

Systematic Review

Air Pollution and Breast Cancer Risk: An Umbrella Review

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Abstract: Breast cancer (BC) is a major global health challenge, responsible for one in four cancer diagnoses and one in six cancer-related deaths worldwide. It is the most frequently diagnosed cancer among women and the primary cause of cancer-related deaths in most countries. Recent studies have suggested a potential link between exposure to ambient air pollutants—such as nitrogen dioxide (NO₂) and particulate matter (PM₁₀ and PM_{2.5})—and an increased risk of breast cancer. However, the existing evidence remains inconclusive. This umbrella review, conducted according to PRISMA guidelines, aimed to evaluate the strength and reliability of epidemiological evidence concerning this association. All seven meta-analyses included in this review reported a relative risk greater than 1 for exposure to the three pollutants, though findings varied in terms of heterogeneity and publication bias. Notably, the overall analysis indicates that exposure to both NO₂ and PM_{2.5} may be associated with an increased risk of breast cancer incidence, while the evidence linking PM_{2.5} exposure to breast cancer mortality appears to be weaker. The most vulnerable groups were identified as premenopausal European women exposed to NO₂ and PM₁₀, as well as individuals in developed countries exposed to PM_{2.5}. Further research is necessary to examine PM composition and refine exposure assessment methodology. Given the widespread impact of breast cancer as the most common invasive malignancy, incorporating this outcome into environmental health research on air pollution is essential. A clearer understanding of these associations could support more targeted environmental interventions. Importantly, the available evidence suggests that breast cancer prevention can be addressed not only through personal lifestyle changes but also through broad public health policies focused on reducing NO₂, PM₁₀ and PM_{2.5} levels.

Keywords: air pollution; female breast cancer; particulate matter; nitrogen dioxide

1. Introduction

Breast cancer (BC) continues to represent a significant public health concern due to its high prevalence, accounting for roughly 11.6% of all cancer diagnoses worldwide [1,2]. The



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development of BC is influenced by a complex interplay of factors, as there are several types of breast cancer, each with different pathophysiological mechanisms [3,4]. Epidemiological studies have reported various risk factors associated with the development or progression of BC. These include hereditary and genetic factors, such as an individual or family history of BC and/or ovarian cancer, and inherited mutations (i.e., BRCA1, BRCA2, and other BC susceptibility genes). Other important risk factors include early age at menarche, late age at menopause, nulliparity, late age at first birth, obesity, and smoking [5,6], while breastfeeding and physical activity are recognized as protective factors [7–9]. Additionally, the International Agency for Research on Cancer (IARC) has highlighted some environmental and lifestyle-related risk factors, such as alcohol consumption, diethylstilbestrol exposure, hormonal therapies (including combined estrogen–progestin contraceptives and menopausal treatments), and ionizing radiation (X-rays and gamma rays) [10]. Recent studies have increasingly associated environmental pollutants—particularly endocrine-disrupting chemicals (EDCs) like dioxins and phthalates—with early breast development, breastfeeding difficulties, and a heightened risk of breast cancer due to their ability to interfere with the endocrine system and mammary gland development [11–15].

An emerging area of investigation is the potential relationship between ambient air pollution and breast cancer. While current findings vary across studies [16–25], a notable increase in BC cases in U.S. women between 1986 and 2002 has been partly attributed to greater exposure to emissions from traffic and industrial sources [26]. The biological plausibility of a potential connection between air pollution and BC is supported by the International Agency for Research on Cancer (IARC)’s classification of outdoor air pollution as a human carcinogen [27]. Given that approximately 80% of the global urban population is exposed to air pollution levels that exceed the recommended limits set by the World Health Organization (WHO) [28], further exploration of this potential association is crucial. Research has primarily concentrated on three pollutants—nitrogen dioxide (NO₂), and particulate matter (PM₁₀ and PM_{2.5})—as potential contributors to BC risk. This work aims to systematically assess the available literature using the umbrella review approach, which synthesizes findings from existing reviews to clarify areas of agreement, identify methodological limitations, and highlight directions for future investigation [29,30].

2. Materials and Methods

2.1. Umbrella Review Methods

Umbrella reviews, as described by Aromataris et al. [29], aggregate and re-analyze findings from existing systematic reviews to identify potential biases, areas of consensus, and gaps in the literature. As noted by Fusar-Poli [30], this approach offers a higher-level synthesis that enhances the interpretation and relevance of evidence, supporting more informed clinical and policy decisions.

The current umbrella review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) guidelines [31,32], in line with a priori protocol agreed upon by all authors and registered in PROSPERO (CRD42023395494).

2.2. Objective, Inclusion Criteria, and Exclusion Criteria

2.2.1. Review Question

This review seeks to determine whether exposure to air pollution is associated with an increased risk of BC incidence and mortality. Specifically, it examines the potential association between BC risk and exposure to nitrogen dioxide (NO₂) and to particulate matter (PM₁₀ and PM_{2.5}).

2.2.2. Objective

The primary objective of this review is to assess the quality and reliability of existing evidence pertaining to the associations between exposure to NO₂, PM₁₀, and PM_{2.5} and the risk of BC incidence and mortality.

2.2.3. Inclusion Criteria

Meta-analyses investigating the association between air pollution exposure and BC incidence or mortality in women, based on observational studies, were considered. The inclusion criteria were structured according to the Population, Exposure, Comparator, and Outcome (PECO) framework. The specific PECO criteria used for selecting meta-analyses are detailed below.

Participants:

Women in the general population who are at risk of developing BC.

Exposures and comparator:

Meta-analyses considering the exposure to the three pollutants PM₁₀, PM_{2.5}, and NO₂, using various exposure assessment models, and comparing different concentration levels.

Outcomes:

Incidence and mortality risks reported using statistical measures such as the pooled relative risk (RR).

2.2.4. Exclusion Criteria

The following types of studies were excluded: commentary, letters, non-English language studies, studies that did not report relevant BC outcomes, and studies that did not include a meta-analysis.

A study was classified as a meta-analysis if it met the following criteria:

- It clearly detailed the methodology used for systematic review;
- It provided details on the search strategy employed;
- It identified relevant primary studies from at least one database (e.g., PubMed OR Embase);
- It performed a quality appraisal of the include primary studies.

2.3. Search Strategy and Data Extraction

A comprehensive search was conducted in the PubMed (MEDLINE) and Embase databases from inception to December 2024. The search strategy was designed around the key terms “breast cancer” and “air pollutants” and was limited to meta-analyses with a search filter in the title or abstract fields. Table S1 provides the detailed search strategy.

Study selection was independently performed by two authors (CM and CAM), ensuring a rigorous screening process, while data extraction was performed in pairs by all authors (CM and MPo, MPa and MF, LM and SC, EF and CM, and CaM and GT). Extracted data included the following variables: first author, year of publication, study design, total population, number of events, age of the participants, country, confounders, pollutant increase, effect size with the corresponding 95% confidence interval (CI), exposure models, pollutants, exposure time, heterogeneity, and publication bias. We also discuss histological type, TNM stage, hormonal receptors, and menopausal status in the selected studies. To assess the extent of overlap among primary studies listed in each review, a citation matrix was constructed, and the Corrected Covered Area (CCA) was calculated. This metric quantifies the proportion of shared studies across various meta-analyses, providing a standardized measure of redundancy [33]. The formula for the CCA is as follows:

$$CCA = (N - r)/(rc - r)$$

where N represents the total number of citations of primary studies (including repeated citations), r is the count of primary studies, and c is the number of meta-analyses considered. The CCA is then classified as minimal (<5%), moderate (5–10%), or high (>10%), based on the proportion of overlap among studies [33].

2.4. Meta-Analytical Methods Used by the Authors of the Included Studies

To ensure a thorough assessment of the methodologies employed in the meta-analyses included in this umbrella review, we examined several key aspects: stated objectives and research questions, search strategy, eligibility criteria, data extraction process, quality assessment, statistical analysis (effect size measurement, model selection, heterogeneity, and publication bias), sensitivity analysis, registration and protocol adherence, PRISMA reporting, and ethical considerations.

2.5. Quality of Assessment

Two researchers (MP and MF) evaluated the methodological quality of each included meta-analysis using the AMSTAR 2 tool [34]. This 16-item checklist assesses key aspects of meta-analysis, with seven items, marked with an asterisk, designated as critical domains due to their significant impact on overall validity (Table S2). These items may be modified by the authors according to their perspectives on the article under review. In particular, we decided to classify domain 8 (Did the review authors describe the included studies in adequate detail) as ‘critical’, instead of domain 7 (Did the review authors provide a list of excluded studies and justify the exclusions?) of the original list, considering domain 8 a more appropriate measure of the quality of the studies.

Each criterion was evaluated based on whether it was fully met or only partially met. The overall assessment of methodological quality was determined by considering the extent to which criteria were satisfied. If a review exhibited multiple non-critical weaknesses, confidence in its findings was considered lower, which could result in a reclassification from moderate to low confidence. The confidence ratings are summarized in Table 1 [34].

Table 1. Rating overall confidence in the results of the review [34].

Rating	Description
High	No or one non-critical weakness: the meta-analysis provides an accurate and comprehensive summary of the results of the available studies that address the question of interest.
Moderate	More than one non-critical weakness *: the meta-analysis has more than one weakness but no critical flaws; it may provide an accurate summary of the results of the available studies that were included in the review.
Low	One critical flaw with or without non-critical weaknesses: the review has a critical flaw and may not provide an accurate and comprehensive summary of the available studies that address the question of interest.
Critically low	More than one critical flaw with or without non-critical weaknesses: the review has more than one critical flaw and should not be relied on to provide an accurate and comprehensive summary of the available studies.

* Multiple non-critical weaknesses diminish confidence in the review, and it is appropriate to move the overall appraisal down from moderate to low confidence.

2.6. Evaluation of the Strength of Evidence

The strength of the evidence was initially classified into four categories—strong, moderate, modest, and weak—based primarily on the percentage of meta-analyses reporting positive associations, grouped as follows: strong: $\geq 75\%$; moderate: $\geq 50\text{--}75\%$; modest: $\geq 25\text{--}50\%$; and weak: $<25\%$ [35].

This initial classification was then subject to potential downgrading by 1 level based on 3 additional criteria: number of cases (less or greater than 1000); level of heterogeneity, measured using the I^2 statistic (with thresholds at 40%, 60%, and 80% for increasing heterogeneity); and presence of publication bias, assessed via Egger’s test ($p > 0.10$ = low

risk; $p = 0.05$ – 0.10 = moderate risk; and $p < 0.05$ = high risk) or using the trim-and-fill method. Criteria are summarized in Table 2.

Table 2. Criteria used to evaluate the strength of evidence.

Evidence	Positive Association	Cases	Heterogeneity I^2	Publication Bias
Strong	$\geq 75\%$	>1000	0–40%	Absent: Egger’s test $p > 0.10$ or Negligible difference in case of trim-and-fill
Moderate	≥ 50 – 75%	>1000	41–60%	Possible: Egger’s test $p = 0.05$ – <0.10
Modest	≥ 25 – $<50\%$	<1000	61–80%	High: Egger’s test $p \leq 0.05$
Weak	0– $<25\%$	<1000	81–100%	High: Egger’s test $p \leq 0.05$

The strength of the association was based on the pooled relative risks (RRs) from multiple meta-analyses and classified as follows: very strong ($RR > 5$), strong ($RR > 2$), moderate ($RR > 1.5$), modest ($RR > 1.2$), and weak ($RR > 1$). In accordance with the American Statistical Association’s recommendations, a positive relative risk (RR) with a lower confidence interval limit between 0.70 and 1.00 was considered a potential trend warranting further investigation [36–38].

3. Results

3.1. Search Strategy Outcome

The complete process of article collection, screening, and eligibility assessment is shown in Figure 1. The initial search identified 40 studies. After eliminating duplicates, 34 studies remained and were screened based on their title and abstract. Of these, 15 were excluded for not meeting the inclusion criteria, leaving 19 full-text articles that were considered potentially eligible for inclusion. Of these, eight studies were not meta-analyses, one study was a mapping review, two studies had no inherent topic, and one study had no data stratified for sex. Finally, seven meta-analyses were included in this umbrella review: [39–45]. Excluded articles and the reasons for exclusion are reported in Table S3.

3.2. Quality Assessment and Bias

The seven studies evaluated received scores ranging from 10.5 to 14 based on the number of positive/yes ratings. Specifically, five studies were classified as having “moderate” quality, one as “low” quality, and another as “critically low” quality (Table 3). Additionally, all confounding factors considered within each study were analyzed and systematically categorized (Table S4). Regarding publication bias, Gabet et al., 2021 [39], Guo et al., 2021 [40], and Wei et al., 2021 [43] used a funnel plot, an Egger’s test and trim-and-fill analysis to investigate publication bias. Zhang et al., 2019 [41], Praud et al., 2023 [44], and Arif et al., 2024 [45] used a funnel plot and an Egger’s test, while Yu et al., 2021 [42] used only a funnel plot.

3.3. Associations Between NO_2 , PM_{10} , and $PM_{2.5}$ Exposure and Breast Cancer

Tables 4 and 5 summarize the key characteristics of the seven studies examining the associations between exposure to these three pollutants and breast cancer incidence and mortality, respectively. Table S5 summarizes the primary studies included in each meta-analysis for individual pollutants and outcomes, along with their corresponding CCA index. Tables S6–S8 provide a summary of the increases in pollutant levels, the exposure assessment models used, sensitivity analyses, heterogeneity, and potential publication bias for the meta-analyses included in this umbrella review, categorized by NO_2 , $PM_{2.5}$, and PM_{10} , respectively.

PRISMA 2020 flow diagram for new systematic reviews which included searches of databases, registers and other sources

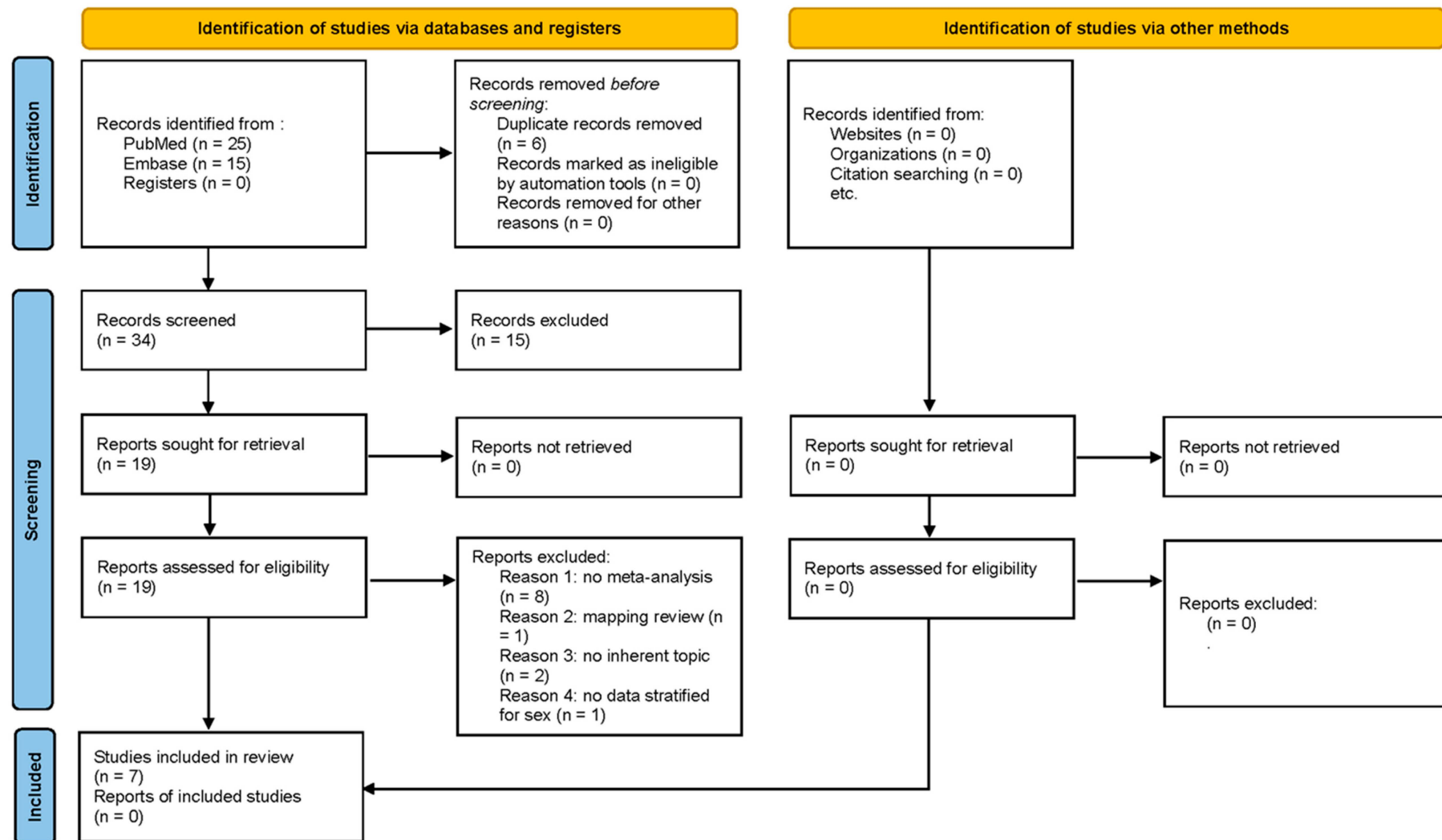


Figure 1. PRISMA flow chart describing the selection of included articles. Adapted by Page et al. 2021 [32].

Table 3. Quality assessment and risk of bias in meta-analysis using AMSTAR 2 [34].

	Gabet et al., 2021 [39]	Guo et al., 2021 [40]	Zhang et al., 2019 [41]	Yu et al., 2021 [42]	Wei et al., 2021 [43]	Praud et al., 2023 [44]	Arif et al., 2024 [45]
1. Did the research questions and inclusion criteria for the review include the components of PICO/PECOS?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
2. * Did the report of the review contain an explicit statement that the review methods were established prior to the conduct of the review and did the report justify any significant deviations from the protocol?	Yes	Yes	Yes	Partial Yes	Yes	Yes	Partial Yes
3. Did the review authors explain their selection of the study designs for inclusion in the review?	Yes	No	Yes	Yes	Yes	Yes	Yes
4. * Did the review authors use a comprehensive literature search strategy?	No	Yes	Yes	Yes	Yes	Yes	Yes
5. Did the review authors perform study selection in duplicate?	Yes	No	No	Yes	Yes	Yes	Yes
6. Did the review authors perform data extraction in duplicate?	No	Yes	Yes	Yes	No	No	No
7. Did the review authors provide a list of excluded studies and justify the exclusions?	No	No	No	No	No	No	No
8. * Did the review authors describe the included studies in adequate detail?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
9. * Did the review authors use a satisfactory technique for assessing the risk of bias (RoB) in individual studies that were included in the review?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
10. Did the review authors report on the sources of funding for the studies included in the review?	Yes	Yes	Yes	Yes	Yes	No	No
11. * If meta-analysis was performed, did the review authors use appropriate methods for statistical combination of results?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
12. * If meta-analysis was performed, did the review authors assess the potential impact of RoB in individual studies on the results of the meta-analysis or other evidence synthesis?	Yes	Yes	Yes	Yes	Yes	Yes	No
13. * Did the review authors account for RoB in primary studies when interpreting/discussing the results of the review?	Yes	Yes	Yes	Yes	Yes	Yes	No
14. Did the review authors provide a satisfactory explanation for, and discussion of, any heterogeneity observed in the results of the review?	Yes	Yes	Yes	No	Yes	Yes	Yes
15. * If they performed quantitative synthesis, did the review authors carry out an adequate investigation of publication bias (small study bias) and discuss its likely impact on the results of the review?	Yes	Yes	Yes	Yes	Yes	Yes	Yes
16. Did the review authors report any potential sources of conflict of interest, including any funding they received for conducting the review?	No	No	No	No	No	Yes	Yes
Total of yes	13/16 (81.2%)	13/16 (81.2%)	14/16 (87.5%)	14/16 (87.5%)	14/16 (87.5%)	13/16 (81.2%)	10.5/16
Rating overall confidence	Low	Moderate	Moderate	Moderate	Moderate	Moderate	Critically low

The domains considered critical are identified using an asterisk.

Table 4. Associations between NO₂, PM₁₀, and PM_{2.5} exposure and BC incidence.

Pollutant	Author, Year	Design of Included Studies	Total Population	Age (Years)	Country	Effect Size RR (95% CI)	Heterogeneity I ² *	Publication Bias **
NO ₂	Gabet et al., 2021 [39]	6 cohort, 4 case-control	3,914,690	25–75	1 Germany, 2 Sweden, 5 Canada, 2 Denmark, 2 France, 1 Netherlands, 1 UK, 1 Spain, 2 Italy, 1 Norway, 2 USA, 1 Austria	1.03 (1.01–1.05)	24	0.018
	Wei et al., 2021 [43]	11 cohort, 3 case-control	4,002,546	35–85	3 Denmark, 4 USA, 1 Europe, 1 Germany, 1 Israel, 5 Canada	1.02 (1.01–1.04)	46.8	0.024
	Praud et al., 2023 [44]	8 cohort, 5 case-control	128,618	Not reported	3 USA, 5 Canada, 1 Denmark, 2 France, 1 Germany, 1 Europe	1.02 (1.00–1.03)	16.9	0.27
PM ₁₀	Zhang et al., 2019 [41]	8 cohort	592,369	25–65+	5 USA, 2 Denmark, 1 Netherlands, 1 UK, 1 Italy, 1 Norway, 1 Germany	1.05 (0.98–1.12)	72.7	Not Reported
	Yu et al., 2021 [42]	4 cohort	2,107,018	0–90	1 Denmark, 1 USA, 1 Germany	1.05 (0.93–1.19)	68	0.030
	Gabet et al., 2021 [39]	1 case-control, 5 cohort	1,326,524	0–90	1 France, 1 Germany, 1 Sweden, 2 Denmark, 1 Netherlands, 1 UK, 1 Italy, 1 Norway, 3 USA, 1 Austria	1.06 (0.99–1.13)	27.6	0.41
	Guo et al., 2021 [40]	1 case-control, 8 cohort	2,552,761	0–90	5 USA, 1 Canada, 2 Denmark, 1 Italy	1.03 (0.98–1.09)	65.1	0.009
	Wei et al., 2021 [43]	7 cohort	2,290,241	0–90	4 USA, 1 Germany, 1 Denmark, 1 Europe	1.04 (0.98–1.10)	70.3	0.06
	Arif et al., 2024 [45]	10	Not reported	Not reported	4 Europe, 4 Americas	1.14 (0.97–1.30)	84.0	0.00
PM _{2.5}	Yu et al., 2021 [42]	6 cohort	2,871,705	25–85	1 Denmark, 1 USA, 3 Canada	1.03 (0.93–1.13)	63	0.020
	Wei et al., 2021 [43]	11 cohort	11,755,200	0–90	2 Denmark, 6 USA, 1 Europe (Sweden, Norway, Italy, UK, Netherlands, Austria), 3 Canada	1.03 (0.99–1.06)	8.2	0.00023
	Gabet et al., 2021 [39]	1 case-control, 6 cohort	2,848,486	25–85	1 France, 1 Sweden, 2 Canada, 2 Denmark, 1 Netherlands, 1 UK, 1 Italy, 1 Norway, 3 USA, 1 Austria	1.01 (0.93–1.11)	37.4	0.72
	Guo et al., 2021 [40]	11 cohort, 1 case-control, 1 ecological, 1 cross-sectional	6,643,972	25–85	7 USA, 2 Canada, 2 China, 2 Denmark, 1 Italy, 1 Japan	1.04 (0.98–1.10)	17.4	0.293
	Zhang et al., 2019 [41]	11 cohort, 2 ecological	994,551	25–>65	1 Canada, 6 USA, 1 Sweden, 2 Denmark, 1 Netherlands, 1 UK, 1 Austria, 1 France, 3 Italy, 1 Spain, 1 Germany, 1 China, 1 Puerto Rico, 1 Taiwan, 1 Japan	1.02 (0.93–1.11)	30.6	0.218
	Arif et al., 2024 [45]	14	Not reported	Not reported	4 Europe, 7 Americas	1.05 (0.98–1.12)	55.7	0.00

* I² test interpretation: 0–40%: might not be important, 41–60%: may be moderate, 61–80%: may be substantial, 81–100%: may be considerable. ** Egger’s test *p*-value: *p*-value < 0.05 indicates the presence of publication bias.

Table 5. Associations between PM₁₀ and PM_{2.5} exposure and BC mortality.

Pollutant	Author, Year	Study Design	Total Population (nr)	Cases	Age (Years)	Country	Effect Size RR (95% CI)	Heterogeneity I ² *	Publication Bias **
PM _{2.5}	Yu et al., 2021 [42]	4 cohort	756,393	7895	0–90	3 USA, 1 Hong Kong 1 Canada, 6 USA, 1 Sweden, 2 Denmark, 1 Netherlands, 1 UK, 1 Austria, 1 France, 3 Italy, 1 Spain, 1 Germany, 1 China, 1 Puerto Rico, 1 Taiwan, 1 Japan	1.18 (0.81–1.73)	70	0.02
	Zhang et al., 2019 [41]	5 cohort, 2 ecological	913,779	5439	25–65+	7 USA, 2 Canada, 2 China, 2 Denmark, 1 Italy, 1 Japan	1.17 (1.05–1.30)	73.1	0.122
	Guo et al., 2021 [40]	3 cohort, 1 case-control, 1 ecological, 1 cross-sectional	692,257	51,661	25 to 85	Not reported	1.20 (0.92–1.48)	52.5	0.12
	Arif et al., 2024 [45]	Not reported	Not reported	Not reported	Not reported	Not reported	1.17 (1.07–1.27)	55.2	0.04
PM ₁₀	Zhang et al., 2019 [41]	cohort/case- control	264,064		25–55+	USA	1.11 (1.02–1.21)	0.0	
	Guo et al., 2021 [40]	cohort/case- control	264,064		25–55+	USA	1.07 (0.93–1.20)	56.4	Not NA

* I² test interpretation: 0–40%: might not be important, 41–60%: may be moderate, 61–80%: may be substantial, 81–100%: may be considerable. ** Egger’s test *p*-value: *p*-value < 0.05 indicates the presence of publication bias.

3.3.1. NO₂ Exposure and BC Incidence

The association between NO₂ exposure and BC incidence is addressed in only three of the seven selected studies: Gabet et al. [39], Wei et al. [43], and Praud et al. [44]. All three studies reported a positive association with relative risks (RRs) of 1.03 (95% CI: 1.01–1.05), 1.02 (95% CI: 1.02–1.04), and 1.015 (95% CI: 1.003–1.028), respectively, per 10 µg/m³ NO₂ increase. The meta-analyses included 16 primary studies [46–61] showing a very high level of overlap (CCA = 66%) [33] and heterogeneity ranging from 16% to 46.8%.

Wei et al. [43] conducted four subgroup analyses based on study design, geographical region, menopausal status, and estrogen/progesterone receptor (ER/PR) status. Risk was higher in case-control studies and among Asian populations. No differences in risk were observed based on hormonal receptor status. In other subgroup analyses, risk estimates remained unchanged, while heterogeneity was lower among studies conducted in North America and Europe, as well as in postmenopausal women and receptor-positive and receptor-negative groups. Gabet et al. [39] also conducted multiple subgroup analyses, showing stable association across study design, population characteristics, level of adjustment, and analytical approaches. Variations emerged depending on how exposure was characterized. Risk estimates ranged from 1.01 in studies using residential history for exposure assessment to 1.06 in studies involving participants recruited from 2000 onward and were higher in premenopausal women (1.04, 95% CI 0.94–1.16). Regarding hormone receptor status, the relative risk (RR) was 1.045 (0.980–1.114) for estrogen-receptor-positive/progesterone-receptor-positive (ER+/PR+) tumors and 0.987 (0.885–1.101) for estrogen-receptor-negative/progesterone-receptor-negative (ER-/PR-) tumors. Similarly, Praud et al. [44] found an increased risk among European populations and ER+/PR+ tumors, with negligible heterogeneity. Sensitivity analyses are detailed in Table S6.

The overall findings suggest a moderate positive association between NO₂ exposure and BC incidence, despite substantial study overlap.

3.3.2. PM₁₀ Exposure and BC Incidence

The association between PM₁₀ exposure and BC incidence is addressed in six studies. The reported relative risks (RRs) across studies were as follows: 1.06 (95% CI: 0.99–1.13) [39], 1.03 (95% CI: 0.98–1.09) [40], 1.05 (95% CI: 0.98–1.12) [41], 1.05 (95% CI: 0.93–1.19) [42], 1.04 (95% CI: 0.98–1.10) [43], and 1.14 (95% CI: 0.97–1.30) [45]. All six meta-analyses reported a positive association with the lower confidence interval limit exceeding 0.93, well above the 0.70 threshold suggested by the American Statistical Association to indicate a positive trend [36]. The meta-analyses included nine primary studies [25,46,47,49,52,56,58,62,63] showing a very high level of overlap (CCA = 56%) [33] and high heterogeneity (from 27.6% to 84.0%). Five studies conducted subgroup analyses to explore heterogeneity (Table S7). Zhang et al. [41] performed three subgroup analyses, adjusting for invasive breast cancer and estrogen/progesterone receptor status. No significant differences in risk estimates were observed, though heterogeneity decreased across all subgroups. Wei et al. [43] analyzed factors such as study design, geographic location, menopausal status, and estrogen/progesterone receptor status, with the European population showing higher risk (RR = 1.16, 95% CI: 1.07–1.25). Gabet et al. [39] performed extensive subgroup analyses, noting reductions in both risk estimates and heterogeneity when considering major reproductive factors, residential history, hormone-receptor-positive cases, menopausal status, North American cohorts, and studies using home addresses for exposure assessment. Increased heterogeneity was reported for hormone-receptor-negative cases. Studies published since 2000, and those using modelling for exposure assessment, reported higher risk estimates alongside increased heterogeneity. Guo et al. [40] observed higher risk estimates in studies with follow-up periods shorter than 11 years and greater heterogeneity in

studies conducted in North America, and in those published before 2017. Arif et al. [45] reported a higher risk measure in European studies compared to those from the Americas with low heterogeneity (<0.01%). Yu et al. [42] did not perform subgroup analyses. The overall findings suggest a weak positive trend between PM₁₀ exposure and BC incidence, highlighting the necessity of further research.

3.3.3. PM_{2.5} Exposure and BC Incidence

The association between PM_{2.5} exposure and BC incidence is addressed in six of the seven selected meta-analyses. These studies reported relative risk (RR) estimates of 1.03 (95% CI: 0.93–1.13) [39], 1.01 (95% CI: 0.93–1.11) [40], 1.042 (95% CI: 0.987–1.108) [41], 1.02 (95% CI: 0.93–1.11) [42], 1.03 (95% CI: 0.99–1.06) [43], and 1.05 (95% CI: 0.98–1.12) [45]. All studies reported a positive association, with the lower bound of the confidence interval exceeding 0.93—well above the 0.70 threshold suggested by the American Statistical Association [36]. The meta-analyses included 18 primary studies [22,25,46–49,56,58–60,62–68] showing high overlap (CCA = 38%) and heterogeneity between 8.2% and 55.7%.

Five studies conducted subgroup analyses (Table S7). Zhang et al. [41] performed three subgroup analyses, examining invasive breast cancer and estrogen/progesterone receptor status. Their results indicated similar risk estimates across subgroups, with a reduction in heterogeneity. Wei et al. [43] conducted four subgroup analyses, considering study design, geographic region, menopausal status, and receptor type. While risk estimates remained stable, heterogeneity decreased in some cases, particularly among European populations and receptor status subgroups. Gabet et al. [39] reported lower risk estimates and reduced heterogeneity for reproductive factors, residential history, hormonal status, and North American populations. Both risk estimates and heterogeneity increased in studies conducted in Europe and in those using exposure modeling and home address data. Guo et al. [40] analyzed subgroups based on follow-up duration (greater or less than 11 years), geographic region, and publication period (before or after 2017). They found no significant changes in risk estimates but observed lower heterogeneity in studies with follow-up periods exceeding 11 years, those conducted in North America, and those published before 2017. Arif et al. [45] reported lower risk estimates in the Americas but higher heterogeneity compared to Europe. Yu et al. [42] did not perform any subgroup analyses. The overall findings suggest a positive moderate potential trend between PM_{2.5} exposure and BC incidence, pointing to the need for additional studies and more in-depth analysis in future work.

3.3.4. PM_{2.5} Exposure and BC Mortality

The association between PM_{2.5} exposure and BC mortality is addressed in only four of the seven selected studies [40–42,45]. The meta-analyses reported relative risk (RR) estimates of 1.20 (95% CI: 0.92–1.48) [40], 1.17 (95% CI: 1.05–1.30) [41], 1.18 (95% CI: 0.81–1.73) [42], and 1.17 (95% CI: 1.07–1.27) [45]. Two meta-analyses reported a positive association, with the lower bound of the confidence interval exceeding 1. In the other two studies, the lower bounds exceeded 0.81—higher than the threshold of 0.70 suggested by the American Statistical Association [36]. The meta-analyses included 10 primary studies, [49,63,69–76] with high overlap (CCA = 30%) and heterogeneity from 52.5% to 73.1%.

Subgroup analyses were conducted in only two studies [41,42] and details are reported in Table S7. Zhang et al. [41] found increased risk in cohort studies and higher exposure levels, though heterogeneity remained high (Table S4). Guo et al. [40] reported higher risk in studies with shorter follow-up periods and in developing countries, while heterogeneity increased with longer follow-up durations (>11 years), older publications, and in Asian

cohorts and research from developing countries. Yu et al. [42] and Arif et al. [45] did not conduct subgroup analyses.

Overall, findings suggest a positive modest trend between PM_{2.5} exposure and BC mortality, highlighting the necessity for additional studies.

3.3.5. PM₁₀ Exposure and BC Mortality

Only two meta-analyses [40,41] evaluated the relationship between PM₁₀ exposure and BC mortality, both including the same two primary studies [69,70], but assigning them different weights in the pooled relative risk estimates. The two meta-analyses reported an RR of 1.11 (95% CI: 1.02–1.21) with no heterogeneity ($I^2 = 0\%$) and an RR of 1.07 (95% CI: 0.93–1.20) with moderate heterogeneity ($I^2 = 56.4\%$), respectively. The association between PM₁₀ exposure and BC mortality was classified as “weak”. Guo et al. [40] performed subgroup analyses, noting higher risks with shorter follow-up periods and earlier publication dates, but found no major differences in other subgroups. Zhang et al. [41] did not perform subgroup analyses due to lack of heterogeneity. Given the limited number of studies, further investigation is needed.

4. Discussion

This umbrella review synthesizes evidence on the associations between exposure to nitrogen dioxide (NO₂), fine particulate matter (PM_{2.5}), and coarse particulate matter (PM₁₀) and the risk of breast cancer (BC) incidence and mortality in women. All included meta-analyses reported pooled relative risks (RRs) greater than 1 for each pollutant–outcome pair, with lower bounds of confidence intervals (CIs) exceeding 0.70, although with varying levels of heterogeneity. Among the pollutants examined, NO₂ showed sufficiently conclusive evidence of a weak positive association with BC incidence. For PM_{2.5} and PM₁₀, the findings suggest a positive trend for both incidence and mortality outcomes, with the trend being more pronounced for PM_{2.5} and incidence. The most vulnerable groups included premenopausal European women exposed to NO₂ and PM₁₀ and individuals in developed countries exposed to PM_{2.5}. A recent cohort study in the U.S. study [77] found an 8% increase in estrogen-receptor-positive BC per 10 µg/m³ increase in PM_{2.5}.

The carcinogenic effects of NO₂ may be driven by both direct and indirect mechanisms. NO₂ exposure has been associated with reduced DNA methylation, which can alter gene expression and contribute to genomic instability—a key factor in cancer progression [78]. Epigenomic studies have identified lower DNA methylation levels in blood samples years before BC diagnosis [79]. Additionally, as a major component of vehicle emissions and other high-temperature fossil fuel combustion, NO₂ can be a marker of traffic-related and industrial air pollution, including harmful substances like heavy metals, PAHs, and benzene, which have known genotoxic, mutagenic, and endocrine-disrupting effects [80–88], supporting the association between NO₂ exposure and breast cancer risk. Accordingly, the IARC classifies diesel emissions as carcinogenic (Group 1) and gasoline exhaust as possibly carcinogenic (Group 2B) [89]. The carcinogenic potential of particulate matter (PM) is less defined due to variations in composition and particle size, which depend on emission sources and regional air quality [90]. Certain PM components—black carbon, elemental carbon, and sulfates—are recognized as risk factors [65,91,92], along with airborne metals like mercury, cadmium, and lead, which are linked to increased postmenopausal BC risk [93–95].

Epidemiological and toxicological studies suggest two key mechanisms for PM-related cancer risk: (1) oxidative stress from reactive oxygen species (ROS), causing DNA damage [96,97] and (2) inflammation, with the release of cytokines like TNF-α and IL-1α that may promote tumor progression [98]. Furthermore, evidence indicates a dose–response relationship between PM exposure, elevated C-reactive protein (CRP) levels, and breast cancer risk [99]. Finally, BC mortality is influenced not only by environmental exposures

but also by healthcare-related factors such as delays in diagnosis, limited treatment access, and barriers to early detection.

Strengths and Limitations

This umbrella review presents several strengths. To our knowledge, a comprehensive synthesis evaluating the links between NO₂, PM_{2.5}, and PM₁₀ exposure and breast cancer risk has not yet been conducted. By providing a broad perspective on air pollution as a public health concern, this review highlights its potential role in breast cancer, the most commonly diagnosed malignancy worldwide, with incidence peaking between the ages of 60 and 70. A rigorous methodology was applied, including a structured literature search across two scientific databases, independent study selection and data extraction by two researchers, and quality assessment using AMSTAR 2.

However, some limitations exist. First, the included meta-analyses exhibited considerable heterogeneity, which weakens causal inferences. This variability stems from differences in geographic regions, time periods, data collection methods, and exposure assessments. While subgroup analyses attempted to address this issue, they remained limited by their reliance on general study characteristics. More refined stratifications—such as histologic and molecular subtypes, mammographic breast density, occupational history, and individual-level exposure data—were unavailable, as these factors were not considered in the original studies. This heterogeneity is further complicated by physiological changes that occur during critical periods of mammary tissue development and hormone signaling, such as puberty, pregnancy, and menopause. These stages, known as windows of susceptibility (WOS), may represent heightened vulnerability to chemical exposures. Assessing the impact of such exposures during these critical windows is essential for accurately evaluating their contributions to breast cancer risk [100–104]. Understanding pollutant impacts during these periods is essential for effective prevention [99–104]. A further limitation is the high overlap among the included meta-analyses, as reflected by the high Corrected Covered Area (CCA) index (30–66%). This overlap indicates that the synthesized evidence is not entirely independent. Although alternative methods like network meta-analysis or excluding redundant reviews are suggested [105], we included all of them due to their unique subgroup analyses. Still, this highlights the need for more primary studies.

Another concern pertains to how exposure is measured. Estimating individual exposure to air pollution is a key challenge in environmental health research, especially in regions with multiple pollution sources and uneven pollutant distribution. The accuracy of these estimates can vary depending on the method used—for instance, whether data come from air quality monitoring stations, land-use regression (LUR) models, or other modeling techniques. In addition, differences in the length and timing of exposure assessments between studies can introduce inconsistencies. These variations may lead to exposure misclassification, which can influence the reliability of the results and complicate comparisons across studies [106]. Many studies assessed only one pollutant, ignoring potential combined effects, and few analyzed PM composition, even though toxicity varies by component [107,108]. Furthermore, most meta-analyses assumed a linear relationship between air pollution and BC risk, without exploring potential nonlinear effects. Residual confounding is also a concern, as not all studies adjusted for socioeconomic, behavioral, or genetic factors. Lastly, publication bias may influence results. Although funnel plots and Egger's tests were used, three authors also applied trim-and-fill methods, excluding smaller studies to estimate adjusted summary effects based on larger ones [39,40,43].

5. Conclusions

This umbrella review highlights the growing evidence that air pollution, particularly exposure to nitrogen dioxide (NO₂), fine particulate matter (PM_{2.5}), and coarse particulate matter (PM₁₀), may increase the risk of both breast cancer incidence and mortality in women, though findings varied in terms of heterogeneity and publication bias. The most vulnerable groups were identified as premenopausal European women exposed to NO₂ and PM₁₀, as well as individuals in developed countries exposed to PM_{2.5}.

Given that breast cancer remains the most frequently diagnosed invasive malignancy and the second leading cause of cancer-related deaths among women worldwide, it is imperative that future environmental epidemiological research prioritize breast cancer as a critical health outcome when assessing the effects of air pollution.

In particular, further studies are needed to investigate the impact of multi-pollutant exposures and their combined effects on breast cancer risk. Additionally, identifying specific components of PM, such as black carbon, elemental carbon, and various metals, that may drive carcinogenic risks, is crucial. It is also important to assess breast cancer risk across different histologic and molecular subtypes, as the effects of environmental pollutants may vary in their carcinogenicity across different types of cancer. Furthermore, the existing evidence base is adequate to support breast cancer prevention strategies encompassing both individual lifestyle modifications and public health policies and environmental regulations designed to decrease NO₂, PM₁₀, and PM_{2.5} concentrations.

Supplementary Materials: The following supporting information can be downloaded at <https://www.mdpi.com/article/10.3390/environments12050153/s1>: Table S1: Search strategy in PubMed and Embase; Table S2: AMSTAR2 check list. Table S3: Excluded articles and reasons. Table S4: Confounders included in the study analysis. Table S5: Primary studies included in each review. Table S6: Sensitivity analysis and publication bias for NO₂ exposure. Table S7: Sensitivity analysis and publication bias for PM_{2.5} exposure. Table S8: Sensitivity analysis and publication bias for PM₁₀ exposure.

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Abbreviations

The following abbreviations are used in this manuscript:

BC Breast cancer

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