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Effect of lead exposure on respiratory health: a systematic review and meta-analysis

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Abstract
In recent years, the incidence of respiratory diseases such as asthma and pneumonia has increased significantly. However, the effect of lead (Pb) pollution on the respiratory system remains unclear. The aim of this study was to evaluate the effects of exposure to environmental and occupational Pb on respiratory health. Articles published in PubMed and Web of Science before September 2023 were systematically searched. The overall adjusted odds ratio (OR) and 95% confidence intervals (CIs) for the association between Pb exposure and respiratory diseases were extracted from each relevant article. The random effects model was applied to analyze the overall pooled effect estimates. Among the 36,373 search results, 36 related articles were screened for meta-analysis. The results of the meta-analysis suggested that Pb exposure increased the risk of respiratory diseases: OR = 1.12 (95% CIs: 1.05, 1.18). The funnel plot, Egger's and Begg's tests showed no publication bias. Sensitivity analysis confirmed that the meta-analysis was statistically reliable and stable. Environmental and occupational Pb exposure is associated with an increased risk of respiratory diseases including asthma. The study highlights the importance of further research on the harmful effects of Pb and the urgency of mitigating air pollution.

Keywords Pb exposure · Respiratory health · Meta-analysis · Systematic review

Introduction

Air pollution is harmful to human health and increases the global economic burden of environmental diseases (Nunes et al. 2021). Long-term exposure to atmospheric particulate matter (PM), especially fine $PM_{2.5}$ (aerodynamic diameter ≤ 2.5 μm) can lead to high mortality and morbidity (McGuinn et al. 2019; Shi et al. 2021). Although the content of trace elements in $PM_{2.5}$ is low, some of them are usually highly toxic, non-degradable in the environment, and easy to bioaccumulate (Hua et al. 2023). Among them, lead (Pb), as a cumulative toxicant, is listed by the World Health Organization (WHO) as one of the top ten chemicals of public health concern (WHO 2022). WHO also estimates that Pb exposure caused 21.7 million years of disability and death globally in 2019 because of long-term health effects (WHO 2022). Pb in the environment mainly comes from sources such as non-ferrous metal mining and smelting, coal combustion, and past Pb-containing gasoline combustion (Cui et al. 2023). In addition, Pb is widely used in industries such as paints, batteries, plastics, printing, pigments, ceramics, and cosmetics (Tarvainen et al. 2023).

People are exposed to Pb through occupational and environmental exposures, which mainly include inhalation of Pb particles from burning Pb-containing materials and ingestion of Pb-contaminated dust, water, and food. Pb can be transported to various tissues and organs through blood circulation, which is known to affect almost all human organ systems (Liu et al. 2024). Pb that enters the body is absorbed through the digestive system and affects the function of the reproductive, liver, endocrine, immune, and gastrointestinal systems (Swaringen et al. 2022). Studies have shown that Pb poisoning leads to cognitive decline, growth retardation, and decreased immunity in children (Galiciolli et al. 2022).

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At present, acute and chronic respiratory infections are the leading causes of morbidity and mortality worldwide (Reiner et al. 2019). Collaborators (2017) analyzed the global burden of disease system in 2015 and found that lower respiratory tract infections caused 20.74 million deaths and 103 million disability-adjusted life-years. Exposure to Pb-contaminated environments is significantly associated with respiratory infections in preschool children and increases the incidence of obstructive pulmonary disease and bronchial reactivity (Li et al. 2020; Rabito et al. 2011; Rokadia and Agarwal 2013). However, some studies have obtained different conclusions. For example, Lee et al. (2020) showed that chronic obstructive pulmonary disease (COPD) was not related with blood lead levels after adjusting for possible confounding variables such as smoking and occupation. The relationship between Pb exposure and respiratory diseases remains to be firmly determined. Therefore, more reliable estimates are required to assess the effects of Pb exposure on respiratory diseases.

A previous review analyzed the effects of Pb and zinc exposure on asthma in the general population from 2000 to 2018 (Darabi et al. 2023). However, only four published articles were reviewed in this review to assess the effect of blood Pb levels on asthma. It is difficult to establish a relationship between Pb exposure and respiratory disease because the available evidence is incomplete and there is only one endpoint for respiratory disease outcomes. Recently, there has been a rapid increase in the number of studies on Pb exposure and respiratory diseases and an increase in interest from the scientific community, which requires the latest review of the evidence. Therefore, we aimed to conduct a systematic review and meta-analysis of published evidence on the association between Pb exposure and respiratory disease-related outcomes in the general population. We followed all applicable PRISMA guidelines for system review and meta-analysis (Crawford et al. 2023).

Materials and methods

Search strategy

Literature on Pb exposure and respiratory diseases was searched in PubMed and Web of Science online databases. We limited the search to studies published up to September 29, 2023. Keywords such as Pb and respiratory diseases were used to retrieve relevant literature, as detailed in Table S1.

Eligibility criteria

After removing duplicate articles using automated identification, we screened titles and abstracts to filter articles that did not meet the inclusion criteria. The full text of the remaining literature was independently reviewed by two reviewers (CT and DKX). In case of inconsistent opinions, the third reviewer (WHH) reviews the manuscript based on the inclusion and exclusion criteria, and the final decision was made after discussion by the third reviewer. Details of the inclusion and exclusion criteria are given below:

Inclusion criteria

- Cohort, cross-sectional, and case-control studies.
- The outcome was symptoms of respiratory disease.
- Studies reported the effect of Pb on respiratory diseases quantitatively, and outcomes included effect estimates, such as rate ratio (RR), odds ratio (OR), hazard ratio (HR), and 95% confidence intervals (CIs).
- Peer-reviewed studies.

Exclusion criteria

- Duplicate studies.
- Conference abstracts, experimental studies, and grey literature.
- Studies that did not report effect measure or 95% CIs, variance, or standard error.
- Case report, case-control, time-series, and case-crossover designs.
- Study populations that were limited by specific medical conditions.
- research on animals.

Risk of bias

There are no commonly used tools to evaluate the risk of bias (RoB) at the level of systematic review of observational study of environmental exposure (Cai et al. 2021). We used the tool developed by the WHO to assess RoB in observational air pollution epidemiological studies (WHO 2020). The RoB assessment tool evaluates the following six areas: confounding, selection bias, exposure assessment, outcome assessment, missing data, and selective reporting. For each domain, RoB is evaluated as "low", "moderate" or "high". Each domain was divided into 1 to 4 subdomains, with a total of 13 subdomains. The RoB for each study is the

highest rating for an individual subdomain. The RoB assessment for each qualified study was performed independently by two authors (CT and DKX). Differences were resolved through discussion. In case of disagreement, the third author (WHH) assists in the unification.

Data extraction and synthesis

Two authors jointly completed the data extraction, one author (CT) extracted the information, and the other author (DKX) checked the information. Information extracted included the main authors, publication year, study area, design, study population characteristics, exposure assessment method, exposure levels, health outcomes, effect estimates, correlation coefficients, and corresponding 95% CIs. For all the above indicators, we considered an article to be "unclear" if it did not contain an explicit conclusion. If there is no report in the article, we considered it "not reported".

Meta-analysis

Meta-analysis was performed using the statistical software STATA 17. We extracted effect values and their associated 95% CIs as the base data to assess the effect of Pb exposure on respiratory diseases. Most articles used OR value and 95% CIs to indicate the effect. If the original article reported the β value and SE (standard error), the formulas exp(β) and exp(β ± 1.96*SE) were applied to calculate the OR and its 95% CIs (Borroni et al. 2022). Random effects model was used to obtain pooled OR and to calculate the heterogeneity of any study. $P < 0.05$ or $I^2 > 50\%$ indicates heterogeneity (Cheng et al. 2022). Subgroup analyses were performed based on factors such as region, age, sample, and study design. Funnel plots, Egger's and Begg's tests were applied to assess publication bias (Wu et al. 2021). To evaluate potential unstable factors in the meta-analysis, sensitivity analysis were performed by removing one study at a time (Zhang et al. 2023).

Results

Search results

The PRISMA flowchart illustrates the literature screening process of this study (Fig. 1). Of the initial 36,373 studies, the titles and abstracts of 25,001 studies were considered. Subsequently, 1828 articles were carefully read, 76 articles were included in qualitative synthesis, and 36 articles were included in the meta-analysis. We list the main features and

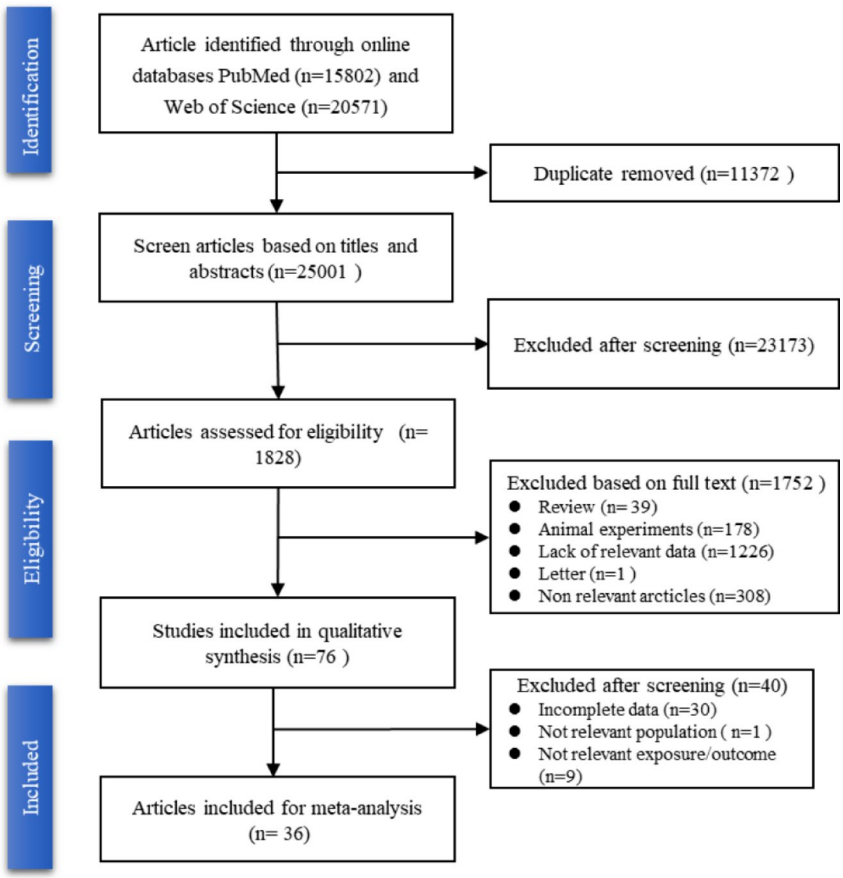

Fig. 1 Flow chart of study selection

results of each study in Table 1. Of the 36 selected studies, 16 were from North America, 16 were from Asia, and 4 were from Europe. Twenty-two were cross-sectionally designs, followed by 10 cohort studies and 4 case-control studies.

Characteristics of the eligible studies

The 36 included studies had different study period, exposure characteristic, and population characteristics. There were 286,077 participants in 36 studies. The duration of investigation ranged from a few months (short-term) to a year or more (long-term). Twenty-four studies investigated long-term health indicators, 6 studies investigated short-term health outcomes, and 6 studies did not specify the study period.

The study evaluated the outcome of one or more different respiratory diseases, specifically: asthma ($n=26$), wheezing ($n=8$), cough ($n=4$), dyspnea/shortness of breath ($n=3$), respiratory infections/syncytial virus ($n=3$), throat discomfort ($n=3$), chronic bronchitis ($n=2$), and obstructive lung disease ($n=2$). Most of the studies measured blood Pb concentrations in the subjects. In addition, one study measured prenatal urinary Pb, three studies measured prenatal maternal blood Pb, two studies measured umbilical cord Pb, and two studies measured Pb in total suspended particulates.

Risk of bias assessment

Figure 2 shows the RoB assessment for the included studies. Table S2 shows the detailed analysis of the six areas based on RoB. It was found that 7 studies had high RoB, 6 studies had low RoB, and the other studies were assessed as having moderate RoB. In terms of RoB, confounding factor is the weakest domain. Among them, 16 studies did not adjust for BMI and 5 studies did not adjust for any confounding factors (Bener et al. 2001; Khazdair et al. 2012; Kuntawee et al. 2020; Mitsui-Iwama et al. 2019; Myers et al. 2002).

Results of the meta-analysis

Figure 3 and Table S3 show the pooled effect estimations and heterogeneity of each endpoint. Although heterogeneity existed across studies and varied by study area and evaluation method, the results showed that Pb exposure was associated with respiratory diseases (OR = 1.12, 95% CIs: 1.05, 1.18). Figure S1 shows the results of sensitivity analysis. Exclusion of individual studies did not significantly affect the results, revealing that the analysis was statistically reliable and stable.

For the different endpoints, Pb exposure had no significant effect on wheeze (OR = 1.05, 95% CIs: 0.93, 1.20), cough (OR = 1.11, 95% CIs: 0.87, 1.41), respiratory symptoms (OR = 1.69, 95% CIs: 0.81, 3.54), shortness of breath (OR = 1.66, 95% CIs: 0.61, 4.50), throat discomfort (OR = 1.53, 95% CIs: 0.78, 3.01), bronchial symptoms (OR = 0.95, 95% CIs: 0.70, 1.28), and lung disease (OR = 1.59, 95% CIs: 0.97, 2.58). Pb exposure was associated with asthma (OR = 1.12, 95% CIs: 1.02, 1.23).

Figure 4 shows the subgroup analyses. Significant associations of Pb exposure with respiratory diseases were observed in North America (OR = 1.11, 95% CIs: 1.02, 1.22) and Asia (OR = 1.16, 95% CIs: 1.05, 1.28). Regarding the age subgroup analyses, Pb pollution was associated with increase respiratory disease in the age groups < 6 years (OR = 1.17, 95% CIs: 1.00, 1.38) and > 18 years (OR = 1.15, 95% CIs: 1.03, 1.29). For the different Pb samples, the OR was 1.27 (95% CIs: 1.03, 1.56) for Pb in $PM_{2.5}$, 1.31 (95% CIs: 1.04, 1.66) for maternal blood Pb, and 1.12 (95% CIs: 1.05, 1.19) for blood Pb. Subgroup analysis of the study design showed that Pb pollution was significantly associated with respiratory diseases (OR = 1.18, 95% CIs: 1.10, 1.26) in the cross-sectional.

Publication bias

The funnel plots of all included studies were visually symmetrical (Figure S2), indicating no publication bias. The Egger's ($P=0.07$) and Begg's ($P=0.81$) tests were both greater than 0.05, confirming the absence of publication bias for the endpoint of respiratory diseases.

Discussion

In this meta-analysis, 36 population-based articles were identified to assess the impact of Pb exposure on respiratory disease. In general, the available evidence indicates that Pb exposure is significantly associated with respiratory diseases (OR = 1.12, 95% CIs: 1.05, 1.18). Twenty-six studies investigating the association between Pb exposure and asthma indicated some evidence of a positive relationship. The authors conducted a large population-based birth cohort study and found that pregnant woman and infant cumulative exposure to low levels of Pb may increase pediatric asthma risk (Hsieh et al. 2021). Similarly, Koh et al. (2019) found that serum Pb levels were associated with self-reported asthma and atopic dermatitis in subjects at least 19 years of age. Farkhondeh et al. (2015) summarized the possible mechanisms of Pb-induced asthma, including Pb-induced inflammation and oxidative stress, as well as effects on the immune system.

Eight studies have investigated the association between Pb exposure and the incidence of wheezing. A birth cohort

Table 1 Characteristics of articles included

Reference	Location	Study period	Study design	Sample size	Mean age (SD) or range (years)	Exposure sample	Exposure range or mean	Results	Main findings
Yau et al. (2023)	China	2016 to 2018	Cross-sectional study	1906	≥ 20	Blood Pb	0.5–37.6 μg/dL	Lung fibrotic changes: OR = 1.36 (1.01,1.82)	Blood Pb was associated with lung fibrotic change.
Ruan et al. (2022)	China	2013 to 2016	Cohort study	628	3.96 ± 0.47	Maternal urinary Pb	0.322 μg/dL	Wheeze: OR = 1.24 (0.69, 2.25)	Prenatal Pb exposure was not associated with wheeze.
McRae et al. (2022)	Mexico	July 2007 to February 2011	Cohort study	633	4–5 and 6–7	Maternal blood Pb	2.88 μg/dL	Ever Wheeze: OR = 1.97 (1.05, 3.67) Asthma: OR = 0.84 (0.27, 2.62)	Blood Pb was associated with ever wheeze.
Hsieh et al. (2021)	China	2004 to 2014	Cohort study	171,281	3.41 ± 1.78	Simulated ambient Pb	0.495 ng/m^3	Asthma: HR = 1.28 (1.07–1.53) HR = 0.82 (0.68–0.97)	Prenatal Pb exposure was associated with asthma.
Feiler et al. (2021)	US	July 1994 to January 1995	Cohort study	222	0.5-6	Blood Pb	0–50 μg/dL	Asthma: RR = 1.03 (0.98, 1.09)	Child blood Pb levels was not associated with diagnosed asthma.
Pesce et al. (2021)	French	2003 to 2006	Cohort study	651	8	Maternal blood Pb and cord blood Pb	19.1 and 14.5 μg/dL	Asthma: HR = 1.25 (0.71, 2.20) HR = 0.74 (0.41, 1.33)	Maternal Pb blood levels were not associated with asthma.
Zammit et al. (2020)	Italy	March 2012 to March 2013	Cross-sectional study	2400	11–14	Pb in $PM_{2.5}$	0.9–10 ng/m^3	Asthma: OR = 1.1 (1.04, 1.15)	Pb in $PM_{2.5}$ was associated with doctor-diagnosed asthma.
Kuntawee et al. (2020)	Thailand	May to July 2017	Cross-sectional study	102	47–68	Blood Pb	0.48 μg/dL	Asthma: OR = 0.79 (0.36, 1.73)	Blood Pb was not associated with asthma.
Li et al. (2020)	China	September 2017 to October 2018	Case-control study	219	3–7	Blood Pb	0.90–6.19 μg/dL	Recurrent respiratory infections: RR = 2.33 (1.23, 4.41)	Blood Pb was associated with respiratory infections in preschool children.
Cornwell et al. (2020)	US	2001 to 2016	Cross-sectional study	14,751	1–11	Blood Pb	-	Asthma: PR = 1.09 (0.76, 1.59)	There was no association of blood Pb and asthma.
Oktapodas Feiler et al. (2020)	US	2012 to 2017	Case-control Study	617	< 4	Blood Pb	0–70 μg/dL	Respiratory Syncytial Virus: OR = 0.95 (0.6, 1.49)	Blood Pb was not associated with respiratory syncytial virus.
Mitsui-Iwama et al. (2019)	Japan	October 2016 to April 2017	Cross-sectional study	446	0–68	Urinary Pb	Control:1.81 Case:3.45 pmol/Cr	Wheeze: OR = 3.16 (0.68, 14.6)	Urinary Pb increased in those with current wheeze.
Koh et al. (2019)	Korean	2005 to 2016	Cross-sectional study	16,089	≥ 19	Serum Pb	-	Asthma: OR = 1.1 (1.02, 1.17)	Serum Pb level was associated with self-reported asthma.
Yang et al. (2019)	US	2007 to 2012	Cross-sectional study	13,888	20–79	Serum Pb	0.154 vs. 0.165 μg/L	Asthma: OR = 1.67 (1.1, 2.55)	High serum lead level was associated with current asthma.
Wu et al. (2019)	US	2007 to 2012	Cross-sectional study	5866	2–15	Blood Pb	-	Asthma: OR = 1.08 (1, 1.16), Wheezing or whistling: OR = 0.99 (0.92, 1.08)	Blood Pb was associated with asthma and not associated with current wheezing or whistling.

Table 1 (continued)

Reference	Location	Study period	Study design	Sample size	Mean age (SD) or range (years)	Exposure sample	Exposure range or mean	Results	Main findings
Kang and Kim (2019)	Korean	2010 to 2013	Cross-sectional study	1478	10–19	Blood Pb	1.33 μg/dL	Asthma: OR = 1.94 (1.06, 3.57)	Blood Pb was associated with asthma.
Shaheen and Pan (2019)	US	1999 to 2016	Cross-sectional study	22,885	1–15	Blood Pb	-	Asthma: OR = 1.4 (1.2, 1.7)	Blood Pb was associated with asthma in children.
Wang et al. (2017)	China	2011	Cross-sectional study	930	5.74 ± 0.77	Blood Pb	1.86 ± 1.21 μg/dL	Asthma: OR = 5.5 (1.69, 17.94)	Blood Pb was associated with asthma.
Park et al. (2016)	Korean	2010 to 2012	Cross-sectional study	5912	≥ 19	Blood Pb	0.42–17.71 μg/dL	Asthma: OR = 1.67 (1.1, 2.55)	Pb exposure may increase risk of asthma.
Huang et al. (2016)	China	October 2010 to January 2012	Case-control study	1102	> 18	Urinary Pb	1.93 vs. 1.39 μg/g	Asthma: OR = 0.52 (0.42, 0.63)	Asthma prevalence was negatively associated with urinary Pb.
Zeng et al. (2016)	China	December 2012 to January 2013	Cross-sectional study	470	3–8	Blood Pb	6.24 vs. 4.75 μg/dL	Wheeze: OR = 0.64 (0.32, 1.27) Dyspnea: OR = 0.64 (0.23, 1.79) Cough: OR = 0.95 (0.6, 1.52) Phlegm: OR = 1.2 (0.72, 2.01) Asthma: OR = 9.5 (1.16, 77.49)	Pb exposure was associated with respiratory symptoms and asthma.
Nguyen et al. (2016)	Japan	4 January 2011 to 30 June 2011	Cohort study	87	56.8–64.8	Pb in Total Suspended Particulates	0-37.33 ng/m^3	Cough: OR = 1.04 (0.96, 1.13)	No relationship was observed between Pb and cough.
Wells et al. (2014)	US	2005 to 2006	Cross-sectional study	1430	4–12	Blood Pb	1.13 μg/dL	Asthma: OR = 1.07 (0.86, 1.33)	There was no relationship between Pb and asthma.
Rabito et al. (2013)	US	-	Cohort study	1297	9	Blood Pb	-	Asthma: OR = 0.91 (0.55, 1.48)	Pb exposure was not associated with asthma.
Rokadia and Agarwal (2013)	US	2007 to 2010	Cross-sectional study	9575	42.3 vs. 54.4	Serum Pb	1.18 vs. 1.73 μg/dL	Obstructive lung disease: OR = 2.37 (1,08, 5.19)	Obstructive lung disease was significantly associated with serum Pb.
Mendy et al. (2012)	US	2007 to 2008	Cross-sectional study	1857	≥ 20	Urinary Pb	0.59 μg/g	Asthma: OR = 0.72 (0.46, 1.12) Chronic bronchitis: OR = 0.68 (0.36, 1.29)	Asthma and chronic bronchitis were not related to Pb.
Khazdair et al. (2012)	Iran	-	Cross-sectional study	208	34.13 vs. 31.31	Serum and urine Pb	urine Pb: 23.56 vs. 76.84 serum Pb: 72.51 vs. 370.50	Sputum: OR = 6.04 (1.71, 21.30) Cough: OR = 3.80 (1.35, 10.66) Wheezing: OR = 1.41 (0.39, 5.16) Tightness of breath: OR = 5.48 (2.16, 13.91)	Pb exposure workers have higher risk of respiratory diseases.
Pugh Smith and Nriagu (2011)	US	-	Cross-sectional study	356	0–14	Blood Pb	-	Asthma: OR = 7.52 (1.32, 42.9)	The level of blood Pb in asthmatic children is five times higher than that in non-asthmatic children.

Table 1 (continued)

Reference	Location	Study period	Study design	Sample size	Mean age (SD) or range (years)	Exposure sample	Exposure range or mean	Results	Main findings
Rabito et al. (2011)	US	October 2007 to October 2008	Cohort study	73	18–50	Blood Pb	0.6 to 38.4 μg/dL	Respiratory symptoms: OR = 2.91 (0.94, 9.06)	Construction work was associated with increase of respiratory symptoms and blood Pb levels.
Motosue et al. (2009)	US	-	Cross-sectional study	682	-	-	-	Asthma: OR = 1.86 (1.01, 3.43)	Blood Pb level was significantly associated with asthma in the low-income Hispanic population.
Min et al. (2008)	Korean	-	Cross-sectional study	523	19 to 58	Blood Pb	2.96 ± 1.59 μg/dL	Increased bronchial responsiveness: β = 0.01797, SE = 0.007	Blood Pb was associated with increased bronchial responsiveness.
Joseph Christine et al. (2005)	US	1995 to 1998	Cross-sectional study	4634	1.2	Blood Pb	-	Prevalent asthma: OR = 1.2 (0.9, 1.5)	Among African Americans, blood Pb level was not associated with asthma.
Palkovicova et al. (2004)	Slovakia	-	Cohort study	304	0–5	Placental Pb	0.023 vs. 0.039 mg/g	Asthma: OR = 2.61 (1.19, 5.69)	Placental Pb concentrations was associated with asthma.
Shaheen et al. (2004)	UK	1991 to 1992	Cohort study	2173	2.5–3.5	Umbilical cord Pb	0.0270 ppb	Wheezing: OR = 1.05 (0.88, 1.25)	No significant associations were found between wheezing and umbilical cord Pb.
Myers et al. (2002)	US	1999	Case-control study	202	26.6 vs. 24.2 months	Blood Pb	control: < 0.2 μmol/L case: ≥1.2 μmol/L	Asthma: OR = 0.7 (0.4, 1.3)	There was no relationship between placental Pb and asthma.
Bener et al. (2001)	United Arab Emirates	February to June 1999	Cross-sectional study	200	control: 35.5 case: 34.6	Blood Pb	19.8 vs. 77.5 μg/dL	Throat discomfort: RR = 1.06 (0.67, 1.66) Cough: RR = 1.11 (0.82, 1.51) Shortness of breath: RR = 1.33 (0.96, 1.86) Wheeze: RR = 1.08 (0.79, 1.48) Asthma: RR = 1.75 (1.36, 2.26)	The occurrence of some respiratory symptoms may be related to Pb exposure in industrial workers.

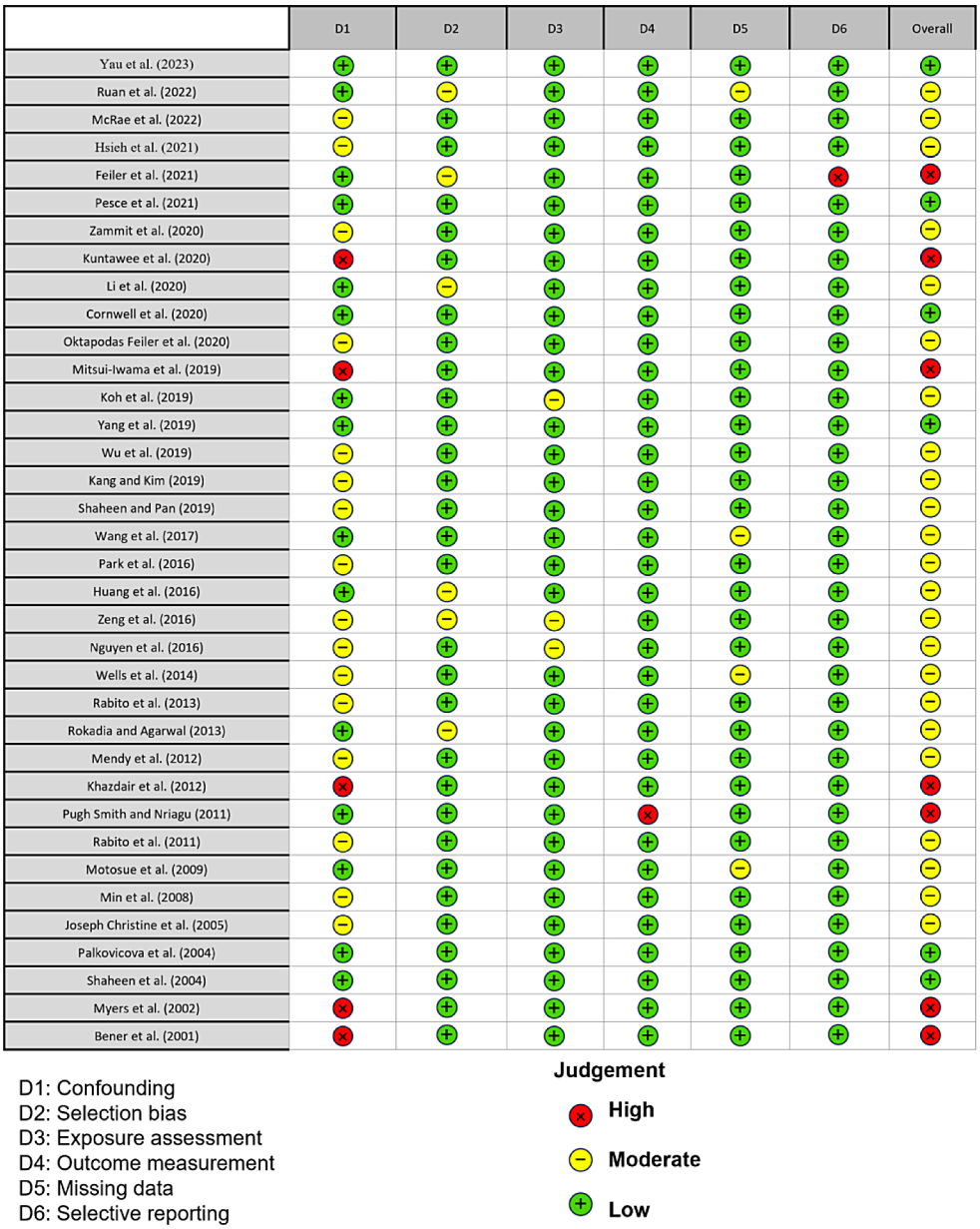

Fig. 2 Traffic light plot of RoB assessment

study in Mexico City found a significant association between Pb exposure and ever wheeze (McRae et al. 2022). Conversely, they did not find an association between prenatal Pb exposure and early childhood wheeze (OR = 1.24, 95% CIs: 0.69, 2.25) (Ruan et al. 2022). There were four studies that explored the effect of long-term exposure to Pb on the risk of cough. Khazdair et al. (2012) suggested a significant positive effect of Pb exposure on cough, whereas 3 associations were non-significant. Regarding the outcome endpoints of respiratory symptoms, shortness of breath, throat discomfort, bronchial symptoms, and lung disease, no clear associations with Pb exposure were found in our meta-analyses. Evidence on some of these outcome endpoints is very sparse, reported in only two studies.

The harm of Pb on the respiratory system should not be ignored, especially for children under 6 years old (OR = 1.17, 95% CIs: 1.00, 1.38) and adults older than 18 years (OR = 1.15, 95% CIs: 1.03, 1.29). Similarly, Taylor et al. (2019) found that Pb emissions from smelting posed a significant risk of harm to children's health, including respiratory diseases, IQ, academic performance, and social behavior problems. Asthmatic children with high blood Pb were more likely to have eosinophilia, higher total IgE levels (83.3%), and more severe asthma symptoms (Mohammed et al. 2015). Zheng et al. (2013) found that children in Guiyu Town, one of the largest e-waste disposal centers in China, had significantly lower forced vital capacity (FVC) than the lowest age group in the reference region.

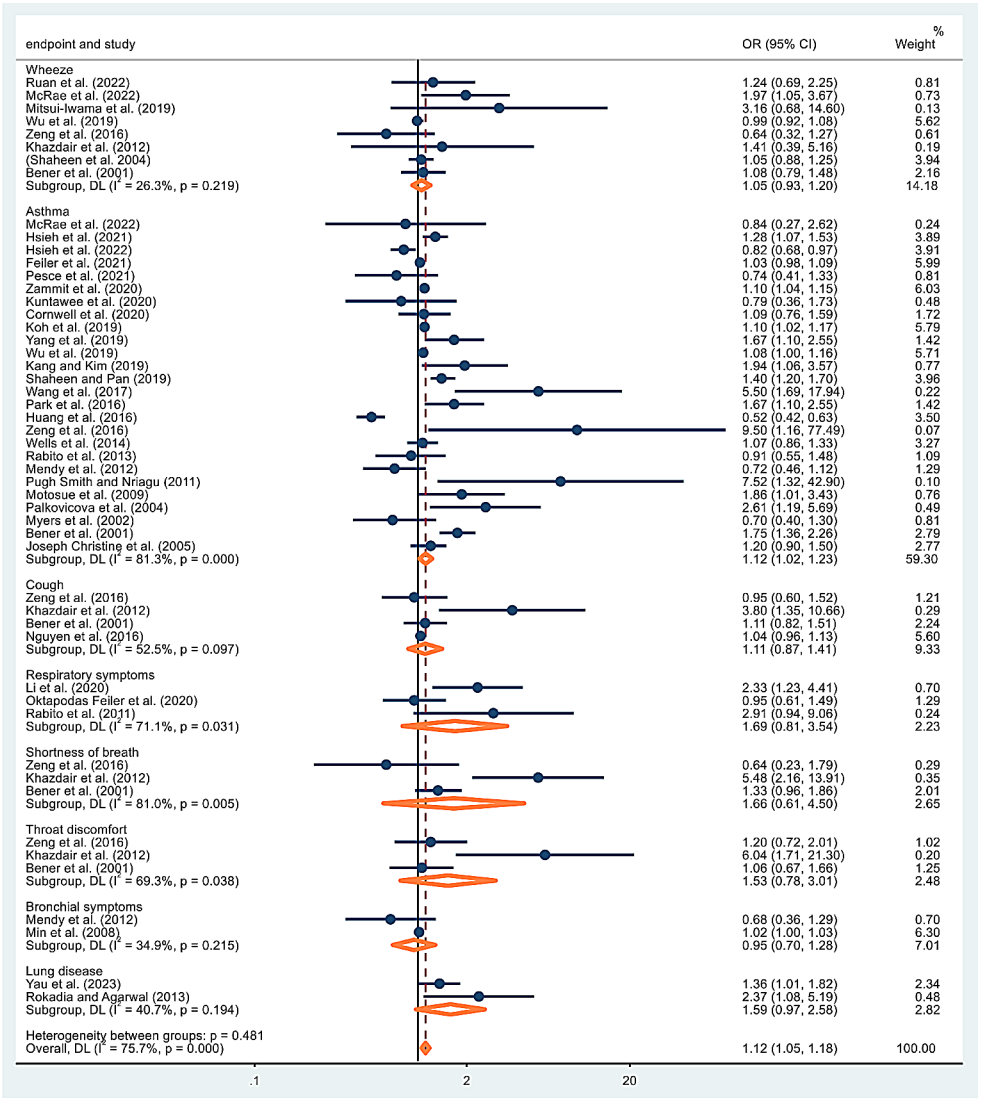

Fig. 3 Forest plot of Pb exposure and respiratory diseases

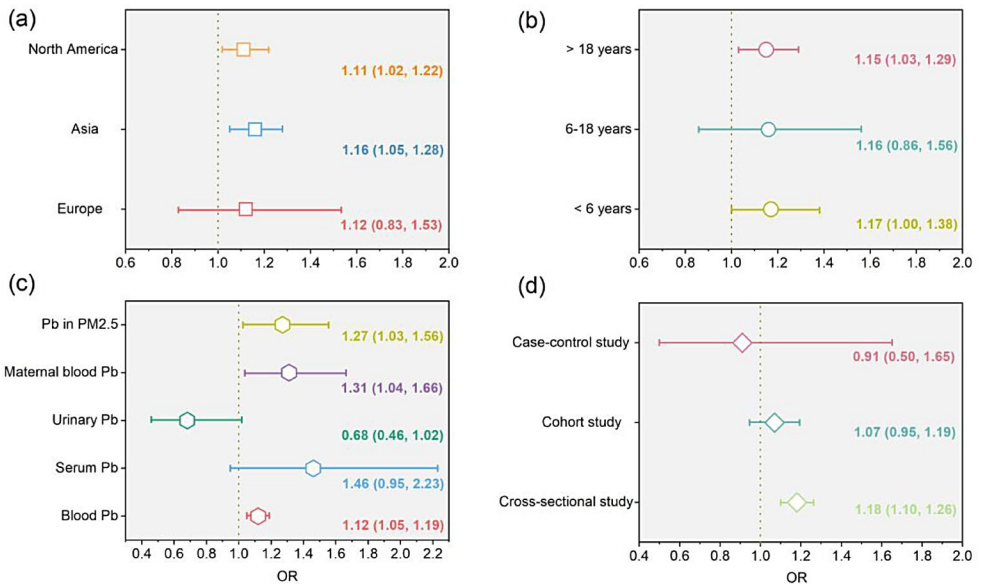

Fig. 4 Subgroup analysis for respiratory diseases adjusted based on the study area **(a)**, age **(b)**, samples **(c)**, and study design **(d)**

Prenatal Pb exposure in pregnant women can also lead to a significant increase in the incidence of asthma, wheezing, and obstructive bronchitis in children (Palkovicova et al. 2004). Children are a more vulnerable and sensitive group due to their physical incompetence compared to adults. In addition, children have unique exposure pathways (mother-to-child transmission), high-risk behaviors (hand-to-mouth contact), larger surface areas of respiratory capacity and body weight, and lower toxin clearance rates, resulting in a high risk of exposure (Grant et al. 2013). The sequelae of childhood Pb exposure may persist into adolescence and adulthood (David et al. 2022). Jurdziak et al. (2015) and Khazdair et al. (2012) found that Pb exposure is more likely to lead to respiratory symptoms such as chest pressure, sputum, wheezing, and decreased vital capacity parameters (e.g., FVC) in adults. Adults can be exposed to Pb through a variety of routes, including occupational settings, smoking, transportation, and environmental exposures. Long-term bioaccumulation and high-dose exposure to Pb may lead to mitochondrial damage and lipid accumulation in lung cells, thereby accelerating lung tissue damage (Lin et al. 2024). In addition, Wen et al. (2023) found a positive correlation between blood lead and blood eosinophil counts in adults with asthma. Higher blood eosinophil counts have been shown to be a risk factor for future asthma attacks in adults with persistent asthma.

A growing number of reports provide preliminary evidence for the mechanisms of Pb toxicity to the respiratory system. Pb may indirectly damage interstitial lung cells and tissues by triggering inflammatory response and contribute to COPD (Cabral et al. 2015). Dobrakowski et al. (2016a) investigated the correlation between occupational Pb accumulation and oxidative stress in 36 males. Dobrakowski et al. (2016b) found that occupational Pb can lead to changes in the activity of enzymes related to the antioxidant defense system, thereby inducing increased oxidative stress. However, it is unclear whether a slight increase in oxidative stress markers increases the risk of future clinical outcomes. Zeng et al. (2017) further confirmed that heavy metal Pb can produce reactive oxygen species (ROS), resulting in lung cell damage, alveolar collapse, atelectasis, and alveolar ventilation dysfunction. Oxidants can also cause airway inflammation and airway hyperresponsiveness, which is a major symptoms of asthma (Duan et al. 2022). Wei et al. (2020) in a 4-year longitudinal follow-up of 1243 workers, found that high Pb exposure increased biomarkers of oxidative stress, leading to lipid peroxidation and redox imbalance, as well as changes in the morphology and function of lung epithelial cells.

Recent articles have indicated that Pb interferes with the redox balance of the body and that immunotoxicity may be the causes of respiratory diseases. The pathological mechanisms of Pb immunotoxicity are mainly characterized by changes in the concentration of Th1 lymphocytes, IgE, and some cytokines (Jurdziak et al. 2015). The imbalance between Th1 and Th2 lymphocytes under Pb exposure may be responsible for the stimulation of production, thereby increasing the risk of atopic response and asthma (Gao et al. 2007). Kalahasthi et al. (2022) reviewed 40 studies that found significantly higher levels of impaired immune and inflammatory markers in occupational Pb-exposed populations. High-quality articles are required to strengthen the understanding of Pb in the mechanism of immunotoxins and to reveal its association with respiratory diseases. Many gaps remain in the understanding of Pb exposure and respiratory function in children. Zeng et al. (2017) found that children living in exposed areas had higher blood Pb and lower levels of hemoglobin, hematocrit, and lung function. However, the effects and mechanisms of low hemoglobin on respiratory diseases are still unclear.

Despite the current ban on leaded gasoline and paint, Pb pollution continues to increase the burden of disease, especially in low- and middle-income countries (Attina Teresa and Trasande 2013). The main sources of Pb include deteriorating paint, dust, soil, air, drugs, cosmetics, toys, adulterated food, and workplace etc. (Swaringen et al. 2022). Emissions from Pb smelters can contaminate aerosols, soil, and dust, resulting in increased concentrations of Pb in human blood, which is the main source of Pb pollution. Currently, recycling of lead-acid batteries and poorly controlled electronics are becoming major sources of Pb pollution (Earl et al. 2016). A survey by the Indian Chamber of Commerce and Industry revealed that 76% of e-waste workers suffer from respiratory diseases (including asthma, breathing difficulties, and coughing) (Sharma 2015). Due to lower labor costs and weak government regulation, approximately 80% of e-waste from developed countries is illegally exported to developing countries, especially China and India (Awasthi et al. 2016). Previous literature has indicated that heavy metal pollution from e-waste in India has spread to the surrounding environment through informal activities (Awasthi et al. 2016). Even if Pb emissions are controlled, the historical accumulation of Pb pollution in aerosols, soil, and dust can lead to elevated blood Pb levels (Taylor et al. 2019). In the subgroup study of study regions, Pb exposure levels in North America and Asia were associated with respiratory diseases. It is recommended to use special masks to filter $PM_{2.5}$ in areas with Pb pollution to minimize direct oral inhalation.

The health effects of Pb poisoning are irreversible, so it is important to control sources of Pb to minimize Pb exposure. In 2008, the United States Environmental Protection Agency developed a stricter standard that the average Pb concentration in the air should not exceed 0.15 $\mu g/m^3$ over

a rolling period of three months. However, even blood Pb levels < 5 μg/dL (the reference level for initiating public health action in the United States) can have deleterious health effects in children (Caldwell et al. 2017). The WHO report states that the safety level of Pb exposure is not clear (WHO 2018).

Pb poisoning remains a major environmental pollution issue and there are many measures to reduce Pb exposure. In addition to policies restricting the use of Pb in industry, measures at the individual and household-level mainly include preventing exposure to Pb paint and reasonable disposal of Pb-acid batteries (Jahir et al. 2021). In addition, changing nutritional status and increasing dietary iron and calcium levels and intake of vitamin B1, B2, B6, and B9 can offset the adverse effects of Pb (Reuben et al. 2017). In the case of severe Pb poisoning, medical and follow-up treatment are needed and chelating therapy is commonly used (Tirima et al. 2018). Previous studies have shown that public education on knowledge of Pb poisoning is an effective preventive measure (Nussbaumer-Streit et al. 2016). Active prevention combined with case management measures can effectively eliminate the increase of blood Pb level. Many efforts have been made to prevent and control Pb hazards, but there is still a lack of effective strategies to control the consumption of Pb-contaminated products (Pfadenhauer et al. 2016).

Strengths and limitations

As we know, this is the first meta-analysis of the relationship between Pb exposure and respiratory diseases. All respiratory outcomes, including respiratory diseases, respiratory symptoms, and respiratory function assessments, were included in the analysis. The evidence of this study is comprehensive. The review followed the guidelines for systematic reviews and meta-analysis and developed stricter inclusion and exclusion criteria, so the evidence and results of this study are reliable. The included studies covered most age groups of the population and covered 12 countries. Some of these studies had large sample sizes and were conducted over a long research time, which increases the credibility of the research evidence.

There are some limitations to this study. First, the included studies were published in English, which may have led to reporting bias. Second, the number of studies on the outcome endpoints such as chronic bronchitis and obstructive lung disease is small, which did not allow us to perform meta-regression to test the effects of several regulatory factors on the set effect and the heterogeneity explained by these characteristics (Ziou et al. 2022). Third, medical record review may only register severe respiratory diseases. In the study of minors, some questionnaires and interviews were derived from adult agents or self-reports, and memories and misunderstandings of the questions may lead to deviations in the results. Most of the included studies were cross-sectional studies and did not allow causal inferences.

Conclusion

Our systematic review and meta-analysis found some evidence of the relationship between exposure to Pb-contaminated environments and respiratory diseases. The results showed that Pb exposure increased the risk of respiratory diseases (OR = 1.12, 95% CIs: 1.05, 1.18). Subgroup analysis indicated that Pb exposure was significantly associated with respiratory diseases in the Asian and North American studies, but not in the European studies. Pb exposure increased the risk of respiratory diseases in the age groups under 6 years and over 18 years. Our meta-analysis provides evidence for a positive association between Pb exposure and the development of respiratory diseases. However, the existing literature is limited, with most of them being cross-sectional studies lacking high-quality exposure measures. Further studies are needed to strengthen the evidence system and to determine the exact mechanism by which Pb exposure affects the functioning of respiratory disease.

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Declarations

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