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REVIEW ARTICLE

Relationship between particulate matter exposure and female breast cancer incidence and mortality: a systematic review and meta-analysis

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Abstract

Objectives The associations of PM with the risk and prognosis of breast cancer have not been determined. This systematic review aimed to provide an updated understanding of the relationship between PM exposure level and breast cancer incidence and mortality.

Methods Articles from Web of Science and PubMed databases were methodically inspected until March 8, 2020. In final, 15 studies were kept for analysis, which provided necessary information to estimate the impact of PM on breast cancer risk and prognosis. These studies were combined for quantitative analyses to evaluate the effect of per 10 μg /m^3 increment exposure of $PM_{2.5}$ (< 2.5 μm in aerodynamic diameter) and PM_{10} (< 10 μm in aerodynamic diameter) using random-effects model.

Results $PM_{2.5}$ exposure was associated with increased breast cancer mortality (relative risk [RR] = 1.09; 95% confidence interval [CI]: 1.02, 1.16; $P_{Q\text{-test}} = 0.158$). No association of $PM_{2.5}$ (1.02; 0.97, 1.18; 0.308) and PM_{10} (1.03; 0.98, 1.09; 0.009) with the increase incidence of breast cancer was observed. Stratified analysis suggested that $PM_{2.5}$ was associated with the increase mortality of breast cancer (1.10; 1.03, 1.17; 0.529) in subgroup of developed country. PM_{10} was associated with breast cancer incidence based on studies published after 2017 (1.08; 1.00, 1.15; 0.157) and European studies (1.15; 1.06, 1.25; 0.502).

Conclusions Our study indicated that $PM_{2.5}$ exposure was related to breast cancer mortality. Further researches in this field are needed to validate the conclusion.

Keywords Air pollution · PM · Breast cancer · Mortality · Incidence

Abbreviations

N/A	Not applicable
RR	Relative risk
CI	Confidence interval
SD	Standard deviation
OR	Odds ratio
HR	Hazard ratio

Qing Guo and Xi Wang contributed equally to this work.

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NOS Newcastle–Ottawa scale
CRP C-reactive protein
IL-6 Interleukin-6
IQR Interquartile range
ER Estrogen receptor

Introduction

Breast cancer has developed into one of the most prevalent cancers threatening the survival of women worldwide. According to the latest report released by the American Cancer Society, new incidence of breast cancer in 2019 will rank first among all female cancer ($n = 268{,}600$), accounting for 30%, and the mortality of breast cancer will be the second highest ($n = 41{,}760$), accounting for 15% in America (Siegel et al. 2019).

Air pollution exposure is closely linked to a variety of cancer risks, and the International Agency for Research on Cancer (IARC) Working Group incorporated PM into human carcinogens in 2013 (Loomis et al. 2013). $PM_{2.5}$ exposure can cause oxidative damage to DNA and increase the expression level of oxidation markers such as 8-hydroxydeoxyguanosine (8-OHdG) and nuclear 7, 8-Dihydro-8-Oxo-2′-deoxyguanosine (8-oxo-DG) (Soberanes et al. 2012; Vattanasit et al. 2014). Cellular responses include release of inflammatory mediators, activation of transcription factors, kinase cascades, etc. ultimately lead to cell apoptosis or damage, which may be one of the potential mechanisms of PM on carcinogenesis (Ghio et al. 2012). In the past decades, accumulating epidemiological studies have demonstrated that PM exposure increases lung cancer incidence and mortality (Guo et al. 2016; Pun et al. 2017; Raaschou-Nielsen et al. 2013, 2016; Villeneuve et al. 2015), as well as other diseases (e.g., gastric cancer, dermatomyositis, heart failure (Shah et al. 2013; Weinmayr et al. 2018; Zantos et al. 1994)). It has been found that individuals living in urban areas are at higher risk of breast cancer and have higher $PM_{2.5}$ exposure (Brody et al. 2009). Mammograms show extremely and heterogeneously dense breasts in women exposed to high $PM_{2.5}$, which is an independent risk factor for breast cancer that has been extensively studied (Yaghjyan et al. 2017). In addition, PM has the effect of increasing the number of DNA strand breaks and reducing estrogen activity in MCF-7 and T47D-KBluc human breast cancer cells (Chen et al. 2013). The associations between breast cancer and PM_{10} (< 10 μm in aerodynamic diameter) or $PM_{2.5}$ (< 2.5 μm in aerodynamic diameter) exposure have been explored; however, the conclusions are still inconsistent (Datzmann et al. 2018; Hart et al. 2016; Hu et al. 2013; Reding et al. 2015). For example, Hu et al 2013 found that $PM_{2.5}$ and PM_{10} was significantly associated with breast cancer mortality. However, Hart et al 2016 did not observe the similar relationship between $PM_{2.5}$, PM_{10} and breast cancer risk.

Even though there are no animal studies on PM exposure and breast cancer risk, some scholars have conducted related cell line studies and found that hydroxylated polycyclic aromatic hydrocarbons (hydroxy-PAHs) contained in PM can interact with estrogen receptors, which is one of the possible mechanisms that PM is associated with breast cancer risk (Wenger et al. 2009). To gain a more comprehensive understanding of PM and breast cancer risk, and provide relevant evidence for future public health issues, we conducted this meta-analysis to investigate the association between incidence and mortality of breast cancer and PM, mainly for coarse particles (PM_{10}) and fine particles ($PM_{2.5}$).

Methods

Literature search

We manually retrieved PubMed and Web of Science databases using two sets of keywords in the title or abstract: (1) "air pollution", "particulate matter"; (2) "breast neoplasms", "breast tumors", "breast cancer", "breast carcinomas", and the language of results were limited to English. Comprehensive search criteria and procedures are listed in Supplement A. Besides, we additionally identified references of eligible studies and relevant reviews to check possible articles. We have restricted the literature to human studies published before March 8, 2020.

Inclusion and exclusion criteria

Studies evaluating the association between breast cancer incidence, mortality and PM exposure were incorporated into this systematic review. The included literature must provide quantitative estimates of $PM_{2.5}$ or PM_{10} exposure and breast cancer incidence or mortality, and reported odds ratio (OR) or hazard ratio (HR) or relative risk (RR) with a 95% confidence interval (CI). Literature that reported the association between breast cancer risk and other contaminants instead of PM were excluded. Furthermore, no sufficient data or the data provided cannot be converted to PM exposure standardized increment (per 10 $\mu g/m^3$) were excluded.

Regarding multiple publications containing overlapping study populations, we selected the publication with the longest follow-up years and the largest number of cases. Review articles were excluded. We conducted initial screening by browsing the title and abstract of retrieved articles, followed by full-text reading for eligibility, with the reasons for exclusion recorded.

Data extraction

The following data from eligible studies were extracted: country, the first author, publication year, the number of events and participants, follow-up years, mean age or range of age, pollutants ($PM_{2.5}$ and PM_{10}), outcome (incidence or mortality), adjusted risk estimates ([RR] or [HR]) and their 95% Cl, study type, study source, PMID, exposure assessment method, exposure distribution, adjusted risk factors. If the information above was not available, we contacted the author for further information in the original study. To ensure the accuracy of the derived data, the entire process was performed independently by two authors (Qing Guo and Xi Wang).

Quality assessment

The included cohort studies and case–control studies were evaluated for methodological quality by Newcastle–Ottawa scale (NOS), a nine-point scoring system for meta-analysis (Stang 2010) (Supplement B), which is comprised of three segments: selection, comparability, and outcome. High-quality studies have scores of 7–9, medium-quality studies have 4–6, and low-quality studies have scores of 4 or less (Wang et al. 2019; Xiong et al. 2019).

Statistical analyses

The HRs in the cohort studies were approximated to RRs, and unified risk estimates in this meta-analysis were reported as RRs. A generalized linear model was applied to examine the relationship between PM exposure and breast cancer risk (Liao et al. 2011; Pope et al. 2011; Yin et al. 2017). To convert all study estimates into standardized increments of PM exposure (per 10 μg/m^3), we used the following formula to recalculate risk estimates for each study (Shah et al. 2013):

$$RR_{(standardized)} = RR_{(original)}^{Increment\ (10)/Increment\ (original)}$$

where the RR was indicated as a continuous measure of interquartile range, the increment in IQR was used instead (Kim et al. 2018). The random-effects model was employed to combine RRs and 95% CI to allow between-study and within-study heterogeneity contribute to the variance (Borenstein et al. 2010; Hamra et al. 2014). We used I^2 values (Low: 25%; moderate: 50%; high: 75%) and Q test (statistically significant when P value < 0.1) to evaluate the heterogeneity (Higgins et al. 2003). Further, we performed subgroup analyses based on geographic location, country development, publication year, and follow-up time, respectively. To avoid the potential publication bias, Egger's tests were created, with the two-sided P value < 0.05. Besides, we provided forest and funnel plots to visually present the impact of a single study on the overall results, and performed trim and fill analyses in case of asymmetrical funnel plots. All statistical analyses in this systematic review were performed by Stata software version 12.0.

Results

Literature selection

A total of 1787 articles (105 from PubMed, 1667 from Web of Science and 15 from reference lists) were identified. After excluding the duplicates, titles and abstracts of 1,682 studies were reviewed. Screening out the irrelevant researches, we inspected the full-text of 35 possible studies for the eligibility subsequently, of which 20 studies were excluded for some reason: (1) fifteen studies have no correlation with this systematic review; (2) there were three reviews; (3) one study provided relevant data but could not be converted into valid information. Ancona et al. (2015) conducted research on PM_{10} from incinerators and breast cancer risk. The exposure level of PM_{10} was quite low (0.027 n/m^3 between the 95th and 5th percentiles), and the study found no association between PM_{10} and breast cancer mortality (HR = 0.97; 95% CI 0.71–1.33). When this data was standardized using the corresponding formula, the estimate was 0 (95% CI 0–∞). (4) Tagliabue et al (2016)'s target population was a group of women who had been diagnosed with breast cancer before the study begins. Based on this, the relationship between $PM_{2.5}$ exposure and breast cancer mortality was studied. This is inconsistent with the inclusion criteria, and its research data will lead to a positive result of the combined estimates in this systematic review. The screening process was shown in Supplementary Fig. 1. Finally, 15 articles [12 cohort studies (Andersen et al. 2017a; Andersen et al. 2017b; Bai et al. 2019; Cheng et al. 2019; Datzmann et al. 2018; DuPre et al. 2019; Hart et al. 2016; Reding et al. 2015; To et al. 2015; Turner et al. 2017; White et al. 2019; Wong et al. 2016), 2 case–control studies (Hu et al. 2013; Hung et al. 2011) and 1 cross-sectional study (Iwai et al. 2005)] covering 6,265,721 participants were included in the meta-analysis.

Overall meta-estimates

The characteristics of the included 15 articles are summarized in Table 1: 9 of the included studies were conducted in North America, 3 in Europe, and 3 in Asia. The follow-up duration for each study was longer than 4 years. Detailed information on PM exposure measurement of the included literature in Supplementary Table 4. The estimated effect values for a single study are shown in Supplementary Fig. 2 and Supplementary Fig. 3, and the pooled results are

Table 1 Detailed information of studies included in this meta-analysis

Study ID	References	Country	Published year	Age	No. of events (mortality or incidence)	Total population	Increment ($\mu g/m^3$)	RR (95% CI)	Mean follow-up period	Study type	Study	PMID
1	Hu et al. (2013)	United States	2013	> 55	N/A (mortality)	255,128	5 10	$PM_{2.5}$: 1.86 (1.12, 3.10) PM_{10}: 1.13 (1.02, 1.25)	10	Case–control	Surveillance Epidemiology and End Results	23592372
2	Reding et al. (2015)	United States	2015	35–74	1749 (incidence)	49,340	3.6 5.8	$PM_{2.5}$: 1.03 (0.96, 1.11) PM_{10}: 0.99 (0.98, 1.00)	4.95	Cohort	Sister Study Cohort	26464427
3	To et al. (2015)	Canada	2015	40–59	2789 (incidence)	29,549	10	$PM_{2.5}$: 1.24 (0.99, 1.55)	28.64	Cohort	Canadian National Breast Screening Study	25863281
4	Wong et al. (2016)	China	2016	> 65	111 (mortality)	60,273	10	$PM_{2.5}$: 1.8 (1.26, 2.55)	10.3	Cohort	N/A	27197138
5	Hart et al. (2016)	United States	2016	25–42	3416 (incidence)	115,921	10	$PM_{2.5}$: 0.90 (0.79, 1.03) PM_{10}: 1.00 (0.93, 1.07)	18	Cohort	Nurses' Health Study II Cohort	27257091
6	Andersen, Ravnskjaer et al. (2017a)	Denmark	2017	> 44	1145 (incidence)	22,877	3.3 2.9	$PM_{2.5}$: 1.00 (0.91, 1.09) PM_{10}: 1.02 (0.94, 1.11)	16	Cohort	Danish Nurse Cohort	27913396
7	Andersen, Stafoggia et al. (2017b)	Denmark	2017	> 55	3612 (incidence)	74,750	5 10	$PM_{2.5}$: 1.08 (0.77, 1.51) PM_{10}: 1.07 (0.89, 1.30)	12.4	Cohort	European Study of Cohorts for Air Pollution Effects, Transport related Air Pollution and Health impacts—Integrated Methodologies for Assessing Particulate Matter	29033383
8	Turner et al. (2017)	United States	2017	> 30	3844 (mortality)	623,048	4.4	$PM_{2.5}$: 1.03 (0.97, 1.08)	22	Cohort	Cancer Prevention Study II	28886601

Table 1 (continued)

Study ID	References	Country	Published year	Age	No. of events (mortality or incidence)	Total population	Increment ($\mu g/m^3$)	RR (95% CI)	Mean follow-up period	Study type	Study	PMID
9	Datzmann et al. (2018)	Italy	2018	ALL	9577 (incidence)	1,918,449	10	PM_{10}: 1.19 (1.09, 1.31)	4	Cohort	AOK PLUS	29884153
10	DuPre et al. (2019)	United States	2019	30–55, 25–42	1211 (mortality)	8,936	10 10	$PM_{2.5}$: 1.09 (0.87, 1.36) PM_{10}: 1.05 (0.89, 1.24)	13.25, 12.0	Cohort	Nurses' Health Study Cohort, Nurses' Health Study II Cohort	30647065
11	Bai et al. (2019)	Canada	2019	35–85	91,146 (incidence)	2,564,340	5.3	$PM_{2.5}$: 1.01 (0.99, 1.02)	14	Cohort	Ontario Population Health and Environment Cohort	31304979
12	Cheng et al. (2019)	United States	2019	45–75	2726 (incidence) 2729 (incidence)	57,589	10 10	$PM_{2.5}$: 1.10 (0.85, 1.42) PM_{10}: 1.05 (0.95, 1.16)	14.7	Cohort	Multiethnic Cohort	30924138
13	White et al. (2019)	United States	2019	35–74	2820 (incidence)	49,771	3.6 5.8	$PM_{2.5}$: 1.04 (0.98- 1.10) PM_{10}: 1.01 (0.97- 1.05)	8.4	Cohort	United States-wide prospective cohort	31596602
14	Hung et al. (2011)	China	2011	61 stations	N/A (mortality)	N/A	Quartiles <30.39 30.48–39.41 39.48–51.10	$PM_{2.5}$: 1.00 1.12 (0.96–1.32) 1.19 (1.03–1.38)	9	Ecological	N/A	N/A
15	Iwai et al. (2005)	Japan	2005	47 prefectures 13 large cities	9171 (mortality)	435,750	10	$PM_{2.5}$: 1.12 (1.04, 1.20)	N/A	Cross-sectional study	N/A	16053935

N/A not applicable, *RR* relative risk, *CI* confidence interval

grouped by the outcome (incidence or mortality) related to $PM_{2.5}$ and PM_{10}. All estimates represent changes in breast cancer incidence and mortality associated with per 10 μg/m^3 increment exposure in PM.

In this meta-analysis, $PM_{2.5}$ was related to the incidence of breast cancer (RR = 1.09; 95% CI 1.02–1.16; I^2 = 17.4%; $P_{Q\text{-test}}$ = 0.158). No significant association between breast cancer mortality and $PM_{2.5}$ was observed (1.20; 0.92–1.48; 52.5%; 0.078). The estimates of incidence and mortality for breast cancer with PM_{10} were 1.03 (0.98–1.09; 65.1%; 0.009) and 1.07 (0.93–1.20; 56.4%; 0.130), respectively.

We analyzed the relationship between $PM_{2.5}$ and the incidence and mortality of breast cancer (Table 2). The meta-estimates for $PM_{2.5}$ and breast cancer incidence by follow-up period (< 11 years and ≥ 11 years) were 1.11 (95% CI 0.97–1.25), 1.01 (0.94–1.08), respectively. By continent, the meta-estimates for North America and Europe were 1.03 (0.96–1.11) and 0.99 (0.75–1.23), respectively. When the meta-analyses were performed by the publication time (before 2017 and after 2017), the meta-estimates were 1.05 (0.84–1.25) and 1.02 (0.99–1.05), respectively. The relationship between $PM_{2.5}$ and breast cancer mortality was also conducted. With regard to follow-up period (< 11 years and ≥ 11 years), the meta-estimates were 1.42 (0.70–2.13), 1.06 (0.94–1.17), respectively. And for the continent, the meta-estimates were 1.06 (0.94–1.18) for North America and 1.11 (1.00–1.21) for Asia. The meta-estimates were 1.10(1.03–1.17) for developed country and 1.36(0.65–2.07) for developing country. The significant relationship between $PM_{2.5}$ exposure and breast cancer mortality was found in the subgroup for the publication time before 2017 (1.11; 1.00–1.22), and the meta-estimates showed 1.06 (0.94–1.17) for studies published after 2017.

As shown in Table 3, the meta-estimates of PM_{10} and breast cancer incidence were 1.05 (0.95–1.15) for follow-up period (< 11 years) and 1.02 (0.97–1.08) (≥ 11 years). When analyzed by geographic location (North America and Europe), the meta-estimates were 0.98 (0.97–1.00), 1.15 (1.06–1.25), respectively. And for the publication time, the meta-estimates were 0.98 (0.97–1.00) for the group of before 2017 and 1.08 (1.00–1.15) for the group of after 2017. There are 2 studies included that explored the association between PM_{10} and breast cancer mortality. And the meta-estimates were 1.13 (1.02–1.25) for follow-up period (< 11 years) and 0.99 (0.86–1.14) (≥ 11 years), same as the meta-estimates for publication time (before 2017 and after 2017).

Table 2 Estimates of breast cancer risk associated with a 10 μg/m^3 increment exposure in $PM_{2.5}$

Exposure	RR (95% CI)	I^2 (*P* value)	Studies included (by ID)
Incidence	1.04 (0.98, 1, 10)	17.4% (0.293)	2, 3, 5, 6, 7, 11, 12, 13
Follow-up period			
< 11 years	1.11 (0.97, 1.25)	0.0% (0.837)	2, 13
≥ 11 years	1.01 (0.94, 1.08)	25.5% (0.243)	3, 5, 6, 7, 11, 12
Continent			
North America	1.03 (0.96, 1.11)	38.6% (0.149)	2, 3, 5, 11, 12, 13
Europe	0.99 (0.75, 1.23)	0.0% (0.656)	6, 7
Publication			
Before 2017	1.05 (0.84, 1.25)	66.3% (0.051)	2, 3, 5
After 2017	1.02 (0.99, 1.05)	0.0% (0.856)	6, 7, 11, 12, 13
Mortality	1.20 (0.92, 1, 48)	52.5% (0.078)	1, 4, 8, 10, 14, 15
Follow-up period			
< 11 years	1.42 (0.70, 2.13)	68.2% (0.043)	1, 4, 14
≥ 11 years	1.06 (0.94, 1.17)	0.0% (0.642)	8, 10
Continent			
North America	1.06 (0.94, 1.17)	0.0% (0.476)	1, 8, 10
Asia	1.11 (1.00, 1.21)	68.6% (0.042)	4, 14, 15
Country			
Developed Country	1.10 (1.03, 1.17)	0.0% (0.529)	1, 8, 10, 15
Developing Country	1.36 (0.65, 2.07)	80.1% (0.025)	4, 14
Publication			
Before 2017	1.11 (1.00, 1.22)	60.6% (0.055)	1, 4, 14, 15
After 2017	1.06 (0.94, 1.17)	0.0% (0.642)	8, 10

RR relative risk, *CI* confidence interval

Table 3 Estimates of breast cancer risk associated with a 10 μg/m^3 increment exposure in PM_{10}

Exposure	RR (95% CI)	I^2 (P-Value)	Studies included (by ID)
Incidence	1.03 (0.98, 1.09)	65.1% (0.009)	2, 5, 6, 7, 9, 12, 13
Follow-up period			
< 11 years	1.05 (0.95, 1.15)	86.4% (0.001)	2, 9, 13
≥ 11 years	1.02 (0.97, 1.08)	0.0% (0.809)	5, 6, 7, 12
Continent			
North America	0.98 (0.97, 1.00)	0.2% (0.391)	2, 5, 12, 13
Europe	1.15 (1.06, 1.25)	0.0% (0.502)	6, 7, 9
Publication			
Before 2017	0.98 (0.97, 1.00)	0.0% (0.584)	2, 5
After 2017	1.08 (1.00, 1.15)	39.7% (0.157)	6, 7, 9, 12, 13
Mortality	1.07 (0.93, 1.20)	56.4% (0.130)	1, 10
Follow-up period			
< 11 years	1.13 (1.02, 1.25)	–	1
≥ 11 years	0.99 (0.86, 1.14)	–	10
Publication			
Before 2017	1.13 (1.02, 1.25)	–	1
After 2017	0.99 (0.86, 1.14)	–	10

RR relative risk, *CI* confidence interval

Sensitivity analyses

We conducted sensitivity analyses of PM ($PM_{2.5}$, PM_{10}) exposure and breast cancer incidence and mortality. After omitting the study by Datzmann et al. (2018), it indicated that the heterogeneity changed from $P = 0.009$, $I^2 = 65.1\%$ to $P = 0.547$, $I^2 = 0.0\%$ in the study of PM_{10} and breast cancer incidence. This study is a large sample semi-individual cohort study, which may be the explanation for heterogeneity. After deleting the study by Wong et al. (2016) in the analysis of $PM_{2.5}$ and breast cancer mortality, the P value increased from 0.158 to 0.537, and I^2 changed from 37.2% to 0.0%. HRs were used in the study by Wong et al. (2016); however, we utilized RRs in this meta-analysis (He et al. 2017).

Publication bias

Asymmetry was found in the funnel plots (Supplementary Fig. 4, (b), (c)) in the preliminary visual judgment of publication bias. Therefore, Egger's tests were conducted to provide more specific information in the analysis of breast cancer incidence and mortality with PM ($PM_{2.5}$, PM_{10}), and we did not find publication bias. In the analysis of $PM_{2.5}$ exposure and breast cancer incidence and mortality, Egger's tests showed $P = 0.407$, $P = 0.120$, respectively. With regard to the association between PM_{10} exposure and breast cancer incidence, the P value in Egger's tests was 0.063. No publication bias of the relationship between PM_{10} and breast cancer mortality was performed owing to the limitation of the number of relevant studies ($n = 2$).

Discussion

The meta-analysis included 15 studies, twelve of which were cohort studies, two were case–control studies and one was a cross-sectional study, covering populations in 10 countries. The results suggest that $PM_{2.5}$ exposure is related to breast cancer mortality. No association of $PM_{2.5}$ and PM_{10} with the increase incidence of breast cancer was found. It should be pointed out that Hung et al. studied the relationship between PAH and breast cancer mortality using $PM_{2.5}$ as an indicator, which could make the relationship between breast cancer and $PM_{2.5}$ exposure more positive.

In the subgroup analyses, when was stratified by follow-up period, geographic location, country development, and publication year, most of the results were similar. Regarding the breast cancer risk associated with $PM_{2.5}$ exposure, it showed that the meta-estimates of the Asian population or developing countries were higher than that of the North American population or developed countries. In terms of the relationship between PM_{10} and breast cancer risk, the meta-estimates for breast cancer incidence in the European population were significantly higher than those in North America. Moreover, compared with studies published after 2017, studies published before 2017 found a lower correlation between PM_{10} exposure and breast cancer incidence, which is different from the findings of $PM_{2.5}$ mentioned above.

According to the difference of aerodynamic diameter, PM contains ultrafine particles ($PM_{0.1}$), fine particles ($PM_{2.5}$), and coarse particles (PM_{10}), in which $PM_{2.5}$ and PM_{10} are widely concerned in relation to public health (Pope et al. 2011). However, $PM_{2.5}$ and $PM_{0.1}$ cause more damage,

probably because they are more easy to pass through the barrier of the body and eventually penetrate to the alveoli (Valavanidis et al. 2008). Both long-term exposure and short-term exposure to PM can have adverse effects on human beings, which has developed into a serious public health problem (Brunekreef and Holgate 2002).

The reason for a higher cancer risk caused by exposure to PM has not been fully elucidated. Possible mechanisms are as follows: (1) PM entering the body induces the production of reactive oxygen species, which causes oxidative stress reaction and ultimately leads to DNA damage (Crobeddu et al. 2017; Risom et al. 2005). In addition, studies have found that $PM_{2.5}$ is associated with lipid peroxidation, especially in newborns, which may also be a potential reason for PM to threaten human health (Ambroz et al. 2016). (2) Another possible mechanism involves an inflammatory response. Reactive oxygen intermediates can cause inflammation by activating the expression of TNF-α, IL-1α(Rahman and MacNee 1998). It has been found that PM can cause an increase in the expression level of C-reactive protein (CRP) in the body and can be used as an indicator of inflammation (Donaldson et al. 2001). In a cohort study of older adults, after more than 12 weeks of measurement, it was found that interleukin-6 (IL-6) was associated with exposure to traffic-related contaminants (Zhang et al. 2016a). Changes in the levels of these inflammation-related substances are indicative of the possibility of inflammation compared to normal bodies. (3) The physical properties and chemical composition of $PM_{2.5}$ related to carcinogenesis are unfavorable, and will change with season and geographical location (Bell et al. 2007). Studies have found that $PM_{2.5}$ can carry heavy metals (vanadium, nickel, etc.), organics, black carbon, nitrates, polycyclic aromatic hydrocarbons (PAH) and other substances (Mannucci et al. 2019; White et al. 2019). Prolonged exposure to PAH increases the risk of breast cancer in women (Lee et al. 2019; Stults and Wei 2018). PAH with lipophilic properties can be stored in the adipose tissue of the breast and combine with DNA to form the PAH-DNA adduct, which changes the structure and function of DNA (Agudo et al. 2017; Morris and Seifter 1992; Shen et al. 2017; Zhang et al. 2019). Another possible mechanism has been found in animal experiments, PAH may enhance its carcinogenicity and cytotoxicity by affecting the estrogen metabolic pathway (Kummer et al. 2008; Zhang et al. 2016b).

The literature included in this meta-analysis has identified breast cancer risk factors as potential confounders. Among the adjusted models for breast cancer morbidity or mortality outcomes, we chose the model that takes into account the most comprehensive risk factors. Detailed information of risk factors that have been adjusted in the included studies are shown in Supplementary Table 5. A meta-analysis based on retrospective and prospective studies showed that both active and passive smoking increased breast cancer risk (Macacu et al. 2015). Female estrogen and progesterone (ER and PR) receptors have also been found to be associated with breast cancer risk. In a cohort study of Japan, long-term smoking patients with ER+ or PR+ before menopause increased the mortality of breast cancer (Kakugawa et al. 2015). Studies have found that pesticide exposure is related to breast cancer risk, which may be associated with some of the chemicals carried by PM (Engel et al. 2005). And as mentioned above, PM is easily present in adipose tissue. We therefore speculate that obese women are at higher risk of breast cancer if the other conditions are the same or similar (Niehoff et al. 2017). Unhealthy behaviors such as motor vehicle pollutant exposure, smoking, potato consumption during breastfeeding may also lead to an increase in heavy metals (Pb, Cd) of breast milk, therefore, it is particularly important to monitor the pollutant exposure and develop healthy lifestyles (Garcia-Esquinas et al. 2011; Rebelo and Caldas 2016).

The main advantages of this systematic review can be divided into three aspects. First, we set the incidence and mortality rates as different outcomes. We analyzed $PM_{2.5}$ and PM_{10}, which are currently recognized as having a greater impact on human health. With the progression of the disease stage, the risk of breast cancer death will increase significantly. However, early screening can reduce breast cancer mortality (Tagliabue et al. 2016). A prospective study involving women from more than 10 countries in Europe found that alcohol intake increased breast cancer risk and was positively correlated with drinking time (Romieu et al. 2015). A healthy lifestyle index score (HLIS) is negatively correlated with breast cancer, which indicates that the lifestyle of postmenopausal women is a likely factor influencing breast cancer risk (McKenzie et al. 2015). In this study, we found the association between $PM_{2.5}$ exposure and breast cancer mortality, but not in breast cancer incidence. PM itself or its conjugates can produce molecular and cellular damage to breast tissues, but there are no relevant studies to clarify the molecular biological mechanism of PM exposure not related to breast cancer incidence. We believe that there are many reasons for the increase in breast cancer mortality. In addition to the biological role of PM, it is also related to the basic physiological state of the exposed population. This may be an explanation for the difference in results, and more future research is needed to confirm this view.

Second, we did not make restrictions on the publication time and geographic location of the literature, thus, we can expansively assess the incidence and mortality of breast cancer caused by PM exposure without time and space constraints. Third, the previous review qualitatively discussed the relationship between PM and breast cancer risk but did not yield quantitative results (White et al. 2018). However, we integrated data from existing relevant

literature and used random effects models to estimate RR of cancer incidence/mortality and PM. Therefore, this study presents more exhaustive and effective information to date.

There are still some limitations of this meta-analysis that need to be noted: (1) Regarding breast cancer risk and PM exposure, although there is already a certain amount of research, more data is needed. Based on pre-defined inclusion/exclusion principles, this article contains 15 studies, but more robust statistical results require the support of a large amount of information. (2) This study conducted subgroup analyses, but due to the limitations of the number of articles and the content of these researches, it can only be restricted to the basic information level of the literature, and there are no more in-depth analyses of breast cancer risk. (3) The study does not distinguish between indoor pollution and outdoor pollution.

Conclusions

This study identified an association between $PM_{2.5}$ exposure and breast cancer mortality. The atmospheric PM can come from multiple sources, and traffic-related exhaust emissions cannot be ignored. In the process of development, modern society needs to be alert to the human health risks caused by PM. Further, more large-scale studies and biological studies should be conducted to explore ways PM contributes to—or more likely is a surrogate of true risk factors for—breast cancer mortality.

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Availability of data and materials The datasets created and analyzed during the current study are available from the corresponding author by reasonable request.

Compliance with ethical standards

Conflict of interest The authors report no conflicts of interest.

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