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RESEARCH ARTICLE

# Adverse effects of short-term personal exposure to fine particulate matter on the lung function of patients with chronic obstructive pulmonary disease and asthma: a longitudinal panel study in Beijing, China

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## Abstract

Fine particulate matter ($PM_{2.5}$) is an important environmental factor affecting human health. However, most studies on $PM_{2.5}$ and health have used data from fixed monitoring sites to assess $PM_{2.5}$ exposure, which may have introduced misleading information on the exposure–response relationship. We aimed to assess the effect of short-term personal $PM_{2.5}$ exposure on lung function in patients with chronic obstructive pulmonary disease (COPD) and asthma. To achieve this, we conducted a longitudinal panel study among 37 COPD patients and 45 asthma patients from Beijing, China. The COPD group and the asthma group completed 148 and 180 lung function tests, respectively. We found that in COPD patients, for every 10-μg/m$^3$ increase in $PM_{2.5}$ exposure at lag2, the $FEV_1$, FVC and DLco decreased by −0.014 L (95% CI −0.025, −0.003), −0.025 L (95% CI −0.050, −0.003) and −0.089 mmol/min/kPa (95% CI −0.156, −0.023), respectively. There was also a decrease of −0.023 L/s (95% CI −0.042, −0.003) and −0.017 L/s (95% CI −0.032, −0.002) in MMEF at lag3 and lag03, respectively. In the asthma group, every 10-μg/m$^3$ increase in $PM_{2.5}$ exposure led to a reduction of −0.012 L (95% CI −0.023, −0.001), −0.042 L (95% CI −0.081, −0.003) and −0.061 L/s (95% CI −0.116, −0.004) in the $FEV_1$, FVC and PEF at lag3, respectively. Our findings suggest that $PM_{2.5}$ exposure may primarily affect both airway function and lung diffusion function in COPD patients, and airway function in asthma patients.

**Keywords** Personal $PM_{2.5}$ exposure · Lung function · COPD · Asthma

## Background

Air pollution is a major factor threatening human health worldwide. Fine particulate matter ($PM_{2.5}$), which is composed of particles measuring ≤2.5 μm in aerodynamic diameter, has been recognised as the most important type of air pollutant. While $PM_{2.5}$ is a serious environmental health problem all over the world, it is especially so in China, where $PM_{2.5}$ has become the fourth health risk factor affecting Chinese residents (Zhou et al. 2019a). Studies have shown that $PM_{2.5}$ exposure is associated with premature death and years of life lost (YLL) and that a reduction of $PM_{2.5}$ concentration can reduce the loss of life of residents. A time-series study conducted in 72 Chinese cities showed that a 10-μg/m$^3$ increase in $PM_{2.5}$ was associated with an increase in YLL of 0.43 years, and that a potential life expectancy of 0.14 years could be obtained if the $PM_{2.5}$ concentration was reduced in compliance with the World Health Organization's air quality guideline (25 μg/m$^3$) (Patz et al. 2020). $PM_{2.5}$ has a large surface area and a small particle size and can absorb various toxic and harmful substances causing damage to multiple organs when entering the respiratory system via inhalation

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(Xing et al. 2016). Of the organ systems, the respiratory system is considered to be the most vulnerable to $PM_{2.5}$.

Chronic obstructive pulmonary disease (COPD) and asthma are both chronic respiratory diseases with increasing incidence and have become significant healthcare burdens worldwide. According to the China Pulmonary Health (CPH) study, the prevalence of COPD and asthma in Chinese people aged ≥20 years was 8.6% and 4.2%, respectively (Huang et al. 2019; Wang et al. 2018). COPD and asthma are complex heterogeneous diseases that share many common risk factors, such as genetic susceptibility, smoking and air pollution (Postma et al. 2011). While active smoking is the major preventable risk factor globally, the effect of $PM_{2.5}$ exposure on COPD and asthma cannot be underestimated.

Accumulated epidemiological findings have illustrated that $PM_{2.5}$ exposure is responsible for the increased hospitalisation and mortality of patients with COPD and asthma (Atkinson et al. 2014; Cai et al. 2019; Cakmak et al. 2019; Fan et al. 2016; Li et al. 2016). Lung function is a good indicator of respiratory health and the severity of pulmonary disease and an early predictor of mortality. Both COPD and asthma comprise patients whose lung function have been impaired and are more vulnerable to $PM_{2.5}$ exposure. Therefore, the impact of $PM_{2.5}$ on lung function in these two groups of patients is of great concern. However, the existing results on the acute effects of short-time $PM_{2.5}$ exposure on lung function in patients with COPD and asthma are inconsistent. For example, studies conducted in Italy (Lagorio et al. 2006) and England (Sinharay et al. 2018) found that there were negative associations between short-term exposure to $PM_{2.5}$, forced expiratory volume ($FEV_1$) and forced vital capacity (FVC) in patients with COPD. A panel study conducted in the USA found that a short-term exposure to $PM_{2.5}$ was associated only with a decreased FVC but not $FEV_1$ in COPD patients (Hart et al. 2018). However, several other epidemiological studies have indicated that there is no association between short-term exposure to $PM_{2.5}$ and lung function parameters in COPD or asthma patients (de Hartog et al. 2010; Girardot et al. 2006).

Some of these inconsistencies may be due to differences in $PM_{2.5}$ exposure measurement. In most of the recent epidemiological studies, $PM_{2.5}$ concentration data were obtained from a central monitoring station. Such data were assumed to represent the averaged exposure of the population and were used for the analyses. However, personal $PM_{2.5}$ exposure is strongly influenced by many factors, such as daily activities, lifestyle and microenvironments (Lei et al. 2016). Therefore, whether the $PM_{2.5}$ concentration data of a fixed monitoring site actually represents the real exposure level has been a concern of researchers (Avery et al. 2010; Evangelopoulos et al. 2020; Sarnat et al. 2006). Moreover, some studies have also shown that using $PM_{2.5}$ concentration data from a fixed monitoring site to represent personal $PM_{2.5}$ exposure may lead to bias in the exposure–response relationship (Chen et al. 2019).

Personal exposure monitoring is considered as the current the 'gold standard' for air pollutant exposure assessment. However, few studies have been conducted to assess the effect of personal $PM_{2.5}$ exposure on lung function in patients with COPD and asthma. Therefore, in this longitudinal panel study, we used a personal $PM_{2.5}$ monitoring device to obtain the real-time $PM_{2.5}$ exposure data of participants during daily activities and assess the effect of short-term exposure to personal $PM_{2.5}$ on lung function in patients with COPD and asthma.

## Methods

### Study design

Two parallel longitudinal panel studies were conducted between July 2017 and August 2019—one with COPD patients and the other with asthma patients. To ensure that the patients were followed up once every season, and to include different levels of air pollution empirically, the study was designed to have follow-ups every 3 months, for a total of four visits in a year. The duration of each visit was 3 days, which was comparable with other panel studies in related fields (Bloemsma et al. 2016; de Hartog et al. 2010; Gao et al. 2020; Hart et al. 2018). Personal $PM_{2.5}$ data were recorded, and questionnaires and pulmonary function tests were completed at each visit. Briefly, on the first day 10:00 of the visit, each participant was asked to wear a personal $PM_{2.5}$ monitoring device. On the fourth day at 10 AM, they returned the equipment, performed the pulmonary function test and completed the questionnaire survey. The survey included demographic information such as sex, age, occupation and educational levels as well as the disease course and the presence of comorbidities at the first visit. Moreover, data on medications, respiratory symptoms and number of acute exacerbations between visits were collected at each visit. All procedures were explained to the participants, and informed consent from each participant was obtained in writing. The Ethics Committee of the China–Japan Friendship Hospital approved the research protocol (2017-19). This study was conducted in accordance with the Declaration of Helsinki.

### Participants

The participants were recruited from the outpatient department of the China–Japan Friendship Hospital. The inclusion criteria for the COPD patients were aged 45–75 years and physician-diagnosed COPD in line with the Global Initiatives for Chronic Obstructive Pulmonary Disease guidelines, with a ratio of $FEV_1$ to FVC of less than 70% after post-bronchodilator spirometry (Vogelmeier et al. 2017). The inclusion criteria for the asthma patients were aged 18–75 years and physician-diagnosed asthma according to the Global

Asthma Prevention Initiative guidelines, with a $FEV_1$ reversibility of >12% and 200 mL after post-bronchodilator spirometry (Bousquet and Humbert 2015).

The exclusion criteria were designed to exclude patients whose lifestyle or complications would have had a significant impact on their lung function, or who would not be able to complete the pulmonary function tests, or all four visits. The exclusion criteria for both COPD and asthma patients were as follows: (1) currently smoking or had quit smoking for no more than 6 months; (2) the presence of complications and comorbidities, such as malignant tumours, severe cardiovascular and cerebrovascular diseases, hepatic and renal insufficiency and active tuberculosis; (3) had undergone assessments on the effects of epilepsy or psychiatric diseases; (4) had undergone chest, abdominal or eye surgeries in the last three months; (5) pregnant and lactating women.

## Environmental data

We used the MicroPEM Personal Exposure Monitor (version 3.2; RTI International, USA), which has been widely used in personal $PM_{2.5}$ exposure assessments, to obtain personal $PM_{2.5}$ exposure data (Lei et al. 2016; Wang et al. 2017; Ye et al. 2020). The MicroPEM is a light, compact, low-noise and portable personal exposure monitoring device with a rubber tube at the top to collect the air from the wearer's breathing area. It consists of an onboard micro-nephelometer that can simultaneously obtain real-time $PM_{2.5}$ data as well as real-time temperature and humidity data through temperature and humidity sensors (Du et al. 2019).

Exposure data were recorded every 10 s with the flow rate of 0.5 L/min (calibrated with a flowmeter). The MicroPEM was placed in a small backpack, and the participant was instructed to carry it all the time and to keep the rubber tube close to the mouth and nose breathing area during the monitoring period. The wearing of the MicroPEM started at 10:00 on the first day, and the monitoring ended at 10:00 on the fourth day. All participants were instructed individually on how to use the equipment.

To assess the impact of other air pollutants on the acute effects of $PM_{2.5}$ on lung function, we also collected concentrations of other air pollutants from Beijing Municipal Environmental Protection Bureau (http://www.bjepb.gov.cn/) and the monitoring site closest to the patients' home address was selected. The 24-h mean concentrations of sulphur dioxide ($SO_2$), nitrogen dioxide ($NO_2$), carbon monoxide (CO) and maximum 8-h average concentration of ozone ($O_3$) were adopted.

## Pulmonary function testing

On the fourth day of the follow-up, a pulmonary function test was performed using a MasterScreen spirometer (Jaeger, Germany). Technicians involved in the study had been professionally trained, and the same spirometer was used throughout the study. Moreover, participants were asked not to change their medication before a pulmonary function test. Before the pulmonary function test, the participant's age, sex, height, weight, temperature; atmospheric pressure; and other indicators were entered in a computer, and the corresponding predictive value was generated automatically by the computer. At least three tests were performed each time, and the time interval between each measurement was ensured to be maintained >5 min. The optimal forced expiratory flow–volume curve was taken as the final result.

The spirometry parameters were as follows: $FEV_1$, FVC and the ratio of $FEV_1$/FVC; diffusing capacity of the lungs for carbon monoxide (DLco); peak expiratory flow (PEF); vital capacity (VC); total lung volume (TLC); maximal mid-expiratory flow (MMEF).

## Statistical analysis

Lung function, daily $PM_{2.5}$ concentrations, relative humidity and temperature data were analysed using SPSS software version 22.0 (IBM Corp., Armonk, NY, USA) and expressed as means ± SDs. For the correlation analysis, since these variables are repeated measurements of individuals, and each individual served as his/her own control, we analysed the effect of $PM_{2.5}$ exposure on lung function by using a linear mixed-effect (LME) model. The model allowed each person's observations at different time points to be used as their own point of comparison, and provided the advantage of interpreting the correlations of multiple repeated measurements by including a random intercept for each person (Bondell et al. 2010).

In our LME model, the lung function parameters were approximately normally distributed (Supplementary Fig. 1) and were regarded as response variables. The $PM_{2.5}$ was considered as the fixed-effect variable and the patient's identity number was introduced as the random-effect variable. This model also used sex, age, body mass index (BMI), disease duration, past smoking status, temperature and relative humidity as fixed-effect covariates to control for potential confounding effects. For temperature and relative humidity, we conducted a linear correlation test before the regression analysis. The results showed that the influence of temperature and relative humidity on lung function was non-linearly correlated (Supplementary Table 1). Therefore, non-linear control of temperature and relative humidity was adopted in the subsequent analysis. To control the potential nonlinear relationship, we adjusted for the nonlinear and delayed effects of weather conditions on lung function by fitting natural cubic splines with a *d.f.* of 3 for the 3-day moving average air temperature and relative humidity.

First, we analysed the single-day lag effect and the cumulative lag effect with reference to other panel studies in related

fields (Gao et al. 2020; Hart et al. 2018). We evaluated the single-day lag effect, which referred to the effect on the current day of the lung function test (lag0), then 1 day before testing (lag1), 2 days before testing (lag2) and 3 days before testing (lag3). We then evaluated the cumulative lag effect, which referred to the relationship between the moving averages (lag01, lag02 and lag03). The lag time distribution of this study is shown in Supplementary Fig. 2. Then we fitted the two-pollutant model to assess whether the effects of $PM_{2.5}$ are dependent on simultaneous exposure to other air pollutants. Finally, we used the LME model to complete a stratified analysis to evaluate the effect of previous smoking history (former smoker or never smoker) on the results.

The LME model analysis was conducted using the R software (version 4.0.0) with the package 'lmertest'. The results were expressed as the value of lung function changes and its 95% CIs for every 10-μg/m$^3$ increase in $PM_{2.5}$ concentration. The statistical significance was set at a $P$ value of <0.05.

## Results

### Descriptive statistics

We included 37 patients with COPD and 45 patients with asthma. The baseline demographic and clinical characteristics are described in Table 1. Most of the participants in the COPD group were men (62.2%), whereas most of the participants in the asthma group were women (75.6%). The mean age was 63.0±9.0 and 55.7±12.3 years in the COPD group and asthma group, respectively, and the corresponding number of never smokers was 21 (56.8%) and 35 (77.8%), respectively. Comorbidities included cerebrovascular disease, hypertension, diabetes, peptic ulcer and connective tissue disease. In terms of medication use, compound preparations of inhaled corticosteroids and long-acting beta-2 agonist (ICS + LABA) were the most commonly used drugs in both groups. One month before enrolment in this study, one COPD patient (2.7%) and two asthma patients (4.4%) were treated with oral glucocorticoid therapy due to acute exacerbation.

Table 2 summarises the descriptive statistics for daily $PM_{2.5}$ concentrations, meteorological variables and lung function throughout the follow-up period. The average $PM_{2.5}$ concentrations during the study were 43.92 ± 42.70 μg/m$^3$ and 46.77 ± 44.97 μg/m$^3$ for the COPD and asthma groups, respectively, which were beyond the threshold values recommended by the World Health Organization (WHO), in which the suitable $PM_{2.5}$ concentration for human health were defined as no more than 10 μg/m$^3$ (WHO 2005). The corresponding $FEV_1$/FVC ratios were 56.53 ± 8.14 ml and 75.82 ± 6.39 ml for the COPD and asthma groups, respectively.

As for each follow-up visit, the descriptive statistics of lung function and daily $PM_{2.5}$ exposure data are shown in Supplementary Table 2 for the COPD group and Supplementary Table 3 for the asthma group.

**Table 1** Characteristics of participants at baseline.

| Variable | COPD ($n$ = 37) | Asthma ($n$ = 45) |
|---|---|---|
| Gender (male) | 23 (62.2%) | 11 (24.4%) |
| Age (years) | 63.0±9.0 | 55.7±12.3 |
| BMI (kg/m$^2$) | 26.3±4.9 | 25.2±3.9 |
| Smoking | | |
| Never smoker | 21 (56.8%) | 35 (77.8%) |
| Former smoker | 16 (43.2%) | 10 (23.2%) |
| Pack-years | 27.2±6.1 | 18.0±4.5 |
| Disease duration (years) | 5.1±6.2 | 11.7±9.8 |
| Comorbidity | | |
| Cerebrovascular disease | 8 (21.6%) | 8 (17.8%) |
| Connective tissue disease | 0 (0.0%) | 2 (4.4%) |
| Hypertension | 6 (16.2%) | 9 (20.0%) |
| Peptic ulcer | 3 (8.1%) | 6 (13.3%) |
| Diabetes | 5 (13.5%) | 5 (11.1%) |
| History of pollen or drug allergy | 7 (18.9%) | 13 (28.9%) |
| CAT/ACT score | 13.8±8.6 | 21.7±3.3 |
| Medication use[a] | | |
| ICS | 3 (8.1%) | 3 (6.7%) |
| LAMA | 15 (40.5%) | 0 (0.0%) |
| ICS + LABA | 32 (86.5%) | 45 (100%) |
| SABA | 2 (5.4%) | 5 (11.1%) |
| SAMA | 7 (5.4%) | 1 (2.2%) |
| Oral glucocorticoids | 1 (2.7%) | 2 (4.4%) |

Data are presented as means ± SD or counts and percentages. Pack-years refers to cigarettes smoked per day divided by 20 and then multiplied by smoking years

COPD, chronic obstructive pulmonary disease; BMI, body mass index; CAT, COPD assessment test; ACT, asthma control test; ICS, inhaled corticosteroids; LABA, long-acting beta-2 agonist; LAMA, long-acting muscarinic antagonist; SABA, short-acting beta-2 agonist; SAMA, short-acting muscarinic antagonist

[a] Self-report of prescriptions 1 month prior to enrolment to the study

### Effects of $PM_{2.5}$ exposure on lung function

For the single-day lag model analysis in the COPD group, we found statistically significant adverse effects of personal $PM_{2.5}$ exposure on $FEV_1$, FVC, DLco and MMEF. As shown in Fig. 1, for every 10-μg/m$^3$ increase of $PM_{2.5}$ exposure in lag2, $FEV_1$, FVC and DLco decreased by −0.014 L (95% CI −0.025, −0.003), −0.025 L (95% CI −0.050, −0.003) and −0.089 mmol/min/kPa (95% CI −0.156, −0.023), respectively. A per 10-μg/m$^3$ increase in $PM_{2.5}$ exposure at lag3 was related to a reduction in MMEF of −0.023 L/s (95% CI −0.042, −0.003). In the cumulative lag analysis, a per 10-μg/

**Table 2** Descriptive statistics for daily $PM_{2.5}$ concentrations, weather conditions and lung function during the study period (July 2017 to August 2019)

| | COPD | | Asthma | |
|---|---|---|---|---|
| | Mean ± SD | Min–Max | Mean ± SD | Min–Max |
| $PM_{2.5}$-lag0 (μg/m$^3$) | 42.67 ±41.92 | 1.56–265.85 | 45.9±47.07 | 1.47–289.11 |
| $PM_{2.5}$-lag1 (μg/m$^3$) | 44.52 ±40.32 | 4.75–200.07 | 47.5±38.28 | 2.75–225.06 |
| $PM_{2.5}$-lag2 (μg/m$^3$) | 43.86 ±40.75 | 3.81–200.07 | 46.72 ±38.17 | 3.99–201.42 |
| $PM_{2.5}$-lag3 (μg/m$^3$) | 44.24 ±44.49 | 4.18–290.69 | 49.08 ±55.17 | 2.91–467.27 |
| $PM_{2.5}$-average (μg/m$^3$) | 43.92 ±42.70 | 1.56–290.69 | 46.77 ±44.97 | 1.47–289.11 |
| Temperature (°C) | 24.15±3.72 | 14.48–34.49 | 23.37±4.57 | 19.98–33.54 |
| Relative humidity (%) | 36.71 ±12.52 | 10.28–71.15 | 39.45 ±13.44 | 7.46–72.12 |
| $FEV_1$/FVC | 56.53±8.14 | 34.55–75.33 | 75.82±6.39 | 53.20–92.19 |
| FVC (L) | 2.48±0.74 | 1.08–4.16 | 2.94±0.80 | 1.60–5.14 |
| VC (L) | 2.75±0.76 | 1.37–4.43 | 3.05±0.80 | 1.86–5.40 |
| TLC (L) | 5.04±1.09 | 3.10–7.88 | 5.03±0.82 | 2.68–7.71 |
| DLco (mmol/min/kPa) | 6.38±2.02 | 1.15–13.41 | 7.10±1.59 | 1.30–13.10 |
| $FEV_1$ (L) | 1.41±0.48 | 0.52–2.44 | 2.19±0.65 | 0.89–4.15 |
| PEF (L/s) | 4.20±1.52 | 0.25–7.66 | 6.09±1.71 | 2.68–10.74 |
| MMEF (L/s) | 0.98±0.44 | 0.16–2.15 | 2.25±1.17 | 0.43–8.04 |

Lag numbers indicate days prior to pulmonary function test (e.g. lag0 is the day of testing)

Min, the minimum value; Max, the maximum value; $PM_{2.5}$, particulate matter with aerodynamic diameters ≤2.5 μm; FVC, forced vital capacity; $FEV_1$, forced expiratory volume in the first second; $FEV_1$/FVC, the ratio of forced expiratory volume in one second and forced vital capacity; VC, vital capacity; TLC, total lung volume; DLco, diffusing capacity of the lung for carbon monoxide; PEF, peak expiratory flow; MMEF, maximum mid-expiratory flow

m$^3$ increase in $PM_{2.5}$ exposure was associated with a MMEF decrease of −0.017 L/s (95% CI −0.032, −0.002) at lag03.

For the asthma group in the single-day lag model, short-term personal $PM_{2.5}$ exposure was negatively associated with $FEV_1$, FVC and PEF, and the association was significant for all three variables at late lag (lag3). As shown in Fig. 2, for every 10-μg/m$^3$ increase in $PM_{2.5}$, exposure at lag3 was associated with a decrease in $FEV_1$, FVC and PEF of −0.012 L (95% CI −0.023, −0.001), −0.042 L (95% CI −0.081, −0.003) and −0.061 L/s (95% CI −0.116, −0.004), respectively. For the cumulative lag analysis, a per 10-μg/m$^3$ increase in $PM_{2.5}$ exposure was associated with a PEF decrease of −0.122 L/s (95% CI −0.232, −0.011) at lag03.

Table 3 shows the results on two-pollutant models in COPD group. The associations of $PM_{2.5}$ and $FEV_1$ remained statistically significant when controlling for the effects of other air pollutants. The estimated effects of $PM_{2.5}$ on FVC turned out to be statistically insignificant after adjustment of $SO_2$ and $NO_2$. Similarly, the estimated effects of $PM_{2.5}$ on DLco and MMEF turned out to be statistically insignificant after adjustment of CO. The results on two-pollutant model in asthma group are shown in Table 4. The associations of $PM_{2.5}$ and FVC remained statistically significant when controlling for the effects of other air pollutants. The estimated effects of $PM_{2.5}$ on $FEV_1$ turned out to be statistically insignificant after adjustment of $NO_2$ and $SO_2$. The estimated effects of $PM_{2.5}$ on PEF turned out to be statistically insignificant after adjustment of $SO_2$.

In the stratified analysis, the results showed that the effect of personal $PM_{2.5}$ exposure on the lung function parameters was more evident in former smokers than never smokers (Supplementary Table 4).

## Discussion

In this longitudinal panel study, we used data from the MicroPEM to explore the effect of short-term personal $PM_{2.5}$ exposure on lung function in patients with COPD and asthma. We observed that short-term personal $PM_{2.5}$ exposure was associated with a decreased $FEV_1$, FVC, DLco and MMEF in patients with COPD and a lower $FEV_1$, FVC and PEF in patients with asthma. Personal exposure monitoring is a more precise way of measuring the $PM_{2.5}$ concentration that

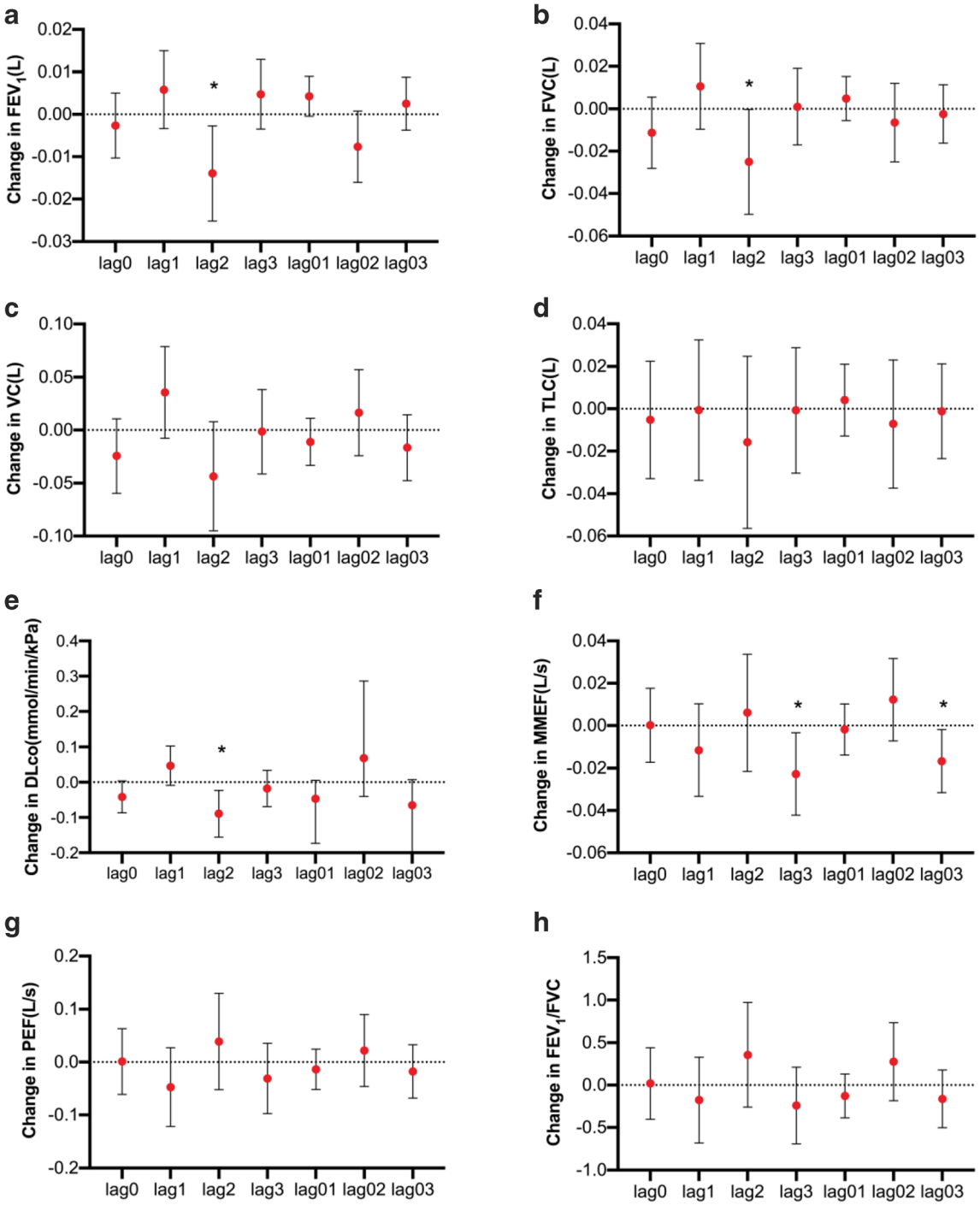


**Fig. 1** Changes in lung function associated with every 10-μg/m$^3$ increase in personal $PM_{2.5}$ exposure of patients with chronic obstructive pulmonary disease. The *x*-axis represents the different time windows and the *y*-axis the corresponding changes (means and 95% CIs). *$P$ <0.05. $PM_{2.5}$, particulate matter with aerodynamic diameters of ≤2.5 μm; FVC, forced vital capacity; $FEV_1$, forced expiratory volume in 1 s; $FEV_1$/FVC, ratio of forced expiratory volume in 1 s to forced vital capacity; VC, vital capacity; TLC, total lung volume; DLco, diffusing capacity of the lungs for carbon monoxide; PEF, peak expiratory flow; MMEF, maximal mid-expiratory flow.

people receive daily in a microenvironment and is considered the 'gold standard' for air pollution exposure assessment (Lei et al. 2016). To our knowledge, this study is one of the few studies that have assessed the effect of $PM_{2.5}$ in patients with COPD and asthma using personal $PM_{2.5}$ exposure data.

Studies have shown that $PM_{2.5}$ can cause respiratory inflammation and injury, which is related to a variety of respiratory diseases, such as chronic respiratory diseases, pneumonia, acute lower respiratory infection and lung cancer (Ciabattini et al. 2020; Gharibi et al. 2019; Liu et al. 2021; Sheikh et al.

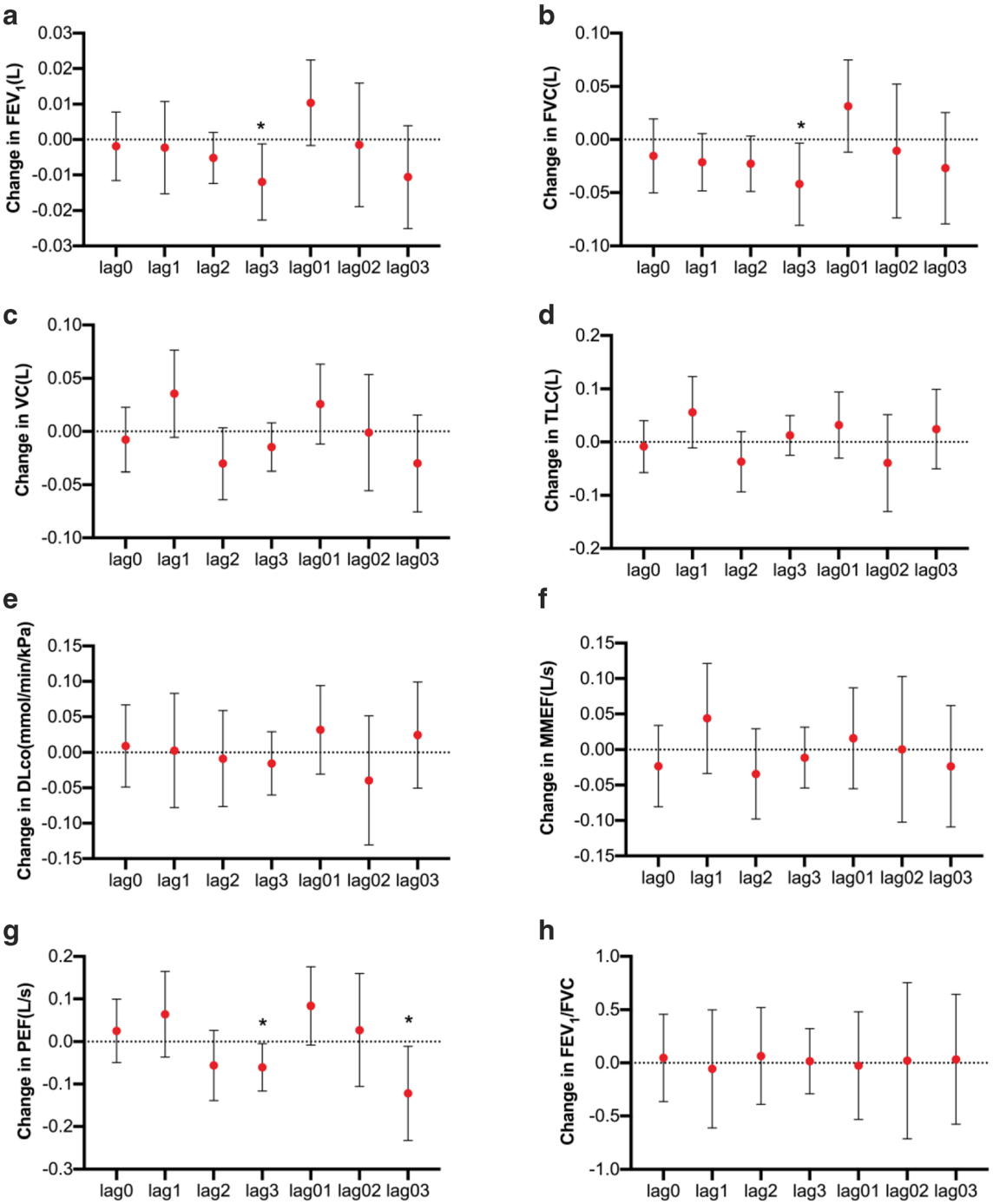


**Fig. 2** Changes in lung function associated with every 10-μg/m$^3$ increase in personal $PM_{2.5}$ exposure of patients with asthma. The *x*-axis represents the different time windows and the *y*-axis the corresponding changes (means and 95% CIs). *$P$ <0.05. $PM_{2.5}$, particulate matter with aerodynamic diameters of ≤2.5 μm; FVC, forced vital capacity; $FEV_1$, forced expiratory volume in 1 s; $FEV_1$/FVC, ratio of forced expiratory volume in 1 s to forced vital capacity; VC, vital capacity; TLC, total lung volume; DLco, diffusing capacity of the lungs for carbon monoxide; PEF, peak expiratory flow; MMEF, maximal mid-expiratory flow

2019; Mehta et al. 2013). COPD and asthma are the most common chronic respiratory diseases. Both patients with COPD and asthma are susceptible to air pollution. Spirometry is widely used as a non-invasive, rapid and economical detection method that can assess the severity of respiratory diseases objectively and to evaluate the impact of air pollutants on cardiopulmonary health.

Our results showed that increased personal $PM_{2.5}$ exposure led to a decrease in $FEV_1$, FVC, DLco and MMEF in patients with COPD. Moreover, increased personal $PM_{2.5}$ exposure affected $FEV_1$, FVC and PEF in asthma patients, adversely. However, no statistically significant difference between short-term $PM_{2.5}$ exposure and TLC, VC and $FEV_1$/FVC in both

**Table 3** The effect of $PM_{2.5}$ on lung function in patients with COPD in the two-pollutant models

| | $FEV_1$ (lag2) | FVC (lag2) | DLco (lag2) | MMEF (lag3) |
|---|---|---|---|---|
| $PM_{2.5}$ | −0.014 (−0.025, −0.003)* | −0.025 (−0.050, −0003)* | −0.089 (−0.156, −0.023)* | −0.023 (−0.042, −0.003)* |
| +CO | −0.014 (−0.026, −0.002)* | −0.029 (−0.054, −0.003)* | −0.073 (−0.169, 0.024) | −0.032 (−0.065, 0.001) |
| $+NO_2$ | −0.013 (−0.025, −0.002)* | −0.019 (−0.045, 0.008) | −0.081 (−0.154, −0.007)* | −0.026 (−0.048, −0.003)* |
| $+SO_2$ | −0.014 (−0.025, −0.003)* | −0.014 (−0.044, 0.015) | −0.110 (−0.19, −0.029)* | −0.031 (−0.055, −0.007)* |
| $+O_3$ | −0.014 (−0.025, −0.003)* | −0.025 (−0.05, −0.001)* | −0.085 (−0.15, −0.019)* | −0.023 (−0.042, −0.003)* |

Data are presented as changes with 95% CIs in lung function. Lag numbers indicate days prior to pulmonary function test (e.g. lag0 is the day of testing)

*$p < 0.05$

$PM_{2.5}$, particulate matter with aerodynamic diameters ≤2.5μm; $SO_2$, sulphur dioxide; $NO_2$, nitrogen dioxide; CO, carbon monoxide; $O_3$, ozone; FVC, forced vital capacity; $FEV_1$, forced expiratory volume in the first second; DLco, diffusing capacity of the lung for carbon monoxide; MMEF, maximal mid-expiratory flow

COPD and asthma populations was observed. $FEV_1$ can reflect the degree of airflow limitation in the large airway, while MMEF and PEF are good indicators of small airway dysfunction (de Hartog et al. 2010; Pellegrino 2005). Our findings indicate that short-term personal $PM_{2.5}$ exposure may primarily affect the airway and lung diffusion function in COPD patients, and the airway function, rather than the lung diffusion function, in asthma patients.

The degree of decrease in the $FEV_1$ and FVC in our study was higher than that in a panel study involving 125 patients with COPD in the USA, which found that when indoor $PM_{2.5}$ exposure increased by a per IQR of 5.8 μg/m$^3$, the $FEV_1$ and FVC decreased by 0.004 L and 0.021 L, respectively (Hart et al. 2018). The decrease in $FEV_1$ in our study was lower than that in another panel study in Seattle, USA, which showed that for every 10-μg/m$^3$ increase in $PM_{2.5}$, the $FEV_1$ decreased by 0.078 L and 0.046 L in COPD and asthma patients, respectively (Trenga et al. 2006). International guidelines recommend that patients with asthma have their PEF measured daily to assess asthma control and reduce acute attacks. This study found that personal $PM_{2.5}$ exposure had an acute effect on PEF in asthma patients. A panel study in Seattle found a negative correlation between personal $PM_{2.5}$ exposure and PEF in children with asthma, which is consistent with the results of this study (Trenga et al. 2006).

However, our results were also inconsistent with those of some previous studies. A panel study with 64 COPD patients conducted in Beijing showed that $PM_{2.5}$ exposure affected the FVC% but not $FEV_1$% (Gao et al. 2020). Another panel study conducted in Italy also found an inverse effect between $PM_{2.5}$ exposure and $FEV_1$ and FVC in COPD patients; however, no association was observed in asthma patients (Lagorio et al. 2006). Moreover, another panel study conducted in Europe showed that there was no effect of $PM_{2.5}$ and the $FEV_1$, FVC, or PEF in both asthma and COPD patients (de Hartog et al. 2010). The Oxford Street and Hyde Park study that included 60 patients with moderate to severe asthma found there were no consistent associations between $PM_{2.5}$ and $FEV_1$, FVC and MMEF (McCreanor et al. 2007). Similarly, a panel study from the UK, which included 16 patients with COPD, found that neither personal $PM_{2.5}$ exposure nor $PM_{2.5}$ exposure from atmospheric monitoring station was associated with $FEV_1$ (Brauer et al. 2001). These inconsistencies may be due to the differences in exposure measurement methods, sample size, race and microenvironments.

**Table 4** The acute effect of $PM_{2.5}$ on lung function in patients with asthma in the two-pollutant models

| | $FEV_1$ (lag3) | FVC (lag3) | PEF (lag3) |
|---|---|---|---|
| $PM_{2.5}$ | −0.012 (−0.023, −0.001)* | −0.042 (−0.081, −0.003)* | −0.061 (−0.116, −0.005)* |
| +CO | −0.011 (−0.022, −0.001)* | −0.041 (−0.080, −0.002)* | −0.063 (−0.12, −0.006)* |
| $+NO_2$ | −0.010 (−0.021, 0.001) | −0.044 (−0.085, −0.004)* | −0.081 (−0.155, −0.006)* |
| $+SO_2$ | −0.011 (−0.021, 0.001) | −0.041 (−0.08, −0.001)* | −0.009 (−0.095, 0.077) |
| $+O_3$ | −0.012 (−0.022, −0.001)* | −0.043 (−0.082, −0.004)* | −0.063 (−0.119, −0.007)* |

Data are presented as changes with 95% CIs in lung function. Lag numbers indicate days prior to pulmonary function test (e.g. lag0 is the day of testing)

*$p < 0.05$

$PM_{2.5}$, particulate matter with aerodynamic diameters ≤2.5μm; $SO_2$, sulphur dioxide; $NO_2$, nitrogen dioxide; CO, carbon monoxide; $O_3$, ozone; FVC, forced vital capacity; $FEV_1$, forced expiratory volume in the first second; DLco, diffusing capacity of the lung for carbon monoxide; PEF, peak expiratory flow

The available research on the association of $PM_{2.5}$ and MMEF and DLco is limited. Our results are consistent with a study completed in the USA, which found that increased $PM_{2.5}$ concentrations were correlated with a decreased MMEF% (−0.66; 95% CI −1.07, −0.24) in patients with asthma (Vempilly et al. 2013). The CPH study used MMEF as one of the indicators of small airway dysfunction and found that $PM_{2.5}$ is a major preventable risk factor for small airway dysfunction (Xiao et al. 2020). DLco is a gas transmission measurement method that reflects the complex interaction between gas and alveolar capillaries and is one of the best predictors of emphysema and impaired lung function (Nambu et al. 2015). In addition, the decreased DLco is strongly associated with reduced physical activity, increased frequency of acute exacerbation and a higher risk of mortality in patients with COPD. DLco may be regarded as a tool for the multidimensional assessment of COPD in the future (Balasubramanian et al. 2019). Currently, there are few studies that focus on the association between $PM_{2.5}$ exposure and the diffusion function in COPD or asthma patients. Further research is necessary to confirm our findings.

There are several possible biological mechanisms for the association between $PM_{2.5}$ exposure and lung function. The first is the mechanism of oxidative damage. $PM_{2.5}$ is a strong oxidant, which can induce oxidative stress and inflammation in the respiratory tract and lungs, leading to the apoptosis of lung epithelial cells, airway remodelling and impaired respiratory function (Xing et al. 2016; Zhao et al. 2019; Zhou et al. 2019b). Studies have also found that $PM_{2.5}$ can directly stimulate the vagus nerve of the respiratory tract, causing bronchial spasm and contraction, and increasing respiratory resistance, leading to the decline of lung function. Besides, other studies have shown that $PM_{2.5}$ can cause respiratory defence responses, including increased mucus secretion, impaired ciliary system clearance and bronchial hyperresponsiveness (Anderson et al. 2003). This evidence is consistent with our findings on the acute effect between $PM_{2.5}$ and lung function. However, it should be noted that our research does not reveal the mechanism related to $PM_{2.5}$ that may be active in these processes, so further research is needed to verify any causality.

This study had several important strengths. First, we used an individual sampler to obtain real-time individual $PM_{2.5}$ exposure data that closely reflected the real exposure level and enabled us to evaluate the effect of $PM_{2.5}$ exposure and lung function more accurately. Second, because smoking is an important factor affecting lung function, all patients included in our study were either non-smokers or smokers who quit smoking for more than half a year, and this decision helped in eliminating the significant confounding effect of smoking. Third, compared with other studies, we have analysed more lung function indexes, such as lung volume index TLC, airflow limitation and airway function indexes, including MMEF, PEF, $FEV_1$ and FVC, and the lung diffusion function index DLco, which demonstrated the effect of short-term $PM_{2.5}$ exposure on lung function comprehensively.

There were also some limitations to our study. First, due to the small diameter of the filter membrane of the monitoring equipment, we were not able to detect the chemical elements of $PM_{2.5}$ particles. Second, in the two-pollutant model, exposure data for $NO_2$, CO, $SO_2$ and $O_3$ are derived from environmental monitoring stations rather than individual monitoring. Therefore, individual monitoring of other air pollutants is needed in the future to more accurately assess the impact of air pollution on lung function.

## Conclusions

Despite its limitations, our study suggested that short-term personal $PM_{2.5}$ exposure can decrease the $FEV_1$, FVC, DLco and MMEF in COPD patients and $FEV_1$, FVC and PEF in asthma patients. The results of the two-pollutant model suggest that the effects of $PM_{2.5}$ on $FEV_1$ in COPD patients and FVC in asthma patients are relatively independent, and are not affected by other gaseous pollutants. These findings indicate that $PM_{2.5}$ may primarily affect airway function and lung diffusion function in COPD patients, and airway function, rather than lung diffusion function, in asthma patients. Our findings can provide relevant information for the formulation of public health policies and the development of appropriate interventions to control air pollution, thus bringing benefits to public health. Further studies about the effect of different chemical components of $PM_{2.5}$ on lung function are needed in the future.

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**Authors' contributions** T.Y. and C.W. proposed this study and revised the manuscript. R.D., N.H. and T.Y. completed participants' recruitment and follow-up. R.D. wrote the original draft. H.N., T.Y. and K.H. helped perform the analysis with constructive discussions. H.C. and C.C. collected samples and did quality control. All authors revised the report and approved the final version before submission.

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**Data availability** Data can be obtained from appropriate authors on reasonable request.

## Declarations

**Ethics approval and consent to participate** The study protocol was approved by the Ethics Committee of the China–Japan Friendship Hospital (2017-19). Written informed consent was obtained from each participant.

**Consent for publication** Not applicable.

**Competing interests** Not applicable.

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