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Associations among air pollution, asthma and lung function: a cross-sectional study

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Ambient air pollution affects the respiratory system, but evidence of its impacts on asthma and lung function is lacking. We aimed to evaluate whether ambient air pollutants are associated with asthma prevalence, asthma outcomes, and lung function in adults. A cross-sectional study of 454,921 adults aged 37 to 73 years from the UK Biobank was performed with linear or logistic regression to assess the associations among air pollution and asthma prevalence, current wheezing, asthma hospitalizations and lung function. Each interquartile range (IQR) increase in of $PM_{2.5}$ (odds ratio [OR]: 1.023, 95% confidence interval [CI]: 1.011–1.035), PM_{10} (OR: 1.013, 95% CI: 1.004–1.022), NO_2 (OR: 1.025, 95% CI: 1.013–1.039), and NO_x (OR: 1.019, 95% CI: 1.008–1.029) was significantly associated with asthma prevalence, respectively. Moreover, exposure to air pollution was related to increased odds of current wheezing and asthma-related hospitalization. Among asthmatic participants, each IQR increase in PM_{coarse}, PM_{10}, NO_2, and NO_x was significantly associated with decreases of 5.143 ml, 7.614 ml, 13.266 ml, 9.440 ml, respectively, for the forced expiratory volume in one second and 11.744 ml, 15.637 ml, 13.041 ml, 9.063 ml, respectively, for the forced vital capacity. In a large sample size study of British adults, air pollution was related to increased odds of asthma prevalence. Among the asthmatic population, air pollution was associated with increased odds of current wheezing, hospitalization, and decreased lung function.

Keywords Air pollution, Asthma, Lung functions, Wheezing, Hospitalization, UK Biobank

Abbreviations

BMI	Body mass index
CI	Confidence interval
CO	Carbon monoxide
FEV1	Forced expiratory volume in one second
FVC	Forced vital capacity
IQR	Interquartile range
NO_x	Nitrogen oxides
NO_2	Nitrogen dioxide
OR	Odds ratio
O_3	Ozone
$PM_{2.5}$	Particle matter with diameter < 2.5 μm
PM_{10}	Particle matter with diameter < 10 μm
PM_{coarse}	Particulate matter with diameter between 2.5 μm and 10 μm

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SD	Standard deviation
SO_2	Sulfur dioxide

Asthma is a chronic, immune-mediated inflammatory disorder characterized by reversible airway obstruction with heterogeneity in its clinical presentation[1]. In the United Kingdom, the asthma prevalence was 9.6%, with approximately 93,000 hospital inpatient episodes and 1,160 deaths attributed to asthma in 2016[2]. Moreover, the lifelong treatment of asthma imposed a heavy global economic burden.

It is widely recognized that air pollution exposure has various health effects. A systematic analysis of the 2010 Global Burden of Disease revealed that exposure to ambient particulate matter pollution accounted for 3.1% of global disability-adjusted life-years and that the number of deaths attributable to exposure to particulate matter pollution was 3.2 million[3]. Many studies have explored the associations between air pollution and the prevalence, incidence, control, or mortality of asthma[4–9]. The RHINESSA study with 3,428 participants found that exposure to NO_2, PM_{10} and O_3, in childhood, adolescence and adult was related to increased odds of asthma attacks in the last 12 months, but was not associated with physician-diagnosed asthma[10]. Moreover, exposure of children and adolescents to $PM_{2.5}$ and O_3 was linked with decreased lung function; however, no significant relationship between exposure in adulthood and lung function has been reported[10]. A cross-sectional analysis of 646,731 participants aged ≥ 20 years using DataSHIELD revealed that long-term PM_{10} exposure is related to asthma prevalence[11]. However, the true role of ambient air pollution in asthma remains uncertain due to inconsistent results. Moreover, studies have focused mainly on children, leaving many gaps that still need to be explored.

Herein, we conducted a cross-sectional survey to examine the linkage between air pollution and asthma prevalence, asthma outcome, and lung function measures in a population-based cohort of adults from the UK Biobank. Moreover, we explored whether age, sex, BMI, and smoking status could modify the association.

Methods

Study design and participant

The UK Biobank project recruited over 500,000 participants aged between 37 and 73 years and collected their phenotypic and genetic data with the available online study protocol (https://www.ukbiobank.ac.uk/media/3sbeknnz/ukbiobank_protocol.pdf). The participants completed a self-report questionnaire collecting detailed exposure and personal information, provided biological samples for various types of assays, and underwent physical measurements. All participants provided informed written consent, and the North West Multicenter Research Ethics Committee granted ethical approval (Reference 16/NW/0274; Application Number 84979). All methods were performed in accordance with the Declaration of Helsinki and relevant guidelines, complying with the necessary ethical standards.

In the present study, 502,411 participants were recruited at baseline. 41,299 participants with missing data on air pollution, including $PM_{2.5}$, PM_{coarse}, PM_{10}, NO_x, and NO_2, 5,264 participants without data on smoking status, BMI, sex, and age, and 927 individuals with missing self-reported asthma status were excluded. Ultimately, a total of 454,921 individuals were analyzed for the associations between air pollution and asthma prevalence. Among the 48,896 individuals with asthma, 853 with missing data on self-reported wheezing status were excluded to evaluate the association between air pollution and current wheezing (N = 48,043). To estimate the relationship between air pollution and lung function, we included individuals whose lung function data met the ATS/ERS reproducibility criteria (N = 29,101) and excluded those who had smoked within one hour prior to lung function measurement (N = 288), leaving 28,813 individuals for analysis (Supplementary Material Fig. 1).

Air pollution

The levels of ambient air pollutants including particulate matter (PM) with diameters of < 10 μm (PM_{10}), 2.5–10 μm (PM_{coarse}), and < 2.5 μm ($PM_{2.5}$), nitrogen oxides (NO_x), and nitrogen dioxide (NO_2), were evaluated with land-use regression (LUR) models developed by the European Study of Cohorts for Air Pollution Effects (ESCAPE) project[12,13]. Moreover, the annual average levels of air pollutants in 2010 were defined as individual exposures. LUR models calculate the annual moving average concentrations of air pollutants on the basis of predictor variables derived from geographic information system (GIS) data, including land use, traffic density, and topographical features, at a 100 m × 100 m resolution. The participants were assigned ambient air pollution concentrations based on their residential coordinates within 100 m × 100 m grid cells.

Outcomes

Asthma was defined as physician diagnosed asthma by a self-report questionnaire. Current wheezing was considered as a response of "Yes" to the question with regarding whether the participant have ever had whistling or wheezing in the chest last year. Moreover, individuals with asthma-related hospitalizations were defined as ever having had a hospitalization with a diagnosis with asthma. The International Classification of Diseases Clinical Modification (ICD) code for asthma consisted of ICD-10: J45.

Prebronchodilator lung function including FEV1 and FVC was measured with two or three blows with a spirometer (Vitalograph Pneumotrac 6800). Furthermore, the results were guaranteed to be reproducible and acceptable with a less than 5% difference in the FEV1 and FVC between the first two blows; otherwise, a third blow was necessary. Moreover, to reduce measurement uncertainty, bias, and drift, a list of comprehensive and robust quality procedures was established. The evaluation of lung function was prohibited in the following situations: abdominal or chest surgery, eye surgery, heart attack in the previous 3 months, pregnancy, having a chest infection in the previous month, treatment for tuberculosis, detached retina, and history of collapsed lung.

Statistical analysis

Baseline characteristics are presented as continuous variables with means ± standard deviations (SDs) and as categorical variables with numbers (percentages) according to asthma status. T-tests or chi-square tests were applied to compare the characteristics between participants with or without asthma as appropriate. Pearson correlation coefficients for air pollutants were also calculated. We observed associations between air pollution and asthma, at least one asthma hospitalization, current wheezing, or lung function with logistic or linear regression. All models were adjusted for sex, age, race, education level, smoking status (current, former, or never), body mass index (BMI), and annual household income. When estimating the association between air pollution and asthma, we additionally adjusted for allergy history (including hay fever, allergic rhinitis, or eczema). When exploring associations between air pollution and asthma outcomes or lung function, we further adjusted for asthma medication use (including β2 agonists, anticholinergic medications, inhaled corticosteroids and so on)[14]. Furthermore, models for lung function measures were additionally adjusted for height and height squared. The data were analyzed with SPSS 27 (StataCorp LP, College Station, TX, USA), and two-sided *P* value less than 0.05 was considered significant.

Results

Baseline characteristics

The main characteristics of the 454,921 individuals, including 48,896 subjects with asthma, are shown in Table 1. Compared to participants without asthma, participants with asthma were younger, more likely to be white, female, and obese; and had a lower total household income and lower education level.

The mean (SD) $PM_{2.5}$, PM_{coarse}, PM_{10}, NO_2 and NO_x level were 9.99 μg/m^3 (1.06), 6.43 μg/m^3 (0.90), 16.23 μg/m^3 (1.90), 26.62 μg/m^3 (7.61), and 43.95 μg/m^3 (15.59), respectively. $PM_{2.5}$ was strongly related to NO_2 ($r = 0.865$) and NO_x ($r = 0.848$), but less strongly related to other types of PM. PM_{10} has a strong correlation with PM_{coarse} ($r = 0.817$). NO_2 was also strongly correlated with NO_x ($r = 0.922$) (Table 2).

Air pollution and the prevalence of asthma

The results for ambient air pollution and asthma prevalence are shown in Fig. 1. Each interquartile range (IQR) increase in $PM_{2.5}$ (odds ratio [OR]: 1.023, 95% confidence interval [CI]: 1.011–1.035), PM_{10} (OR: 1.013, 95% CI: 1.004–1.022), NO_2 (OR: 1.025, 95% CI: 1.013–1.039), and NO_x (OR: 1.019, 95% CI: 1.008–1.029) was significantly associated with the increased prevalence of asthma after adjusted for smoking status, education level, sex, age, ethnicity, BMI, household income, and allergy history. When divided into quartiles, air pollutant level in quartile 4, including $PM_{2.5}$ (OR: 1.037, 95% CI: 1.009–1.066), PM_{10} (OR: 1.039, 95% CI: 1.011–1.068), NO_2 (OR: 1.067, 95% CI: 1.038–1.097), and NO_x (OR: 1.048, 95% CI: 1.020–1.078), significantly increased the odds of asthma compared with air pollutant level in quartile 1 (Supplementary Material Table 1). The results showed a similar trend when air pollution exposure was defined by the WHO guidelines (Supplementary Material Table 2).

After stratified by age, sex, smoking status, and BMI, we repeated the analysis to investigate the modification impact of air pollution on asthma by those factors (Supplementary Material Figures 2, 3). The effect size was larger in the participants older than 60 years (Supplementary Material Tables 3, 4) and the population of female (Supplementary Material Tables 5, 6). The effect was more significant in obese individuals (BMI > 30 kg/m^2) than in those with a normal BMI (Supplementary Material Tables 7, 8). Moreover, current smokers presented greater estimated effects than did participants who had never smoked (Supplementary Material Tables 9, 10).

A sensitivity analysis of air pollution and asthma was conducted after excluding participants with missing data on noncritical covariates (including annual household income, race, and education level) ($N = 383,239$) to estimate the stability of the data with similar results (Supplementary Material Table 11).

Air pollution and current wheezing or asthma-related hospitalization

Figure 2 shows the associations between air pollution and at least one asthma-related hospitalization or current wheezing. Exposure to air pollutants, especially $PM_{2.5}$ (OR: 1.029, 95% CI: 1.004–1.054) and NO_x (OR: 1.030, 95% CI: 1.008–1.053) was related to increased odds of current wheezing. The associations between air pollution exposure and asthma hospitalizations were examined, with the following results: $PM_{2.5}$ (OR: 1.058, 95% CI: 1.029–1.087), PM_{10} (OR: 1.022, 95% CI: 1.001–1.044), NO_2 (OR: 1.062, 95% CI: 1.030–1.095) and NO_x (OR: 1.053, 95% CI: 1.028–1.079). The results showed a similar trend after the participants were divided into two groups based on WHO air pollution guidelines or into four groups according to quartiles of air pollution levels (Supplementary Material Tables 12, 13).

Air pollution and lung function measures among asthmatic individuals

Table 3 shows the multivariate analysis of lung function among asthmatic individuals. Among asthmatic participants, each IQR increase in PM_{coarse}, PM_{10}, NO_2, and NO_x was significantly associated with a decrement of 5.143 ml, 7.614 ml, 13.266 ml, 9.440 ml FEV1 and 11.744 ml, 15.637 ml, 13.041 ml, 9.063 ml FVC respectively. The results were similar across sex-stratified analyses, with each IQR increase in air pollution correlated with a decrease in lung function. Moreover, the observed impact was more pronounced and significant in females (Table 3).

Discussion

In a large population-based study of 454,921 adults aged 37 to 73 years, a higher level of air pollution was associated with higher odds of asthma. Moreover, a higher level of air pollution increased the odds of current wheezing and asthma-related hospitalization. Furthermore, an increase in air pollution was correlated with decrease lung function among individuals with asthma.

Variables	Total participants (*N* = 454,291)	Participants with asthma (*N* = 48,896)	Participants without asthma (*N* = 406,025)	*P*
Age (year), Mean ± SD	56.56 ± 8.09	55.54 ± 8.28	56.68 ± 8.06	< 0.001
Age category, N (%)				< 0.001
< 60	256,981 (56.49)	226,889 (55.88)	30,092 (61.54)	
≥ 60	197,940 (43.51)	179,136 (44.12)	18,804 (38.46)	
Sex, N (%)				< 0.001
Female	247,394 (54.38)	218,961 (53.93)	28,433 (58.15%)	
Male	207,527 (45.62)	187,064 (46.07)	20,463 (41.85)	
Average total household income before Tax (€), n (%)				< 0.001
Less than 18,000	87,756 (19.29)	78,250 (19.27)	9506 (19.44)	
18,000 to 30,999	98,741 (21.71)	88,703 (21.85)	10,038 (20.53)	
31,000 to 51,999	100,614 (22.12)	89,770 (22.11)	10,844 (22.18)	
52,000 to 100,000	78,037 (17.15)	69,193 (17.04)	8844 (18.09)	
Greater than 100,000	20,647 (4.54)	18,279 (4.50)	2368 (4.84)	
Missing	69,126 (15.20)	61,830 (15.23)	7296 (14.92)	
Education, N (%)				< 0.001
College or University degree;	143,833 (31.62)	127,273 (31.35)	16,560 (33.87)	
A Levels/AS Levels or equivalent	50,089 (11.01)	44,446 (10.95)	5643 (11.54)	
O Levels/GCSEs/CSE or equivalent	123,659 (27.18)	110,862 (27.30)	12,797 (26.17)	
Other (e.g., NVQ, nursing)	53,151 (11.68)	47,739 (11.76)	5412 (11.07)	
None of above	75,910 (16.69)	68,315 (16.83)	7595 (15.53)	
Missing	8279 (1.82)	7390 (1.82)	889 (1.82)	
Ethnicity, N (%)				0.002
White	428,544 (94.20)	45,911 (93.90)	382,633 (94.24)	
Mixed	2745 (0.60)	391 (0.80)	2354 (0.58)	
Asian or Asian British	8995 (1.98)	1003 (2.05)	7992 (1.97)	
Black or Black British	7551 (1.66)	848 (1.73)	6703 (1.65)	
Chinese	1393 (0.31)	109 (0.22)	1284 (0.32)	
Other ethnic groups	4139 (0.91)	442 (0.90)	3697 (0.91)	
Missing	1554 (0.34)	192 (0.39)	1362 (0.34)	
Smoking status, N (%)				< 0.001
Never	249,057 (54.75)	221,650 (54.59)	27,407 (56.05)	
Previous	158,683 (34.88)	141,604 (34.88)	17,079 (34.93)	
Current	47,181 (10.37)	42,771 (10.53)	4410 (9.02)	
BMI (kg/m^2), Mean (SD)	27.43 ± 4.80	28.07 ± 5.36	27.36 ± 4.74	< 0.001
BMI category, N (%)				< 0.001
Normal (< 25)	150,229 (33.02)	135,578 (33.39)	14,651 (29.96)	
Overweight (25 ~ 30)	193,367 (42.51)	173,286 (42.68)	20,081 (41.07)	
Obesity (30~)	111,325 (24.47)	97,161 (23.93)	14,164 (28.97)	
FEV1 (mL), Mean (SD)		2594.46 ± 739.63		
FVC (mL), Mean (SD)		3574.22 ± 952.15		
FEV1/FVC (%), Mean (SD)		72.54 ± 7.67		
Current wheeze, N (%)		31,140 (64.82)		
Asthma hospitalization, N (%)		10,011 (20.51)		

Table 1. Characteristics of participants from the UK Biobank. *A* Advanced, *AS* Advanced Subsidiary, *BMI*, body mass index (calculated as weight in kilograms divided by height in meters squared), *GCSE* General Certificate of Secondary Education, *O* Ordinary, *FEV1* forced expiratory volume in one second, *FVC* forced vital capacity, *SD* standard deviation.

The adverse effects of air pollutants on the lungs may be explained by autophagy, dysregulated cell immunity, epigenetic modifications, impaired of phagocytosis, inflammatory cell infiltration, disruption of cellular signaling pathways and the airway epithelial barrier, oxidative stress, and parenchymal destruction[4]. However, previous studies have yielded inconsistent results regarding the impact of air pollution on asthma prevalence and lung function. To our knowledge, most studies have focused on young people, resulting in a limited understanding of adults. Children may be more susceptible to detrimental respiratory ambient air pollution than adults are, particularly through its effect on lung development[15]. Asthma in adults is characterized as less atopic and more

Pollution	N	Mean (SD)	Minimum	Maximum	IQR	Pearson correlation coefficients				
		μg/m³	μg/m³	μg/m³	μg/m³	$PM_{2.5}$	PM_{coarse}	PM_{10}	NO_2	NO_x
$PM_{2.5}$	45,4291	9.99 (1.06)	8.17	21.31	1.27	1	0.221	0.532	0.865	0.848
PM_{coarse}	45,4291	6.43 (0.90)	5.57	12.82	0.80		1	0.817	0.200	0.238
PM_{10}	45,4291	16.23 (1.90)	11.78	31.39	1.76			1	0.505	0.512
NO_2	45,4291	26.62 (7.61)	12.93	108.49	9.84				1	0.922
NO_x	45,4291	43.95 (15.59)	19.74	265.94	16.54					1

Table 2. Descriptive statistics of air pollutants. *SD* standard deviation, *IQR* interquartile range, $PM_{2.5}$ particulate matter with diameter < 0.25 μm, PM_{10} particulate matter with diameter < 10 μm, PM_{coarse} particulate matter with diameter between 2.5 μm and 10 μm, NO_2 nitrogen dioxide, NO_x nitrogen oxides.

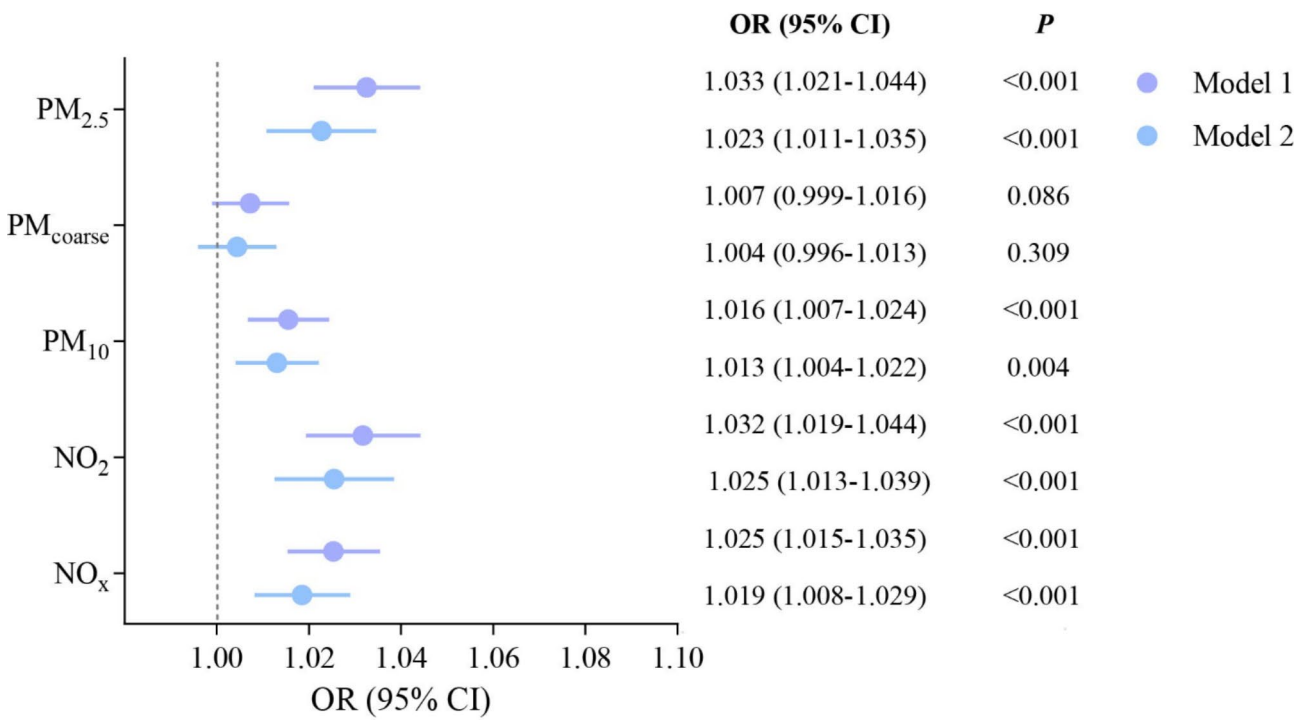

Fig. 1. Associations between ambient air pollution exposure (per IQR increase) and the odds of asthma. Model 1 was crude. Model 2 was adjusted for age, sex, ethnicity, body mass index, household income, education level, smoking status, and allergy history. The IQRs of $PM_{2.5}$, PM_{coars}, PM_{10}, NO_2, and NO_x were 1.27, 0.80, 1.76, 9.84, and 16.54, respectively. *OR* odds ratio, *CI* confidence interval, *IQR* interquartile range, $PM_{2.5}$ particulate matter with diameter < 0.25 μm, PM_{10} particulate matter with diameter < 10 μm, PM_{coarse} particulate matter with diameter between 2.5 μm and 10 μm, NO_2 nitrogen dioxide, NO_x nitrogen oxides.

complex than asthma in children[16], suggesting a difference between children and adults with asthma and that asthma in adults is more likely to be influenced by factors other than atopy-related factors, such as air pollution[17]. Therefore, the analysis of associations between air pollution and asthma in adults is warranted.

Different studies of different air pollutants have yielded inconsistent results regarding the association between air pollution and asthma prevalence. A retrospective cross-sectional study with 11,585 individuals found that exposure to higher ambient NO_2 but not $PM_{2.5}$, PM_{10}, or SO_2 was associated with increased asthma prevalence among Taiwanese adults[17]. Furthermore, data from three European cohort studies found that PM_{10} and NO_2 was associated with the prevalence of asthma among adults[18]. In the present study, exposure to air pollutants, including $PM_{2.5}$, PM_{10}, NO_2, and NO_x, but not PM_{coarse} significantly increased the prevalence of asthma with greater impacts on older participants, females, obese individuals, and current smokers. The explanations for the sex differences may include the higher prevalence of asthma in females, the influence of female hormones, and anatomical differences between males and females, which may strengthen the association between air pollution and asthma prevalence in women[19]. As individuals age, the natural decrease in lung function and the weakening of the immune system may increase the vulnerability of the respiratory system to air pollution, resulting in a more pronounced effect in older populations[20]. Moreover, a large 16-year follow-up cohort showed that exposure to smoking increased the risk of asthma for both active smokers and passive smokers[21], which helped explain the

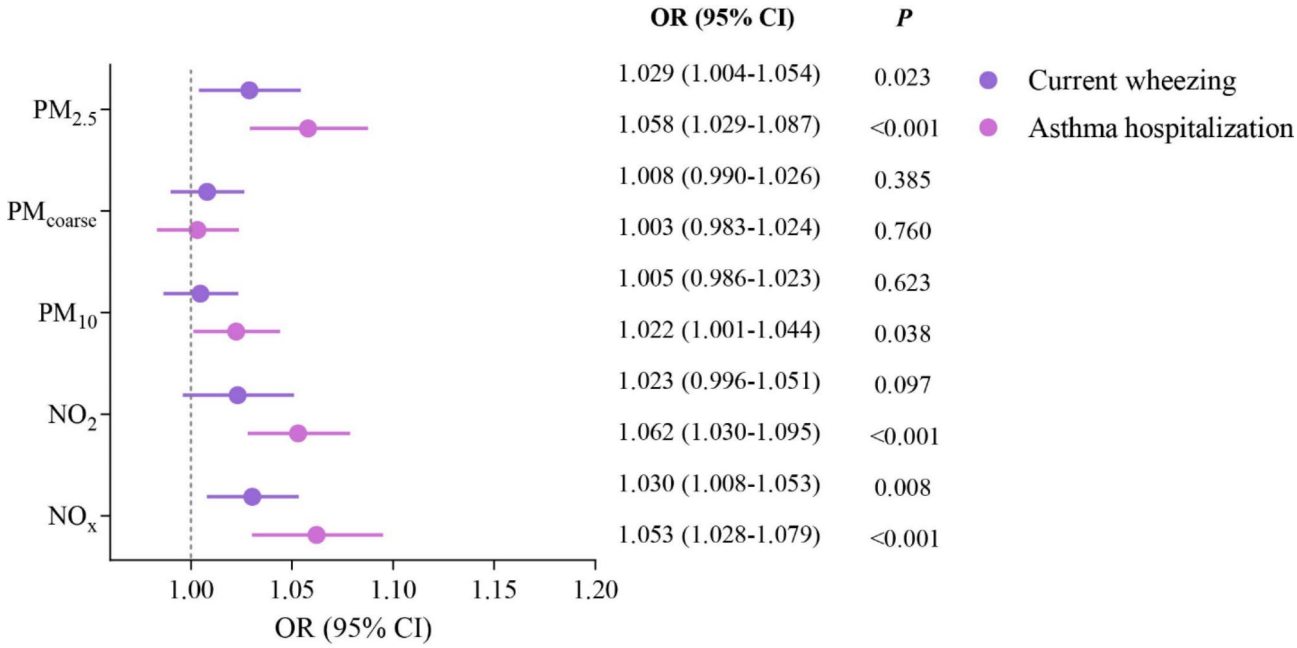

Fig. 2. Associations between ambient air pollution exposure (per IQR increase) and the odds of wheezing or hospitalization among asthma patients. Adjusted for age, sex, ethnicity, body mass index, household income, education level, smoking status, and asthma medication. The IQRs of $PM_{2.5}$, PM_{coars}, PM_{10}, NO_2, and NO_x for current wheezing were 1.27, 0.81, 1.74, 9.98, and 16.76, respectively. The IQRs of $PM_{2.5}$, PM_{coars}, PM_{10}, NO_2, and NO_x for asthma hospitalization were 1.28, 0.81, 1.74, 9.98, and 16.77, respectively. *OR* odds ratio, *CI* confidence interval, *IQR* interquartile range, $PM_{2.5}$ particulate matter with diameter < 0.25 μm, PM_{10} particulate matter with diameter < 10 μm, PM_{coarse} particulate matter with diameter between 2.5 μm and 10 μm, NO_2 nitrogen dioxide, NO_x nitrogen oxides.

results observed in the smoking subgroups. Moreover, it is generally accepted that obesity is also a risk factor for asthma in adults[22], which may make the effects of air pollution on asthma prevalence more pronounced in obese individuals. Owing to the variability of this association among populations, additional research is necessary to confirm the disparities in this relationship and clarify the potential underlying causes.

Air pollution may also affect asthma outcomes, such as current wheezing, asthma-related hospitalization. A systematic review that encompassed 22 studies and utilized a case-crossover design concluded that $PM_{2.5}$, NO_2, CO, and O_3 but not SO_2 or PM_{10} were significantly linked with asthma exacerbation including visits to emergency departments and hospitalizations[11]. Another systematic review of 67 studies observed that short-term exposure to O_3, NO_2, and SO_2 was related to increased odds of asthma exacerbations including hospital admissions and asthma-associated emergency room visits[23]. Similarly, our results revealed that exposure to air pollutants, especially $PM_{2.5}$, NO_2 and NO_x was associated with increased odds of current wheezing and asthma hospitalization, indicating that air pollution may increase the risk of asthma exacerbation.

The associations between air pollution and lung function have also been assessed in previous studies with different conclusions. Some studies have shown that air pollution consisting of $PM_{2.5}$, PM_{10}, and NO_2 was linked to a decrease in lung function[24–26]. However, a Tasmanian Longitudinal Health Study revealed no significant interactions between NO_2 and asthma prevalence and subsequent changes in lung function[27]. In addition, a prospective single-center panel study did not find significant evidence that exposure to air pollution (NO_2/O_3) affects clinical control including lung function among individuals with asthma, however, the sample size was small, with only 32 participants included[8]. Our results found that exposure to $PM_{2.5}$, PM_{coarse}, PM_{10}, NO_2 and NO_x was significantly associated with a decrease in FEV1, FVC, and FEV1/FVC. Moreover, the associations between air pollution and asthma prevalence, current wheezing, hospitalization, and lung function exhibited partial inconsistencies across multiple studies, possibly due to differing measures of air pollutants, diverse populations, or varying definitions of outcomes. Further research, as well as longitudinal studies, is needed to explore the relationships among these factors to investigate whether there is a causal association.

The major strength of this study is that the well-organized and rigorously defined large population-based analysis provided adequate statistical power to evaluate impacts of air pollution on asthma and lung function in adults. Nevertheless, there are several limitations to this study. First, the cross-sectional design limits the value of the results in establishing causal associations. Second, the definition of asthma outcomes was not strict enough due to the form of the subjective questionnaire. Third, estimation of the effects of air pollution focused on individual pollutants, which may not accurately reflect the real-world presence of air pollution mixtures. Fourth, owing to data limitations, postbronchodilator lung function data are not available. Finally, our findings were analyzed among individuals aged 37 to 73 years; therefore, the conclusions may not be generalizable to children and adolescents.

Air pollution (Per IQR)	Total asthma patients (*N* = 28,813)		Female (*N* = 17,450)		Male (*N* = 11,363)	
	β (95% CI)	*P*	β (95% CI)	*P*	β (95% CI)	*P*
$PM_{2.5}$						
FEV1 (mL)	− 7.389 (− 15.303 ~ 0.524)	0.067	− 10.906 (− 19.475 ~ − 2.338)	0.013	− 14.041 (− 29.139 ~ 1.057)	0.068
FVC (mL)	− 5.858 (− 15.141 ~ 3.424)	0.216	− 9.035 (− 19.146 ~ 1.076)	0.080	− 15.335 (− 32.985 ~ 2.315)	0.089
FEV1/FVC (%)	− 0.133 (− 0.234 ~ − 0.033)	0.009	− 0.170 (− 0.290 ~ − 0.050)	0.005	− 0.128 (− 0.303 ~ 0.047)	0.152
PM_{coarse}						
FEV1 (mL)	− 5.143 (− 10.705 ~ 0.420)	0.070	− 3.044 (− 9.128 ~ 3.041)	0.327	− 8.687 (− 19.233 ~ 1.858)	0.106
FVC (mL)	− 11.744 (− 18.267 ~ − 5.221)	< 0.001	− 6.158 (− 13.337 ~ 1.021)	0.093	− 20.182 (− 32.506 ~ − 7.858)	0.001
FEV1/FVC (%)	0.076 (0.005 ~ 0.147)	0.035	0.050 (− 0.035 ~ 0.135)	0.250	0.110 (− 0.012 ~ 0.232)	0.078
PM_{10}						
FEV1 (mL)	− 7.614 (− 13.599 ~ − 1.630)	0.013	− 5.428 (− 11.967 ~ 1.110)	0.104	− 14.531 (− 25.891 ~ − 3.170)	0.012
FVC (mL)	− 15.637 (− 22.655 ~ − 8.618)	< 0.001	− 9.700 (− 17.414 ~ − 1.985)	0.014	− 28.821 (− 42.095 ~ − 15.548)	< 0.001
FEV1/FVC (%)	0.066 (− 0.010 ~ 0.142)	0.090	0.043 (− 0.048 ~ 0.135)	0.352	0.084 (− 0.048 ~ 0.216)	0.210
NO_2						
FEV1 (mL)	− 13.266 (− 21.881 ~ − 4.650)	0.003	− 14.348 (− 23.742 ~ − 4.954)	0.003	− 19.125 (− 35.440 ~ − 2.811)	0.022
FVC (mL)	− 13.041 (− 23.147 ~ − 2.935)	0.011	− 12.994 (− 24.080 ~ − 1.909)	0.022	− 22.428 (− 41.500 ~ − 3.356)	0.021
FEV1/FVC (%)	− 0.146 (− 0.256 ~ − 0.037)	0.009	− 0.175 (− 0.306 ~ − 0.043)	0.009	− 0.132 (− 0.321 ~ 0.057)	0.171
NO_x						
FEV1 (mL)	− 9.440 (− 16.607 ~ − 2.272)	0.010	− 12.421 (− 20.268 ~ − 4.573)	0.002	− 12.824 (− 26.302 ~ 0.655)	0.062
FVC (mL)	− 9.063 (− 17.471 ~ − 0.655)	0.035	− 11.123 (− 20.383 ~ − 1.862)	0.019	− 15.482 (− 31.238 ~ 0.274)	0.054
FEV1/FVC (%)	− 0.118 (− 0.209 ~ − 0.027)	0.011	− 0.164 (− 0.274 ~ − 0.055)	0.003	− 0.084 (− 0.240 ~ 0.073)	0.294

Table 3. Multivariable analysis of ambient air pollution exposure and lung function measures among asthmatic patients. All models were adjusted for age, sex, ethnicity, body mass index, household income, education level, smoking status, asthma medication. Models for FEV1 and FVC were additionally adjusted for height and height squared. The IQRs of $PM_{2.5}$, PM_{coars}, PM_{10}, NO_2, and NO_x were 1.27, 0.80, 1.76, 9.84, and 16.54, respectively. *IQR* interquartile range, $PM_{2.5}$ particulate matter with diameter < 0.25 µm, PM_{10} particulate matter with diameter < 10 µm, PM_{coarse} particulate matter with diameter between 2.5 µm and 10 µm, NO_2 nitrogen dioxide, NO_x nitrogen oxides, *FEV1* forced expiratory volume in one second, *FVC* forced vital capacity.

Conclusion

Our study revealed that increased air pollution was associated with increased asthma prevalence, with greater impacts on older participants, females, obese individuals, and current smokers. Among individuals with asthma, increased air pollution exposure was linked to increased odds of current wheezing, asthma-related hospitalizations, and decreased lung function. To further confirm the causal relationship between air pollution and asthma, longitudinal cohort studies are necessary, particularly in subgroups with large effect sizes.

Data availability

The data underlying this article was provided by UK Biobank under license. Data will be shared on request to the corresponding author with permission of UK Biobank.

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Declarations

Competing interests

The authors declare no competing interests.

Ethics approval

Informed written consents were provided from all the participants, while the ethics approval was granted from the North West Multicenter Research Ethics Committee (Reference 16/NW/0274; Application Number 84979). All methods were performed in accordance with the Declaration of Helsinki and relevant guidelines, complying with the necessary ethical standards.

Additional information

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