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Association between ambient fine particulate matter and preterm birth or term low birth weight: An updated systematic review and meta-analysis☆

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ABSTRACT

An increasing number of studies have been conducted to determine a possible linkage between maternal exposure to ambient fine particulate matter and effects on the developing human fetus that can lead to adverse birth outcomes, but, the present results are not consistent. A total of 23 studies published before July 2016 were collected and analyzed and the mean value of reported exposure to fine particulate matter ($PM_{2.5}$) ranged from 1.82 to 22.11 We found a significantly increased risk of preterm birth with interquartile range increase in $PM_{2.5}$ exposure throughout pregnancy (odds ratio (OR) = 1.03; 95% conditional independence (CI): 1.01−1.05). The pooled OR for the association between $PM_{2.5}$ exposure, per interquartile range increment, and term low birth weight throughout pregnancy was 1.03 (95% CI: 1.02−1.03). The pooled ORs for the association between $PM_{2.5}$ exposure per 10 increment, and term low birth weight and preterm birth were 1.05 (95% CI: 0.98−1.12) and 1.02 (95% CI: 0.93−1.12), respectively throughout pregnancy. There is a significant heterogeneity in most meta-analyses, except for pooled OR per interquartile range increase for term low birth weight throughout pregnancy. We here show that maternal exposure to fine particulate air pollution increases the risk of preterm birth and term low birth weight. However, the effect of exposure time needs to be further explored. In the future, prospective cohort studies and personal exposure measurements needs to be more widely utilized to better characterize the relationship between ambient fine particulate exposure and adverse birth outcomes.

1. Introduction

The fundamental causes of adverse birth outcomes are not well understood, despite growing evidence that environmental factors may play an important role. Air pollution is one of the most concerned environmental factors; evidence of adverse health effects of ambient air pollution has rose dramatically (West et al., 2016). The World Health Organization (WHO) estimated that in 2013, 87% of the global population lived in communities that exceeded the WHO's air quality guideline of a maximum mean ambient fine particulate matter ($PM_{2.5}$) of 10 (Brauer et al., 2016). The Global Burden of Disease (GBD) rated ambient fine particulate matter exposure as the seventh most important risk factor contributing to global mortality (Forouzanfar et al., 2015). Recently, an increasing number of studies have shown that maternal exposure to air pollution can affect the developing fetus, resulting in adverse birth outcomes such as infant death, still birth, term low birth weight

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(TLBW), preterm birth (PTB), and small for gestational age (Lee et al., 2013; Vinikoor-Imler et al., 2014; Dibben and Clemens, 2015). Furthermore, air pollution is also related to adverse respiratory and cardiovascular outcomes in clinical, epidemiological and toxicological studies (Ritz and Wilhelm, 2008; Yang et al., 2013; Rappazzo et al., 2014).

The effects of adverse birth outcomes can persist for a person's entire life, according to Developmental Origins of Health and Disease (Barker et al., 1990, 1993; Hanson and Gluckman, 2011). PTB is the main cause of death in newborn babies, and is associated with a high risk of childhood disability (Howson et al., 2013). Birth weight is an important indicator of fetal growth, development, and nutritional status; low birth weight is also related to low economic and social development of the given country or region. Considered a chronic disease, low birth weight is one of the major risk factors associated with global disease burden (Symanski et al., 2014).

Fine particulate matter refers to a heterogeneous mixture of substances that sometimes includes accumulated heavy metals and toxic organic pollutants such as polycyclic aromatic hydrocarbons (Dejmek et al., 2000); fine particulate matter may affect birth outcomes directly or indirectly. Laurent et al. related TLBW with primary particle concentrations by source and composition, and showed that increased risk of TLBW is associated with several major sources of $PM_{2.5}$ (especially gasoline, wood burning, and commercial meat cooking), and chemical composition in $PM_{2.5}$ (elemental and organic carbon, potassium, iron, chromium, nickel, and titanium, but not lead or arsenic) (Laurent et al., 2014). Basu et al., in California, USA, showed similar results; heavy metal elements, sulfur, sulfate, bromine, and ammonium are associated with reductions in birth weight and an increased risk of TLBW (Basu et al., 2014). Over the past decade, a number of recent studies estimated the association between $PM_{2.5}$ exposure during pregnancy in whole or part and TLBW or PTB. The results are inconsistent and controversial. Some studies found that air pollutants significantly impact birth outcomes, while others failed to find such associations (Laurent et al., 2013; Vinikoor-Imler et al., 2013; Brown et al., 2015; Huang et al., 2015). Huynh et al. executed a matched case–control study of the relationship between PTB and level of $PM_{2.5}$ exposure throughout pregnancy in California from 1999 to 2000. After controlling for demographic factors, the odds ratio (OR) of PTB per 10 of $PM_{2.5}$ was 1.15 (95%; CI: 1.07–1.24). Wu et al. conducted a retrospective cohort study in California from 1997 to 2006, examining the effect of air pollution levels throughout pregnancy on PTB risk in a cohort of 81186 singleton births, and found an association between PTB and $PM_{2.5}$, per 10 μg/m3, was 1.24 (95%; CI: 1.08–1.42). Pereira et al. saw a positive but non-significant association between $PM_{2.5}$ exposure and PTB (OR = 1.01, 95 CI %: 0.93–1.09) in Connecticut, USA. Wilhelm et al., in California, USA, did not find a consistent concentration-dependent relationship between ambient fine particulate matter and PTB (OR = 0.73, 95; CI % = 0.67–0.08). All of the aforementioned studies were conducted in the USA, yet the results of these studies were not consistent. Due to the varied compositions of ambient fine particulate matter, and the limitations of these studies' researching approaches, there is not yet sufficient evidence to establish causality.

To further understanding of the association between air pollution and birth outcomes, we have collected all data presently available and conducted a quantitative meta-analysis on the relationship between $PM_{2.5}$ exposure and term low birth weight or preterm birth. Of note, the pooled ORs include both 10 increases and interquartile range (IQR) incremental increases in $PM_{2.5}$ exposure. Data from more recent studies allowed us to assess the effect by gestational period, to conduct meta-regression and sensitivity analyses, to valuate publication bias, and to measure heterogeneity.

2. Materials and methods

2.1. Inclusion criteria and search strategy

From December 2015 to July 2016, we conducted a search on PUBMED and Cochrane, as well as on China National Knowledge Infrastructure (CNKI) and Wanfang Data Knowledge Service Platform to collect local data from Chinese studies. We limited our search to papers published in English or Chinese. We searched the following terms on PUBMED, based on the terminology used in recent reviews of the subject: "air pollution", "air pollutants", "particular matter", "fine particular matter", "pregnancy outcome", "birth outcomes", "infant, newborn", "birth weight", "infant, low birth weight", "low-birth-weight infant", "premature birth", "infant, premature" and "obstetric labor, premature". Meanwhile, to obtain additional publications, we manually searched the references of each primary study. Publications were also identified in the same manner from review articles.

2.2. Selection criteria

To choose the related articles, we first filtered the titles and abstracts of all studies. Studies were excluded if they were not related fine particulate matter to with low birth weight or preterm birth. Then, from the identified papers, we selected studies meeting the following eligibility criteria: a) inclusion of $PM_{2.5}$ exposure during pregnancy and single live births, b) clear definition of maternal exposure to $PM_{2.5}$ and of birth outcomes, c) pregnancy outcomes of TLBW or PTB, d) low birth weight defined by the World Health Organization as a birth weight of a liveborn infant of 2,499 g or less, including TLBW and preterm low birth weight (however we only included TLBW in this study) e) preterm birth determined as a less than 37 weeks gestational age at delivery, and f) presentation of sample sizes and ORs with 95% confidence intervals for each 10 or IQR increment increase of $PM_{2.5}$ exposure. If more than one study was determined for a given population, only the study that included either the most recent population data or the most up-to-date information (or both) was selected. Studies that met all of the above inclusion criteria were short listed for inclusion in the review.

2.3. Data extraction

Two investigators processed the data from each eligible study independently, using consistent strategy. If a study provided associations between $PM_{2.5}$ exposure and PTB or TLBW, both throughout pregnancy and in trimester-specific periods, all association data was extracted. A number of studies evaluated $PM_{2.5}$ exposure based on multiple sources of air pollution data (Stieb et al., 2016a,b; Kloog et al., 2012, Hannam et al., 2014; Fleischer et al., 2014; Coker et al., 2015; Lavigne et al., 2016; Hyder et al., 2014). We chose estimations based on remote sensing data because this increased sample size and decreased selection bias. Data was extracted systematically from each study using a pre-designed standard data collection form. We collected the following information from all of the studies: author(s), date of publication, study period, location, study design, air pollution sources, exposure measurement, sample size, exposure range, outcome assessment, exposure period, Newcastle-Ottawa Scale (NOS) grade, Agency for Healthcare Research and Quality (AHRQ) grade, and the ORs and 95% CIs used during statistical analyses. The selection process of this study is showed in detail in Fig. 1.

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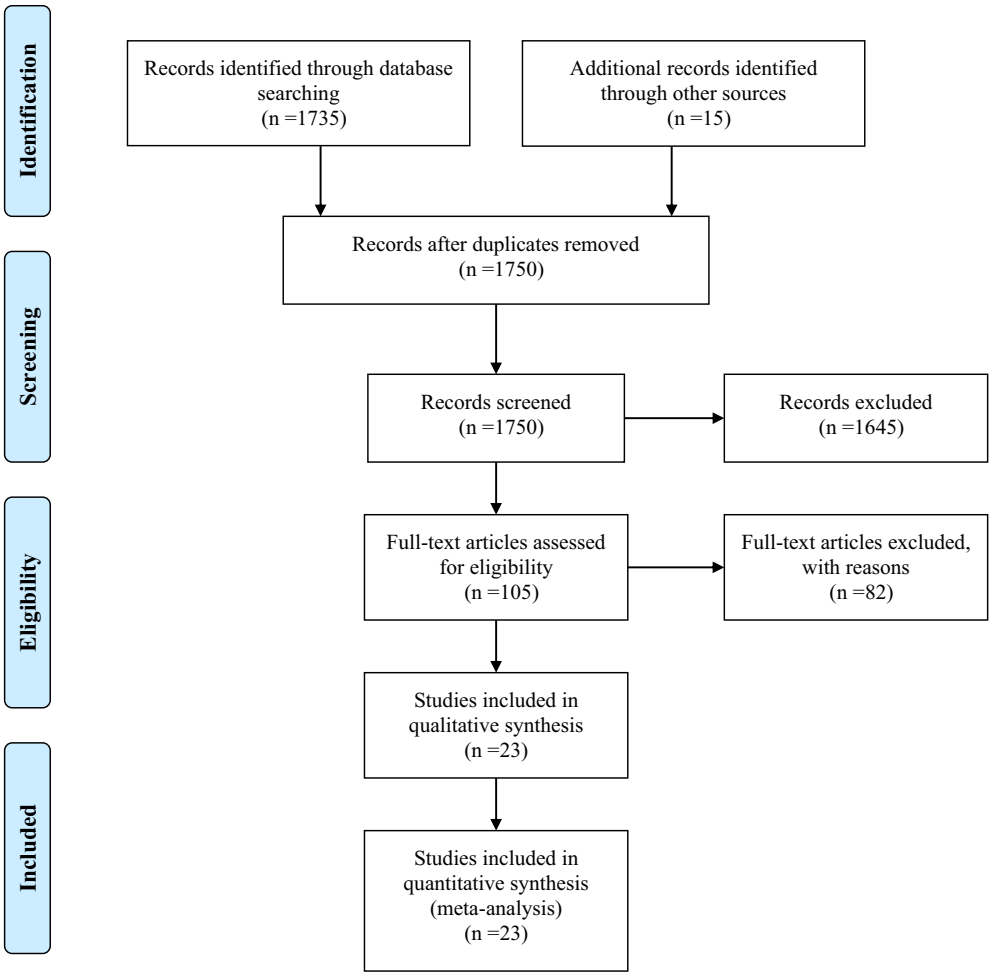

Fig. 1. Flow chat of select studies in meta-analysis.

2.4. Meta-analysis and statistical analysis

Prior to conducting the meta-analysis, we converted all risk estimates into a common exposure unit of 10 μg/m3 or IQR increases in $PM_{2.5}$, which allowed us to quantitatively pool estimates from different studies. We grouped effect estimates by gestational period (first trimester, second trimester, third trimester or entire pregnancy). To calculate the partial regression coefficients (β) and their standard errors (SE), we transformed all ORs and their 95% CIs by logarithms (ln). The systematic review was conducted in accordance with the Meta-analysis of Observational Studies in Epidemiology guidelines demand (Borenstein et al., 2010). Heterogeneity of the 23 studies was assessed using the Q statistic or I^2 statistic, depending on whether fixed or random effects models were used in the pooling of individual studies. Significance of the pooled ORs, relative risks (RR) and/or hazard ratios (HR) was determined by the Z-test, whereby the observed p values less than 0.05 were regarded as statistically significant, using RevMan (Version 5.2).

Publication bias, which can result from the publication of small studies with negative findings, was visually assessed using a funnel plot for asymmetry (Egger et al., 1997). Begg's test as well as Egger's test were also conveyed to further assess publication bias. The meta-analysis was considered to have significant publication bias if $p < 0.05$. These analyses were carried out with Stata (Version 12.0).

3. Results

3.1. Characteristics of the eligible studies

Table 1 summarized the main parameters from each individual study. Twenty-three studies met the inclusion criteria and were included in our review, of which 9 assessed TLBW, 8 assessed PTB, and 6 assessed both TLBW and PTB. Eighteen studies employed a retrospective cohort study, while the remainder were case-control studies (n = 2), prospective cohort studies (n = 1) and cross-sectional studies (n = 2). The majority of the studies were conducted in the USA (n = 17), followed by Canada (n = 2), Europe (n = 3), and other (n = 1). Individual studies analyzed between as few as 3853 and as many as 3,545,177 births. Seven studies used both monitoring network data and remote sensing data to assess $PM_{2.5}$ exposure, while the other sixteen studies employed only ground-based monitoring data. Almost all studies adjusted for sex of infants, mothers' demographic (including age, race/ethnicity, education, socioeconomic status (SES) and smoking status) and pregnancy-related factors (including prenatal care, parity, gestational week and season). The mean reported $PM_{2.5}$ exposure ranged from 1.82 to 22.11 amongst all 23 studies.

3.2. Quantitative data synthesis

Results of our pooled analyses are illustrated in Fig. 2. Forest

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Table 1
Characteristics of the studies included in the meta-analysis.

Author	Location	Study period	Design	Data source	Exposure measurement	Birth (n)	Exposure period	OR (95%CI)		Exposure range (mean (IQR)/μg/m^3)	Outcome assessment
								PTB	TLBW		
Stieb et al., 2016a,b	Canada	1999–2008	retrospective cohort study	MD, RSD	land use regression (LUR) model	2,966,705	WP	0.96 (0.93, 0.99)	1.01 (0.94, 1.08)	8.4 (IQR: 3.6)	PTB, TLBW, SGA
Kloog et al., 2012	Massachusetts, USA	2000–2008	retrospective cohort study	MD, RSD	land use regression (LUR) model	634,244	WP, TS	1.06 (1.01, 1.13)	–	9.6 (IQR: 5.3)	PTB, Birthweight
Hannam et al., 2014	North West England, UK	2004–2008	retrospective cohort study	MD, RSD	spatio-temporal (S-T) model	203,562	WP, TS	0.98 (0.85, 1.12)	–	22.11	PTB, Birthweight, SGA
Ritz et al., 2007	Los Angeles, USA	2003	case-control survey nested within a birth cohort	MD	Zip code geocode model	58,316	WP, TS	1.00 (0.94, 1.07)	–	–	PTB
Fleischer et al., 2014	22 countries	2004–2008	cross-sectional study	MD, RSD	GEOS-Chem model	192,900	WP, TS	0.96 (0.90, 1.02)	1.00 (0.97, 1.03)	–	PTB, TLBW
Huynh et al., 2006	California, USA	1999–2000	matched case –control study	MD	Proximity models (5 miles)	42,692	WP, TS	1.15 (1.15, 1.16)	–	18.0	PTB
Coker et al., 2015	Los Angeles (LA) County, California, USA	1995–2006	retrospective cohort study	MD, RSD	land use regression (LUR) model	1,522,084	WP	–	1.19 (1.02, 1.39)	17.04	TLBW
Morello-Frosch et al., 2010	California, USA	1996–2006	retrospective cohort study	MD	tract and ZIP Code geocode model	3,545,177	WP, TS	–	1.04 (1.02, 1.07)	16.7	Birthweight, TLBW
Lavigne et al., 2016	Ontario, Canada	2005–2012	retrospective cohort study	MD, RSD	chemical transport model, geographically weighted regression (GWR)	239,811	WP, TS	1.04 (1.02, 1.06)	–	9.1 (IQR:2.01)	Birthweight, TLBW, PTB
Dadvand et al., 2014	Barcelona, Spain	2001–2005	retrospective cohort study	MD	land use regression (LUR) model	6438	WP, TS	–	1.17 (0.98, 1.39) T1:1.07 (0.88, 1.29) T2:1.19 (0.97, 1.45) T3:1.24 (1.03, 1.49)	IQR:3.1	TLBW
Ha et al., 2014	Florida, USA	2004–2005	retrospective cohort study	MD	Hierarchical Bayesian models	423,719	WP, TS	1.053 (1.04, 1.07) T1:1.03 (1.02, 1.04) T2:1.12 (1.11, 1.14) T3:1.03 (1.01, 1.04)	T2:1.034 (1.007,1.061)	9.9 (IQR: 2.0)	TLBW, PTB, VPTB
Hyder et al., 2014	Connecticut and Massachusetts, USA	2000–2006	retrospective cohort study	MD, RSD	mixed-effects model	834,332	WP, TS	1.00 (0.96, 1.04) T1:1.00 (0.99, 1.01) T2:1.00 (0.98, 1.02) T3:1.00 (0.98, 1.01)	1.09 (1.02, 1.17) T1:1.05 (1.01, 1.08) T2:1.00 (0.97, 1.03) T3:1.02 (0.99, 1.06)	11.36 (IQR: 1.12)	TLBW, SGA, PTB
Laurent et al., 2013	Los Angeles and Orange counties, Southern California, USA	1997–2006	retrospective cohort study	MD	CALINE 4 dispersion model	105,092	WP	–	0.99 (0.92 1.06)	4.25(IQR: 1.36)	Birthweight, TLBW
Basu et al., 2014	California, USA	2000–2006	retrospective cohort study	MD	Geographic information system (GIS) model	322,981	WP, TS	–	1.01 (0.98, 1.05)	18.7 (IQR: 7.563)	Birthweight, TLBW
Vinikoor-Imler et al., 2014	North Carolina, USA	2003–2005	retrospective cohort study	MD	Community Multi-Scale Air Quality (CMAQ) model	322,981	WP, TS	–	T1: 0.94 (0.91, 0.98) T2: 0.95 (0.92, 0.99) T3: 1.01 (0.97, 1.06)	14.02	TLBW, SGA
Pereira et al., 2014a,b	Connecticut, USA	2000–2006	retrospective cohort study	MD	Proximity models (40 km)	48,208	WP, TS	1.01 (0.93, 1.09) T1: 1.03 (0.98, 1.08) T2: 0.98 (0.93, 1.03) T3: 0.99 (0.94, 1.04)	–	–	PTB
Gray et al., 2014	North Carolina, USA	2002–2006	retrospective cohort study	MD	Spatial hierarchical Bayesian model	457,642	WP	1.01 (0.99, 1.02)	1.02 (0.99, 1.04)	13.6 (IQR: 2.0)	Birthweight, TLBW, PTB, SGA
Ebisu and Bell, 2012	northeastern and mid-Atlantic United States	2000–2007	retrospective cohort study	MD	Proximity models (county-level)	1,207,800	WP, TS	–	2.2 (–0.2, 4.8)	13.41 (IQR: 2.71)	TLBW
Gehring et al., 2011		1996–1997	prospective birth cohort study	MD	Spatio-temporal exposure model	3853	WP, TS	1.22 (0.83, 1.80) T1: 0.98 (0.75, 1.29)	–	20.1 (IQR: 4.6)	Birthweight, PTB

	North, West, and Centre of The Netherlands									
Bell et al., 2007	Connecticut and Massachusetts USA	1999–2002	retrospective cohort study	MD	Proximity models (county-level)	358,504	WP, TS	–	1.054 (1.022, 1.087)	11.9 (IQR: 2.2)	Birthweight, TLBW
Laurent et al., 2014	Los Angeles County, California, USA	2001–2008	retrospective cohort study	MD	Chemical transport modeling (UCD_P model)	960,945	WP	–	1.025 (1.017, 1.033)	IQR: 5.82	TLBW
Pereira et al., 2014a,b	Connecticut, USA	2000–2006	retrospective cohort study	MD	Proximity models	61,688	WP, TS	1.13 (1.00, 1.28) T1: 1.10 (1.03, 1.17) T2: 0.93 (0.87, 0.99) T3: 1.06 (1.00, 1.11)	–	IQR: 2.33 Median: 12.38	PTB
Wu et al., 2009	Los Angeles and Orange Counties, California, USA	1997–2006	retrospective cohort study	MD	CALINE 4 dispersion model	81,186	WP, TS	1.03 (1.01–1.06)	–	1.82 (IQR: 1.35)	PTB, VPTB, MPTB

MD, monitoring network data; RSD, remote sensing data; WP, entire pregnancy period; TS, trimester specific; PTB, preterm birth; MPTB, moderate preterm deliveries; VPTB, very preterm deliveries; TLBW, term low birth weight; SGA, small for gestational age, T1, first trimester; T2, second trimester; T3, third trimester; IQR, interquartile range.

plots showed the estimated effect of $PM_{2.5}$ exposure on TLBW and PTB from the individual studies. Fifteen studies were incorporated into the meta-analysis of the risk of TLBW associated with 10μg/m^3 or IQR increases in $PM_{2.5}$. We estimated a significant increase in TLBW risk associated with overall $PM_{2.5}$ exposure (per IQR increment) throughout pregnancy across all 7 studies (OR = 1.03; 95% CI: 1.02–1.03). The pooled OR values of $PM_{2.5}$ exposure in the first, second and third trimester were 1.00 (95% CI: 0.91–1.11), 1.00 (95% CI: 0.96–1.03) and 1.03 (95% CI: 0.98–1.09), respectively (Table 2 and Fig. 2). Fewer estimates were available for the pooled estimate of OR for TLBW associated with 10μg/m^3 increase (n = 4) than IQR increment. The pooled OR of TLBW associated with 10μg/m^3 throughout pregnancy was 1.05 (95% CI: 0.95–1.12). Most studies indicated increased odds of TLBW with increased $PM_{2.5}$ exposure, but pooled estimates for all three trimesters were not significant. Heterogeneity, as measured by the I^2 value, was high, with the exception of the whole pregnancy period (IQR increment) and third trimester, which were moderate.

A forest plot for the association between 10μg/m^3 or IQR increment increases in $PM_{2.5}$ and PTB is presented in Fig. 2. Pooled ORs based on exposure period are summarized in Table 3. The pooled association estimate was greater for the entire pregnancy period for IQR increments compared to individual trimesters or entire pregnancy periods for 10μg/m^3 increments. We estimated a significant increase in risk of PTB associated with increased overall $PM_{2.5}$ exposure (per IQR increment) during the entire pregnancy across all 8 studies included (OR = 1.03; 95% CI: 1.01–1.05). The pooled OR values of $PM_{2.5}$ exposure in the first, second and third trimester were 1.03 (95% CI: 1.00–1.06), 1.01 (95% CI: 0.93–1.10) and 1.02 (95% CI: 0.99–1.04), respectively. The pooled OR for the relationship between PTB and 10μg/m^3 increment increases was 1.02 (95% CI: 0.93–1.12). The ORs from individual studies on PTB indicated mixed positive and negative associations (see Table 1). Heterogeneity was low for the entire pregnancy period (IQR increment increases) and 3rd trimester, but was high for all other groups.

3.3. Heterogeneity and publication bias

We used the Newcastle-Ottawa Scale (NOS) and Agency for Healthcare Research and Quality (AHRQ) guidelines to evaluate the methodological quality of each of the involved studies (Supplementary Material Tables 1–3). The mean scores ranged from 6 to 8. Several studies evaluated $PM_{2.5}$ exposure value by proximity models and land use regression or dispersion models simultaneously, and these studies calculated more than one effect estimates of $PM_{2.5}$ on birth outcomes (Dibben and Clemens, 2015, Lee et al., 2013; Huang et al., 2015; Laurent et al., 2013, 2014; Fleischer et al., 2014; Hyder et al., 2014). We chose the models with smaller confidence intervals to reduce the bias in exposure assessment. We evaluated the possibility of a publication bias in the 23 studies, and the funnel plot illustrated a symmetrical distribution of the points, suggesting a lack of publication bias; furthermore, no publication bias was found by either Begg's test and Egger's test (Tables 2 and 3).

4. Discussion

This systematic review provides an updated summary of the current scientific evidence of, and quantitatively assesses the overall association between adverse birth outcomes, specifically TLBW and PTB, and maternal $PM_{2.5}$ exposure during pregnancy (per 10μg/m^{3}or IQR increment increase). We found that maternal exposure to $PM_{2.5}$ per IQR increment increase throughout the entire pregnancy is related to an extra risk of PTB and TLBW

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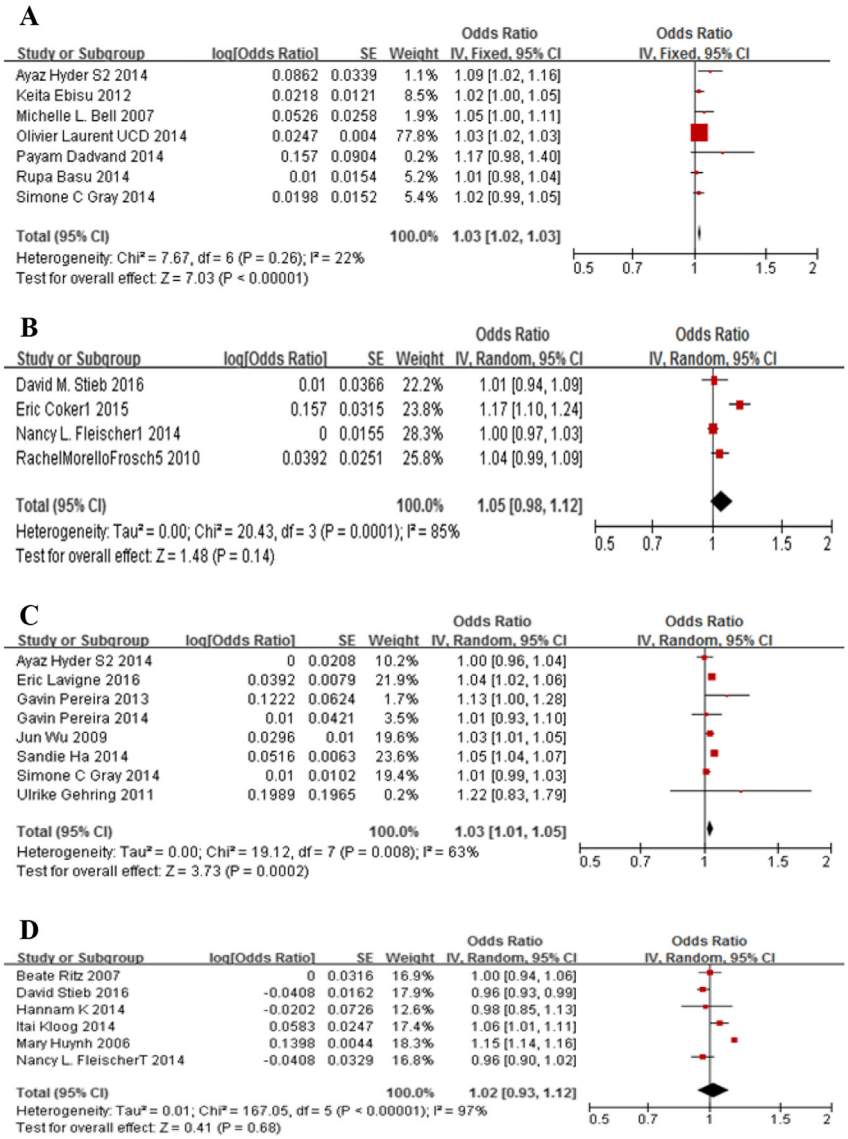

Fig. 2. Forest plot showing the risks of $PM_{2.5}$ expose and adverse birth outcomes during the entire pregnancy period. A: the association between $PM_{2.5}$ (per IQR increase) and term low birth weight; B: the association between $PM_{2.5}$ (per 10 g/m^3 increase) and term low birth weight; C: the association between $PM_{2.5}$ (per IQR increase) and preterm birth; D: the association between $PM_{2.5}$ (per 10 g/m^3 increase) and preterm birth.

(OR = 1.03, 95% CI:1.01–1.05 and OR = 1.03, 95% CI:1.02–1.03, respectively). The pooled ORs for the effect of $PM_{2.5}$ exposure (per 10μg/m^3 increment increase) throughout the entire pregnancy on PTB and TLBW revealed a positive, but not statistically significant, correlation (OR = 1.02, 95 CI %: 0.93–1.12 and OR = 1.05, 95 CI %: 0.98–1.12, respectively).

We identified 14 previous reviews that link $PM_{2.5}$ to TLBW and/or PTB, six studies employed a meta-analysis study, while the remainder were systematic reviews (n = 8). Systematic studies on maternal exposure to particulate matter during pregnancy did not provide consistent evidence of an association with the risk of preterm birth or term low birth weight. And the reported effects

Table 2
Pooled association between $PM_{2.5}$ exposure (per IQR increase or 10μg/m^3 increase) during the pregnancy and TLBW risk (OR, 95%CI) in different subgroups.

Subgroups	NO. of study	P for heterogeneity test	Summary OR (95%CI)	P for hypothesis test	I^2 (%)	P for Egger's test	P for Begg's test
Exposure during the entire pregnancy (10μg/m^3)	4	0.0001	1.05 (0.98, 1.12)	0.14	85	0.409	0.734
Exposure during the entire pregnancy (IQR)	7	0.26	1.03 (1.02, 1.03)*	<0.00001	22	0.42	0.095
First trimester exposure (IQR)	3	<0.0001	1.00 (0.91, 1.11)	0.92	90	0.783	1
Second trimester exposure (IQR)	4	0.001	1.00 (0.96, 1.03)	0.83	81	0.138	0.174
Third trimester exposure (IQR)	3	0.11	1.03 (0.98, 1.09)	0.28	55	0.033	0.296

* means that the result is statistical significance.

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Table 3
Pooled association between $PM_{2.5}$ exposure (per IQR increase or 10μg/m^3 increase) during the pregnancy and PTB risk (OR, 95%CI) in different subgroups.

Subgroups	NO. of study	P for heterogeneity test	Summary OR (95%CI)	P for hypothesis test	I^2 (%)	P for Egger's test	P for Begg's test
Exposure during the entire pregnancy (10μg/m^3)	6	<0.00001	1.02 (0.93, 1.12)	0.68	97	0.055	1
Exposure during the entire pregnancy (IQR)	8	0.008	1.03 (1.01, 1.05)*	0.0002	63	0.970	0.902
First trimester exposure (IQR)	5	0.009	1.03 (1.00, 1.06)	0.07	70	0.244	0.806
Second trimester exposure (IQR)	4	<0.00001	1.01 (0.93, 1.10)	0.83	98	0.265	0.734
Third trimester exposure (IQR)	4	0.06	1.02 (0.99, 1.04)	0.16	59	0.886	1

* means that the result is statistical significance.

associated with exposure at specific stages of pregnancy were inconsistent (Patelarou and Kelly, 2014; Shah and Balkhair, 2011; Bosetti et al., 2010; Ritz and Wilhelm, 2008; Ghosh et al., 2007; Sram et al., 2005; Maisonet et al., 2004; Glinianaia et al., 2004). The odds ratio risk of term low birth weight reported across the meta-analysis studies ranged from 1.02 to 1.09 for an increase exposure from 10. The excess risk of preterm birth ranged from odds ratio 1.05 to 1.15 (per 10μg/m^3 increment) across the studies (see Table 4). The present findings are coherent with the results reported in these previous meta-analyses. However, we had shorter 95% confidence intervals and smaller heterogeneity by using IQR increment increase as selected standard.

Epidemiological evidence links maternal exposure to ambient fine particular matter and birth outcomes by inducing systemic oxidative stress and inflammation, both of which are putative risk factors for low birth weight and preterm birth (Shah and Balkhair, 2011). By assessing biomarkers' levels of pregnant women's biological samples from cohort studies (both prospective and retrospective designs), researchers found that oxidative stress and inflammation can result in endocrine disruption, increase in maternal susceptibility to infections, placental inflammation, or impairment of placentation (Janssen et al., 2012, 2015; Saenen et al., 2016). Exposure to air pollutants is negatively associated with testosterone levels and high levels of exposure to particulate matters (PM_{10} and $PM_{2.5}$), may increase the prevalence of cells with immature chromatin. Placental mitochondria dysfunction is a potential mechanism through which prenatal maternal exposure to fine particular matter may cause low birth weight (van den Hooven et al., 2012). During the pregnancy, the placenta supports the nourishment, growth and development of the fetus, and a low placenta weight is related to birth weight (Salafia et al., 2006; Webster et al., 2008; Stieb et al., 2012; Dadvand et al., 2013). During pregnancy, maternal $PM_{2.5}$ exposure may be an important risk factor for intrauterine inflammation which could affect the growth, development and function of the placenta (Nachman et al., 2016). Some researches based on biological samples collected through birth cohort studies show that particulate air pollution affects the ability of the placenta by influencing mitochondria within the cells (Clemente et al., 2016; Saenen et al., 2016). Previous experimental studies in animals have recapitulated the outcomes from epidemiological studies. Exposure of pregnant female mice to ambient levels of air pollution during different gestational periods, as compared to controls, resulted in decreased weights of both placental and fetal, the sex ratio interference and changes of placental functional morphology (LICHTENFELS et al., 2007; Rocha E Silva et al., 2008; Veras et al., 2008; Damaceno-Rodrigues et al., 2009). In addition, inflammation at the sit of the placenta has been shown to expose rats under to $PM_{2.5}$ (de Melo et al., 2015). However, the mechanisms underlying the relationship between exposure to air pollution and adverse birth outcomes remains to be fully elucidated and requires further study.

We found a positive but non-significant relationship between $PM_{2.5}$ exposure period during only first, second or third trimester and PTB per IQR increase. On the other hand, only third trimester exposure was associated with higher TLBW (although not statistically significant either). The deficiency of statistical significance may be due to the lower number of studies that included trimester-specific data. Of note, previous studies found that birth weight is more consistently correlated to maternal exposure to $PM_{2.5}$, than PTB (Lavigne et al., 2016). Our results are not consistent with this finding, likely due to our selected birth weight outcome measures. One of the reasons why the data is disparate is that both TLBW and PTB are multifactorial, and influenced by a wide range of factors (including maternal age, education, race/ethnicity, SES, smoking status, maternal prenatal care, or parity, gestational week, season or sex of the infant). Most of these factors are rigid and unavoidable. The majority of studies adjusted for these common confounding variables. However, the data is irregular due to the variation in available sources of maternal and infant information. One such variable, maternal smoking is a confirmative risk factor for adverse pregnancy outcomes such as stillbirth, low birth weight and embryos or fetuses being small for gestational age. Nonetheless, not all studies included/acknowledged smoking as a confounding variable. Furthermore, the specific definitions of maternal smoking are multitudinous. Finally, birth outcomes are not only affected by maternal factors; parental exposure to air pollution may also play a role. More comprehensive and detailed birth records would help scientists control for such confounding variables.

Previous systematic reviews on air pollution and birth outcomes rarely included studies using exposure models incorporating satellite data. However, our meta-analysis included all exposure models, including monitoring of network data, remote sensing data, or both, and we were inclined to choose exposure-estimate model, which used satellite data as exposure source. Compared to ground monitor data, remote sensing data reduces exposure error and expands the study area, which not only increases sample size and generalizability but also decreases selection bias. Furthermore, satellite data has better spatial and temporal continuity than traditional monitor-based data. Satellite remote sensing continues to evolves and progress; in fact, there is already higher spatial resolution data (such as 3 × 3 km and 1 × 1 km) available today than was used in these studies, which will further reduce exposure error and enable us to more precisely estimate daily exposure by location (Kloog et al., 2012; Forouzanfar et al., 2015; West et al., 2016) Nevertheless, the use of any model predicting individual exposure inevitably introduces estimating error; future studies should employ individual direct exposure measurements to obtain more precise and accurate data.

The selection of study population, adjusted factors, air pollution data, and exposure estimation model varied among studies, and this is likely a source of heterogeneity. Furthermore, all of the studies' exposure estimation models used outdoor air pollution levels to calculate personal exposure. However, indoor air pollution varies and is vital to our discussion. Although we recognize that

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Table 4
Related results of the previous meta-analysis reviews.

First Author	Numbers of Studies Included	Outcome	Pollutant	OR (95%CI, $PM_{2.5}$ per 10μg/m^3 increment)	
				Preterm Birth	Term Low Birth Weight
Lamichhane et al., 2015	44	birth weight, preterm birth	PM_{10}, $PM_{2.5}$	1.14 (1.06–1.22)	
Sun et al., 2015	18	preterm birth	$PM_{2.5}$	1.13 (1.03–1.24) T1: 1.08 (0.92–1.26) T2: 1.09 (0.82–1.44) T3: 1.08 (0.99–1.17)	
Sun et al., 2016	32	birth weight, low birth weight	$PM_{2.5}$		1.090 (1.032, 1.150) T1: 1.026 (0.930, 1.130) T2: 1.035 (0.952, 1.125) T3: 1.035 (0.960, 1.585)
Nieuwenhuijsen et al., 2013	16	stillbirth, birth weight, preterm birth, small for gestational age, fetal growth, congenital anomalies,	PM_{10}, $PM_{2.5}$, NO_2,SO_2, Ozone	1.15 (1.14–1.16)	
Stieb et al., 2012	62	low birth weight, preterm birth, small for gestational age	CO, NO_2, Ozone, PM_{10}, $PM_{2.5}$, SO_2	1.05 (0.98, 1.13)	1.05 (0.99, 1.12)
Sapkota et al., 2012	20	low birth weight, preterm birth	PM_{10}, $PM_{2.5}$	1.15 (1.15, 1.16) T1: 1.04 (0.73, 1.34) T3: 1.07 (1.00, 1.15)	1.02 (0.99, 1.05)

T1, first trimester; T2, second trimester; T3, third trimester.

study region, the study design, and exposure assessment method could be sources of heterogeneity, we did not analyze them in this review owing to the restricted number of studies. Another variable is the fact that all of the included studies used different adjusting variables. Some vital variables, like smoking, were not included in the adjusted model. Due to our exclusion criteria, the number of included studies was limited. Furthermore, we only considered single pollutant models, because there was high heterogeneity between included studies in a subgroup analyses. Finally, a better understanding of the concentration-response association between air pollution and adverse birth outcome would be extremely valuable. We found there to be no publication bias based on an Egger's test, or a Begg's test. Nevertheless, owing to the limited sample size, we note that our study results should be interpreted with caution.

5. Conclusions

This meta-analysis reveals a clear association between $PM_{2.5}$ exposure throughout pregnancy and risks of preterm birth and term low birth weight, further supporting the hypothesis that $PM_{2.5}$ exposure increases the risk of adverse birth outcomes. Additionally, we have identified potential sources of the heterogeneity including epidemiologic research design, exposure assessment source, exposure estimation model, and study region. These findings provide an essential foundation for more in-depth meta-analysis of the relationship between air pollution and birth outcomes in the future.

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Appendix A. Supplementary data

Supplementary data related to this article can be found at http://dx.doi.org/10.1016/j.envpol.2017.03.055.

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