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Review Article

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The risk of type 2 diabetes and its association with NO_2 pollution (An update systematic review and meta-analysis of cohort studies)

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ABSTRACT

The purpose of this review was to examine the relationship between NO_2 exposure and the increased likelihood of Diabetes Mellitus (DM) or Type 2 Diabetes (T2D). We conducted the systematic review of cohort studies published between 2015-2025 by carrying out searches in several electronic databases. Nine cohort were included in this meta-analysis. The pooled HR was 1.09 (95% CI = 1.04-1.15) for the impact of NO_2 exposure towards the increasing likelihood of DM. In conclusion, the risk of T2D was increasing because the exposure of NO_2.

Keywords:
Air pollution; Diabetes mellitus; Nitrogen dioxide (NO_2); Type 2 diabetes

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Review

Type 2 diabetes (T2D) is one of the fastest-growing non-communicable diseases worldwide and represents a major cause of morbidity, premature mortality, and healthcare expenditure. The global prevalence continues to rise, with projections estimating approximately 784 million adults living with diabetes by 2045, more than 95% of whom have T2D [1, 2]. While obesity, unhealthy diet, and physical inactivity are established risk factors, increasing evidence suggests that environmental exposures also contribute to T2D development.

T2D is associated with high health-care and social expenses, premature death, and morbidity. T2D is growing globally, particularly in low- and middle-income nations, owing to epidemiologic changes such as nutrition shifts, urbanization, and sedentary lifestyles [3]. The increased trend in early-onset T2D: various risk factors contribute to the rising trend of early-onset T2D, highlighting the disease's complex nature [4].

A recent meta-analysis indicated an increased likelihood of T2D because of breathing in airborne contaminants such as Nitrogen dioxide (NO_2) or Particulate Matter (PM) [5]. Study by Burkart et al., (2022) also highlighting the relationship between T2D and air pollution which reported that air pollution has been accountable for about a fifth of the international burden of T2D and induced a greater attributable burden at the community level than either using cigarettes or inactivity [6].

Among traffic-related air pollutants, NO_2 has received increasing attention because it is a reliable indicator of traffic emissions and long-term urban air pollution exposure [7, 8]. Chronic NO_2 exposure has been associated with oxidative stress [9, 10], systemic inflammation [10–12], endothelial dysfunction [10, 12], and insulin resistance [10, 12], all of which are plausible biological mechanisms contributing to T2D development. Although particulate matter has been extensively investigated, accumulating epidemiological evidence also suggests that NO_2 may independently increase T2D risk.

Previous systematic reviews evaluated the association between overall air pollution and T2D but largely combined multiple pollutants ($PM_{2.5}$, PM_{10}, NO_2, ozone, etc.) without specifically synthesizing evidence for NO_2. Furthermore, several large prospective cohort studies published during the last decade were unavailable to earlier meta-analyses, potentially changing the pooled estimate.

An updated meta-analysis is essential to guide public health policy because NO_2 levels are rising in developing countries and stronger air quality restrictions are being discussed in high-income countries.

Unlike previous reviews that evaluated multiple pollutants collectively, this study generates pollutant-specific pooled estimates, examines heterogeneity across study characteristics, and provides updated evidence for environmental health policy and diabetes prevention. Therefore, this study provides an updated systematic review and meta-analysis focusing exclusively on long-term NO_2 exposure and T2D risk by incorporating studies published between 2015 and 2025.

Literature search strategy

This systematic review and meta-analysis followed a pre-registered protocol in the International Prospective Register of Systematic Reviews (PROSPERO CRD420251109667). The review was conducted in accordance with the Preferred Reporting Items for Systematic

Reviews and Meta-Analyses (PRISMA) 2020 guidelines.

A comprehensive literature search was performed in three electronic databases: PubMed, ScienceDirect, and Google Scholar. Studies published from January 1, 2015, to December 31, 2025, were considered. The search combined controlled vocabulary (where available) and free-text terms related to nitrogen dioxide exposure and type 2 diabetes. The search strategy was using keywords as follows: ("nitrogen dioxide" OR NO_2) AND ("type 2 diabetes" OR "type 2 diabetes mellitus" OR "diabetes mellitus") AND (cohort OR longitudinal OR prospective) AND ("hazard ratio" OR HR).

Inclusion and exclusion criteria

Studies were eligible if they met the following criteria: (1) employed a prospective or retrospective cohort design; (2) included participants from the general population without type 2 diabetes at baseline; (3) evaluated long-term NO_2 exposure as the primary exposure; (4) reported incident type 2 diabetes or diabetes mellitus as the outcome; (5) provided hazard ratios (HRs) with corresponding 95% confidence intervals or sufficient data for effect-size calculation; and (6) were published in peer-reviewed journals in English.

Studies were excluded if they (1) involved highly specific populations (e.g., patients with pre-existing chronic diseases or occupational cohorts); (2) used a cross-sectional, case-control, ecological, or experimental design; (3) were reviews, editorials, conference abstracts, letters, or dissertations; (4) lacked full-text availability or sufficient methodological information; or (5) did not report an extractable hazard ratio.

Study selection and quality assessment

Two reviewers (TH & ET) independently screened titles and abstracts for eligibility. Full texts of potentially eligible articles were subsequently assessed according to the predefined inclusion and exclusion criteria. Any disagreement was resolved through discussion, and when necessary, by consultation with a third reviewer.

Methodological quality was evaluated independently by two reviewers (AA & RF) using the Critical Appraisal Skills Programme (CASP) Cohort Study Checklist. The checklist assessed study validity, selection bias, exposure and outcome measurement, confounding, follow-up, and the overall reliability of the reported findings.

Data analysis

The analysis in this study was carried out using the JASP 0.19.3.0 software. The analysis was carried out on 271,030,978 respondents with no T2D in baseline. The results of the analysis are presented in the forest plot and the possibility of bias is presented in the funnel plot.

Results

Characteristics of studies

The literature search identified 614 records from three electronic databases: PubMed (n = 265), ScienceDirect (n = 34), and Google Scholar (n = 315). After duplicate removal, 50 unique records remained for title and abstract screening. Fifteen articles underwent full-text assessment; nine met the eligibility criteria and were included in the systematic review and meta-analysis. The study selection process followed the PRISMA 2020 guidelines and is presented in Fig. 1.

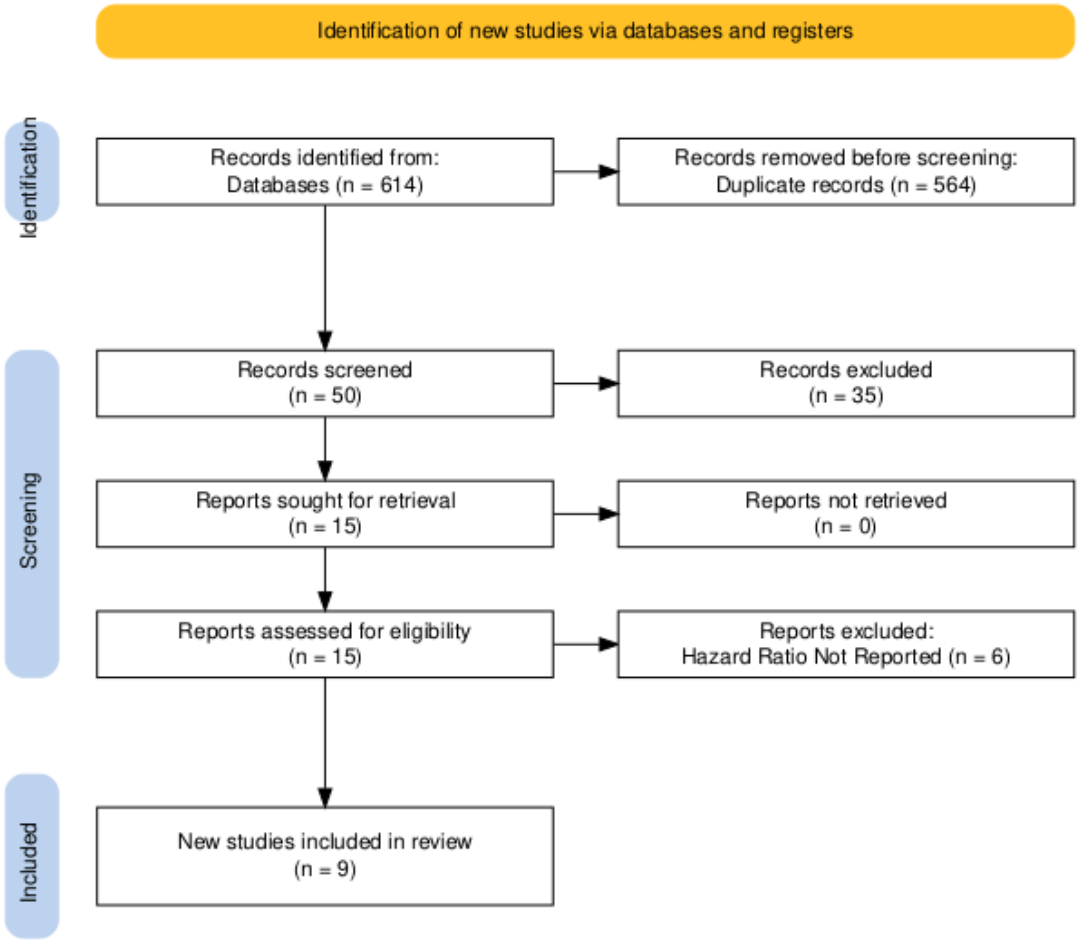

Fig. 1. PRISMA Diagram of the effect of NO_2 on T2D

The included studies comprised 9 cohort datasets conducted in the United Kingdom (n = 4), Canada (n = 2), and the United States, China, and Korea (n = 1 each). Sample sizes ranged from 14,667 to 4,800,000 participants. Owing to differences in adjustment for potential confounders across studies, the meta-analysis focused on the association between long-term NO_2 exposure and incident type 2 diabetes. The characteristics of the included studies are summarized in Table 3. All nine studies were appraised following guidelines of STROBE statement checklist for cohort study (see Table 1). For the results of appraisal see Table 2.

Table 1. Appraisal of studies used STROBE statement checklist for cohort study

Item No	Checklist
1	In the title or abstract, utilize a term that is often used to describe the study's design. Give a thorough and impartial description of the research and findings in the abstract.
2	Describe the investigation's scientific foundation and justification.
3	Indicate precise goals, along with any predetermined hypotheses.
4	Start the report by outlining the essential components of the study design.
5	Detail the environment, sites, and pertinent dates, as well as recruiting, encounter, monitoring, and data collecting periods.
6	Indicate the sources and methods utilized to choose participants, along with the eligibility requirements. Describe the follow-up techniques. For paired research, involve comparing criteria and the quantity of affected and unaffected..
7	All outcomes, exposures, predictors, possible confounders, and effect modifiers should be precisely defined. If relevant, provide diagnostic criteria.
8	Provide data sources and specifics on the assessment (measurement) techniques for each variable of interest. If there are multiple groups, describe how the assessment procedures are comparable.
9	Explain any initiatives to deal with any bias sources.
10	Explain how the study size was arrived at
11	Explain how the quantitative variables were handled in the analyses. If applicable, describe the chosen classifications and the reasoning behind them.
12	Explain every statistical technique, including confounding control techniques. Explain any techniques used to look at interactions and subgroups. Discuss the steps taken to address missing data. If appropriate, describe how the loss to follow-up was handled.

Table 1. Continued

Item No	Checklist
13	List the number of participants at each step of the study, such as those who may be eligible, were evaluated for eligibility, were found to be eligible, were included in the study, finished the follow-up, and were analyzed. At each level, provide an explanation for your lack of engagement. Think about using a flow diagram.
14	Describe the study participants' demographic, clinical, and social features as well as their exposures and any confounders. Indicate how many participants have missing data for each variable of interest. Provide a summary of the follow-up time, including the average and total amount.
15	Summary measurements or result event counts over time should be reported.
16	Give estimates and their accuracy (e.g., 95% confidence interval) for both unadjusted and, if applicable, confounder-adjusted estimates. Indicate which confounders were considered and why. When categorizing continuous variables, report the boundaries of each category. If applicable, think about converting relative risk estimates into absolute risk for a significant amount of time.
17	Discuss additional investigations that were conducted, such as sensitivity analyses and subgroup and interaction analysis.
18	Outline the main findings in relation to the study's goals.
19	Discuss about the study's limitations while addressing potential sources of bias or imprecision. Talk about the extent and direction of any possible prejudice.
20	Give a preliminary overall interpretation of the results, including the objectives, limitations, range of analysis, results from related studies, and other relevant information.
21	Address about the study's external validity and generalizability.

Table 2. Critical appraisal for studies on the risk of T2D and its association with NO_2 pollution using strobe strobe statement checklist for cohort study

Referene	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18	19	20	21	22
[13]	✓	✓	x	x	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
[14]	✓	✓	x	x	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
[15]	✓	✓	x	x	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
[16]	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
[17]	✓	✓	x	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
[18]	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
[19]	✓	✓	x	x	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
[20]	✓	✓	x	x	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓
[21]	✓	✓	x	x	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓	✓

Meta-Analysis

Fig. 2 presents the forest plot of the pooled analysis. A Bayesian random-effects meta-analysis was performed to account for between-study heterogeneity. The pooled hazard ratio (HR) was 1.09 (95% CI: 1.04–1.15), indicating a statistically significant positive association between long-term NO_2 exposure and the risk of type 2 diabetes. Individual study estimates were consistently above the null value (HR > 1.0), although the magnitude of the association varied across studies. Between-study heterogeneity was addressed using the random-effects model.

Publication bias

Egger's test indicated significant funnel plot asymmetry ($z = 3.793$, $p < 0.001$), with an estimated bias coefficient of 1.037 (95% CI: 1.008–1.066) (Fig. 3). These findings suggest the presence of small-study effects; however, funnel plot asymmetry does not necessarily indicate publication bias. Alternative explanations, including between-study heterogeneity, methodological differences, or chance, should also be considered. Therefore, the pooled effect estimate should be interpreted with caution, as the possibility of publication bias or other sources of asymmetry cannot be excluded.

Table 3. Characteristics of studies used in current systematic review

Authors (year)	Sample size and population	Cohort databases	Study period	Assessment of NO_2 instrument	Findings	Adjustment factors
[13]	183,290 participants aged 30 to 100 years in Toronto, Canada	The Ontario Population Health and Environment Cohort (ONPHEC)	1 April 1996 until 31 December 2012	Land use regression model	There is a relationship between NO_2 and incident of diabetes (HR 1.06; CI 95% 1.05–1.07)	Ultrafine particles and $PM_{2.5}$.
[14]	78, 230 participants aged 40-70 years in United Kingdom	UK Biobank	2006–2021	Land use regression model	NO_2 significantly increased the T2D risk (HR 1.11; CI 95% 1.04–1.18)	Gender, age, ethnicity, employment status, education level, average total household income, physical activity, smoking status, alcohol status, polygenic risk score for T2D, body mass index, and waist circumstance.
[15]	77, 278 participants aged 40-70 years in United Kingdom	UK Biobank	2006–2021	Land use regression model	One interquartile range increase of NO_2 raised the T2D risk (HR 1.14; CI	Ethnicity, gender, age, residential area, education level, employment status, average total household

Table 3. Continued

Authors (year)	Sample size and population	Cohort databases	Study period	Assessment of NO_2 instrument	Findings	Adjustment factors
					95% 1.06–1.21)	income, daily dietary energy intake, smoking status, alcohol status, weekly physical activity, systolic blood pressure, diastolic blood pressure, body mass index, , polygenic risk score for T2D, and dietary approaches to stop hypertension diet (DASH) score.
[16]	502,412 aged 40–69 years participants aged over 40 in China	UK Biobank	2009-2020	Land use regression models	The exposure to NO_2 were significantly associated with an elevated risk of incident T2D (HR 1.07; CI 95% 1.02–1.11)	Polygenic risk score for type 2 diabetes, ethnicity, education, economic status, Townsend deprivation index, assessment centre, length of time at residence, smoking status, alcohol intake frequency,

Table 3. Continued

Authors (year)	Sample size and population	Cohort databases	Study period	Assessment of NO_2 instrument	Findings	Adjustment factors
						sedentary time, sleep duration, physical activity, and healthy diet score.
[17]	156,490 participants aged 37–73 in United Kingdom	UK Biobank	2006-March 31, 2021	Land use regression models	There was a association between NO_2 and diabetes mellitus (HR 1.04; CI 95% 1.02–1.05)	Gender, ethnicity, age, level of education, average household income, and lifestyle variables such the waist-to-hip ratio, physical activity, smoking history, alcohol use, supplements for vitamins, protein consumption, poly-unsaturated fat consumption, total sugar consumption, and dietary fiber intake.
[18]	4,800,000 participants aged ≥35	Ontario Population Health and Environment	January 1, 2001-December 31, 2015	Land use regression model	There is a relationship between NO_2 and incident of	Covariables include age, sex, SES, geography, and access to

Table 3. Continued

Authors (year)	Sample size and population	Cohort databases	Study period	Assessment of NO_2 instrument	Findings	Adjustment factors
	Ontario, Canada	Cohort (ONPHEC)			diabetes (HR 1.042; CI 95% 1.033–1.051)	healthcare. Every model is stratified by domicile in the Greater Toronto Area.
[19]	14,667 participants aged 40-69 in Korea	Cardiovascular Disease Association Study (CAVAS)	2005-2016	Community multiscale air quality models	NO_2 concentrations were found to be associated with incident diabetes (HR 1.29; CI 95% 1.12–1.49)	Gender, every month household income, level of education, alcohol and smoking habits, regular exercise, body mass index, and Mini Dietary Assessment index score.
[20]	264,859,458 participants aged 65 years and older in the contiguous United States	National U.S. cohort (Medicare Chronic Conditions Warehouse)	2000-2016	Highly spatio-temporally resolved prediction models (neural network, random forest, and gradient boosting)	The HR for first diabetes occurrence was 1.055 (CI 95% 1.05-1.06) for 5 ppb increase in NO_2.	Medicaid eligibility, age, sex, race, date of death, median household income, population density, percentage of home renters, percentage of residents without a high school degree, and

Table 3. Continued

Authors (year)	Sample size and population	Cohort databases	Study period	Assessment of NO_2 instrument	Findings	Adjustment factors
						percentage of the population self-identified as Black and Hispanic.
[21]	359,153 participants aged 40–69 in United Kingdom	UK Biobank	April 2007 -2018	Land use Regression models	Compared with low levels of air pollution, the HRs for risk of T2D for high levels of NO_2 were 1.19 (CI 95% 1.14-1.24)	Race, education, household income, smoking and alcohol use, body mass index, consumption of fruits and vegetables, family history of diabetes, hypertension, heart disease, depression, cancer, and physical activity.

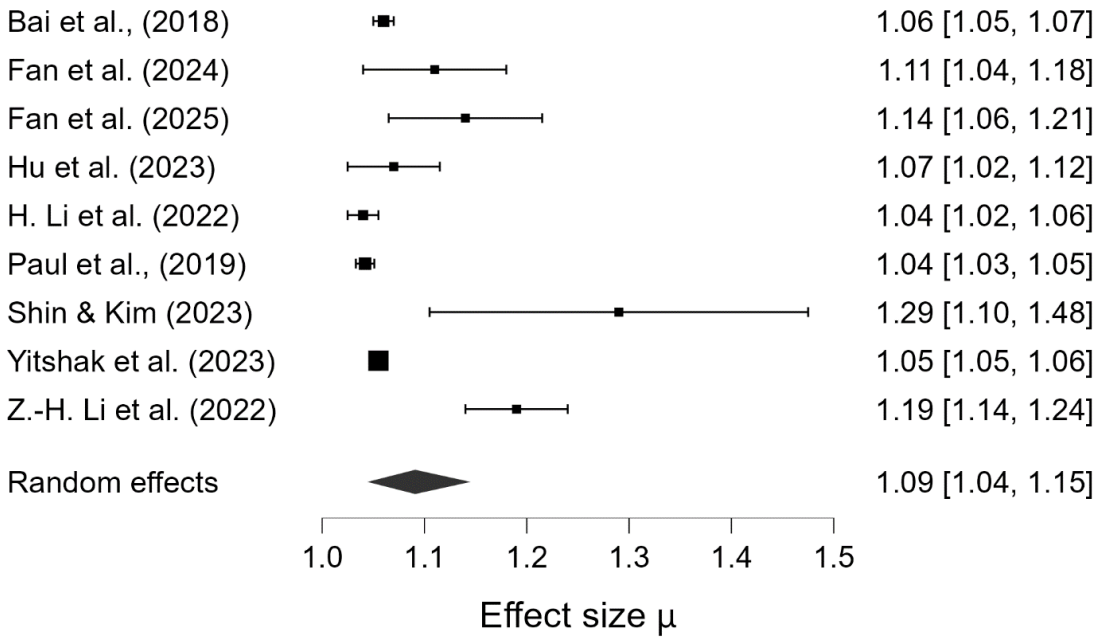

Fig. 2. Forest Plot of the effect of NO_2 on T2D

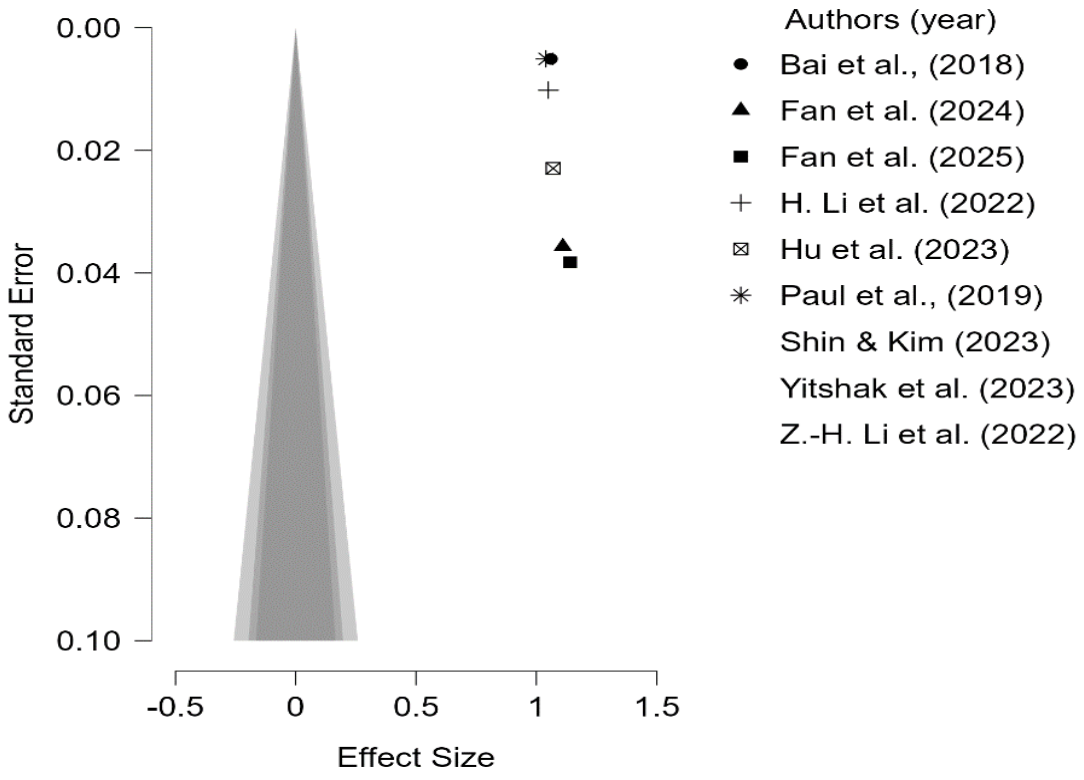

Fig. 3. Funnel Plot of the effect of NO_2 on T2D

Discussion

This meta-analysis found that higher long-term NO_2 exposure was associated with an increased risk of developing T2D.

This result is in line with an earlier comprehensive review and meta-analysis that reported a positive association between exposure to NO_2 and a higher risk of diabetes mellitus [9]. Our findings also are consistent with those of Parasin et al. (2025), who also reported a positive association between long-term NO_2 exposure and diabetes risk [9]. However, the pooled effect estimate in the present review was derived exclusively from cohort studies, thereby reducing the influence of study designs that are more susceptible to selection and recall bias. This methodological difference may provide greater confidence in the temporal relationship between NO_2 exposure and incident T2D, although causal inference remains limited because all included studies were observational.

Previous meta-analysis of cohort also stated the same conclusion to this current study [22]. Earlier meta-analyses used risk ratio or odd ratio as their primary effect size. In contrast, our analysis incorporates hazard ratio derived from more recent statistical methodologies. This shift aligns with updated epidemiological guidelines for exposure-outcome assessments, which may improve comparability with recent epidemiological studies. Nevertheless, direct comparison with previous pooled estimates should be interpreted cautiously because differences in effect measures, exposure assessment, confounder adjustment, and study populations may contribute to variation in the reported associations.

Another important difference is the inclusion of cohort studies published between 2015 and 2025. Several of these recent studies included larger populations, longer follow-up periods, and improved exposure assessment using high-resolution spatiotemporal models. Consequently, the present meta-analysis reflects the most current epidemiological evidence and may provide a more precise estimate of the association than earlier reviews.

According to Rajagopalan et al., (2012) and Brook et al., (2010), air pollution and diabetes are likely related because of oxidative stress and/or systemic inflammation brought on by particles and/or NO_2, which in turn affects metabolic pathways [23, 24].

As a free radical, NO_2 can weaken tissue antioxidant defenses, which can lead to inflammation and damage, as demonstrated by several in vitro test systems. As exposed to 26 230 µg/m3 (13.95 ppm) of nitrogen dioxide, human blood plasma quickly lost ascorbic acid, uric acid, and protein thiol groups. Alpha-tocopherol (vitamin E) and lipid peroxidation were also reduced [25].

Animal studies have supported the plausibility of diabetes linkages by demonstrating that controlled exposure to fine particles enhances insulin resistance and other physiological markers crucial for the development of diabetes [26, 27].

This study has several limitations. First, subgroup analyses according to participant characteristics, such as sex, age, or geographic region, could not be performed because these data were not consistently reported across

the included studies. Consequently, potential effect modification by demographic or regional factors could not be evaluated. Second, variation in exposure assessment methods, adjustment for confounding variables, and NO_2 exposure metrics across studies may have contributed to between-study heterogeneity. Finally, evidence of funnel plot asymmetry suggests that small-study effects cannot be ruled out, and the pooled estimates should therefore be interpreted with caution.

Despite these limitations, restricting the meta-analysis to cohort studies strengthens the temporal inference between long-term NO_2 exposure and incident diabetes and provides robust evidence to inform environmental health research and air quality policy.

Conclusion

In conclusion, this updated systematic review and meta-analysis suggests that long-term NO_2 exposure is associated with an increased risk of type 2 diabetes in adults. However, this association should be interpreted cautiously because evidence of funnel plot asymmetry and small-study effects was observed, which may have influenced the pooled effect estimate. Therefore, although the findings support a potential relationship between NO_2 exposure and type 2 diabetes, they do not establish causality. Further large-scale, high-quality prospective cohort studies with standardized exposure assessment are needed to confirm the magnitude and consistency of this association. From a public health perspective, reducing long-term NO_2 exposure may contribute to diabetes prevention as part of broader strategies targeting established risk factors.

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Competing interests

The study's authors affirm that they have no competing interests.

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Ethical considerations

Ethical issues (Including plagiarism, Informed Consent, misconduct, data fabrication and/or falsification, double publication and/or submission, redundancy, etc) have been completely observed by the authors.

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