Influence of PM$_1$ exposure on total and cause-specific respiratory diseases: a systematic review and meta-analysis

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Abstract
An increasing number of studies examined the potential effects of PM$_1$ (submicronic particulate matter with an aerodynamic diameter $\leq 1 \mu m$) on the risk of respiratory diseases; however, the results have been inconclusive. This study aimed to determine the overall association between PM$_1$ with total and cause-specific respiratory diseases. A systematic review and meta-analysis was conducted with 68 related articles retrieved, and six articles met the full inclusion criteria for the final analysis. For a 10 $\mu g/m^3$ increase in PM$_1$, the pooled odds ratio (OR) was 1.05 (95% CI 0.98–1.12) for total respiratory diseases, 1.25 (95% CI 1.00–1.56) for asthma, and 1.07 (95% CI 1.04–1.10) for pneumonia with the $I^2$ value of 87%, 70%, and 0%, respectively. Subgroup analyses showed that long-term exposure to PM$_1$ was associated with increased risk of asthma (OR 1.47, 95% CI 1.33–1.63) with an $I^2$ value of 0%, while short-term exposure to PM$_1$ was not associated with asthma (OR 1.07, 95% CI 0.89–1.27) with the $I^2$ value of 0%. Egger’s test showed that publication bias existed ($P = 0.041$); however, the funnel plot was symmetrical with the inclusion of the moderator. In conclusion, elevated levels of PM$_1$ may increase morbidity in total and cause-specific respiratory diseases in the population.

Keywords Air pollution · PM$_1$ · Respiratory disease · Asthma · Pneumonia · Meta-analysis

Introduction
Respiratory diseases are the leading causes of morbidity and mortality worldwide (GBD 2019 Diseases and Injuries Collaborators 2020). In recent decades, chronic respiratory diseases, such as chronic obstructive pulmonary disease (COPD) and asthma, have attracted increasing attention (Wang et al. 2018; Huang et al. 2019). In 2019, the numbers of people with COPD and asthma were 212 million and 262 million worldwide, respectively (GBD 2019 Diseases and Injuries Collaborators 2020). Therefore, further study on the risk factors is necessary to minimize the morbidity of respiratory diseases and to improve prevention and guidelines for treating respiratory diseases.

Ambient particulate matter pollution has been a severe public health issue worldwide (Kim et al. 2019; Wang et al. 2021a). Although there has been improvement in air quality over recent decades in some countries, more than 90% of the global population lives in areas with air quality exceeding guidelines (Evanlopoulos et al. 2020). In 2019, ambient particulate matter pollution led about 1.4 million deaths in China (GBD 2019 Diseases and Injuries Collaborators 2020). The acute and long-term effects of ambient air pollution on human health are well known. Some epidemiological studies showed that the degree of exposure to ambient particulate matter (PM) is associated with daily mortality, mainly from cardiovascular and respiratory diseases (Liu et al. 2019a; Tian et al. 2020). Previous epidemiological studies have
focused on the adverse effects of fine particulate matter (PM$_{2.5}$, particulate matter with an aerodynamic diameter $\leq$ 2.5 $\mu$m) and inhalable particulate matter (PM$_{10}$, particulate matter with an aerodynamic diameter $\leq$ 10 $\mu$m) (Doiron et al. 2019; Yang et al. 2020). Exposure to PM$_{2.5}$ and PM$_{10}$ has been proven to be associated with respiratory diseases (Yao et al. 2020; Gurung et al. 2017; Pun et al. 2017; Sicard et al. 2019; Cao et al. 2021).

The most recent research indicated that PM$_1$ (submicron particulate matter with an aerodynamic diameter $\leq$ 1 $\mu$m) contributed 77–86% of the PM$_{2.5}$ concentration in China (Chen et al. 2018). However, it remains unknown whether PM$_1$ or PM$_{1-2.5}$ induced the adverse effects of PM$_{2.5}$. It has been stated that the size of PM has a negative correlation with the level of its toxicity in the lungs, which means that PM$_1$ provide more detrimental effects than PM$_{2.5}$ (Hamra et al. 2014; Valavanidis et al. 2008). However, limited evidence was found for the association between particulate matter and respiratory diseases, especially on PM$_1$.

According to previous epidemiological studies, the exposure to PM$_1$ contributing to the development of respiratory diseases remains uncertain (Zhang et al. 2021). To assess the effects of exposure to PM$_1$ quantitatively and accurately on the respiratory diseases, we conducted a systematic review and meta-analysis on all relevant studies published thus far.

**Methods**

**Literature search strategy**

This review was conducted according to the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement and the Meta-analysis of Observational Studies in Epidemiology (MOOSE) guidelines (Moher et al. 2009; Stroup et al. 2000). Three authors systematically searched PubMed (1966 to Apr 2021), Embase (1950 to Apr 2021), and the Cochrane Library (2000 to Apr 2021) for studies on the associations between PM$_1$ and respiratory diseases. The full search strategies are described in the supplementary data (Appendix 1). We also examined the references of the selected papers and reviews for additional pertinent data.

**Inclusion and exclusion criteria**

The flowchart of studies through the review process is shown in Fig. 1. Studies included in this meta-analysis met the following criteria: (1) epidemiological studies investigated the association between exposure to PM$_1$ and morbidity of respiratory diseases; (2) the subjects of the study were the general human population, regardless of age, geographical areas, and occupations of the population; (3) studies that quantitatively showed the results of estimation of exposure to ambient outdoor PM$_1$; (4) respiratory diseases in relation to exposure to PM$_1$ were selected according to the 10th Revision of the International Classification of Diseases: total respiratory diseases (ICD-10: J00-J99) and cause-specific diseases including upper respiratory tract infections (URTJ, J00-J06), pneumonia (J18), obstructive pulmonary diseases (COPD, J40-J44 and J47), and asthma (J45-J46); (5) provided the effect size of prevalence, hospital admission or emergency visit of respiratory diseases per 10 or interquartile range (IQR) $\mu$g/m$^3$ increase in PM$_1$ concentration: regression coefficient, percentage change ($PR$), excess rate ($ER$), risk ratio ($RR$), hazard ratio ($HR$) and odds ratio ($OR$), standard error ($SE$), and/or 95% confidence interval (CI); and (6) were published in English with full text. The exclusion criteria were as follows: (1) did not conform to the inclusion criteria or (2) reviews, commentaries, or communications; or (3) the subjects of the study were patients with comorbid diseases. If studies were published based on overlapping data, the most recent article with comprehensive data was included.

**Risk of bias and quality assessment**

The methodological quality and risk of individual studies were assessed independently by two authors (Yaoyu Hu and Mengqiu Wu) using the criteria recommended by BioMed Central for study assessment (Luong et al. 2019). This recommendation contains 20 items for several study designs. The details of the score are as follows:

1. Overall, scores higher than 75% are considered high quality, and scores lower than 50% are not included.
2. Single questions: (1) there are only two options (yes or no): 1 is high quality, and 0 is low quality; (2) there is a problem if there are two high-quality studies and one low-quality study; (3) if there are three problems, the highest one is high quality, the middle one is medium quality, and the second one is low quality. In addition, we used RoB (Risk of Bias) for all quality evaluations.

**Data extraction**

The data extraction process was independently conducted by two researchers. The extracted data included citation information (name of the first author, publication year, and location in which the study was carried out); study setting (study design, time span, sample size, age of population, and percentages of males); exposure (pollutants, exposure type, mean concentration, and method used to estimate air pollutant levels); and outcome (health outcome/diagnosis, day lags of the effect, unit of concentrations of PM$_1$, confounding factors that were adjusted for and study results). The short-term effect was
defined as < 7 days. For short-term exposure studies with multiple lag estimates, we used an a priori lag selection protocol devised by Atkinson et al. to select one estimate for preventing the overrepresentation of a single study in this meta-analysis (Atkinson et al. 2014).

**Statistical analysis**

We used OR with 95% CI in the prevalence/hospital admission/emergency visit of respiratory diseases as a measure of effect size. All estimates were transformed to a 10 μg/m³ increase in PM₁ concentration to pool results. We used Q-statistics to conduct heterogeneity tests, where \( P < 0.10 \) was considered to be statistically significant. The \( I^2 \) statistics were calculated to represent the percentage of variation observed in studies caused by heterogeneity. An \( I^2 \) value < 50% was generally regarded as low moderate heterogeneity between studies, indicating a fixed-effect model to pool the estimates. \( I^2 \) values > 50%, representing high heterogeneity, indicated a random-effect model. We used forest plots to graphically display results. We assessed publication bias using funnel plots, contour-enhanced meta-analysis funnel plots, Begg’s test, and Egger’s weighted linear regression.

A subgroup analysis based on the exposure type of PM₁ (short term and long term) was conducted. All tests were two-sided, and \( P < 0.05 \) was considered statistically significant, except in the heterogeneity test \( (P < 0.10) \). Transformation of effect size and meta-analysis was performed using MATLAB version 2018 and R version 4.0.5, respectively.

**Results**

**Search findings and study characteristics**

Sixty-eight studies were identified through the literature search, and 21 studies were eligible for full-text evaluation. Among them, 6 studies (Zhang et al. 2021; Wang et al. 2021b; Zhang et al. 2020; Yue et al. 2020; Luong et al. 2016; Michaud et al. 2004), published between 2004 and 2021, met our full inclusion criteria and were finally analyzed. The flowchart of this review shows the detailed process of selection (Fig. 1). The basic characteristics of the literature in the meta-analysis are summarized in Table 1. The included studies were performed in various regions (China, 4; Vietnam, 1; America, 1), including cross-sectional, time series, and case-crossover studies. In the included studies, PM₁ was defined as fine particulate matter with an aerodynamic diameter ≤ 1 μm. The daily mean concentrations of PM₁ across the 6 studies were 26.9 μg/m³. The outcomes of respiratory diseases included
Table 1: Summary of six articles included in the systematic review.

| ID  | Authors (year)       | Location       | Study design (time-span) | Population (years old) | Study size | Exposures (μg/m³) | Unit of increment (μg/m³) | Outcome | Effect (95% CI) | Controlled variables | Health outcome |
|-----|----------------------|-----------------|--------------------------|------------------------|------------|-------------------|--------------------------|---------|-----------------|---------------------|-----------------|
| 1   | Wang et al. (2021b)  | Hefei, China    | Time series, 2018–2016   | 15-68                  | 147        | 3.00              | 1.00                     | PM1      | 1.25 (1.00–1.54) | Yes                  | Pneumonia       |
| 2   | Zhang et al. (2021)  | Wuhan, China    | Cross-sectional, 2014–2018 | 3-5                    | 1000       | 7.40              | 1.00                     | PM1      | 1.54 (0.82–2.90) | Yes                  | Asthma Incidence |
| 3   | Zhang et al. (2020)  | Shenzhen, China | Case-crossover, 2015–2016 | All                    | 1000       | 19.00             | 1.00                     | PM1      | 1.12 (0.85–1.47) | Yes                  | Asthma Hospital admission |
| 4   | Yue et al. (2020)    | Liaoning, China | Cross-sectional, 2012-2013 | All                    | 1000       | 54.00             | 42.00                    | PM1      | 1.10 (1.01–1.19) | Yes                  | Asthma Hospital admission |
| 5   | Luong et al. (2016)  | Hanoi, Vietnam  | Case-crossover, 2010-2011 | All                    | 1000       | 19.00             | 1.00                     | PM1      | 1.03 (0.90–1.42) | Yes                  | Asthma Hospital admission |
| 6   | Michaud et al. (2004)| Hawaii, America | Time series, 1997-2001    | NA                     | 4339       | 1.97              | 10.00                    | PM1      | 1.07 (0.89–1.27) | Yes                  | Asthma Emergency department visit |

PC: percentage change; OR: odds ratio; HR: hazards ratio.

Discussion

This is the first meta-analysis to evaluate the impact of PM1 on respiratory diseases and provide an estimate of the impact. We found positive associations between PM1 and total and cause-specific respiratory diseases. PM1 is a health-damaging particle because it has chemical components and can penetrate deep inside the lungs to aggravate existing asthma or contribute to chronic bronchitis development (Fuertes et al. 2014;
Luong et al. found that an increase in PM$_1$ concentration would decrease pulmonary function (Luong et al. 2016). In addition, our meta-analyses showed that asthma was most strongly associated with PM$_1$, which was consistent with the limited literature available on fine particles (Liu et al. 2020; Hassanvand et al. 2015). We found that the funnel graph was asymmetric, and Egger’s test observed publication bias. However, with the addition of a moderator, we found that the funnel diagram is symmetrical. This indicates that most of the heterogeneity comes from different types of research designs.

It is not easy to assess the long-term impact of PM$_1$ on human health because long-term prospective observation and research require many resources and much effort.

**Fig. 2** Assessment of the risk of bias in the included studies

**Fig. 3** Forest plots for the association between PM$_1$ and respiratory diseases

(a) Total respiratory diseases

| Study      | log(OR) | SE     | Odds Ratio | OR   | 95% CI      | Weight |
|------------|---------|--------|------------|------|-------------|--------|
| Zhang-2020 | 0.09    | 0.0234 |            | 1.09 | [1.04; 1.14] | 44.3%  |
| Luong-2016 | 0.02    | 0.0058 |            | 1.02 | [1.01; 1.03] | 55.7%  |

Random effects model

Heterogeneity: $I^2 = 87\%$, $t^2 = 0.0020$, $p < 0.01$

(b) Asthma

| Study      | log(OR) | SE     | Odds Ratio | OR   | 95% CI      | Weight |
|------------|---------|--------|------------|------|-------------|--------|
| Zhang-2021 | 0.43    | 0.3213 |            | 1.54 | [0.82; 2.96] | 9.6%   |
| Zhang-2020 | 0.11    | 0.1397 |            | 1.12 | [0.85; 1.47] | 25.0%  |
| Yu-2020    | 0.38    | 0.0620 |            | 1.47 | [1.33; 1.63] | 37.1%  |
| Michaud-2004 | 0.03  | 0.1163 |            | 1.03 | [0.82; 1.28] | 28.3%  |

Random effects model

Heterogeneity: $I^2 = 70\%$, $t^2 = 0.0321$, $p = 0.02$

(c) Pneumonia

| Study      | log(OR) | SE     | Odds Ratio | OR   | 95% CI      | Weight |
|------------|---------|--------|------------|------|-------------|--------|
| Wang-2021  | 0.07    | 0.0144 |            | 1.07 | [1.04; 1.10] | 91.0%  |
| Zhang-2020 | 0.11    | 0.0457 |            | 1.12 | [1.02; 1.22] | 9.0%   |

Fixed effect model

Heterogeneity: $I^2 = 0\%$, $t^2 = 0.0$, $p = 0.32$
Special techniques are needed to measure and estimate the exposure level of air pollutants over a wide range of areas. The few available studies identified through our literature search reflect considerable difficulties in designing such a study to investigate the impact of PM$_1$ on respiratory diseases. In addition, the results of insufficient research proved to be inconsistent. Therefore, we believe that systematic review and meta-analysis would be a good choice for a more reliable estimation of the long-term effects of PM$_1$ on respiratory diseases.

Despite growing experimental evidence on the toxicity of PM$_1$, whether they truly contribute to the development of clinically manifested respiratory diseases is another question. A previous analysis showed a significant correlation between PM$_1$ exposure and the incidence of respiratory diseases (Yang et al. 2018). However, regarding respiratory diseases, whether PM$_1$ exposure can lead to the development of the disease has been controversial. Several reports have shown a higher prevalence of acute exacerbation of respiratory diseases, and odds have reported conflicting results. Previous studies on the long-term effects of PM$_1$ on respiratory diseases produced inconsistent results (Zhang et al. 2021). This inconsistency of previous studies may be based on differences in host factors that are difficult to quantify. For example, host susceptibility to air pollutants can vary widely due to genetic factors or other environmental factors, such as tobacco smoking status (Ward-Caviness 2019; Lyall et al. 2017).

The main finding of our study was a significant association between the incidence of respiratory diseases and exposure to PM$_1$. However, the explanation for this result is limited because the funnel diagram is asymmetric. This is not only because of the small research influence but also because more than 10,000 participants were involved in the research, which showed relatively wide confidence intervals (Zhang et al. 2021; Wang et al. 2021b). Furthermore, the asymmetry in the funnel plot may be due to the type of study design.
Although the trim-and-fill analysis showed that the pooled OR was not significantly influenced by this funnel plot asymmetry, further well-designed observational studies are still needed to better clarify the association between PM$_1$ and respiratory disease development.

The issue of heterogeneity between the studies must be addressed to appreciate our results more precisely. First, these studies have different follow-up times and durations according to different study designs. Second, different studies focus on different regions, which may lead to more significant. In addition, although there is a lack of knowledge regarding how long it takes for respiratory diseases to develop from exposure to PM$_1$, it is assumed that more prolonged exposure may be more harmful (Yu et al. 2020; Guan et al. 2016). This is in line with a study showing that the duration of tobacco smoking has a more substantial effect than the daily amount of cigarette consumption on the development of COPD (Bhatt et al. 2018). The lengths of the follow-up period of each study may influence the evaluation of the effect. In addition, there are differences in the methods used to estimate air pollutant levels, including ground-based monitoring stations or the space-time extremely randomized trees model. A recently adopted method uses satellite data to estimate air quality to improve the spatial and temporary resolution of air quality modeling (Wang et al. 2019). With satellite-based data, future research is expected to make it easier and more accurate to evaluate the health impact of PM$_1$.

Even though the exact biological mechanism for the association between PM$_1$ and respiratory diseases are not entirely clear, several studies suggested that inhalation of PM may result in inflammation and oxidative stress (Zou et al. 2020; Wang et al. 2020; Valavanidis et al. 2008). Small particles, especially PM$_1$, can more easily enter and deposit in the deeper respiratory tract. After internalized by respiratory epithelial cells, PM$_1$ can trigger oxidative stress and inflammatory responses (Valavanidis et al. 2008; Yang et al. 2017). Existing research indicates that the pro-inflammatory response may play important roles in the effect of PM$_1$ on lung function (Mazzarella et al. 2012).

One of our study’s strengths is the efforts made throughout the design and the systematic review to ensure its validity, including the incorporating risk of bias assessment. Another strength is to search all relevant literature and to make an in-depth, transparent, and repeatable evaluation of the evidence from studies focused on PM$_1$ exposures as a potential cause of respiratory diseases. It is a timely contribution to a rapidly evolving field that could inform future research’s focus and design to improve its utility. However, the present study has limitations. First, the number of included studies was small, and they did not cover all countries in the world. This limitation requires further research worldwide to assess the impact of PM$_1$ on respiratory diseases, which would contribute to an updated systematic review and meta-analysis. Second, most of the studies included in the meta-analysis were conducted in China, with fewer studies in high-income developed countries and low-income developing countries. Studies have shown that countries with different incomes have different air pollutant levels (Quansah et al. 2017; Liu et al. 2019b; Baumgartner et al. 2020; Naidja et al. 2018). Therefore, we need more national data to understand the health effects of PM$_1$ more clearly. Finally, our meta-analysis is based on different observational research designs, including cross-sectional, case-crossover, and time series. Therefore, individual studies may be affected by uncontrolled time-varying deviations, which we could not test.
Conclusion

The present systematic review and meta-analysis demonstrated that the pooled effect of a 10 μg/m³ increase in PM₁ on total respiratory diseases was not statistically significant (OR 1.05, 95% CI 0.98–1.12). There was a marginal association between a 10 μg/m³ increase in PM₁ and asthma (OR 1.25, 95% CI 1.00–1.56). A 10 μg/m³ increase in PM₁ was positively associated with pneumonia (OR 1.07, 95% CI 1.04–1.10). Our research helps evaluate the current literature to understand the public health impact better worldwide. However, the amount of research conducted globally is still minimal, so more research needs to be carried out in different regions.

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Data availability Not applicable

Declarations

Ethics approval Not applicable

Consent to participate Not applicable

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Competing interests The authors declare no competing interests.

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