Ambient air pollution, lung function and COPD: cross-sectional analysis from the WHO Study of AGEing and adult health wave 1

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ABSTRACT

Background Long-term exposure to ambient air pollution leads to respiratory morbidity and mortality; however, the evidence of the effect on lung function and chronic obstructive pulmonary disease (COPD) in older adult populations is inconsistent.

Objective To address this knowledge gap, we investigated the associations between particulate matter (PM), nitrogen dioxide (NO2) exposure and lung function, as well as COPD prevalence, in older Chinese adults.

Methods We used data from the WHO Study on global AGEing and adult health (SAGE) China Wave 1, which includes 11,693 participants from 64 townships in China. A cross-sectional analysis explored the association between satellite-based air pollution exposure estimates (PM with an aerodynamic diameter of ≤10 μm [PM10], ≤2.5 μm [PM2.5] and NO2) and forced expiratory volume in one second (FEV1), forced vital capacity (FVC), the FEV1/FVC ratio and COPD (defined as post-bronchodilator FEV1/FVC < 0.70). Data on lung function changes were further stratified by COPD status.

Results Higher exposure to each pollutant was associated with lower lung function. An IQR (26.1 μg/m3) increase in PM10 was associated with lower FEV1 (−71.88 mL, 95% CI −92.13 to −51.64) and FEV1/FVC (−2.81, 95% CI −3.37 to −2.25). For NO2, an IQR increment of 26.8 μg/m3 was associated with decreases in FEV1 (−60.12 mL, 95% CI −82.40 to −36.23) and FVC (−32.33 mL, 95% CI −56.35 to −8.32). A 31.2 μg/m3 IQR increase in PM2.5 was linked to reduced FEV1 (−8.86 mL, 95% CI −5.40 to 23.11) and FEV1/FVC (−1.85, 95% CI −2.24 to −1.46). These associations were stronger for participants with COPD. Also, COPD prevalence was linked to higher levels of PM10 (POR 1.04, 95% CI 0.98 to 1.11) and NO2 (POR 1.04, 95% CI 1.26 to 1.43), PM2.5 (POR 1.24, 95% CI 1.18 to 1.29) and NO2 (POR 1.26, 95% CI 1.21 to 1.35).

Conclusion Ambient air pollution was associated with lower lung function, especially in individuals with COPD, and increased COPD prevalence in older Chinese adults.

INTRODUCTION

Air pollution represents a significant public health threat, causing over 4 million premature deaths worldwide.1 The full list of medical conditions either mediated or caused directly by ambient air pollution is long and is a subject of active research.2 Yet some of the first and best-established health effects attributed to air pollution are those affecting the respiratory system.1 According to data from the Global Burden of Disease study, 43% of lung diseases, including lung cancer, chronic obstructive pulmonary disease (COPD) and lower respiratory infections, are attributed to elevated levels of air pollutants leading to more than 1.8 million annual deaths.3 COPD is one of the leading causes of death and disability globally,4 with an estimated worldwide prevalence of 10%.5 It has been established that both indoor and outdoor air pollution worsen COPD.6 7 8

A large proportion of deaths related to both ambient air pollution and airway diseases...
Outcome.\textsuperscript{17,18} The heterogeneity of the results from to find any significant links between the exposure and worsening in some publications,\textsuperscript{15,16} whereas others failed to meet the air quality guidelines recommended by the WHO.\textsuperscript{12} Older adults are known to be more susceptible to the detrimental health effects of air pollution.\textsuperscript{13} This age group has a higher incidence and prevalence of chronic, progressive lung disorders, such as COPD.\textsuperscript{8,14} These factors make older adults particularly susceptible to suffering from rapid urbanisation and increased air pollution emissions.

Although the association between ambient air pollution and impaired lung function as well as COPD seems logical and intuitive given the clear associations with a broad range of respiratory mortality and morbidity outcomes, the data regarding the type of pollutants and clinical endpoints have not been consistently measured. For example, air pollutants have been associated with various degrees of lung function impairment and COPD worsening in some publications,\textsuperscript{15,16} whereas others failed to find any significant links between the exposure and the outcome.\textsuperscript{17,18} The heterogeneity of the results from previous studies can be partially explained by variations in population sampling methods, exposure estimation techniques, and measurement and control of important confounders. The lack of studies coming from developing countries, including China, is one of the most significant gaps in the data. To address these limitations in the existing literature, we aimed to investigate the long-term effects of outdoor air pollution on the lung function and COPD prevalence in a nationally representative population of older Chinese adults.

METHODS

Study population

The WHO Study on global AGEing and adult health (SAGE) is a nationally representative, cross-sectional survey of China. Full details on the study methodology are available elsewhere.\textsuperscript{15} Briefly, in the SAGE China survey, information was collected from Chinese adult respondents through an interview between 2007 and 2010.\textsuperscript{15,16} As a result of a multistage random sampling strategy, a total of 11,693 adult participants, aged 50 years and older, were ultimately included in the analysis (Figure S1).

Patient involvement

The study design, analysis, interpretation of the results and the writing or editing of this research paper were made without patient involvement.

Outcome measurement

As part of SAGE China Wave 1, lung function tests were performed using a MIR SpiroDoc Diagnostic Portable Spirometer (Medical International Research, Rome, Italy). Participants were instructed to wear a nose clip, and tests were conducted in a sitting position until three satisfactory spirometric measurements were obtained.\textsuperscript{20} Forced vital capacity (FVC), forced expiratory volume in one second (FEV\textsubscript{1}) and the FEV\textsubscript{1}/FVC ratio were calculated from the averages of the three readings. COPD was defined as a post-bronchodilator FEV\textsubscript{1}/FVC less than 70%.\textsuperscript{21}

Exposure assessment

Data concentrations of particulate matter (PM) and nitrogen dioxide (NO\textsubscript{2}) were predicted through models using combined data from satellite remote sensing, meteorology, land use information and ground monitoring on the pollutants from stations throughout China. From 2004 to 2016, measurements of pollutants’ daily ground-level concentrations were obtained from 1479 stations of the China National Environmental Monitoring Centre. A detailed description of PM and NO\textsubscript{2} predictions from these models has been published previously.\textsuperscript{22–24}

Using the monitor data, 10-fold validation was applied to evaluate the predictive ability of the models for both PM and NO\textsubscript{2}. The adjusted coefficient of determination (R\textsuperscript{2}) and root-mean-squared error are presented in online supplemental table S1.

Exposure to long-term outdoor air pollution was defined as the average concentration of PM and NO\textsubscript{2} for the 3 years (2005–2007). Lastly, we geocoded the community locations of participants and linked these to the estimated annual concentrations of PM and NO\textsubscript{2}.

Covariates

Demographic, behavioural and socioeconomic confounders and potential effect modifiers were identified a priori through a literature search.\textsuperscript{25,26} Demographic confounders included age, sex, body mass index (BMI) and marital status. Behavioural variables were smoking status (never, current, former), and tobacco consumption (mean daily tobacco consumption), alcohol use (never or ever), fruit and vegetable consumption (sufficient and insufficient), and physical activity (low, moderate and high). Socioeconomic variables involved education level and household income. The level of education based on the participants’ self-reporting was divided into the following categories: (1) no formal education, (2) primary school, (3) middle school, (4) college or higher. Household income was categorised into two levels (low or high) using a median income of 15,000 Chinese yuan (¥) as a threshold. Additionally, we considered the place of residence as either urban or rural. Domestic fuel type
was included as an indicator of indoor air pollution. Two fuel types were mainly used: clean fuels, including electricity and natural gas, and unclean fuels, such as coal, wood, dung and agricultural residues.

**Statistical analysis**

We examined the association between lung function metrics and an IQR increase in 3-year moving averages of PM$_{10}$, PM$_{2.5}$, and NO$_2$ in single pollutant models. To assess for potential dependence in lung function measurements for participants in the same community, data clustering was accounted using a two-level linear regression model, where participants were considered as the first-level unit and the community as the second-level unit. The effect estimates were expressed as absolute differences in lung function measures associated with each IQR (μg/m$^3$) increase in ambient PM$_{2.5}$, PM$_{10}$, or NO$_2$ concentrations.

For models using COPD as an outcome, logistic regression may overestimate the prevalence ratio, given a high prevalence of COPD in the study sample (19.5%). Therefore, we used a modified Poisson regression with robust error variance to directly estimate the prevalence OR of COPD. In fully adjusted models, we controlled for age, sex, BMI, smoking status (never, current, former), tobacco consumption, physical activity, education level, daily fruit and vegetable intake, alcohol use, place of residence, type of fuel used at home and median annual household income. Additionally, several variables were investigated as potential effect modifiers (age, sex, smoking and asthma history, and household income). Interactions were assessed by including multiplicative terms between pollutant variables and several potential effect modifiers into the models. Significance of effect modification was determined if the $p$ value for the hypothesis test of the interaction was $<0.01$.

Sensitivity analyses were performed. First, average pollutants concentrations were used for 1 and 5 years before the baseline survey to ensure that our results were not being driven primarily by our selection of an exposure window. Second, we excluded the participants with cardiovascular diseases and additionally adjusted for city-specific covariates, such as gross domestic product (GDP) and percentage of the population living in urban areas. All analyses were conducted using STATA V.15 (StatCorp, College Station, Texas, USA) and $p$ value $<0.05$ was used to determine statistical significance.

**RESULTS**

Characteristics for participants with complete data in fully adjusted lung function models are summarised in table 1. The mean age of participants with COPD was 64 years, and 61 for participants without COPD. About 51% of the participants were female, and the majority were living in rural areas (57%), reported never smoking (65%), and came from households earning less than ¥15,000 annually (54%). Some 3.5% of study subjects had been previously diagnosed with asthma, and 52% were currently using unclean fuel at their homes. Lastly, COPD prevalence was 19.5%.

Online supplemental table S2, figures S1–S3 show the distribution of residential ambient air pollution concentrations. Mean (±SD) annual estimates of PM$_{10}$, PM$_{2.5}$, and NO$_2$ were 91.11 (±28.95 μg/m$^3$), 54.02 (±17.02 μg/m$^3$) and 28.97 (±22.42 μg/m$^3$), respectively. NO$_2$ concentrations were highly correlated with PM$_{2.5}$ ($r=0.92$), but less so with PM$_{10}$.

**Lung function and air pollution: full cohort**

In the full cohort, including both non-COPD and COPD participants, an IQR increase in the 3-year moving average of all pollutants showed adverse associations with lung function (table 2). In adjusted models, an IQR increase in PM$_{10}$ exposure was associated with lower FEV$_1$ ($−29.08$ mL, 95% CI $−43.26$ to $−14.89$) and FEV$_1$/FVC ratio ($−1.85$, 95% CI $−2.24$ to $−1.46$). For each IQR increase in NO$_2$, lower FEV$_1$ ($−60.12$ mL, 95% CI $−84.00$ to $−36.23$) and FVC ($−32.33$ mL, 95% CI $−56.35$ to $−8.32$) was observed. Furthermore, results showed negative associations between PM$_{2.5}$ concentrations and lung function, with stronger effects on FEV$_1$ than FVC. The FEV$_1$/FVC ratio showed no association with ambient NO$_2$ exposure. For all pollutants, significant effect modification by BMI and lower household income was identified (all $p_{interact} <0.001$). For NO$_2$ and PM$_{2.5}$, older age (>65 years) was linked with lower FEV$_1$. Associations were further investigated in non-COPD and COPD participants separately.

**COPD and air pollution**

Consistent with our findings for lung function, we found an IQR increase in the 3-year moving average of PM$_{2.5}$ and PM$_{10}$ to be associated with 35% and 24% increased prevalence odds ratio (POR) of COPD (POR 1.35, 95% CI 1.26 to 1.43; POR 1.24, 95% CI 1.18 to 1.29), respectively, when COPD status was treated as a binary outcome (figure 1). A nominally positive, non-significant, association was observed for NO$_2$. Effect modification for PM$_{2.5}$ was seen in former smokers ($p=0.04$).

**Lung function and air pollution: non-COPD participants**

In participants without COPD, an IQR increase in each pollutant showed significant inverse associations with FEV$_1$ (PM$_{10}$ $−25.31$, β 95% CI $−36.81$ to $−13.8$; PM$_{2.5}$ $−53.81$, β 95% CI $−107.09$ to $−0.53$; and NO$_2$ $−74.88$, β 95% CI $−146.61$ to $−3.15$). However, an IQR increment in 3-year moving averages of PM$_{10}$, PM$_{2.5}$ and NO$_2$ was not significantly associated with FVC (table 3).

**Lung function and air pollution: COPD participants**

In participants with COPD, all pollutants were statistically significantly associated with decrements in FEV$_1$ and FVC. Additionally, the magnitude of association was more substantial than that found in non-COPD participants (table 3), with an IQR increase in PM$_{10}$,
PM$_{2.5}$ and NO$_2$ associated with FEV$_1$ −25.31 mL (95% CI −36.81 to −13.80), −53.81 mL (95% CI −107.09 to −0.53) and −74.88 mL (95% CI −146.61 to −3.15), respectively. Smaller magnitudes of association were observed in FVC measurement (table 3).

Results of PM$_{10}$, PM$_{2.5}$ and NO$_2$ subgroup analyses for COPD and FEV$_1$ are shown in tables 4 and 5, respectively. FEV$_1$-stratified analyses showed stronger associations for PM$_{10}$ and NO$_2$ in older participants, subjects from lower-income households, and individuals with a history of asthma. The same effect modification patterns were observed for FVC-stratified analyses (data not shown).

Participants from lower-income households had mean FEV$_1$ levels three-to-five times lower than higher-income households.

### Table 1: Population baseline characteristics

|                        | No COPD±SD or % | COPD±SD or % | Total population | Difference p value |
|------------------------|-----------------|--------------|------------------|-------------------|
| NO$_2$ (µg/m$^3$)      | 31.20 (12.86)   | 30.11 (10.07)| 30.99 (12.36)    | 0.23              |
| PM$_{2.5}$ (µg/m$^3$)  | 55.38 (0.81)    | 58.65 (0.80 )| 56.02 (14.88)    | 0.10              |
| PM$_{10}$ (µg/m$^3$)   | 94.46 (1.19)    | 101.92 (1.39 )| 95.93 (27.61)    | 0.02              |
| Age (years)            | 61.05 (8.57)    | 64.45 (9.59 )| 61.72 (8.89)     | 0.16              |
| FVC (L)                | 2.34 (0.78)     | 2.30 (0.94)  | 2.33 (0.81)      | 0.05              |
| FEV$_1$ (L)            | 2.06 (0.02)     | 1.47 (0.03)  | 1.95 (0.78)      | 0.007             |
| FEV$_1$/FVC (%)        | 87.87 (8.28)    | 50.46 (20.02)| 80.49 (18.88)    | 0.03              |
| Height (cm)            | 159.45 (8.73)   | 158.63 (8.68 )| 159.29 (8.72)    | 0.56              |
| BMI                    | 24.26 (4.97)    | 23.60 (4.36) | 24.13 (4.87)     | 0.81              |
| Sex                    | 0.57            | 0.57         | 0.57             |                   |
| Male                   | 4848 (49.24)    | 1207 (49.92 )| 6055 (49.37)     |                   |
| Female                 | 4998 (50.76)    | 1210 (50.08 )| 6209 (50.63)     |                   |
| Place or residence     | 0.85            | 0.85         | 0.85             |                   |
| Urban                  | 4173 (42.38)    | 1040 (43.02)| 5213 (42.51)     |                   |
| Rural                  | 5673 (57.62)    | 1377 (56.98 )| 7051 (57.49)     |                   |
| Physical activity      | 0.004           | 0.004        | 0.004            |                   |
| Low                    | 2623 (76.97)    | 784.7 (23.03)| 3407 (27.79)     |                   |
| Moderate               | 2597 (81.43)    | 592.1 (18.57)| 3189 (26.01)     |                   |
| High                   | 4624 (81.64)    | 1040 (18.36)| 5664 (46.2)      |                   |
| Smoking status         | 0.34            | 0.34         | 0.34             |                   |
| Never                  | 6248 (80.86)    | 1479 (19.14 )| 7728 (63.28)     |                   |
| Current                | 2943 (79.65)    | 752 (20.35)  | 3695 (30.26)     |                   |
| Former                 | 620.4 (78.57)   | 169.2 (21.43)| 789.6 (6.466)    |                   |
| Asthma status          | 0.002           | 0.002        | 0.002            |                   |
| No                     | 9452 (80.92)    | 2228 (19.08)| 11692 (96.44)    |                   |
| Yes                    | 282.7 (65.49)   | 148.9 (34.51)| 431.6 (3.56)     |                   |
| Annual household income (¥) | 0.002 | 0.002 | 0.002 |                   |
| ≤15000                 | 5065 (52.66)    | 1424 (60.89)| 6489 (54.27)     |                   |
| >15000                 | 4554 (47.34)    | 914.6 (39.11)| 5468 (45.73)     |                   |
| Fuel used at home      | 0.44            | 0.44         | 0.44             |                   |
| Clean                  | 4787 (81.21)    | 1107 (18.79)| 5894 (48.34)     |                   |
| Unclean                | 5013 (79.57)    | 1287 (20.43)| 6300 (51.66)     |                   |
| Level of education     | 0.07            | 0.07         | 0.07             |                   |
| No formal education    | 4071 (41.61)    | 1080 (42.31)| 5152 (42.31)     |                   |
| Primary school         | 2047 (20.92)    | 502.1 (20.93)| 2549 (20.93)     |                   |
| Middle school          | 2127 (21.74)    | 418.4 (20.93)| 2545 (20.91)     |                   |
| College or higher      | 1540 (15.74)    | 390.6 (15.85)| 1930 (15.85)     |                   |

BMI, body mass index; FEV$_1$, forced expiratory volume in 1 second; FVC, forced vital capacity; NO$_2$, nitrogen dioxide; PM$_{10}$, particulate matter with an aerodynamic diameter ≤10 µm; PM$_{2.5}$, particulate matter with an aerodynamic diameter ≤2.5 µm.
Reduced FEV1 was linked to all three pollutants; PM 2.5 was associated with lower FEV1/FVC ratio, while NO2 was associated with lower prevalence of COPD among older Chinese adults. Impaired lung function, particulate matter with an aerodynamic diameter less than or equal to 10 µm, PM10 was associated with increased prevalence of COPD, whereas we evaluated older adults from China. Also, the estimates among participants with COPD, suggesting that this may be a particularly susceptible group to the deleterious pulmonary effects of air pollution.

COPD prevalence

Air pollution has been recognised as a likely risk factor for COPD, although only a small number of studies have investigated this association in China. A cross-sectional study of almost 6000 residents of Guangdong province showed statistically significant associations between elevated levels of PM2.5 and PM10, and increased prevalence of COPD. For daily mean PM2.5 >75 mg/m³ and PM10 >50 to ≤150 mg/m³ the adjusted OR for COPD prevalence was 2.530 (95% CI 1.280 to 5.001) and 2.442 (95% CI 1.449 to 4.117), respectively. Accordingly, the present population-based analysis showed 35% (POR 1.35, 95% CI 1.26 to 1.43) and 24% (POR 1.24, 95% CI 1.18 to 1.29) increase in COPD prevalence due to high exposure to PM2.5 and PM10, respectively. Presumably, the difference in prevalence is due to distinct distribution of study populations. Likewise, in a large-scale (n=285 046) longitudinal cohort with 13 years of follow-up, Guo et al found a higher incidence of COPD among those exposed to higher PM2.5 concentrations.

The negative effect of the long-term exposures to PM2.5 and PM10 is further supported by the results from Western countries; however, findings regarding COPD and air pollution are not consistent. For example, a meta-analysis of four large European cohorts did not demonstrate any significant associations between prevalence and incidence of COPD and different air pollutants. We found no significant associations of COPD prevalence with NO2, which contradicts the findings from the UK Biobank study and a US cohort study.

This heterogeneity in findings may be explained by different outcome and exposure assessments, or uncontrolled confounding in some studies. Most of the evidence supporting the positive association between NO2 and COPD incidence or prevalence is derived from research on the general population of adults from Western countries, whereas we evaluated older adults from China. Also, the

### Table 2 Associations of lung function and ambient air pollution exposure

| Pollutant | FEV1 (mL) β (95% CI) | FVC (mL) β (95% CI) | FEV1/FVC (%) β (95% CI) |
|-----------|----------------------|----------------------|-------------------------|
| PM10      | –29.08 (–43.26 to 14.89)* | –8.86 (–5.40 to 23.11) | –1.85 (–2.24 to –1.46)* |
| PM2.5     | –71.88 (–92.13 to 51.64)* | –11.26 (–31.64 to 9.11) | –2.81 (–3.37 to –2.25)* |
| NO2       | –60.12 (–84.00 to 36.23)* | –32.33 (–56.35 to 8.32)* | –0.39 (–1.05 to 0.28) |

*p < 0.05. Models adjusted for age, sex, smoking status, daily tobacco consumption, physical activity, education, BMI, alcohol, place of residence, household income, type of indoor fuel use, daily fruit and vegetables consumption. IQR PM10: 3 years: 31.2 µg/m³; IQR PM2.5: 3 years: 26.1 µg/m³; IQR NO2: 3 years: 26.8 µg/m³. BMI, body mass index; NO2, nitrogen dioxide; PM10, particulate matter with an aerodynamic diameter less than or equal to 10 µm; PM2.5, particulate matter with an aerodynamic diameter less than or equal to 2.5 µm.
A large longitudinal Taiwanese cohort showed a 0.21% decrease in FEV1/FVC with 5-year exposure to PM10, 53.81 (95% CI: 107.09 to 0.53) in PM2.5, and 74.88 (95% CI: 146.61 to 3.15) in NO2. The present results also indicated direct correlations between lower-income households (≤¥15,000) and decreased FEV1/FVC for all three pollutants. This finding illustrates the enhanced effects of air pollution on low-income populations, which can be justified by higher exposure to common health risk factors, such as tobacco smoking, obesity, or gender, with poorer living conditions, poorer nutrition, and more frequent childhood infections and worse access to healthcare services. The discrepancies in the publications mentioned above may arise from several possible explanations. First, studies varied in the populations assessed and the geographic regions studied. The adjustment for potential confounders was not consistent in all studies, and it is not entirely clear to what extent it could have influenced the outcomes. Second, the exposure assessment of the pollutants differed among cohorts, which might have also impacted the results. Third, in some publications, the heterogeneities between included studies and subgroups could have significantly contributed to the statistical insignificance. Fourth, differences in applied statistical methods (with their respective limitations) might also have played a role in variation in the outcome assessment. Fifth, additive effects of co-pollutants, which is highly likely to vary across world regions, could have influenced the magnitude of the associations within the various endpoints. Last, and possibly most importantly, almost no study assessed different phenotypes of COPD, which can be a critical factor for data stratification.
COPD versus non-COPD

The negative impact of increased exposure to air pollutants on the respiratory function was more pronounced in participants with COPD compared with those without the disease. One of the primary explanations for this finding is the fact that individuals with COPD have already decreased pulmonary physiological function.39 This group has a higher risk of developing other respiratory diseases, including airway infections and lung cancer, than the general population.21 Moreover, individuals with COPD often have other chronic comorbidities, such as cardiovascular disease, as well as mental or metabolic disorders.40 Studies showed that patients with COPD have elevated levels of specific inflammatory markers at the

| Characteristics | PM$_{2.5}$ (95% CI) | PM$_{10}$ (95% CI) | NO$_x$ (95% CI) |
|-----------------|---------------------|-------------------|-----------------|
| **Sex**         |                     |                   |                 |
| Male            | −78.92              | −31.58            | −42.65          |
|                 | (−113.58 to 44.25)  | (−55.93 to 7.23)  | (−83.35 to 1.94)|
| Female          | −63.22              | −25.84            | −73.12          |
|                 | (−87.08 to 39.37)   | (−42.55 to 9.12)  | (−101.05 to 45.19)|
| Interaction p value | 0.27               | 0.828             | 0.49           |
| **Age**         |                     |                   |                 |
| <65 years old   | −84.92              | −42.90            | −35.34          |
|                 | (−111.68 to 58.16)  | (−61.91 to 23.88) | (−65.92 to 4.76)|
| ≥65 years old   | −62.14              | −16.51            | −102.16         |
|                 | (−94.85 to 29.43)   | (−39.04 to 6.03)  | (−141.92 to 62.39)|
| Interaction p value | 0.15               | 0.05              | 0.04           |
| **History of asthma** |                 |                   |                 |
| Yes             | −113.49             | −36.61            | −173.72         |
|                 | (−214.69 to 12.29)  | (−106.47 to 33.26)| (−309.50 to 37.94)|
| No              | −72.72              | −29.76            | −61.51          |
|                 | (−93.90 to 51.55)   | (−44.61 to 14.91) | (−86.12 to 36.91)|
| Interaction p value | 0.8                | 0.95              | 0.30           |
| **Smoker**      |                     |                   |                 |
| Former          | −95.89              | −27.13            | −83.35          |
|                 | (−205.73 to 13.95)  | (−105.84 to 51.58)| (−192.65 to 25.95)|
| Current         | −84.93              | −34.17            | −28.24          |
|                 | (−129.63 to 40.22)  | (−65.76 to 2.58)  | (−81.68 to 25.20)|
| Never           | −60.06              | −24.72            | −64.58          |
|                 | (−83.10 to 37.02)   | (−40.78 to 8.65)  | (−91.69 to 37.46)|
| Interaction p value | 0.13               | 0.78              | 0.92           |
| **Total household income (¥)** |                 |                   |                 |
| >15000          | −44.79              | −10.38            | −26.02          |
|                 | (−76.06 to 13.53)   | (−31.29 to 10.54) | (−15.80 to 67.84)|
| ≤15000          | −106.52             | −57.25            | −107.92         |
|                 | (−135.13 to 77.92)  | (−77.88 to 36.62) | (−137.83 to 78.02)|
| Interaction p value | <0.001             | <0.001            | <0.001         |
| **BMI**         |                     |                   |                 |
| Low (<18.5 kg/m$^2$) | −28.30             | −12.34            | −5.68           |
|                 | (−124.22 to 67.62)  | (−23.23 to 1.45)  | (−107.05 to 95.68)|
| Normal (18.5–23.9 kg/m$^2$) | −62.70             | −50.76            | −73.63          |
|                 | (−93.76 to 31.64)   | (−71.26 to 30.26) | (−110.23 to 37.01)|
| Overweight (24.0–27.9 kg/m$^2$) | −27.29             | −73.39            | −99.76          |
|                 | (−52.75 to 1.83)    | (−110.13 to 36.64)| (−141.55 to 57.98)|
| Obese (≥28.0 kg/m$^2$) | 6.83               | −27.17            | −40.18          |
|                 | (−32.57 to 46.22)   | (−85.75 to 31.42) | (−109.58 to 29.21)|
| Interaction p value | <0.001             | <0.001            | <0.001         |

BMI, body mass index.
baseline,31 which may play an essential role in the mechanisms and progression of the disease.41

To our knowledge, this is the first study to report the detrimental effects of long-term ambient air pollution on individuals with COPD in comparison to people without COPD. Previous research reported enhanced adverse effects from air pollutants on lung function in individuals with pre-existing chronic conditions, such as heart disease, asthma, diabetes, obesity or smoking.29 36 Additionally, exposure to air pollution was demonstrated to increase the frequency of COPD exacerbations42 and mortality.8 An interesting finding was reported by Dorion et al, where subjects with occupations considered at risk for COPD (eg, coal mine operatives, chemical and Table 5  COPD subgroup analyses for PM10, PM2.5 and NO2

| Characteristics          | PM10 (95% CI) | PM2.5 (95% CI) | NO2 (95% CI) |
|--------------------------|---------------|---------------|--------------|
| Sex                      |               |               |              |
| Male                     | 1.25 (1.17 to 1.33) | 1.40 (1.27 to 1.54) | 1.09 (0.99 to 1.20) |
| Female                   | 1.24 (1.17 to 1.32) | 1.32 (1.21 to 1.44) | 1.02 (0.93 to 1.11) |
| Interaction p value       | 0.94          | 0.52          | 0.91          |
| Age                      |               |               |              |
| <65 years old            | 1.30 (1.21 to 1.39) | 1.47 (1.33 to 1.62) | 1.02 (0.93 to 1.13) |
| ≥65 years old            | 1.20 (1.13 to 1.27) | 1.28 (1.18 to 1.39) | 1.07 (0.98 to 1.17) |
| Interaction p value       | 0.15          | 0.21          | 0.05          |
| History of asthma        |               |               |              |
| Yes                      | 1.38 (1.18 to 1.61) | 1.66 (1.33 to 2.08) | 1.86 (1.39 to 2.48) |
| No                       | 1.23 (1.17 to 1.29) | 1.33 (1.25 to 1.43) | 1.01 (0.94 to 1.08) |
| Interaction p value       | 0.22          | 0.16          | 0.003         |
| Smoking status           |               |               |              |
| Former                   | 1.40 (1.17 to 1.67) | 1.67 (1.28 to 2.17) | 1.24 (0.99 to 1.55) |
| Current                  | 1.24 (1.14 to 1.36) | 1.40 (1.24 to 1.59) | 1.00 (0.88 to 1.15) |
| Never                    | 1.23 (1.16 to 1.29) | 1.31 (1.21 to 1.42) | 1.03 (0.95 to 1.12) |
| Interaction p value       | 0.09          | 0.04          | 0.57          |
| Total household income (¥) |             |               |              |
| >15,000                  | 1.24 (1.17 to 1.31) | 1.36 (1.24 to 1.49) | 1.00 (0.90 to 1.11) |
| ≤15,000                  | 1.27 (1.18 to 1.36) | 1.41 (1.27 to 1.56) | 1.00 (1.00 to 1.19) |
| Interaction p value       | 0.27          | 0.67          | 0.31          |
| BMI                      |               |               |              |
| Low (<18.5 kg/m²)        | 1.36 (1.17 to 1.57) | 1.47 (1.19 to 1.81) | 1.11 (1.01 to 1.22) |
| Normal (18.5–23.9 kg/m²) | 1.28 (1.20 to 1.36) | 1.42 (1.30 to 1.55) | 1.15 (1.02 to 1.30) |
| Overweight (24.0–27.9 kg/m²) | 1.29 (1.19 to 1.40) | 1.45 (1.28 to 1.64) | 0.85 (0.68 to 1.07) |
| Obese (≥28.0 kg/m²)      | 1.13 (0.98 to 1.31) | 1.11 (0.91 to 1.38) | 0.97 (0.80 to 1.18) |
| Interaction p value       | 0.27          | 0.20          | 0.11          |

BMI, body mass index; COPD, chronic obstructive pulmonary disease.
related process operatives) had three times lower levels of FEV\textsubscript{1} and FVC per each unit increase in PM\textsubscript{10}, NO\textsubscript{2} compared with those with other occupations.\textsuperscript{26} Forbes \textit{et al} reported the effect modification for PM\textsubscript{10}, NO\textsubscript{2} and SO\textsubscript{2} when excluding patients with asthma, emphysema or bronchitis compared with the general cohorts.\textsuperscript{36} However, no direct comparison between these groups was available.

Strengths and limitations

This study has several limitations. First, analyses are limited by the cross-sectional study design and offer little information on the longitudinal effects of evaluated pollutants. Second, differential physical activity patterns, changes in participant addresses and discriminatory infiltration of outdoor pollution to the indoor environment may have contributed to misclassification of exposure levels. Other potential confounding factors, including secondhand tobacco smoking, various environmental components and the impact of other air pollutants, were not assessed. Finally, further studies are warranted to investigate the effects of particulates of different sizes as well as other gaseous pollutants. These limitations are offset by some important strengths. The results of the current study are based on representative populations of adults from eight different provinces in China, whereas most previously published work has been focused on specific areas of the country. Additionally, we were able to adjust for many important confounders and estimated exposure using high-quality spatiotemporal air pollution exposure models.

Future research and conclusions

Although the link between outdoor air pollution and impaired respiratory function has been previously explored, discrepancies in the findings related to the type of pollutants and specific lung function parameters remain. Moreover, there is still a lack of data coming from low-income and middle-income countries. The populations of these countries are of particular interest as they are more often affected by both severe outdoor air pollution and chronic lung diseases, including COPD. The elderly population in Asian countries, including China, is one of the most vulnerable groups in this regard. Air pollution and COPD will continue to be major public health problems worldwide. Considering the ever-ageing global population, future research should focus on older participants with robust study design and powerful statistical models. The influence of COPD phenotype should also be investigated in future studies.

In conclusion, this study contributes to the current evidence regarding the association between ambient air pollution and decreased lung function, as well as increased prevalence of COPD, among adults from developing countries. Our findings further indicate that individuals with COPD are more vulnerable to high levels of air pollutants with a higher risk of decline in respiratory function.

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Competing interests
None declared.

Patient consent for publication
Not required.

Ethics approval
This study was approved by the Ethics Committee of the Chinese Centre for Disease Control and Prevention (approval number 200601).

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Not commissioned; externally peer reviewed.

Data availability statement
Data may be obtained from a third party and are not publicly available. We used data from the WHO Study on global AEng and adult health (SAGE) to analyse and report the findings. Data access policy is available on https://apps.who.int/healthinfo/systems/surveydata/index.php/catalog/13.

Supplemental material
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REFERENCES
1 World Health Organization. Ambient air pollution: a global assessment of exposure and burden of disease, 2016. Available: https://apps.who.intiris/bitstream/handle/10665/250141/9789241511353-eng.pdf?sequence=1
2 Dastoorpoor M, Idani E, Goudarzi G, et al. Acute effects of air pollution on spontaneous abortion, premature delivery, and stillbirth in Ahvaz, Iran: a time-series study. \textit{Environ Sci Pollut Res Int} 2018;25:5447–58.
3 Elbarbary M, Honda T, Morgan G, et al. Ambient air pollution exposure association with anaemia prevalence and haemoglobin levels in Chinese older adults. \textit{Int J Environ Res Public Health} 2020;17:3209.
4 Neisi A, Vosoughi M, Idani E, et al. Comparison of normal and dusty day impacts on fractional exhaled nitric oxide and lung
function in healthy children in Ahvaz, Iran. *Environ Sci Pollut Res Int* 2017;24:12360–71.
5. GBD 2017 Disease and Injury Incidence and Prevalence Collaborators. Global, regional, and national incidence, prevalence, and years lived with disability for 354 diseases and injuries for 195 countries and territories, 1990–2017: a systematic analysis for the global burden of disease study 2017. *Lancet* 2018;392:1789–858.
6. GBD 2015 Mortality and Causes of Death Collaborators. Global, regional, and national life expectancy, all-cause mortality, and cause-specific mortality for 249 causes of death, 1980-2015: a systematic analysis for the global burden of disease study 2015. *Lancet* 2016;388:1459–544.
7. Gan WQ, FitzGerald JM, Carlsen C, et al. Associations of ambient air pollution with chronic obstructive pulmonary disease hospitalization and mortality. *Am J Respir Crit Care Med* 2017;197:72–7.
8. Soriano JB, Abajobir AA, Abate KH, et al. Global, regional, and national deaths, prevalence, disability-adjusted life years, and years lived with disability for chronic obstructive pulmonary disease and asthma, 1990-2015: a systematic analysis for the global burden of disease study 2015. *Lancet Respir Med* 2017;5:691–706.
9. Mannucci PM, Franchini M. Health effects of ambient air pollution in developing countries. *Int J Environ Res Public Health* 2017;14:1048.
10. Chan CK, Yao X. Air pollution in mega cities in China. *Atmos Environ* 2008;42:1–42.
11. World Health Organisation. WHO global ambient air quality database. ambient air Qual. Database (update 2018), 2018. Available: https://whoairquality.shinyapps.io/AmbientAirQualityDataset/
12. Rohde RA, Muller RA. Air pollution in China: mapping of concentrations and sources. *PloS One* 2015;10:e0135749.
13. Simonii M, Baldacci S, Maio S, et al. Adverse effects of outdoor pollution in the elderly. *J Thorac Dis* 2015;7:34–45.
14. Bentayeb M, Simoni M, Baiz N, et al. Adverse respiratory effects of outdoor air pollution in the elderly. *Int J Tuberc Lung Dis* 2012;16:1149–61.
15. Annesi-Maesano I. Air pollution and chronic obstructive pulmonary disease exacerbations: when prevention becomes feasible. *Am J Respir Crit Care Med* 2019;199:547–8.
16. Edginton S, O’Sullivan DE, King W, et al. Effect of outdoor particulate air pollution on FEV1 in healthy adults: a systematic review and meta-analysis. *Occup Environ Med* 2019;76:583–91.
17. Frischer T, Studnicka M, Gartner C, et al. Lung function growth and ambient ozone: a three-year population study in school children. *Am J Respir Crit Care Med* 1999;160:390–6.
18. Moore E, Chatzidiakou L, Kuku M-O, et al. Global associations between air pollutants and chronic obstructive pulmonary disease hospitalizations. A systematic review. *Ann Am Thorac Soc* 2016;13:1814–27.
19. Kowal P, Chatterji S, Naidoo N, et al. Data resource profile: the world Health organization study on global ageing and adult health (SAGE). *Int J Epidemiol* 2012;41:1639–49.
20. Moore VC. Spirometry: step by step. *Breathe* 2012;8:232–40.
21. Agarwal R, Chakrabarti A, Denning DW, et al. Global Strategy for the Diagnosis, Management, and Prevention of Chronic Obstructive Pulmonary Disease - 2020 Report.
22. Chen G, Li S, Knibbs LD, et al. A machine learning method to estimate PM2.5 concentrations across China with remote sensing, meteorological and land use information. *Sci Total Environ* 2018;636:52–60.
23. Chen G, Wang Y, Li S, et al. Spatiotemporal patterns of PM10 concentrations over China during 2005-2016: A satellite-based estimation using the random forests approach. *Environ Pollut* 2018;242:605–13.
24. Zhan Y, Luo Y, Deng X, et al. Satellite-Based Estimates of Daily NO2 Exposure in China Using Hybrid Random Forest and Spatiotemporal Kriging Model. *Environ Sci Technol* 2018;52:4180–9.
25. Lamichhané BK, Leem JH, Kim HC. Associations between ambient particulate matter and nitrogen dioxide and chronic obstructive pulmonary diseases in adults and effect modification by demographic and lifestyle factors. *Int J Environ Res Public Health* 2018;15:363.
26. Wheeler BJ, Ben-Shlomo Y. Environmental air quality, socioeconomic status, and respiratory health: a linkage analysis of routine data from the health survey for England. *J Epidemiol Community Health* 2005;59:948–54.
27. Liu S, Zhou Y, Liu S, et al. Association between exposure to ambient particulate matter and chronic obstructive pulmonary disease: results from a cross-sectional study in China. *Thorax* 2017;72:788–95.
28. Guo C, Zhang Z, Lau AKH, et al. Effect of long-term exposure to fine particulate matter on lung function decline and risk of chronic obstructive pulmonary disease in Taiwan: a longitudinal, cohort study. *Lancet Planet Health* 2018;2:e114–25.
29. Doiron D, de Hoogh K, Probst-Hensch N, et al. Air pollution, lung function and COPD: results from the population-based UK Biobank study. *Eur Respir J* 2019;54:1802140.
30. Wang M, Aaron CP, Madrigno J, et al. Association between long-term exposure to ambient air pollution and change in quantitatively assessed emphysema and lung function. *JAMA* 2019;322:546–56.
31. Schikowski T, Adam M, Marcon A, et al. Association of ambient air pollution with the prevalence and incidence of COPD. *Eur Respir J* 2014;44:614–26.
32. Zhang Z, Wang J, Lu W. Exposure to nitrogen dioxide and chronic obstructive pulmonary disease (COPD) in adults: a systematic review and meta-analysis. *Environ Sci Pollut Res Int* 2018;25:15133–45.
33. Johnson JD, Theurer WM. A stepwise approach to the interpretation of pulmonary function tests. *Am Fam Physician* 2014;89:359–66.
34. Lin H, Guo Y, Di Q, et al. Consumption of fruit and vegetables might mitigate the adverse effects of ambient PM2.5 on lung function among adults. *Environ Res* 2018;160:77–82.
35. Adam M, Schikowski T, Lippert D, et al. Adult lung function and long-term air pollution exposure, escape: a multicentre cohort study and meta-analysis. *Eur Respir J* 2015;45:38–50.
36. Forbes JL, Kapetanakis V, Rudnicka AR, et al. Chronic exposure to outdoor air pollution and lung function in adults. *Thorax* 2006;64:657–63.
37. Ackermann-Liebrich U, Leuenberger P, Schwartz J, et al. Lung function and long term exposure to air pollutants in Switzerland: study on air pollution and lung diseases in adults (SAPALDIA) team. *Am J Respir Crit Care Med* 1997;155:122–9.
38. O’Neill MS, Jernett M, Kavachi I, et al. Health, wealth, and air pollution: advancing theory and methods. *Environ Health Perspect* 2003;111:1861–70.
39. O’Donnell DE, Laveneziana R, Webb K, et al. Chronic obstructive pulmonary disease: clinical integrative physiology. *Clin Chest Med* 2014;35:51–69.
40. Cavaillès A, Brinchault-Thibodeau D, de Hoogh K, et al. The value of blood cytokines and chemokines in assessing COPD. *Respir Res* 2015;16:77.
41. Bradford E, Jacobson S, Varasteh J, et al. The value of blood cytokines and chemokines in assessing COPD. *Respir Res* 2017;18:180.
42. DeVries R, Kriebel D, Sama S. Outdoor air pollution and COPD-Related emergency department visits, hospital admissions, and mortality: a meta-analysis. *COPD* 2017;14:113–21.
Correction: Ambient air pollution, lung function and COPD: cross-sectional analysis from the WHO Study of AGEing and adult health wave 1

Elbarbary M, Oganesyan A, Honda T, et al. Ambient air pollution, lung function and COPD: cross-sectional analysis from the WHO Study of AGEing and adult health wave 1. BMJ Open Resp Res 2020;7:e000684.

The authors want to alert readers to the following correction made to the published version. The typos in the abstract section has been corrected as follows:

1. The participents in the method section has been corrected to 11,693.
2. In results ‘ml’ has been removed from FEV1 /FVC (–2.81, 95%CI –3.37 to –2.25) and FEV1 /FVC (–1.85, 95%CI –2.24 to –1.46).

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