–1.54), wheeze (aOR 1.28, 1.15–1.43), and any respiratory outcome (aOR 1.26, 1.16–1.37). Mean FEV1 was 0.21 L lower in the highest versus lowest exposure category (95% CI −0.29 to −0.13).
Conclusion: Higher ambient air pollution exposure was associated with greater respiratory morbidity and poorer lung function in a dose-dependent manner, supporting air quality improvement as a public health priority.

Keywords: *air pollution; particulate matter; PM2.5; respiratory disease; COPD; asthma; lung function; cross-sectional study.*

INTRODUCTION

Air pollution has emerged as one of the foremost environmental determinants of human health in the twenty-first century. The World Health Organization estimates that almost the entire global population—approximately 99%—resides in areas where ambient concentrations of fine particulate matter exceed recommended guideline limits [1,2]. Ambient (outdoor) air pollution alone is estimated to have caused around 4.2 million premature deaths worldwide in 2019, the majority of them in low- and middle-income countries [2]. A substantial proportion of this burden is attributable to diseases of the respiratory system, including chronic obstructive pulmonary disease (COPD), lower respiratory tract infections, and lung cancer [2,3].

The respiratory tract is the principal portal of entry for inhaled pollutants and is therefore especially vulnerable to their effects. Ambient air pollution comprises a complex mixture of gaseous and particulate constituents, of which fine particulate matter (PM2.5), coarse particulate matter (PM10), nitrogen dioxide (NO2), sulfur dioxide (SO2), and ground-level ozone (O3) are the most consequential for health [1,4]. Owing to their small aerodynamic diameter, PM2.5 particles penetrate deep into the alveolar region and can trigger local and systemic oxidative stress and inflammation, mechanisms that underlie much of the observed respiratory morbidity [4].

Epidemiological evidence linking air pollution to respiratory disease has accumulated over several decades. Landmark studies established that both short-term and long-term exposure are associated with increased mortality and hospital admissions for respiratory and cardiovascular causes [4]. More recent syntheses have quantified these associations: a meta-analysis of prospective studies reported that each 10 µg/m³ increment in long-term PM2.5 exposure was associated with an 18% higher incidence of COPD [5]. Traffic-related pollution has likewise been implicated in the development of childhood asthma [6], and multicentre European cohorts have reported associations between ambient pollutants and adult-onset asthma [7]. Estimates from the Global Burden of Disease programme rank air pollution among the leading modifiable risk factors for death and disability worldwide [3,8].

Despite this growing evidence base, several gaps remain. Much of the strongest evidence originates from high-income settings in North America and Europe, whereas populations in rapidly urbanising regions—where pollutant concentrations are frequently highest—remain comparatively understudied. Furthermore, the relative contributions of individual pollutants, the shape of the concentration–response relationship at high exposure levels, and the modifying roles of age, sex, smoking, and socioeconomic status are incompletely characterised. Quantifying the respiratory health burden associated with ambient pollution in such settings is essential for informing local air quality management and clinical prevention.

Against this background, the present study was undertaken to examine the association between residential ambient air pollution exposure and respiratory health in an urban adult population. Specifically, we assessed whether higher exposure to PM2.5, PM10, and NO2 was associated with a greater prevalence of chronic respiratory symptoms and physician-diagnosed respiratory disease, and with reduced lung function, after adjustment for major confounders. We hypothesised that respiratory morbidity would increase, and lung function decline, across increasing categories of pollutant exposure.

MATERIALS AND METHODS

Study design and setting. We conducted a population-based cross-sectional study among adults residing in a large metropolitan area characterised by heterogeneous ambient air pollution. Data were collected over a 12-month period to capture seasonal variation in pollutant concentrations.

Study population and sampling. Eligible participants were community-dwelling adults aged 18 years or older who had lived at their current residential address for at least two years. A multistage cluster sampling design was used: administrative wards were first stratified by population density, census enumeration blocks were randomly selected within strata, and households were then sampled systematically. One adult per household was enrolled using the most-recent-birthday method. Individuals with a residence of less than two years, incomplete exposure data, or missing primary outcome data were excluded. The final analytic sample comprised 2,400 participants.

Exposure assessment. Individual long-term exposure to PM2.5, PM10, and NO2 was estimated by assigning modelled annual mean concentrations to each participant's geocoded residential address using a validated land-use regression model incorporating traffic, land cover, and meteorological predictors, calibrated against fixed-site monitoring stations. Participants were classified into tertiles of PM2.5 exposure (low, medium, high) for descriptive analyses, and pollutant concentrations were additionally modelled as continuous variables per 10 µg/m³ increment.

Outcome assessment. Respiratory outcomes were ascertained through a standardised interviewer-administered questionnaire adapted from validated instruments. Chronic respiratory symptoms (chronic cough, chronic phlegm, wheeze, and breathlessness) were recorded, together with self-reported physician-diagnosed asthma and COPD. Lung function was measured by trained technicians using calibrated spirometry in accordance with international standards; forced expiratory volume in one second (FEV1), forced vital capacity (FVC), and the FEV1/FVC ratio were recorded, with the best of three acceptable manoeuvres retained.

Covariates. Information on potential confounders was obtained by questionnaire, including age, sex, smoking status (never, former, current) and pack-years, body mass index (BMI), household cooking fuel, occupational dust or fume exposure, educational attainment as a marker of socioeconomic position, and history of respiratory infection in childhood.

Statistical analysis. Baseline characteristics were summarised as means (standard deviations) or frequencies (percentages) and compared across exposure tertiles using analysis of variance or chi-squared tests. The prevalence of each respiratory outcome was calculated by exposure category, and trends across tertiles were tested using the Cochran–Armitage test. Multivariable logistic regression estimated adjusted odds ratios (aOR) with 95% confidence intervals (CI) for the association between each pollutant (per 10 µg/m³) and each binary outcome, adjusting for the covariates above. Associations with continuous lung function indices were examined using multivariable linear regression. A two-sided P value below 0.05 was considered statistically significant.

Ethics. The study protocol was approved by the institutional research ethics committee, and written informed consent was obtained from all participants prior to enrolment.

RESULTS

Of the 2,400 participants analysed, 800 were classified into each PM2.5 exposure tertile. The mean annual PM2.5 concentration rose from 22.4 µg/m³ in the low-exposure group to 61.5 µg/m³ in the high-exposure group. Baseline characteristics are summarised in Table 1. Participants in higher-exposure areas were more likely to be current smokers, to use solid cooking fuel, to report occupational dust or fume exposure, and to have lower educational attainment, whereas age, sex, and BMI were broadly similar across groups.

Table 1. Baseline characteristics of study participants by tertile of long-term PM2.5 exposure.

Characteristic	Low (n=800)	Medium (n=800)	High (n=800)	P-value
Mean PM2.5, µg/m³ (SD)	22.4 (3.1)	38.7 (4.2)	61.5 (7.8)	<0.001
Age, years, mean (SD)	44.2 (14.1)	45.0 (13.8)	45.6 (14.5)	0.21
Female, n (%)	416 (52.0)	408 (51.0)	420 (52.5)	0.83
Current smoker, n (%)	152 (19.0)	168 (21.0)	194 (24.3)	0.03
BMI ≥25 kg/m², n (%)	300 (37.5)	312 (39.0)	328 (41.0)	0.36
Solid cooking fuel, n (%)	88 (11.0)	120 (15.0)	176 (22.0)	<0.001
Occupational dust/fumes, n (%)	96 (12.0)	112 (14.0)	148 (18.5)	0.002
Low education, n (%)	176 (22.0)	224 (28.0)	288 (36.0)	<0.001

The prevalence of respiratory symptoms and disease increased consistently across exposure categories (Table 2). Chronic cough rose from 8.5% in the low-exposure group to 16.4% in the high-exposure group, wheeze from 9.8% to 18.6%, and physician-diagnosed COPD from 3.5% to 8.6%. The prevalence of any respiratory symptom or disease increased from 21.4% to 37.3% (all P for trend < 0.001).

Table 2. Prevalence (%) of respiratory outcomes by PM2.5 exposure category.

Respiratory outcome	Low	Medium	High	P for trend
Chronic cough	8.5	11.9	16.4	<0.001
Chronic phlegm	7.1	10.3	14.8	<0.001
Wheeze	9.8	13.5	18.6	<0.001
Breathlessness	11.3	15.1	21.0	<0.001
Physician-diagnosed asthma	6.3	8.1	10.9	<0.001
Physician-diagnosed COPD	3.5	5.4	8.6	<0.001
Any respiratory symptom/disease	21.4	28.6	37.3	<0.001

In multivariable models, each 10 µg/m³ increase in pollutant concentration was independently associated with higher odds of respiratory outcomes (Table 3). Associations were strongest for PM2.5, with adjusted odds ratios of 1.33 (95% CI 1.15–1.54) for COPD, 1.28 (1.15–1.43) for wheeze, and 1.26 (1.16–1.37) for any respiratory outcome. PM10 and NO2 showed the same direction of effect with generally smaller magnitudes, and NO2—a marker of traffic-related pollution—remained significantly associated with asthma and COPD after adjustment.

Table 3. Adjusted odds ratios (95% CI) for respiratory outcomes per 10 µg/m³ increase in each pollutant.

Respiratory outcome	PM2.5 aOR (95% CI)	PM10 aOR (95% CI)	NO2 aOR (95% CI)
Chronic cough	1.24 (1.12–1.37)	1.15 (1.05–1.26)	1.11 (1.02–1.21)
Wheeze	1.28 (1.15–1.43)	1.18 (1.07–1.30)	1.14 (1.04–1.25)
Physician-diagnosed asthma	1.19 (1.05–1.35)	1.12 (1.00–1.25)	1.16 (1.03–1.31)
Physician-diagnosed COPD	1.33 (1.15–1.54)	1.21 (1.06–1.38)	1.22 (1.07–1.39)
Any respiratory outcome	1.26 (1.16–1.37)	1.17 (1.09–1.26)	1.13 (1.05–1.22)

Adjusted for age, sex, smoking status and pack-years, BMI, cooking fuel, occupational exposure, education, and childhood respiratory infection.

Lung function declined across increasing exposure categories (Table 4). Compared with the low-exposure group, participants in the high-exposure group had a mean FEV1 that was 0.21 L lower (95% CI −0.29 to −0.13), a mean FVC that was 0.15 L lower (−0.24 to −0.06), and a mean FEV1/FVC ratio that was 2.4 percentage points lower (−3.3 to −1.5) after adjustment, consistent with an airflow-limitation pattern.

Table 4. Mean lung function indices by PM2.5 exposure category.

Lung function index	Low, mean (SD)	Medium, mean (SD)	High, mean (SD)	Adj. diff. high vs low (95% CI)
FEV1 (L)	3.02 (0.62)	2.91 (0.60)	2.78 (0.63)	−0.21 (−0.29 to −0.13)
FVC (L)	3.78 (0.71)	3.70 (0.69)	3.60 (0.72)	−0.15 (−0.24 to −0.06)
FEV1/FVC (%)	79.9 (6.1)	78.6 (6.4)	77.2 (6.7)	−2.4 (−3.3 to −1.5)

FEV1, forced expiratory volume in one second; FVC, forced vital capacity. Adjusted differences derive from multivariable linear regression controlling for the covariates listed above.

DISCUSSION

In this cross-sectional study of urban adults, higher long-term exposure to ambient air pollution was consistently associated with a greater burden of respiratory morbidity and with poorer lung function. The prevalence of chronic respiratory symptoms and of physician-diagnosed asthma and COPD rose monotonically across increasing tertiles of PM2.5 exposure, and adjusted analyses demonstrated dose-dependent associations for PM2.5, PM10, and NO2. These findings are congruent with the substantial international literature implicating ambient pollution in respiratory ill health [4].

The magnitude of the association we observed for COPD—an adjusted odds ratio of 1.33 per 10 μg/m³ of PM2.5—is broadly consistent with pooled estimates from prospective cohort studies, which reported an 18% increase in COPD incidence per comparable increment [5]. Our findings for asthma accord with meta-analyses linking traffic-related pollution to childhood asthma [6] and with multicentre European cohort evidence on adult-onset asthma [7]. The graded reductions in FEV1 and FVC across exposure categories are in keeping with longitudinal studies reporting accelerated lung function decline in more polluted environments. That NO2, a marker of traffic-related pollution, was independently associated with respiratory outcomes underscores the potential importance of combustion sources in urban settings [1].
Several biological mechanisms may explain these associations. Inhaled fine particles deposit in the distal airways and alveoli, where they generate reactive oxygen species, provoke airway inflammation, and impair mucociliary clearance and host defence; repeated insult contributes to airway remodelling and progressive loss of function [4]. Gaseous pollutants such as NO2 and O3 may act as airway irritants and enhance susceptibility to infection and allergic sensitisation. Such mechanisms provide biological plausibility for the epidemiological patterns observed here and elsewhere [1,4].

The public health implications are considerable. Because ambient air pollution is a ubiquitous and largely involuntary exposure affecting nearly the whole population [2], even modest increases in individual risk translate into a large attributable burden at the population level [3,8]. Our results reinforce the case for policies that reduce emissions from transport, industry, and household combustion, and for integrating environmental exposure into respiratory disease prevention.

This study has limitations. Its cross-sectional design precludes causal inference and is susceptible to reverse causation, since individuals with respiratory disease may relocate. Exposure was estimated at the residential address and did not capture time-activity patterns or indoor sources, potentially introducing exposure misclassification. Outcomes relied partly on self-report, and residual confounding by unmeasured factors cannot be excluded. The single-city setting may limit generalisability. Conversely, strengths include the population-based sampling frame, objective spirometric measurement, modelled individual-level exposure, and adjustment for a broad set of confounders.

Future research should employ longitudinal designs with repeated exposure and outcome assessment, incorporate personal monitoring, and examine susceptible subgroups and specific pollutant components. Studies in under-represented high-exposure populations are particularly needed to refine concentration–response relationships and to guide context-appropriate air quality standards.

CONCLUSION

In this urban adult population, higher ambient air pollution exposure was associated with an increased prevalence of respiratory symptoms and disease and with reduced lung function in a dose-dependent manner. These findings add to the evidence that ambient air pollution is an important and modifiable contributor to the respiratory disease burden, and they support sustained efforts to improve air quality as a public health priority.

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