Risk of particulate matter on birth outcomes in relation to maternal socio-economic factors: a systematic review

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Abstract

A growing number of studies provide evidence of an association between exposure to maternal air pollution during pregnancy and adverse birth outcomes including low birth weight (LBW) and preterm birth. Prevention of these health effects of air pollution is critical to reducing the adverse infant outcomes, which can have impacts throughout the life course. However, there is no consensus on whether the association between air pollution exposure and birth outcomes varies by maternal risk factors including demographic characteristics and socio-economic status (SES). Such information is vital to understand potential environmental health disparities. Our search found 859 unique studies, of which 45 studies met our inclusion criteria (January 2000–July 2019). We systematically reviewed the 45 identified epidemiologic studies and summarized the results on effect modifications by maternal race/ethnicity, educational attainment, income, and area-level SES. We considered adverse birth outcomes of preterm birth, LBW, small for gestational age (SGA), and stillbirth. Suggestive evidence of higher risk of particulate matter (PM) in infants of African–American/black mothers than infants of other women was found for preterm birth and LBW. We found weak evidence that PM risk was higher for infants of mothers with lower educational attainment for preterm birth and LBW. Due to the small study numbers, we were unable to conclude whether effect modification is present for income, occupation, and area-level SES, and additional research is needed. Furthermore, adverse birth outcomes such as SGA and stillbirth need more study to understand potential environmental justice issues regarding the impact of PM exposure during pregnancy on birth outcomes.

1. Introduction

Birth outcomes such as low birth weight (LBW) (<2500 g) and preterm birth (PTB) (live birth before 37 weeks of pregnancy), like cardiovascular and respiratory diseases, have been identified as risks associated with exposure to ambient air pollution. These outcomes can further lead to neonatal mortality and physical morbidity (Class et al 2014). Low birth weight and preterm birth have serious health consequences through the life course, including developmental disability, obesity, hypertension, diabetes, heart disease, and psychiatric problems (Brauer et al 2008, Blumenshine et al 2010). The World Health Organization estimated that in 2014 approximately 10.6% of all live births globally were preterm (Chawanpaiboon et al 2019), which highlights the need of enhanced understanding of maternal and infant health risk factors.

Numerous epidemiologic studies have investigated the link between prenatal exposure to air pollution and adverse birth outcomes. Some previous review studies reported that evidence is sufficient for such relationships; a review based on 62 epidemiologic studies reported that the evidence is robust for the relationship between birth outcomes including LBW and PTB and ambient concentration of particulate matter (PM), CO, and NO2, while the evidence was less consistent for ozone and SO2 (Stieb et al 2012). Another review study suggested evidence of a potential
effect of exposure to PM, especially PM with aerodynamic diameter no larger than 2.5 μm (PM_{2.5}), on LBW (Bonzini et al 2010). Although review studies are few, some studies suggested that the evidence of the impact of PM was the most robust for LBW compared to other outcomes such as small for gestational age (SGA, birth weight <10th percentile for gestational age and sex) or PTB (Śrám et al 2005, Stieb et al 2012). These previous findings imply that preventing the health effects of air pollution is critical to improving infant health and to reducing the disease burden from adverse birth outcomes.

Studies have documented and quantified racial/ethnic inequity in the level of exposure to fine PM and consumption of goods and services in the United States (Tessum et al 2019). Several studies in other countries such as China have shown that people with lower socio-economic status (SES) are at higher risk of exposure to air pollution (Li et al 2018, Ma et al 2019). Other studies found that risk of PM differs across regions and populations due to demographic or socio-economic factors (Bell et al 2014). Review articles have mainly focused on examination of different associations between air pollution and adverse birth outcomes regarding particular exposure periods (e.g. trimester), air pollutants, and type of exposure variables (e.g. continuous, dichotomous, categorical) (Stieb et al 2012, Jacobs et al 2017). Consensus does not exist on effect modification of the relationship between air pollution and birth outcomes by maternal risk factors including demographic variables and SES. Examining which populations are most impacted by associations between PM and adverse birth outcomes can have implications for the strategies to reduce the disparities from air pollution.

We conducted a systematic review for published epidemiologic studies and summarized evidence of potential effect modification of maternal race/ethnicity and/or SES factors on the associations between PM exposure during pregnancy and risk of adverse birth outcomes including LBW, SGA, PTB, and stillbirth. Our review illuminates current research gaps and can inform decision-making in public health and environmental justice concerns.

2. Materials and methods

2.1. Literature search

Search terms and inclusion/exclusion criteria were designed to identify population-based studies for effect modification of race/ethnicity and/or SES on PM exposure and target birth outcomes. Literature searches were conducted for epidemiologic studies including non-randomized designs (i.e. cohort study, case-control study, cross-sectional study, economic study, time-series analysis) that examined the impact of PM with aerodynamic no larger than 10 μm (PM_{10}) or PM_{2.5}, on birth outcomes published from January 2000 to July 2019. We limited our search for papers published in English. Targeted birth outcomes were PTB, LBW, SGA, and stillbirth. We conducted the searches in the Medline/PUBMED (National Institutes of Health 2019) database. The titles, abstracts, and keywords were searched using the combination of following terms in order to identify the required articles: ‘air pollution’, ‘PM_{10}’, ‘PM_{2.5}’, ‘air pollutant’, ‘particulate’, ‘case control’, ‘cohort’, ‘pregnancy’, ‘birth outcome’, ‘stillbirth’, ‘birth weight’, ‘low birth weight’, ‘preterm birth’, ‘effect modification’, ‘SES’, ‘socio-economic’, ‘income’, and ‘race’. The truncation filter ‘asterisks (*)’ were used with the search terms to search all terms that begin with a certain combination of letters but can have any combination of letters representing the asterisk (e.g. modi* can be modification, modify, etc). The specific search strategies used in Medline/PUBMED searches are shown in our PICOS Worksheet and Search Strategy (see online supplementary materials available online at stacks.iop.org/ERL/14/123004/mmedia). Studies identified in each search strategy were combined and deduplicated.

We screened the titles and abstracts to exclude studies that are not related to our research question. The included studies fulfilled all of the following inclusion criteria: (1) examined maternal exposure to PM (PM_{10}, PM_{2.5} and/or smaller PM) during pregnancy; (2) included at least one of the birth outcomes of PTB, LBW, SGA, and stillbirth; and (3) provided risk estimations of PM and birth outcomes modified by maternal race/ethnicity and/or SES. Exclusion criteria included (1) studies not addressing PM exposure or target birth outcomes; (2) studies not reporting effect modification; (3) studies not performed in human populations; (4) conference abstracts, brief research paper without study details, review, commentaries, or pilot studies; and (5) epidemiologic studies not falling into non-randomized study (e.g. disease mapping study). A flow chart of study identification steps is shown figure 1. We reviewed the whole text of each identified study deserving full-text review and systematic review, as shown in figure 1.

2.2. Data extraction

For the identified relevant studies, we extracted the following information: study location, study duration, population size, study design, pollutants considered, exposure time windows, type of birth outcomes, type of effect modifiers and definition, type of statistical models for effect modifications (e.g. application of interaction terms or stratification), increment of pollution for estimates of the association (e.g. 10 μg m^{-3}), risk estimates and associated uncertainty (e.g. odds ratios (ORs) or relative risks (RRs) and 95% CIs), maternal and/or paternal risk factors considered for adjustment, and data source for exposure. Two investigators independently processed the data extraction from each study using the same data extraction
form. Effect modifiers were identified when a study provided different risk estimates of the relationship between PM and birth outcomes for the level or categories of maternal risk factors. When the study reported differences by maternal race/ethnicity or SES only for PM exposure level or prevalence of birth outcomes, but did not investigate potential differences in the exposure-response relationships between PM and the risk of birth outcomes, no effect modifier of the association between PM and adverse birth outcome was confirmed from such studies. While we consider multiple exposure time windows for the current systematic review, we extracted associations, if available, for exposure throughout whole pregnancy or trimester-specific periods. We identified each potential SES modifier as individual-level maternal SES or area-level SES. If a study provided stratified estimates for the association between exposure and birth outcomes by level of SES, we extracted each of stratified estimates. We determined that not enough studies were available to conduct quantitative meta-analysis due to the small number of identified studies for each SES factor and their sub-groups and varied study designs. Instead, we provide a narrative summary and qualitative assessment.

2.3. Systematic review
The systematic review was performed with consideration of guideline tools for assessing quality of population-based health studies and the methods applied by previous review studies (Bell et al 2014). To summarize study characteristics, we referred to quality criteria from the assessment tools of the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE), the Quality Assessment Tool for review articles by the Health Evidence (Health Evidence 2018), Cochrane guideline (Handbook version 5.1) (Higgins and Green 2011), and the Quality Assessment Tool for Quantitative Studies (National Collaborating Centre for Methods and Tools 2017) regarding the characteristics of primary studies and our research question of systematic review. As a result, we summarized study characteristics of identified studies for the following areas: description of research design and population size, sources of exposure and health data, clear definition of all exposure variables,
health outcomes, race/ethnicity and SES factors, description of exposure time window and statistical models (e.g. logistic model, linear regression) use of sensitivity analysis and presentation of associated results (e.g. tables, figures), and exposure assessment methods (e.g. nearest monitors, modeling estimates). For description of study design, we identified whether a given study stated use of the following methods: ecologic, time-series, cross-sectional, cohort, case-control, or case-crossover. Then, we assigned the study design based on the health data source and statistical analysis used in each study. A study was assigned the category of ‘cohort’ study only when study participants were followed for the period of pregnancy. When the health outcomes of infants were identified using a pre-existing health data source such as birth registry, the study was identified as a ‘case-control’ study in this review, although the study in question considered its study population as a birth cohort. We recognize that terms in epidemiology are not used with perfect consistency, and our goal is to use them consistently within this review.

We summarized the evidence of a potential modified association between PM exposure and birth outcomes for each identified effect modifier using methods adapted from the established categorization methods by Institute of Medicine committees (Fulco et al 2000) and applied by the US Congress and previous studies (Bell et al 2013). We used categories of no, weak, suggestive, and strong evidence of evidence of effect modification. The assignment of these categories for each effect modifier was conducted based on the quantity and quality of the reported results. The quality of results was based on the statistical uncertainty for the relationships between exposure to PM and birth outcomes by status of effect modifiers, although we recognize that overall quality reflects many other aspects of a study. It should be noted that the summarized strengths of effect modification are not a conclusive synthesis of evidence but a summary of the existing state of scientific evidence and the relative comparisons among the identified 45 studies and the effect modifiers.

3. Results

3.1. Characteristics of the eligible studies
The literature search identified 1291 research papers indexed from 1 January 2000 to 7 July 2019, with 859 unique papers. After exclusion, 45 papers met our inclusion criteria. Table 1 summarizes key study features of the included studies. The majority of identified studies was based on regions in the US (33 of the 45 studies). Of the 45 studies, 23 studies addressed term birth weight as either a continuous measurement (i.e. considering birth weight as a continuous variable) or dichotomous measurement (i.e. term low birth weight (TLBW)), 24 addressed PTB, 11 addressed SGA, and 3 addressed stillbirth (table 2). Some studies considered multiple health outcomes. PM$_{2.5}$ was the exposure metric for 38 studies with 15 studies considering PM$_{10}$, and 3 study considered the coarse fraction of PM, defined as PM$_{10}$—PM$_{2.5}$. Finer particles than PM$_{2.5}$ were considered in 2 studies (PM$_{1.0}$ in 1 study and PM$_{0.1}$ in 1 study) (table 2). In terms of study design, 75.6% of the studies (34 studies) conducted case-control analysis, while 20.0% (9 studies) were based on a cohort design (table 2) (Rudra et al 2011, Lee et al 2013, Pereira et al 2014a, 2014b, Schembri et al 2015, Stieb et al 2016, Wang et al 2018, Sheridan et al 2019, Stieb et al 2019).

Of the 45 studies that examined effect modification by maternal race/ethnicity and/or SES, the most commonly studied effect modifier was race/ethnicity with 31 studies. One study used race/ethnicity of infants as a potential effect modifier. The other identified effect modifiers were area-level integrated SES (e.g. European deprivation index) by 5 studies, mothers’ educational attainment by 17 studies, mothers’ occupation (e.g. worked as farmers versus other job workers) by 2 studies, and income or poverty level by 9 studies (table 2).

About 64.4% of the 45 studies analyzed birth outcome risks associated with PM exposure during specific trimesters, while 64.4% of the 45 studies focused on the whole pregnancy. Other exposure periods such as daily or annual exposure was considered for 13 studies, and 2 studies did not specify the exposure period.

Various confounding variables of maternal risk factors were addressed in the estimation between PM and birth outcomes in the identified studies. About 57.8% (26 studies) of the identified studies adjusted for mother’s race/ethnicity, 46.7% (21 studies) for maternal smoking use during pregnancy, 31.1% (14 studies) for mothers’ marital status, and 46.7% (21 studies) for sex of infant, while most studies (75.6%, 34 studies) adjusted for maternal age. Maternal educational attainment, which can be considered a proxy for SES, was adjusted for in more than half of the studies as well (57.8%, 26 studies).

3.2. Summary of effect modification
The summary of scientific evidence for potential effect modification of the PM-birth outcome association by the selected effect modifiers is shown in the figure 2 and supplementary table S1. For race/ethnicity, all studies investigated effect modification based on the race/ethnicity of the mother except for one study (Smith et al 2017). The US was the most represented country among the studies focusing on race/ethnicity. Among 14 studies that focused on LBW and maternal race/ethnicity, 9 studies reported statistically significant risks from PM exposure, with higher risk for infants of African American/black mothers compared to others. Two other studies found that risks for PM exposure, estimated separately by racial/ethnic
Table 1. Studies included in the systematic review.

| Study No. | Study | Location | Study period | Population size (No. births) | Study design | Effect modifiers | Air pollutant exposures | Health outcomes |
|-----------|-------|----------|--------------|-------------------------------|-------------|------------------|------------------------|-----------------|
| [1]       | Basu et al (2014) | California, US | 2000–2006 | 646 296 | Case-control | Race/ethnicity, age | PM$_{2.5}$, chemicals | BW, TLBW |
| [2]       | Bell et al (2007) | Connecticut and Massachusetts, US | 1999–2002 | 358 304 | Case-control | Race/ethnicity | PM$_{2.5}$, PM$_{10}$, SO$_2$, NO$_2$, CO | BW |
| [3]       | Benmarhnia et al (2017) | California, US | 2005–2010 | 1066 783 | Case-control | Race/ethnicity, maternal SES (education, age, medical enrollment, missing paternal information), neighborhood SES status (unemployment rate, poverty, linguistic minority, educational attainment) | PM$_{2.5}$, NO$_2$ | PTB, VPTB, SGA |
| [4]       | Coker et al (2016) | Los Angeles County, California, US | 2000–2006 | 804 726 | Case-control | Neighborhood-SES level | PM$_{2.5}$ | TLBW |
| [5]       | Darrow et al (2009) | Atlanta, US | 1994–2004 | 476 489 | Time-series | Race/ethnicity, maternal education | PM$_{10}$, PM$_{2.5}$, NO$_2$, SO$_2$, O$_3$, CO | PTB |
| [6]       | Ebisu and Bell (2012) | Northeastern and mid-Atlantic US | 2000–2007 | 1207 800 | Case-control | Race/ethnicity | PM$_{2.5}$, PM$_{10}$ | TLBW |
| [7]       | Enders et al (2019) | California, US | 2002–2013 | 2719 596 | Case-control | Race/ethnicity, maternal education, neighborhood poverty rate (PM$_{2.5}$–10) | PM$_{2.5}$ | TLBW |
| [8]       | Erickson et al (2016) | British Columbia, Canada | 2001–2006 | 231,929 | Case-control | Neighborhood-level SES index, Asian immigrant density, urbanicity | PM$_{2.5}$ | BW |
| [9]       | Faiz et al (2013) | New Jersey, US | 1998–2004 | 1719 | Case-control | Race/ethnicity, maternal education, maternal age, smoking, prenatal care | PM$_{2.5}$, SO$_2$, NO$_2$, CO | Stillbirth |
| [10]      | Faiz et al (2012) | New Jersey, US | 1998–2004 | 756 562 | Case-control | Race/ethnicity, maternal education | PM$_{2.5}$, SO$_2$, NO$_2$, CO | Stillbirth |
| [11]      | Geer et al (2012) | Texas, US | 1998–2004 | 1548 904 | Case-control | Race/ethnicity | PM$_{10}$, PM$_{2.5}$, SO$_2$, NO$_2$, CO, O$_3$ | BW |
| [12]      | Green et al (2015) | California, US | 1999–2009 | 3026 269 | Case-control | Race/ethnicity, maternal education | PM$_{2.5}$, SO$_2$, NO$_2$, CO | Stillbirth |
| [13]      | Hao et al (2016) | Georgia, US | 2002–2006 | 511 658 | Case-control | Race/ethnicity, maternal education, area-level poverty level, urbanicity | PM$_{2.5}$, NO$_2$, CO, O$_3$ | PTB |
| [14]      | Laurent et al (2014) | Los Angeles County, California, US | 2001–2008 | 960 945 | Case-control | Race/ethnicity, maternal education, area-level income, hypertension, diabetes, preeclampsia | PM$_{2.5}$, PM$_{0.1}$, NO$_2$, O$_3$ | TLBW |
| [15]      | Le et al (2012) | Detroit, Michigan, US | 1990–2001 | 164 905 | Case-control | Race/ethnicity, maternal education | PM$_{10}$, NO$_2$, SO$_2$, O$_3$, CO | PTB, SGA |
| [16]      | Lee et al (2013) | Allegheny County, Pennsylvania, US | 1997–2002 | 34 705 | Cohort | Race/ethnicity | PM$_{2.5}$, PM$_{10}$, O$_3$ | PTB, SGA |
| [17]      | Madsen et al (2010) | Norway | 1989–2002 | 25 229 | Case-control | Western ethnicity of mother | PM$_{10}$, PM$_{2.5}$, NO$_2$ | BW, SGA |
Table 1. (Continued.)

| Study No. | Study | Location | Study period | Population size | Study design | Effect modifiers | Air pollutant exposures | Health outcomes |
|-----------|-------|----------|--------------|-----------------|--------------|-------------------|------------------------|-----------------|
| [18]      | Morelli et al (2016) | Grenoble and Lyon urban areas, France | 2012 | 2790 000 | Ecologic | European Deprivation Index (EDI) | PM$_{2.5}$ | TLBW |
| [19]      | Morello-Frosch et al (2010) | California, US | 1996–2006 | 3545 177 | Case-control | Race/ethnicity | PM$_{2.5}$, PM$_{10}$, PM coarse (PM$_{2.5-10}$), NO$_2$, SO$_2$, O$_3$, CO | BW |
| [20]      | Ng et al (2017) | California, US | 2002–2009 | 1050 330 | Case-control | Race/ethnicity, maternal education | PM$_{2.5}$, PM$_{10}$, PM coarse (PM$_{2.5-10}$), NO$_2$, SO$_2$, O$_3$, CO | BW |
| [21]      | Padula et al (2014) | California, US | 2000–2006 | 263 204 | Case-control | Race/ethnicity, neighborhood SES status, maternal education | PM$_{2.5}$, PM$_{10}$, CO, NO$_2$ | PTB |
| [22]      | Parker et al (2005) | California, US | 2000 | 18 247 | Case-control | Race/ethnicity, maternal education | PM$_{2.5}$ | PTB |
| [23]      | Pereira et al (2016) | Rochester, New York State, US | 2004–2012 | 7121 | Case-control | Race/ethnicity | PM$_{2.5}$ | PTB |
| [24]      | Pereira et al (2014b) | Connecticut, US | 2000–2006 | 48 208 | Cohort | Race/ethnicity | PM$_{2.5}$ | PTB |
| [25]      | Pereira et al (2014a) | Connecticut, US | 2000–2006 | 271 204 | Cohort | Race/ethnicity | PM$_{2.5}$ | PTB |
| [26]      | Ponce et al (2005) | California, US | 1994–1996 | 59 700 | Case-control | Neighborhood-level SES | Distance-weighted traffic density | PM$_{2.5}$ | PTB, VPTB |
| [27]      | Rappazzo et al (2014) | New Jersey, Ohio, and Pennsylvania in US | 2000–2005 | 1940 213 | Case-control | Race/ethnicity | PM$_{2.5}$ | PTB |
| [28]      | Ritz and Yu (1999) | Southern California, US | 1989–1993 | 97 158 | Case-control | Race/ethnicity, maternal education | PM$_{10}$, CO, NO$_2$, O$_3$ | PTB |
| [29]      | Rudra et al (2011) | Western Washington State, US | 1996–2006 | 3509 (mothers) | Case-control | Maternal employment | PM$_{2.5}$, CO | PTB |
| [30]      | Salihu et al (2012b) | Florida, US | 2000–2007 | 103 961 | Case-control | Race/ethnicity | PM$_{2.5}$, PM$_{10}$, PM coarse (PM$_{2.5-10}$) | TLBW, VLBW, PTB, VPTB, SGA |
| [31]      | Salihu et al (2012a) | Hillsborough County, Florida, US | 2000–2007 | 12 356 | Case-control | Race/ethnicity | PM$_{2.5}$, PM$_{10}$ | TLBW, VLBW, PTB, VPTB, SGA |
| [32]      | Schembari et al (2013) | Bradford City, England | 2007–2010 | 9067 | Cohort | Race/ethnicity | PM$_{2.5}$, PM$_{10}$, NO$_x$, NO$_2$, O$_3$ | BW |
| [33]      | Schifano et al (2013) | Rome, Italy | 2001–2010 | 234 945 | Time-series | Race/ethnicity, maternal education, age, underlying clinical risk factors | PM$_{10}$, NO$_2$, O$_3$ | PTB |
| [34]      | Sheridan et al (2019) | California, US | 2003–2010 | 2293 218 | Cohort | Race/ethnicity | PM$_{2.5}$ | PTB |
| [35]      | Smith et al (2017) | Greater London, UK | 2006–2010 | 540 365 | Case-control | Infant’s race/ethnicity | PM$_{2.5}$, NO$_x$, NO$_2$ | BW, TLBW, SGA |
| [36]      | Stieb et al (2019) | Canada | 1999–2008 | 1001 700 | Cohort | Neighbor income | PM$_{2.5}$, NO$_2$, SO$_2$, CO, O$_3$ | PTB |
| [37]      | Stieb et al (2016) | Canada | 1999–2008 | 2928 515 | | | PM$_{2.5}$, NO$_2$ | BW, TLBW, PTB, SGA |
| Study No. | Study | Location | Study period | Population size (No. births) | Study design | Effect modifiers | Air pollutant exposures | Health outcomes |
|----------|-------|----------|--------------|------------------------------|--------------|------------------|-----------------------|-----------------|
| [38]  | Stieb et al (2015) | Canada | 1999–2008 | 2966 705 | Case-control and cohort (2 different health datasets were used) | Maternal place of birth (within and outside Canada), area-level income | PM<sub>2.5</sub> | TLBW, PTB, SGA |
| [39]  | Tu et al (2016) | Atlanta, Georgia, US | 2000 | 105 818 | Case-control | Maternal place of birth (within and outside Canada), area-level income | PM<sub>2.5</sub>, O<sub>3</sub> | BW, TLBW |
| [40]  | Vinikoor-Imler et al (2014) | North Carolina, US | 2003–2005 | 312 638 | Case-control | Infant gender, area-level income | PM<sub>2.5</sub>, O<sub>3</sub> | TLBW, SGA |
| [41]  | Wang et al (2018) | 30 provinces throughout mainland China | 2013–2014 | 1300 342 | Cohort | Maternal education, occupation, age, household registration, pregnancy BMI, season of conception | PM<sub>1</sub> | PTB |
| [42]  | Winckelmans et al (2015) | Flanders, Belgium | 1999–2009 | 525 635 | Case-control | Maternal education, paternal education, infant sex, parity, maternal age, season of conception | PM<sub>10</sub> | BW, SGA |
| [43]  | Wu et al (2009) | Los Angeles and Orange Counties, California, US | 1997–2006 | 81 186 | Case-control | Race/ethnicity, area-level poverty | PM<sub>2.5</sub>, NOx, NO<sub>2</sub> | PTB |
| [44]  | Wu et al (2016) | California, US | 2001–2008 | 4370 371 | Nested case-control | Race/ethnicity, maternal education, body mass index, gestational weight gain | PM<sub>2.5</sub>, NOx, NO<sub>2</sub> | TLBW, PTB, pre-eclampsia, gestational diabetes mellitus |
| [45]  | Yi et al (2010) | Seoul, South Korea | 2000–2003 | 433 173 | Case-control | Area-level household income | PM<sub>10</sub> | PTB |

* Notes. For the purposes of our systematic review, we refer to this study as a case-control study. The authors refer to the study as a cohort study. The study population was identified from a birth registry. We recognize that terms in epidemiology are not always used with perfect consistency; our goal is to use terms consistently within this review. The 'race/ethnicity' is maternal race/ethnicity, unless otherwise stated. BW: Birth weight measured as continuous variable, TLBW: term low birth weight measured as dichotomous variable, SGA: small for gestational age, PTB: preterm birth, VPTB: very preterm birth, VLBW: very low birth weight measured as dichotomous variable.
| Criterion | Number of studies (%) | Study numbers (refer to table 1) |
|-----------|-----------------------|----------------------------------|
| Research design | | |
| Cohort | 9 (20.0%) | 16, 24, 25, 29, 32, 34, 36, 37, 41 |
| Case-control | 34 (75.6%) | 1–4, 6, 7–15, 17, 19–23, 26–28, 30, 31, 35–37–40, 42–45 |
| Time-series | 2 (4.4%) | 5, 33 |
| Ecologic | 1 (2.2%) | 18 |
| Health outcomes $^a$ | | |
| Birth weight (BW) (continuous) | 12 (26.7%) | 1, 2, 8, 11, 17, 19, 22, 32, 35, 37, 39, 42 |
| TLBW (dichotomous) | 15 (33.3%) | 1, 4, 6, 7, 14, 18, 20, 30, 31, 35, 37–40, 44 |
| Preterm birth | 24 (53.3%) | 3, 5, 13, 15, 16, 21, 23–31, 33, 34, 36–38, 41, 43–45 |
| SGA | 11 (24.4%) | 3, 15, 17, 22, 30, 31, 37, 38, 40, 42 |
| Stillbirth | 3 (6.7%) | 9, 10, 12 |
| Particulate matter exposure $^b$ | | |
| PM$_{2.5}$ | 38 (84.4%) | 1–14, 16–25, 27, 29–32, 34–40, 43, 44 |
| PM$_{10}$ | 15 (33.3%) | 2, 5, 6, 11, 15, 17, 19, 21, 28, 30–33, 42, 45 |
| PM coarse (PM$_{10}$–PM$_{2.5}$) | 3 (6.7%) | 7, 19, 30 |
| PM$_{1}$ | 1 (2.2%) | 41 |
| PM$_{0.1}$ | 1 (2.2%) | 14 |
| Exposure method | | |
| Land use regression model | 7 (15.6%) | 4, 8, 29, 32, 37, 38, 42 |
| Source receptor model | 1 (2.2%) | 20 |
| Interpolated estimates | 5 (11.1%) | 14, 16, 21, 34, 44 |
| Fused model | 2 (4.4%) | 39, 41 |
| Dynamics models | 8 (17.8%) | 13, 17, 18, 27, 35, 40, 43, 44 |
| Average within study area | 6 (13.1%) | 2, 5, 6, 11, 12, 36 |
| Nearest monitor | 14 (31.1%) | 1, 3, 7, 9, 10, 19, 22–25, 28, 30, 31, 45 |
| Single monitoring site | 1 (2.2%) | 17 |
| Not specified | 1 (2.2%) | 33 |
| Exposure period $^c$ | | |
| Whole pregnancy | 29 (64.4%) | 1–2, 6, 7, 10, 11–14, 17, 19, 21–25, 30–32, 34, 35, 37, 38, 41–45 |
| Each trimester | 29 (64.4%) | 1, 2, 6, 7, 10–16, 19–25, 32, 34, 35, 37, 38, 40–45 |
| Others (e.g. annual average of study period, daily) | 13 (28.9%) | 3, 5, 9, 15, 26–29, 33, 36, 38, 39, 42 |
| Not specified | 2 (4.4%) | 8, 18 |
| Effect modifiers $^d$ | | |
| Race/ethnicity of mothers | 31 (68.9%) | 1–3, 5–7, 9–17, 19–25, 27, 28, 30–34, 43, 44 |
| Race/ethnicity of infants | 1 (2.2%) | 35 |
| Educational attainment of mothers | 17 (37.8%) | 3, 5, 9, 10, 12–15, 20–22, 28, 33, 40–42, 44 |
| Area-level household income or poverty level | 9 (20.0%) | 7, 13, 14, 36–39, 43, 45 |
| Occupation of mothers | 2 (4.4%) | 29, 41 |
| Area-level integrated SES | 5 (11.1%) | 4, 8, 18, 21, 26 |
| Sensitivity analyses $^e$ | | |
| Applying different sets of confounding variables in the health risk assessment | 10 (22.2%) | 2, 5, 7, 13, 14, 23, 24, 27, 30, 36 |
| Changing distance of exposure assignment (i.e. buffer size) | 7 (15.6%) | 1, 10, 12, 19, 24, 30, 40 |
| Excluding infants with certain conditions from the analyses (e.g. birth defects, exposure based on work address) | 7 (15.6%) | 7, 17, 21, 24, 32, 34, 35 |
| Stratifying results by birth conditions (e.g. month of conception, cesarean section, parity, smoking status during pregnancy) | 7 (15.6%) | 2, 6, 15–17, 21, 24 |
| Applying different air pollution data for the health risk assessment | 3 (6.7%) | 29, 37, 38 |

$^a$ Notes. Some studies may appear in more than one row.

TLBW = term low birth weight, SGA = small for gestational age.
subgroups, were not significant, but the magnitude of the risks tended to be higher in African American/blacks. The other 3 studies showed no significant effect modification by race/ethnicity. Among 17 studies based on PTB and race/ethnicity, 5 studies found statistically significant risks of PM exposure, with estimated risks generally higher for African American/blacks, whereas 1 study showed significant and higher risk for infants of white mothers. Five other studies presented different magnitude of the risks by race/ethnicity but the risk estimates were not statistically significant so a clear evidence of effect modification was not present. Among those studies, 1 study conducted in Rome showed higher risks of preterm birth in infants of Italian mothers compared to others. While studies generally identified African American/blacks as the most vulnerable to PM-birth outcome associations for PTB and LBW, results were not consistent regarding which race/ethnic group was the next most vulnerable. Among the 18 studies for PM-PTB association, the other 6 studies reported no significant evidence of effect modification of PTB by race/ethnicity. We concluded that there existed suggestive evidence that PM exposure risks for PTB and LBW are higher in infants of African-American/black mothers than in other racial/ethnic groups. Among 8 studies based on SGA and race/ethnicity, 2 studies reported significant and higher risks in African American/blacks, whereas 2 studies showed insignificant risk differences in the relationship between PM and SGA for racial/ethnic subpopulations and 4 studies found no evidence of effect modification by race/ethnicity. We concluded that there existed no current evidence of effect modification by race/ethnicity for SGA. No evidence was found for the effect modification by race/ethnicity for stillbirth, although our conclusion is hindered by the small number of studies, while 1 study reported higher risks in white mothers for the relationship between PM and stillbirth with 2 other studies reporting no significant effect modification.

We identified studies comparing the relationship between PM exposure and birth outcomes by maternal educational attainment for PTB, LBW, SGA, and stillbirth. For PTB, 2 studies found that infants of mothers with less education had higher PM risk, whereas 6 studies did not find such evidence. Among 6 studies based on LBW, 2 studies reported significantly higher PM risks in infants of mothers with less education, whereas 4 studies did not find significant effect modification of PM risk for SGA by maternal education, whereas the 4 studies conducted in California did not find significant effect modification. We concluded that there was no evidence of higher risk of SGA from PM exposure in mothers with less education. The study results for stillbirth modified by maternal education were reported in 1 study showing a tendency of higher risk by lower education level but the results were not statistically significant (Hao et al 2016). Significant effect modification by maternal education was not found in the other 2 studies. Thus, we concluded that there existed no effect modification by maternal education on the relationship between PM exposure and stillbirth.

For effect modification by income, there were 7 studies for PTB, 4 studies for LBW, and 2 studies for SGA. No evidence was found for effect modification.
by income for LBW and PTB as the studies reported no differences in PM risks by income level. We concluded that there is no evidence of effect modification was concluded for SGA, which may relate to the small number of studies.

One study examined the relationship between PTB and PM exposure as modified by mothers’ occupation, reporting higher risks in infants of farmers than other workers. The other study did not find risk differences between mothers who were employed and those who were unemployed during pregnancy. We concluded no evidence of effect modification by occupation for the examined birth outcomes.

For the effect modification by area-level integrated SES levels, there were 2 studies for LBW and 3 studies for PTB. The 2 studies focusing on LBW reported significantly higher risks in regions with lower SES level. In the 3 studies for PTB, the differences in the association between PM exposure and PTB were not statistically significant or the risk differences were not based on statistically comparable risk measurements. In conclusion, there existed no evidence for effect modification by area-level integrated SES levels for PM risk of PTB and LBW.

4. Discussion

While a few previous meta-analyses and systematic reviews have investigated the association between maternal exposure to PM and adverse birth outcomes (Stieb et al 2012, Lamichhane et al 2015, Sun et al 2016, Li et al 2017), little is known about the vulnerability of birth outcomes to air pollution as modified by maternal risk factors. To our knowledge, this is the first study summarizing previously published study results to examine the evidence of different relationships between PM exposure and adverse birth outcomes by maternal risk factors.

We identified knowledge gaps in the evidence for effect modification of maternal race/ethnicity and SES factors. The most commonly examined effect modifier was race/ethnicity. This is likely due to a greater existence of information on race and ethnicity compared to information related to SES in most health data. We found suggestive evidence of effect modification by race/ethnicity for PM risk on PTB and birth weight but not SGA or stillbirth. Several mechanisms could explain health disparities by race/ethnicity. Biological differences in vulnerability to disease and mortality has been suggested as one factor contributing to the racial health disparities (Kawachi et al 2005). Exposure to PM can differ by race/ethnicity. Disproportionate exposure to air pollution among non-white versus white communities has been reported by numerous studies (Pratt et al 2015). For instance, a study using US census tract data showed that household-level PM$_{2.5}$ exposure was lower in neighborhoods with a larger white population across 6 US cities (Jones et al 2014). Race is also an indicator of fewer health-protective behaviors in the United States; whites were more likely to exercise and had more health care use than others, while African American/blacks and Hispanics were more likely to use tobacco and alcohol than whites (Dubay and Lebrun 2008). Race/ethnicity is associated, although not fully correlated with, socioeconomic status, which would affect health behaviors and health outcomes as well. Disparities in birth outcomes risks among racial and ethnic groups may interplay with SES impacts. In the United States, various minority groups, particularly African American/black communities, have been reported to be related to increased levels of a variety of environmental harms in relation to their economic and political vulnerabilities (Lester 2018). Health status such as death rates differs by racial/ethnic minority groups with higher risk in African American/black communities in the United States (Bell et al 2013). The summarized evidence of the disparities in the vulnerability of birth outcomes to PM by race/ethnicity in our review could be related to the SES factors that we reviewed (i.e. income, education), although their effects cannot be disentangled in our analysis. Diversity driven by immigration would put certain groups into more or less vulnerable conditions of the impact of air pollution on birth outcomes as well. A study suggested a weaker exposure-response relationship between PM and birth weight in populations with high density of Asian immigration (Erickson et al 2016), but generalization of the effect modification by immigration is yet unknown due to the small number of studies and genetic, diet, and other differences among Asian and other ethnicities.

We found that educational attainment was the second most studied effect modifier among the selected effect modifiers in our systematic review. Overall, the identified studies suggested that infants of mothers with lower education had higher risk of PTB and LBW associated with maternal exposure to PM, whereas we identified no significant effect modification for SGA or stillbirth. Maternal educational attainment is a known SES factor for vulnerability of birth outcomes associated with air pollution (Yi et al 2010). Different definition of educational attainment among studies and differences in educational systems across countries hinder integration of the effect modification results through quantitative risk combination (i.e. meta-analysis). Categorizations of maternal education level were relatively consistent for the US research among the identified studies and more than half of those studies focused on California (Laurent et al 2014, Padula et al 2014, Wu et al 2016, Ng et al 2017). Among them, 1 study conducted in Georgia had categories of less than high school (<9th), some high school (9–11th), completed high school (12th), some college (<4 years), and completed college or more (≥4 years) (Hao et al 2016), whereas the other 4 studies had categories of less than high school (<9th), high school...
(9–12th), some college (<4 years), and completed college or more (>4 years) (Laurent et al. 2014, Padula et al. 2014, Wu et al. 2016, Ng et al. 2017). Studies in China (Wang et al. 2018) and Rome, Italy (Schifano et al. 2013) categorized education level considering education lower than primary school as previously observed by Wang et al. (2013) or high school (<12 years) and more than high school (>12 years) (Yi et al. 2010, Faiz et al. 2013) or low, medium, and high education (Winckelmans et al. 2015).

Finer categorization of education level appears to be useful to examine if the risk in infants of mothers with mid-level (e.g. secondary) education is higher than the risk in infants of mothers with primary or tertiary education as previously observed by Wang et al (Wang et al. 2018). Even though education categories are not completely consistent among studies, quantitative risk combination would become possible for simple categories of educational attainment (e.g. low versus high education) if more results based on finely resolved educational categories are reported from future studies.

Several studies examined effect modification using indices that combined information of multiple SES factors. The variables integrated into these SES indices were diverse and inconsistent among the identified studies and were measured at either the individual- or area-level. The variables included home ownership, marital status, occupational status, or education level for individual-level information, and median income, income from public assistance, employment rate, age of house, poverty level, or immigrant density for area-level information. Although independent comparison of effect modification by each of these variables or integration of the risk estimates for each variable are not viable among the studies, low level of integrated SES indices generally indicated higher effect estimates for the relationship between maternal exposure to PM and birth outcomes. Further assessments are required for effect modification by SES factors both independently and simultaneously considered with other correlated factors such as sex and race/ethnicity.

While SES factors should be adequately selected and interpreted in terms of plausible mechanisms (Andersen 2006), less attention has been paid to the consideration of more diverse SES variables that may affect the exposure to PM during the pregnancy other than education and income. For instance, job categories (e.g. white-collar versus blue-collar), occupation status and access to prenatal care may relate to general health of infants, but also to exposure patterns of air pollution. Furthermore, the importance of SES over life course as a determinant of health is increasingly recognized (Bell et al. 2005). For infant health, birth outcomes can be determined by long-term adverse health behaviors like smoking or drug use, poor nutrition, inadequate health care, and stress of mothers (Hogan et al. 2012). The SES indicators found in our review are common indicators used in health research of air pollution (Bell et al. 2005). Nonetheless, these indicators do not capture the complex nature of the relationships among SES, exposure to air pollution, and birth outcomes because they define current state (e.g. current year income) but not historical status. Further efforts for understanding lifestyle, time-activity patterns, and exposure patterns of pregnant women through collecting rich maternal information from longitudinal cohort studies will be needed in research investigating environmental justice in relation to health risks of air pollution on birth outcomes.

Most of the 45 studies we reviewed used birth certificate data or birth registry data, while 7 studies utilized birth cohort data (Rudra et al. 2011, Lee et al. 2013, Basu et al. 2014, Schenbari et al. 2015, Wang et al. 2018, Sheridan et al. 2019, Stieb et al. 2019). Some studies using birth certificate data based on electronic databases (Coker et al. 2016, Morello et al. 2016). One study used information at birth that was obtained from the official perinatal forms sent to the responsible non-profit association for perinatal data collection (Winckelmans et al. 2015).

Health studies often rely on fixed-response categories of race/ethnicity from the health data source used, and the fixed-response may fail to explicitly represent complex self-identified race/ethnicity of individuals or communities in the study (Bradby 2003). Further, we found a discrepancy in the definition of race/ethnicity among the studies reporting effect modification by race/ethnicity. The terms ‘non-Hispanic white’ and ‘non-Hispanic black’ was used in 16 studies (Darrow et al. 2009, Morello-Frosch et al. 2010, Rudra et al. 2011, Faiz et al. 2012, Geer et al. 2012, Salihu et al. 2012a, 2012b, Faiz et al. 2013, Basu et al. 2014, Rappazzo et al. 2014, Pereira et al. 2014a, Green et al. 2015, Benmarhnia et al. 2017, Ng et al. 2017, Enders et al. 2019, Sheridan et al. 2019). The terms ‘white’ or ‘black’ without specifying ‘non-Hispanic’ were used in 7 studies (Parker et al. 2005, Bell et al. 2007, Ebisu and Bell 2012, Le et al. 2012, Pereira et al. 2014b, Hao et al. 2016, Smith et al. 2017). The use of ‘African American’ distinguished from white (non-Hispanic) and Hispanic was found in 9 studies (Ritz and Yu 1999, Wu et al. 2009, Ebisu and Bell 2012, Lee et al. 2013, Laurent et al. 2014, Padula et al. 2014, Hao et al. 2016, Pereira et al. 2016, Wu et al. 2016). For the Asian group, the term ‘Asian’ was the most used term in the reviewed studies (13 studies) (Ritz and Yu 1999, Parker et al. 2005, Darrow et al. 2009, Wu et al. 2009, Rudra et al. 2011, Laurent et al. 2014, Padula et al. 2014, Pereira et al. 2014a, 2016, Hao et al. 2016, Wu et al. 2016, Smith et al. 2017, Sheridan et al. 2019), whereas 4 studies (Basu et al. 2014, Green et al. 2015, Ng et al. 2017, Enders et al. 2019) used the term ‘Non-Hispanic Asian’. The uses of different categories and terminologies for race/ethnicity proposes a challenge for direct comparisons of the relationship between air pollution and adverse birth outcomes differed by race/ethnicity among.
epidemiologic studies. Also, the use of broad categories that include many diverse subgroups can obscure differences that may be important for effect modification of air pollution’s health impacts.

Exposure assessment has been conducted using several methods such as through linking individuals’ residential addresses to local monitoring sites or from estimates based on advanced air pollution modeling. Air pollution modeling for individual-level exposure measurement can be divided into two methods: statistical models and dynamics model. Statistical models include land use regression, interpolation, and source-receptor models. Dynamics models include chemical transport, photochemical, and dispersion models. Some models combine multiple model types. The studies meeting our inclusion criteria used the following different methods to estimate exposure to PM: land use regression model, source receptor model, interpolation, fused model, dynamics model (dispersion model, chemical transport model), regional averaging of monitoring data, and use of data from the nearest monitor. A previous meta-analysis (Sun et al 2016) reported that the effect of PM$_{2.5}$ on LBW was higher and more significant when exposure to PM$_{2.5}$ was estimated based on dynamics models rather than observations at the nearest monitoring site from the participant’s address or regional average of monitoring sites within a region. Another aspect of the methods that varied among studies was residential mobility, as women can move during pregnancy, yet many studies assess exposure during pregnancy based on the residence at time of delivery, which can introduce exposure misclassification (Bell and Belanger 2012, Bell et al 2018, Tang et al 2018). Due to the small number of the total studies and wide range of methods included in our study, we did not compare effect modification based on PM associations assessed using different exposure methods, which warrants further study. As exposure methods can differentially affect exposure estimates and their uncertainty by population demographics (Butland et al 2013, Ebisu et al 2014), research on this issue is needed for different regions and subpopulations.

Although partner’s risk factors (e.g. occupation status, education, income, smoking status) likely affect general living quality of the family and the infants, partner’s information was far less considered in the identified studies. Studies have mostly relied on availability of mother’s information, marital status, and community-level SES variables as surrogates for related information. We found that 4 studies among the 45 identified studies considered partner’s information for assessing the impact of PM on birth outcomes such as smoking or race/ethnicity (Bell et al 2007, Winckelmans et al 2015, Wang et al 2018, Enders et al 2019). In many cases, such variables may not be available in the underlying health data used by the studies.

In our review, we found that the number of studies addressing effect modification by maternal race/ethnicity and SES factors was smaller for SGA and stillbirth compared to LBW or PTB. Despite the evidence of the racial/ethnical disparity of stillbirth trends in the U.S. population and etiological studies for causes of stillbirth (Salihu et al 2004), there still exists a lack of studies for racial/ethnical disparity for the relationship between air pollution and stillbirth that could provide important information for improvement of global and local maternal and fetal health. The paucity of studies for stillbirth might be related to the complexity of identifying cases of stillbirth in populations. Data collection for stillbirths is often executed utilizing death certificate data and population-based birth defects surveillance programs (Duke et al 2009, Basu et al 2016). The different terms describing the death of the fetus include miscarriage, spontaneous abortion, and stillbirth, relating to different time points during pregnancy (Munoz et al 2016). Furthermore, the methods of diagnosis and terminologies for stillbirth can vary across different countries and among the states within a country (Munoz et al 2016) although several guidelines for diagnosis and data collections for fetus deaths have been provided (Pathirana et al 2016, Alexander and Zeitlin 2017). The complexity of the definition of stillbirth would challenge accurate detection of stillbirth in populations and comparison of risk trends among epidemiologic studies regarding the different impacts of air pollution on birth outcomes by race/ethnicity and SES. However, our reviewed studies did not provide opportunities for examining the different definitions of stillbirth due to the small study number; thus, this remains an area of needed research.

Limitations of our study include the small number of relevant studies and geographically limited estimates for effect modification of the relationship between air pollution exposure and birth outcomes. Due to the small number of studies, it was not feasible to conduct a quantitative risk summarization; instead we provide a narrative summary of the evidence of effect modification based on the identified studies and our study should be interpreted in this context. Still our study captures and describes the existing literature on this topic.

A strength of this study is that we critically highlight research gaps for the evidence of effect modification by various maternal risk factors covering race/ethnicity and SES. The differences in the PM-adverse birth outcome relationships among subpopulations found in our review imply environmental injustice and provide important information relevant to decision-making for identifying and protecting vulnerable subpopulation.

5. Conclusion

This systematic review on epidemiologic studies was conducted to understand how the associations
between PM exposure and adverse birth outcomes differ by maternal demographic and socio-economic factors. Our review identified higher and more statistically significant magnitude of effect estimates for PM exposure and risk of LBW and PTB for infants of mothers who were African American/black or had lower educational attainment. Epidemiologic studies addressing disparities in the risk of PM by maternal SES have been disproportionately conducted for some types of birth outcomes with more attention on PTB and LBW. Evidence of effect modifications was not clearly present for income, occupation, and area-level SES in the reviewed studies, although our conclusions are limited by the small study numbers, whereas a larger volume of evidence existed for maternal race/ethnicity and educational attainment. Moreover, we found that only limited types of socio-economic factors were examined in previous studies. As a result, we suggest that more studies are required to understand potential effect modification of the risk of SGA and stillbirth due to maternal exposure to PM during pregnancy. Future studies are also needed for other socio-economic factors that can potentially play a role as effect modifiers such as income, job categories, occupation status, and access to prenatal care. Lastly, additional efforts to understand the interplay of race/ethnicity and SES on vulnerability of birth outcomes to air pollution are needed to provide information for identifying vulnerable communities and populations and planning preventive measures.

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Appendix

To summarize study designs that were used in the identified eligible studies, we set our criteria to use terms consistently within this review. Epidemiologic studies on birth outcomes often utilize pre-existing health data such as birth registry or birth certificate data and some authors refer to their study as a retrospective cohort study. However, we categorized this method as case-control study design as those studies identified the case and control groups using birth registry data before they assigned the exposure level to each birth. In other words, this method did not enroll a cohort and follow the population, or choose a cohort based on the exposure status before the outcome status occurs for the cohort. We recognize that the names of epidemiologic study designs can be used in different ways in the literature; our goal here is to use them consistently within this review. We categorized a study as a cohort study only when study participants were followed for the period of pregnancy before the delivery. In table 1, we marked studies that authors referred to as a cohort study but that we identified as a case-control design based on our criteria in this review. For example, Green et al referred to their study as a cohort study (Green et al 2015) but we referred to the study as a case-control study in this review based on the following reasons; the study population was based on all live births and stillbirths obtained from the birth data by the California Office of Vital Statistics and PM2.5 exposure level during the pregnancy was assigned to each stillbirth or non-stillbirth infant. Our criteria and results on the summarized study designs do not imply that epidemiologic studies have been used inaccurate terms. It shows that terms for study designs are not always used with perfect consistency for studying the relationship between air pollution and birth outcomes.

Data availability statement

Any data that support the findings of this study are included within the article. The reviewed articles are listed in the main text and the reference list. The findings of those reviewed articles are detailed in online supplementary material.

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