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Air pollutants–particulate matter $(PM)_{2.5}$ with antenatal exposure leading to adverse obstetrical outcomes of low birth weight and preterm birth: A systematic review and meta-analysis

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Introduction: Particulate matter $(PM)_{2.5}$ exposure affects prenatal health and birth outcomes, including low birth weight (LBW) and preterm delivery (PTD).

Objective: To identify and explore $PM_{2.5}$ exposure on adverse obstetrical effects, including preterm birth and LBW.

Methods: Four hundred and nine studies from 1982 to 2020 were identified in a search of PubMed, Embase, Scopus, Web of Science, and Science Direct. Of the 409 articles, 24 were identified as “qualitatively considered” and 7 were identified as “quantitively eligible” to be included in this meta-analysis. The pooled effect of $PM_{2.5}$ exposure on LBW and PTD was calculated using a random effect model with significant heterogeneity. Seven studies were conducted in the meta-analysis, and the pooled effect of $PM_{2.5}$ exposure on LBW and entire pregnancy was 1.033 (95% CI, 1.025–1.041) with significant high heterogeneity ($I^2 = 96.110$, $P = 0.000$). The pooled effect of $PM_{2.5}$ exposure on PTD and entire pregnancy was 1.024 (95% CI, 1.015–1.033) with significantly different low heterogeneity ($I^2 = 60.036$, $P = 0.082$).

Discussion: Exposure to $PM_{2.5}$ during pregnancy is significantly associated with the risk of LBW, and the risk of PTD is significantly different but consistently associated with $PM_{2.5}$.

Conclusion: Globally, $PM_{2.5}$ exposure is significantly associated with serious pregnancy and birth outcomes worldwide. The emerging risks to prenatal health suggest a need for the government to influence health policies to protect maternal and pediatric health.

Key words: Maternal – Prenatal – Air pollution – $PM_{2.5}$ – Preterm birth – Low birth weight – Intrauterine growth restriction

Introduction

Air pollutant–particulate matter ($PM_{2.5}$) exposure affects prenatal health and birth outcomes. Preterm delivery (PTD) is a when a baby is born at <37 gestational weeks. Every year, 15 million deliveries occur before 37 weeks. Globally, 5% to 18% of deliveries in 184 countries are preterm. Premature births have been the leading cause of mortality among children <5 years of age, contributing to approximately 1 million deaths in 2015.[1] The United Nations' target for 2030 with the Sustainable Development Goal is no more than 12 neonatal deaths per 1,000 live births and no more than 25 deaths per 1,000 live births in children under the age of 5, in all countries worldwide.[2] Birth weight is the most critical indicator of infant growth during birth outcomes. One of the most significant public health problems has been low birth weight. Low birth weight (LBW) is defined as a baby born weighing less than 2,500 g (5.5 lb).[3] Annually, an estimated 20 million births have been delivered as low weight births, ranging from 15% to 20% of all births worldwide. The global target in 2025 is to reduce the rates of low birth weight by 30%, leaving 14 million low-birth weight infants in all deliveries, according to the World Health Organization (WHO).[3]

Several studies have shown increasing associations between $PM_{2.5}$ exposure and PTD with LBW. However, the results of the studies have positive and negative correlations. Particular matter ($PM_{2.5}$) has been recognized as one of the risk factors with a prior history of preterm birth.[4] PTD and LBW are public health issues associated with air pollutants.[5,6] The risk of LBW is associated with $PM_{2.5}$ in the first semester, and $PM_{2.5}$ exposure is not only associated with LBW, but O_3 exposure is positively associated with birth weight in the first and second trimesters.[7] Both LBW and PTD are associated with $PM_{2.5}$, and the most substantial effect of O_3 exposure is increasing the risk of PTD and very preterm birth (VPTD) throughout pregnancy.[8] Between PTD and ambient air pollution (AAP) effects, CO, O_3, PM_{10}, and $PM_{2.5}$ are significantly associated with the second trimester in Wuhan, China.[9] Canadian researchers found that $PM_{2.5}$ exposure and LBW are associated with pregnancy in women who completed postsecondary education.[10] In this study, the meta-analysis explored the association between $PM_{2.5}$ exposure and birth outcomes, including PTD and LBW during different trimesters. By reducing air pollutants global health must reduce the risk of adverse obstetrical outcomes of LBW and PTD.

Patients and Methods

Our study followed the Preferred Reporting Items for Systematic Reviews and Meta-Analysis (PRISMA) guideline,[11] and data were collected into the endnote standard software tool from the library of Asia University. A total of 409 studies from 1982 to 2020 were identified in a search of PubMed, Embase, Scopus, Web of Science, and Science Direct using the following key words: infant, maternal, fetus, neonatal, prenatal, air pollution, $PM_{2.5}$, preterm birth, and low birth weight. Only English publication articles were searched in this study. Of the 409 articles, 24 were identified and qualitatively considered, and 7 articles were quantitively eligible to be included in this meta-analysis.

This study investigated observational studies, including retrospective cohort studies, cohort studies, cross-sectional studies, and case-control studies, and only published English articles were included. Although study titles and abstracts were screened, different interventions or outcomes were initially excluded from this study. The study included the following criteria: $PM_{2.5}$ exposure during the entire pregnancy, obstetric outcomes including PTD, and LBW. Findings of obstetric outcomes were performed as PTD and LBW. Preterm births[1] and common births[3] were defined and measured using WHO guidelines.

This study is appropriate to use for each quantitative and qualitative analysis; the quality assessment was evaluated by the New-Castle-Ottawa (NOS) (see Supplementary Materials Tables 1–3). NOS is divided into cohort, cross-sectional, and case-control studies and rated using a "star system." Scores ranged from zero to 10, with scores indicating quality as very good, 9–10; good, 7–8; satisfactory, 5–6; and unsatisfactory, 0–4.

Each study included authors, publication year, country of analysis, study design, study period, total number of births, number of preterm births, and low birth weights with the intervention of $PM_{2.5}$ exposure. Three professional experts reviewed data extraction. Under written permission, the first and second authors (O.A. and C-J.J., respectively) independently identified and extracted data from all articles. Therefore, ethics committee approval was unnecessary, and secondary data were conducted in a meta-analysis using the comprehensive meta-analysis software. The pooled effect of $PM_{2.5}$ exposure and odds ratio (OR) for LBW, PTD, and 95% confidence intervals (CI) were calculated by comprehensive meta-analysis and using a random effect model with significantly high and significantly different heterogeneity, respectively.

Statistical Analysis

The pooled effect of $PM_{2.5}$ exposure and LBW, PTD was calculated using a random effect model with significant heterogeneity. The heterogeneity is defined as I^2, and the importance of the value of I^2 follows as 0% to 40% might not be important, 30% to 60% may represent moderate heterogeneity; 50% to 90% may represent substantial heterogeneity, and 75% to 100% considerable heterogeneity. The study pooled all estimates between odd ratio (OR) as well as 95% (CI) and only one common exposure unit of effect as 2.5 in the fine particulate matter (PM) from different studies and conducted into meta-analyses and quantitively estimated the association between PTD, LBW, and $PM_{2.5}$ exposure:

1. The meta-analysis pooled all estimations of LBW and PTD with the entire pregnancy.
2. In addition, the meta-analysis pooled all assessments of LBW and PTD with subgroups by first and second trimesters.
3. The Egger test was performed in the funnel plot symmetry.

Results

There were 409 selected studies, and 358 were screened after excluding 51 duplicated studies. Three hundred and one studies were excluded from screening because 35 were systematic reviews, meta-analyses, or case reports, 31 were animal experiments and included indoor pollutant exposure, and 178 were other birth outcomes. Fifty-seven articles were assessed for full-text articles. Thirty-three articles were further excluded: 9 did not provide specific types of pollutants, and 24 defined birth outcomes differently. After full-text articles were excluded, 24 articles were identified and qualitatively considered, and 7 articles were quantitively eligible to be included in this meta-analysis (see Figure 1).

Quantitively, 24 studies were included, of which 11 assessed PTD and LBW.[8,10,12–19] Five studies assessed LBW,[5,20–23] and 8 considered PTD.[9,24–29] Detailed indicators are shown in Table 1.

In this study, quantitatively, 24 studies from 1996 to 2018 were included, with a large sample size of 10,502,332 births from the United States, Canada, China, India, Spain, Australia, Iran, the United Kingdom, and the Netherlands. Fifteen of 24 studies are retrospective cohort studies, 5 are cross-sectional studies, and 4 are case-control studies. Because of the findings, 11 studies evaluated both PTD and LBW, 6 considered LBW, and 7 considered PTD. Of 10,502,332 births, studies presented as few as 1285 births in India[5] and as many as 2,966,705 in Canada.[10] Covariates were parity, maternal age, gestational age, maternal education, maternal race/ethnicity, nationality, registered resident, marital status, employment, occupation, income, birth place, birth year, previous pregnancy, previous delivery, infant and maternal morbidity, and prena-

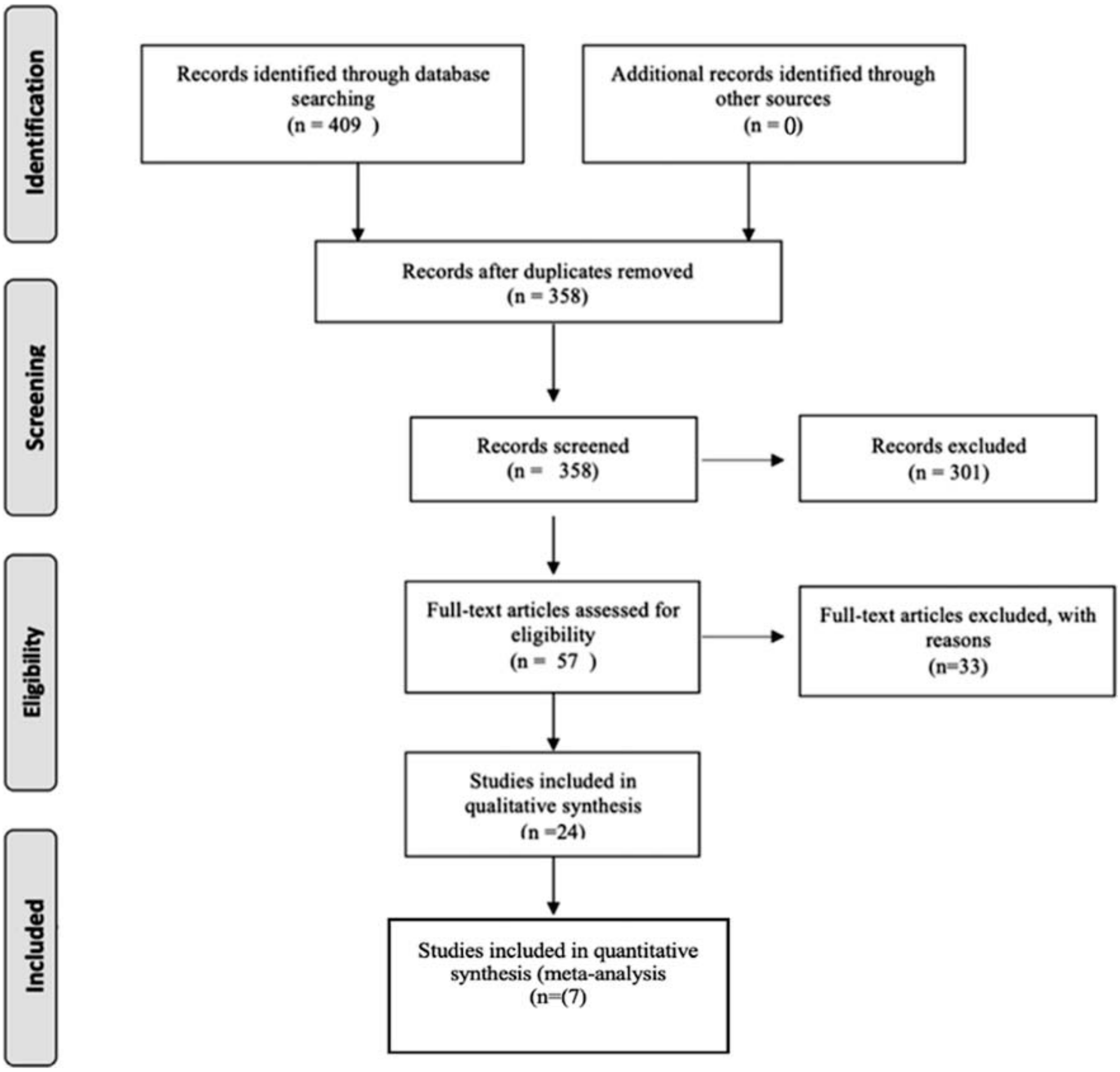


Fig. 1 PRISMA flow chart.

tal visits. One pollutant, $PM_{2.5}$ exposure, varied in different periods of pregnancy, including trimesters.

A total of 7 studies were eligible for inclusion in the meta-analysis, with a significant association between $PM_{2.5}$ exposure and birth outcomes.[6,8,11,12,20,28,30] A summary of pooled effect estimates is shown in Table 2.

The association between exposure in LBW with entire pregnancy was OR = 1.033 (95% CI, 1.025–1.041) with significantly high heterogeneity (I^2 = 96.110%, $P = 0.000$) (see Figure 2A). The significantly different association between exposure in LBW in the first trimester was OR = 1.046 (95% CI, 1.035–1.059) with significantly high heterogeneity (I^2 = 0.000%, $P = 0.744$) (see Figure 2B). The association between exposure in LBW in the second trimester was OR = 1.119 (95% CI, 1.106–1.33) with significantly high heterogeneity (I^2 = 94.147%, $P = 0.000$) (see Figure 2C).

The association between exposure in PTD and entire pregnancy was OR = 1.024 (95% CI, 1.015–1.033) with significantly low heterogeneity (I^2 = 60.036%, $P = 0.082$) (see Figure 3A). Exposure of PTD during the first trimester was OR = 1.036 (95% CI, 1.010–1.062) with significantly different heterogeneity (I^2 = 0.000%, $P = 0.424$) (see Figure 3B). Exposure of PTD during the second trimester was OR = 1.056 (95% CI, 1.030–1.080) with significantly different heterogeneity (I^2 = 0.000%, $P = 0.520$) (see Figure 3C).

Using the Egger test, we did not reach a statistically significant publication bias; the Egger

Table 1 Studies included in the systematic review[a]

Study	Location	Study period	Study design	Population size (No. of births)	Air pollutant exposures	$PM_{2.5}$ exposure period	Birth outcomes
Stieb *et al.* (2016)[10]	Canada	1999–2008	Retrospective cohort study	2,966,705	$PM_{2.5}$ PM_{10}	Entire pregnancy, Trimesters	PTD LBW
Ha *et al.* (2014)[8]	Florida, USA	2004–2005	Retrospective cohort study	423,719	$PM_{2.5}$, O_3	Pregnancy, Trimesters	PTD LBW
Qian *et al.* (2016)[12]	Wuhan, China	2011.06.10–2013.06.09	Nested case-control study	95,911	$PM_{2.5}$, PM_{10}, SO_2, O_3, NO_2, CO	Entire pregnancy, Trimesters	PTD LBW
Liu *et al.* (2019)[6]	Guangdong, China	2014.01.01–2015.12.31	Case-control study	1784	$PM_{2.5}$, PM_{10}, SO_2, O_3, NO_2, CO	Entire pregnancy, Trimesters	PTD LBW
Lavigne *et al.* (2018)[20]	Ontario, Canada	2012–2016	Retrospective cohort study	196,171	$PM_{2.5}$	Entire pregnancy, trimesters	LBW
Wu *et al.* (2017)[21]	Jinan, China	2014–2016	Case-control study	43,855	$PM_{2.5}$, SO_2, NO_2	Entire pregnancy, Trimesters	LBW
Enders *et al.* (2019)[30]	California, USA	2002–2013	Case-control study	2,719,596	$PM_{2.5}$ PM_{10}–$PM_{2.5}$	Pregnancy, Trimesters	LBW
Qian *et al.* (2016)[9]	Wuhan, China	2011–2013	Cohort study	95,911	$PM_{2.5}$, PM_{10}, SO_2, O_3, NO_2, CO	Entire pregnancy, Trimesters	PTD
Balakrishnan *et al.* (2018)[5]	Tamil Nadu, India	2010–2015	Cohort study	1285	$PM_{2.5}$	Pregnancy	LBW
Liang *et al.* (2019)[13]	Chinese cities	2014–2017	Cohort study	1,455,026	$PM_{2.5}$, SO_2, O_3, NO_2	Trimesters	PTD LBW
Arroyo *et al.* (2016)[24]	Madrid, Spain	2001–2009	Cross-sectional study	298,705	$PM_{2.5}$, O_3	Pregnancy	PTD
Arroyo *et al.* (2016)[14]	Madrid, Spain	2001–2009	Cross-sectional study	298,705	$PM_{2.5}$, O_3	Pregnancy	PTD LBW LFD
Liu *et al.* (2017)[15]	Shanghai, China	2013	Cohort study	195,400	$PM_{2.5}$	Pregnancy	PTD LBW
Salihu *et al.* (2012)[16]	Florida, USA	2000–2007	Retrospective cohort study	103,961	$PM_{2.5}$ PM_{10}	Pregnancy	PTD LBW
Chen *et al.* (2018)[17]	Brisbane, Australia	2003–2013	Cross-sectional study	173,720	$PM_{2.5}$, SO_2, O_3, NO_2	Entire pregnancy, Trimesters	PTD LBW
Tu *et al.* (2018)[25]	Georgia, USA	2000	Cross-sectional study	116,112	$PM_{2.5}$, O_3	Pregnancy	PTD
Yuan *et al.* (2020)[18]	Shanghai, China	2013–2016	Cohort study	3692	$PM_{2.5}$	Entire pregnancy, Trimesters	PTD LBW
Sarizadeh *et al.* (2020)[19]	Ahvaz, Iran	2008–2018	Cross-sectional study	150,766	$PM_{2.5}$, PM_{10}, SO_2, O_3, NO_2, CO, NO	Pregnancy	PTD LBW
Liang *et al.* (2019)[26]	Chinese cities	2015–2017	Cohort study	628,439	$PM_{2.5}$, SO_2, O_3, NO_2	Entire pregnancy, Trimesters	PTD
Brauer *et al.* (2008)[22]	Vancouver, British Columbia, Canada	1999–2002	Cohort study	70,249	$PM_{2.5}$, PM_{10}, SO_2, O_3, NO_2, CO, NO	Entire Pregnancy	PTD LBW
Guo *et al.* (2018)[27]	China	2014.01–2014.12	Retrospective cohort study	426,246	$PM_{2.5}$	Entire pregnancy, Trimesters	PTD
Ye *et al.* (2018)[23]	Taizhou, China	2013.01.01–2016.05.31	Retrospective cohort study	26,246	$PM_{2.5}$, PM_{10}, NO_2	Entire pregnancy, Trimesters	LBW
Gehring *et al.* (2011)[28]	North, West, and Centre Netherlands	1996–1997	Retrospective cohort study	3853	$PM_{2.5}$, NO_2	Entire pregnancy, trimesters	PTD
Sun *et al.* (2019)[29]	Zhejiang, China	2013–2017	Prospective cohort study	6275	$PM_{2.5}$, PM_{10}, SO_2, O_3, NO_2, CO	Pregnancy, Trimesters	PTD

LFD, late fetal death.

[a]For our systematic review, we refer to this study as observational.

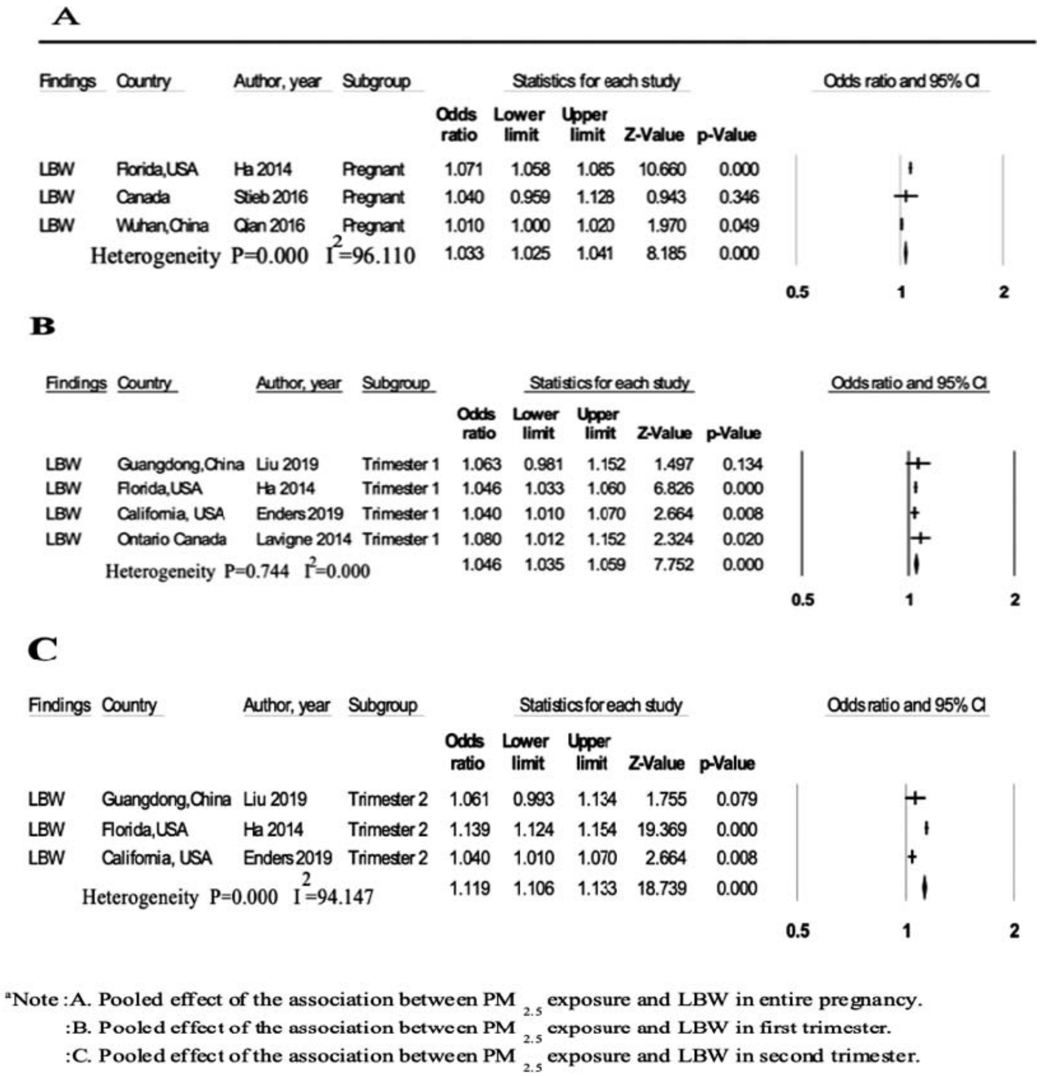


Fig. 2 Forest plot of the association between $PM_{2.5}$ exposure and LBW.

Table 2 Pooled associations between $PM_{2.5}$ exposure between LBW and PTD[a]

Subgroups	No. of studies	*P* value, heterogeneity test	The summary OR (95% CI)	*P* value, hypothesis test	I^2 (%)	*P* value, Egger test[b]
LBW						
Pregnancy	3	0.000	1.033* (1.025–1.041)	0.000	96.110	0.862
First trimester	4	0.744	1.046 (1.035–1.059)	0.000	0.000	0.354
Second trimester	3	0.000	1.119* (1.106–1.133)	0.000	94.147	0.450
PTD						
Pregnancy	3	0.082	1.024 (1.015–1.033)	0.000	60.036	1.000
First trimester	2	0.424	1.036 (1.010–1.062)	0.007	0.000	–
Second trimester	3	0.520	1.055 (1.030–1.080)	0.000	0.000	0.447

[a]All of these subgroup analyses were conducted for the studies that assessed the effects of $PM_{2.5}$ exposure during pregnancy and the first trimester on LBW and PTD risks.

[b]There is no evidence of publication bias using the Egger test.

*$P < 0.05$.

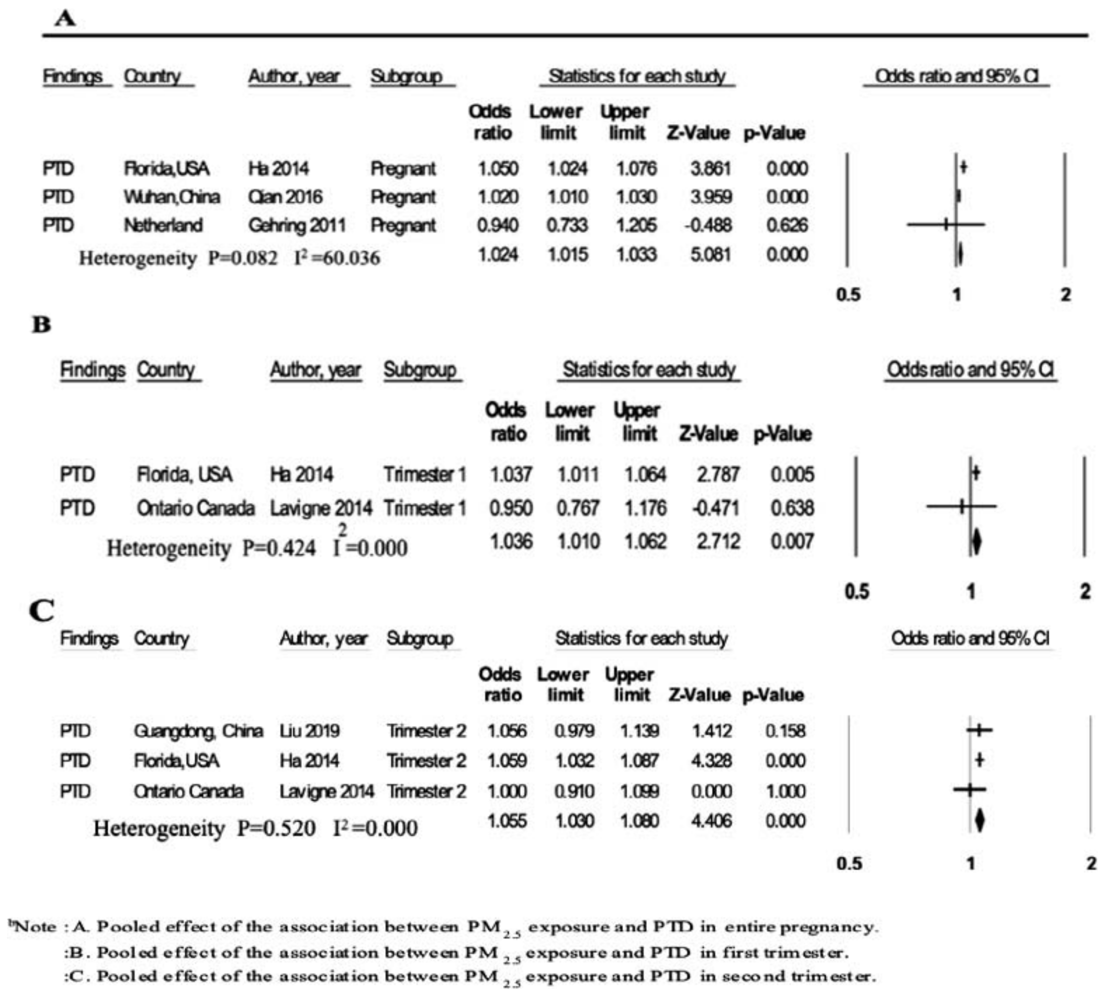


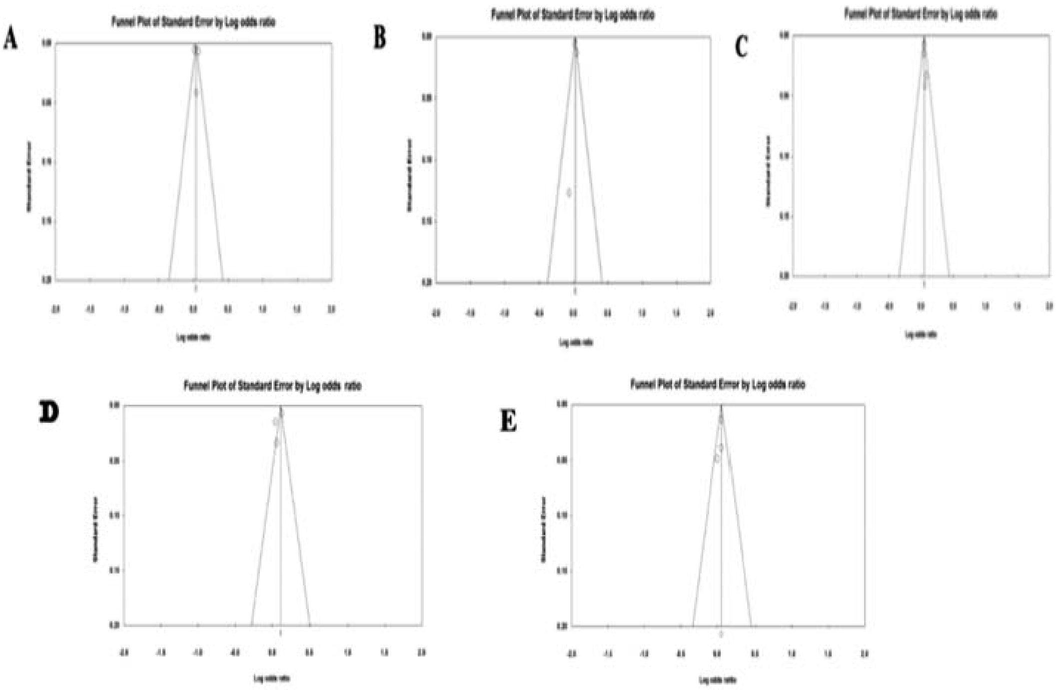
Fig. 3 Forest plot of the association between $PM_{2.5}$ exposure and PTD.

test was performed in the funnel plot symmetry (see Table 2 and Figure 4 A–E).

Discussion

A systematic review was performed to summarize the current scientific findings and quantitatively assess the association between adverse birth outcomes and maternal $PM_{2.5}$ exposure. Meta-estimation was only considered a single pollutant of $PM_{2.5}$, and maternal $PM_{2.5}$ exposure effectively estimated the maternal exposure at the risk of LBW and PTD. Regarding the results from some studies, the high correlation of the multipollutant models makes it more complicated to interpret and disentangle the effect of each pollutant.[31,32] Nevertheless, the findings are even more important in updating the prenatal guidance for adverse birth outcomes.

Maternal exposure to $PM_{2.5}$ is highly related to a risk of LBW during pregnancy and the second trimester. In a New Jersey study,[33] an association between maternal exposure to $PM_{2.5}$ concentration and birth weight increases the risk of LBW in early and late pregnancy. This result was consistent with a chance of fetal growth restriction where $PM_{2.5}$ small for gestational age estimates as first trimester 5.5% and third trimester 3.3% reduction of fetal weight.[33] In addition, a previous study estimated $PM_{2.5}$ concentration in the sixth gestational month was associated with a 10.3 g reduction in birth weight in those pregnancies without any complications.[34] In a Massachusetts and Connecticut study, $PM_{2.5}$ concentration exposure at 37 to 42 gestational weeks was associated with a –14.7 g reduction (–17.1 to –12.3) in birth weight.[35] The effects of $PM_{2.5}$ are not only associated with birth weight but also with head circumference as a change in head circumference



Note A: Funnel Plot of the association between exposure and LBW in entire pregnancy
B: Funnel Plot of the association between exposure and PTD in entire pregnancy
C: Funnel Plot of the association between exposure and LBW in first trimester
D: Funnel Plot of the association between exposure and LBW in second trimester
E: Funnel Plot of the association between exposure and PTD in second trimester

Fig. 4 Funnel plot of the association between exposure in LBW and PTD.

–0.08 (–0.12 to –0.03) cm correlates with a change in birth weight –7 (–17 to 2) g at >37 weeks of gestation.[36] During the Beijing Olympics in 2008, the greater birth weight associated with a lower level of $PM_{2.5}$ concentration in the third trimester of pregnancy was a significant 7% increase in the risk of a low-birth weight baby among term birth in 2018.[37] In the same study, declines in air pollutants during the 2018 Olympic games were associated with a 23-g birth weight increase in pregnancies at 8 gestational months compared with the same calendar date in 2007 or 2009.[38] Maternal exposure to $PM_{2.5}$ is one of the important risk factors for intrauterine inflammation (IUI), a sensitive biomarker for the developing fetus.[39] IUI is a risk factor for adverse birth outcomes, including LBW and PTD, and significantly, the combined presence of maternal inflammatory response and fetal inflammatory response was more strongly associated with extreme preterm birth (<29 weeks).[40]

Another finding from the subgroup regarding the birth outcome concerns a small risk of PTD in our study. In many studies, maternal exposure to $PM_{2.5}$ has been associated with PTD.[22,41–46] However, some studies have found that the effect is positively a small, statistically nonsignificant association of risk of PTD between maternal exposure to $PM_{2.5}$ during pregnancy and the third trimester.[28] Similarly, however, predicted maternal exposure to $PM_{2.5}$ is little evidence of association with preterm delivery risk; an increased risk of preeclampsia is associated with maternal exposure to $PM_{2.5}$ in this setting.[47] Preeclampsia also can lead to preterm delivery and numerous adverse outcomes, including immunologic problems and longer-term motor, cognitive, and fetal growth problems.[48] However, the further finding was not associated with the risk of both LBW and PTD in the first trimester. Although prenatal exposure to $PM_{2.5}$ during pregnancy is significantly associated with the risk of LBW, the risk of PTD is significantly different; however, PTD is consistently associated with $PM_{2.5}$.

There were a few limitations of this study:

1. Some studies made several analytical decisions that caused primary cohort and case-control analyses to differ in important ways other than including the additional covariates.
2. One direct comparison's results significantly differed between the cohort and case-control

study. Although some studies could not conduct the meta-analysis because of weak evidence of adverse effects, these studies were included in a systematic review. Therefore, the third trimester was not appropriate for meta-analysis.
3. Some analyses do not raise numerous questions about the comparability of the data and analysis approaches.

We did not find publication bias based on the Egger test.

Conclusion

This meta-analysis explored that $PM_{2.5}$ exposure is positively associated with birth weight. Another finding from the subgroup regarding the preterm birth outcome concerns a small risk of PTD in this study. PTD is significantly different with exposure to $PM_{2.5}$ and is consistently associated with $PM_{2.5}$. Globally, $PM_{2.5}$ exposure is significantly associated with severe consequences to prenatal care and birth outcomes in pregnancy. Health appears in emerging risks that government needs to influence health policies to pursue maternal and child health.

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