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Outdoor air pollution exposure and the risk of type 2 diabetes mellitus: A systematic umbrella review and meta-analysis

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ABSTRACT

The association between different air pollutants and Type 2 Diabetes Mellitus (T2DM) is a growing topic of interest in public health research. This umbrella review and meta-analysis aimed to consolidate current literature on the association between various outdoor air pollutants and T2DM. Subgroups and dose-response relationships were also analyzed to further quantify the association, especially by the factors such as the type of pollutants, duration of exposure, and geographical variation, etc. A thorough literature search of three databases revealed a total of 71 records for umbrella review and 1524 records for meta-analysis where 8 studies were included in the final review of umbrella review and 46 studies for meta-analysis. The evaluation of the study's quality in umbrella review and meta-analysis were conducted using the AMSTAR 2 criteria and the Newcastle-Ottawa Scale (NOS), respectively. Exposure to Particulate Matter (PM) 2.5, PM10, Nitrogen dioxides (NO2) and Ozone (O3) were significantly associated with the risk of T2DM [OR = 1.12 (95% Confidence Interval (CI): 1.09, 1.15), 1.12 (95% CI: 1.06, 1.18), 1.09 (95%CI: 1.07, 1.12), 1.05 (95%CI: 1.03, 1.08), respectively] and subgroup analysis further revealed that PM2.5, PM10, and NO2 associations were confounded by factors such as ages, study design, regions of exposure and air pollution concentration levels. Lastly, only exposure to PM10 had a significant dose-response relationship with the risk of T2DM (p-value = 0.000). These findings further emphasized the need for standardized methods in conducting air pollution research and additional research on other air pollutants to further explore the relationships between these air pollutants and T2DM.

1. Introduction

One of the biggest threats to global health is air pollution which has a major impact on health among people. There are many different sources of air pollution, including the burning of fossil fuels, automobile emissions, and industrial facilities. Regardless of the duration of exposure, air pollution can have a highly negative impact on human health. Chronic exposure to air pollution can cause cancer, neurological disorders,

Abbreviations: T2DM, Type 2 Diabetes Mellitus; PM2.5, particulate matter with a diameter less than 2.5 μm; PM10, particulate matter with a diameter less than 10μm; NO2, Nitrogen dioxide; NOx, Nitrogen oxides; O3, Ozone; OR, odd ratios; HR, hazard ratio; RRs, risk ratios; 95% CI, 95% confidence intervals.

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cardiovascular diseases, respiratory conditions, and even early death. (National Institute of Environmental Health Sciences, 2001; Manisalidis et al., 2020).

Additionally, the increasing incidence of T2DM, a chronic health condition marked by elevated blood glucose levels and insulin resistance, in regions exposed to high levels of air pollution is becoming a significant health concern in many countries. (International Diabetes Federation, 2021; Bowe et al., 2018). Recent evidence implied that prolonged exposure to air pollution could partially lead to the onset of T2DM later in life. It is believed that the presence of particulate matter such as PM2.5 and other hazardous air pollutants in the blood steam may lead to the inducement of systemic inflammation and the interference with insulin signaling, which resulting in poor glucose metabolism, the development of insulin resistance and consequently the onset of diabetes later in life (Rajagopalan and Brook, 2012; Bowe et al., 2018).

An increasing amount of research indicated that air pollution was positively associated with a higher risk of type 2 diabetes. The importance of air pollutants like PM2.5, PM10 and NO2 were found to be strongly connected to T2DM prevalence, whereas PM2.5 was found to be substantially linked to T2DM incidence. Additionally, it was discovered that prolonged exposure to outdoor air pollution was substantially linked to a higher risk of diabetes complications and death (Eze et al., 2015a; Liu et al., 2019a; Park et al., 2015; Wu et al., 2022; Yang et al., 2020). Nevertheless, despite the convincing data supporting the association between T2DM and air pollution, there are difficulties that need to be addressed. Research studies frequently encountered challenges related to confounding variables and methodological variances, which had influenced the magnitude of the observed relationships and led to bias (Eze et al., 2015a; Liu et al., 2019a; Yang et al., 2020). Several unresolved issues remained in the existing literature. Numerous meta-analyses and systematic reviews found a strong correlation between the incidence of type 2 diabetes and air pollutants such NO2, PM2.5, and PM10 (Eze et al., 2015a; Liu et al., 2019a; Yang et al., 2020). However, these studies often vary in their methodological quality, leading to inconsistent results (Eze et al., 2015a; He et al., 2017). Key issues included the differences in study populations, the air pollution measurement techniques, and the adjustment for confounding variables such socioeconomic status and age, and pre-existing health conditions (Balti et al., 2014; Yang et al., 2020). Furthermore, the types of pollutants studied and the exposure durations reported were not consistent across the literature, limiting the comparability of these results (Wang et al., 2014a; Chen et al., 2022a). The significant variation in the results, which made it challenging to derive reliable conclusions from these investigations, was another significant issue (Liu et al., 2019a; Yang et al., 2020). Some studies reported stronger associations in certain populations or regions, while others failed to find significant effects from the same regions (Eze et al., 2015a; Janghorbani et al., 2014). This variation may be attributed to differences in study design, such as cohort versus cross-sectional studies, or discrepancies in T2DM diagnosis and reports across studies (He et al., 2017; Wang et al., 2014a). These discrepancies made it clear that a more thorough and organized synthesis of the existing data was required.

An umbrella review provided a solution to these challenges by synthesizing evidence across multiple systematic reviews and meta-analyses. It enabled a higher-level evaluation of existing literature, consolidating findings while accounting for variations in study quality and methodology. By critically assessing the methodological rigor and consistency across studies, an umbrella review could help address the heterogeneity in findings and identified gaps that require further investigation. This approach also facilitated the detection of overarching trends and more reliable conclusions, offering a better understanding of the relationship of the relationship between outdoor air pollution and T2DM.

Therefore, this research focused on the correlation between exposure to air pollutants and the probability for developing type 2 diabetes in the general population through synthesizing and analyzing the current body of literature. This will be achieved through two separate complementary methods: an umbrella review of published meta-analyses and a systematic review and meta-analysis of published primary studies. By combining these approaches, the study not only clarified the existing evidence but also highlighted areas where further research is needed to better understand the relationship between outdoor air pollution and T2DM.

2. Material and methods

This research focused on the association between outdoor air pollution exposure and the risk of T2DM. The study consisted of two parts: 1) a systematic umbrella review of published meta-analysis studies on the effect of main air pollutants (i.e., $PM_{2.5}$, PM_{10}, and NO_2) on T2DM and 2) a systematic review and meta-analysis of published primary studies to obtain up-to-date results with more power from a larger sample size.

Our study registered the protocol at PROSPERO (registration no. CRD42023452605) and formatted in compliance with the Synthesis Without Meta-analysis in Systematic Reviews: Reporting Guideline (Campbell et al., 2020) and the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 statement (Page et al., 2021).

2.1. Search strategy

The literature search was conducted from the start through September 30, 2023 with three electronic databases: PubMed, ScienceDirect, and Google Scholar. The literature search has no language restrictions. The following search terms were used for both an umbrella review's and a systematic review & meta-analysis's literature search: 'air pollution', 'particulate matter', 'particles', 'PM2.5', 'PM10', 'black carbon', 'NO2', 'NOx', 'O3', 'diabetes mellitus', 'diabetes', 'type 2 diabetes', 'glucose', 'insulin resistance', 'impaired glucose tolerance', and 'glycated haemoglobin (HbA1c)'. For an umbrella review, the term 'meta-analysis' was also included. Furthermore, a manual search was conducted of the included articles' reference lists and relevant articles.

2.2. Study selection and eligibility criteria

The potential studies were screened for duplication and all duplication studies were removed. Then, the title and the abstract of the remaining studies were reviewed for eligibility. Full-text article reading was also performed in cases the title or the abstract of the potential studies did not provide clear information. Studies were included in an umbrella review and a systematic review & meta-analysis if the studies met the following criteria:

2.2.1. Umbrella review

Studies were incorporated if the published research was: 1) a systematic review with meta-analysis, 2) meta-analysis studies that showed pooled results of selected effect estimates [i.e., Risk Ratios (RRs), Odd Ratios (ORs), or Hazard Ratios (HRs)] with 95% confidence intervals (95% CI), and 3) had a comparison of exposure (exposed or not exposed to outdoor air pollution) with an outcome (T2DM incidence, T2DM prevalence, or risk of T2DM). Studies were excluded if the studies focused on indoor air pollution or conducted in type 1 diabetes mellitus patients, diabetes during pregnancy, or diabetes mortality.

2.2.2. Systematic review and meta-analysis

Studies have been included if the published research was: 1) focusing on the association between T2DM cases (T2DM incidence, T2DM prevalence, or risk of T2DM) and an outdoor air pollution exposure, 2) were cross-sectional, case-control, or cohort studies, and 3) providing the following effect estimates: Relative risks (RRs), Odd ratios (ORs), or

Hazard ratios (HRs), with their corresponding 95% confidence intervals (95% CI). Studies were excluded if the studies focused on indoor air pollution, type 1 diabetes mellitus, gestational diabetes, or diabetes mortality. Review, case reports, letters, registration of trials, or conference abstracts without full-text articles were also excluded from the study.

2.3. Data extraction and quality assessment

Two researchers (NP and NS) separately extracted the data, while a third researcher (SS) decided on the conflicts.

2.3.1. Umbrella review

The eligible studies' information was extracted according to the Joanna Briggs Institute Reviewers' Manual 2014 edition (Aromataris et al., 2014) guideline and A Measurement Tool to Assess Systematic Reviews (AMSTAR) 2 was used to evaluate the studies' methodological quality (Shea et al., 2017). The studies ranged from High to Critically Low.

2.3.2. Systematic review and meta-analysis

The following information was extracted from the eligible studies: the authors, publication year, country, study design, participants' baseline characteristics, study design, air pollutant exposure assessment and estimates, time of exposure/follow-up, DM case diagnosis/definition, and results/main findings.

The studies' methodological quality was assessed using The Newcastle Ottawa Scale (NOS). The scale was based on three criteria [Selection of the study, Comparability of the groups, and Ascertainment of outcome/exposure] with a maximum score of 9 for cohort studies and 10 for cross-sectional studies (Wells et al., 2014). Only studies with a score more than 5 (fair or good quality) were included in this study's final analysis.

2.4. Data synthesis and statistical analysis

2.4.1. Umbrella review

The eligible studies were reviewed in full text to provide a summary of the eligible studies' results. The overall quality of evidence of eligible studies were assessed using the Grading of Recommendations, Assessment, Development, and Evaluations (GRADE) criteria (Langendam et al., 2013). For this study, the criteria was based on one outcome, the diabetes cases among all groups. The strength of evidence [High, Moderate, Low, and Very low] was graded using GRADEpro software version 3.6.1 (McMaster University, 2021) The process was assessed by two independent researchers (NP and NS) and the conflicts were adjudicated by a third researcher (SS). No further analysis was conducted to synthesize the results of these studies.

2.4.2. Systematic review and meta-analysis

The fully adjusted pooled/overall ORs with 95% CI from the primary studies were used to assess the effect of outdoor air pollution exposure on the T2DM risk. If the primary studies reported the effect estimates other than ORs (i.e., HRs and RRs), the effect estimates were converted to ORs for consistency using the following equations (Saokaew et al., 2021):

$$RR = \frac{OR}{(1-r)+(r*OR)} \text{ and } HR = \frac{ln(1-RR*r))}{ln(1-r)}$$

where "r" is the incidence rate of the control group, which was the diabetes cases among the population that was not exposed to outdoor air pollution.

To address the possible differences in included primary studies (e.g., participant's ages, study design, exposure assessment, DM cases diagnosis, etc.), the random-effects model method, which based on the inverse-variance approach, was applied to synthesize the pooled estimates from these primary studies. The z-test with an alpha value of 0.05 was used to test the pooled estimates' significance (Deeks et al., 2023).

The between-study heterogeneity was also performed using Cochran Q statistics and I^2 test, with an alpha value of 0.05. A funnel plot and the Egger test were conducted to test the publication bias. If it was necessary, the trim-and-fill method was also used to further adjust the overall effect estimates (Sterne et al., 2011; Rothstein et al., 2005; Shi and Lin, 2019).

Sensitivity and subgroup analyses were performed using pooling models (random effects model vs. fixed effects model). The study design, area of exposure, ages of participants, DM cases diagnosis, air pollution concentration levels, and the duration of exposures were also evaluated in the subgroup analysis. The subgroup analyses were only conducted for outdoor air pollutants exposure that had included primary studies of more than 10 studies. All analyses were performed using STATA (Stata Corp, 2015) and a p-value of less than 0.05 was considered as statistically significant.

2.4.2.1. Dose-response analysis. The relationship between outdoor air pollutants' concentration levels (i.e., PM2.5, PM10, and NO2) and the risk of T2DM was further explored using a statistical approach called "restricted cubic splines". This method is commonly used for exploring complex, non-linear associations that are often observed in environmental health studies (Harrell, 2010).

Basically, the adjusted pooled OR estimates and the 95% CIs from the primary studies were first converted into its natural logarithm form to be used in the analysis. This was crucial because it helped normalize and reduce the potential skewness in the analysis. Then the weight was assigned to each study converting from its standard error. This weight assignment helped ensuring that the studies with more precise estimates had a greater influence on the overall analysis. Three knots were selected and assigned for the goodness of the model fit (Harrell, 2010; Lusa and Ahlin, 2020). Finally, the weighted linear regression model with the generated spline terms was computed for the analysis with log ORs as a dependent variable and the average air pollutants' concentration levels from the primary studies as an independent variable.

3. Results

3.1. Umbrella review

A total of 71 publications were identified across three databases. After the screening process, 61 publications were excluded due to being duplicated or irrelevant to the study. A total of ten eligible publications were included for full-text reading. Only eight publications met this study's inclusion criteria (Balti et al., 2014; Chen et al., 2022a,b; Eze et al., 2015a,b; He et al., 2017; Janghorbani et al., 2014; Liu et al., 2019a,b; Wang et al., 2014; Yang et al., 2020) and were included in the final analysis (Fig. 1). As a result, a test for publication bias in this umbrella review section was not performed due to small numbers of studies (had less than ten studies).

3.1.1. Characteristics of the included studies

The included studies consisted of seven systematic review and meta-analyses (SRMA) and one meta-analysis (MA) of cohort studies. The air pollutants exposure examined in these studies included PM2.5, PM10, NO2, O3, Sulfur dioxide (SO2), and Sulfate (SO4). The outcomes were reported as either DM incidence or prevalence or a combination of both, with some also reported T2DM deaths in the study. Numbers of primary studies included in these SRMAs, and MA ranged between 8 and 30 studies, with Chen et al. (2022a,b) study having the lowest primary studies included in their analysis and Liu et al., 2019a,b study having the highest primary studies included. Due to the small numbers of primary studies included, one study did convert some of its air pollutant estimates into another air pollutant estimate for consistency (i.e., PM10 to PM2.5 estimates and NOx to NO2 estimates) [Balti et al., 2014].

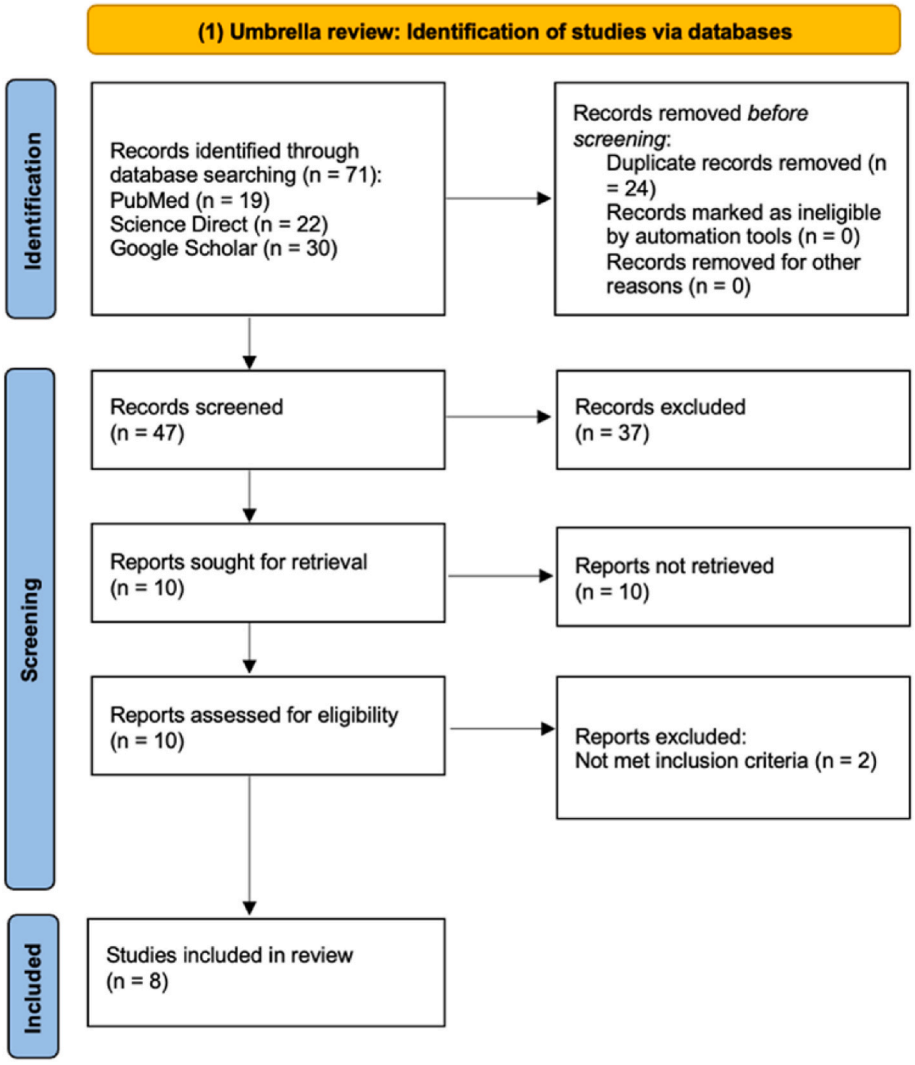

Fig. 1. PRISMA flow diagram of the umbrella review.

Based on AMSTAR 2 criteria, 2 of the included studies were rated high (Balti et al., 2014 & Yang et al., 2020), 5 were rated moderate (Eze et al., 2015a,b; He et al., 2017; Janghorbani et al., 2014; Liu et al., 2019a,b; Wang et al., 2014), and one was rated critically low (Chen et al., 2022a,b). Due to this assessment, most of the studies except one was rated as either High or Moderate in the GRADE ratings, suggesting a good level of confidence in their findings and greatly enhanced the credibility and dependability of their conclusions. Overall, most of the studies had covered the important factors that were adhered to the AMSTAR 2 criteria such as having a clearly defined PICO (Population, Intervention, Comparator, Outcome) structure, comprehensive literature searches, a suitable procedure to assess the risk of bias (RoB), and the mentions of heterogeneity. But some studies were lacking in certain areas such as the lack of sufficient publication bias test and the mention of potential conflicts of interest and funding sources, which are essential in guaranteeing the impartial and transparent of research.

The summarized characteristics of the included studies and the completed details of AMSTAR 2 quality assessment are presented in Table 1 and Supplementary Table S1, respectively.

3.1.2. p.m.2.5

All included studies reported the association between PM2.5 exposure and the risk of T2DM. All of the included studies except two studies (Janghorbani et al., 2014 & Wang et al., 2014) had reported that PM2.5 exposure was significantly associated with T2DM incidence with HRs of either 1.10 or 1.11 [Balti et al. (2014), HR = 1.11 (95% CI: 1.02, 1.20); Yang et al. (2020), HR = 1.10 (95% CI: 1.05, 1.16); and Liu et al. (2019a, b), HR = 1.10 (95%CI: 1.06, 1.14)] and RRs of 1.11 (95%CI: 1.01, 1.21) or 1.14 (95%CI: 1.04, 1.25) [Eze et al., 2015a,b & He et al., 2017, respectively].

For T2DM prevalence, exposure to PM2.5 was also found to be substantially linked to type 2 diabetes prevalence. Two studies (Liu et al., 2019a,b & Chen et al., 2022a,b] both reported an OR of 1.10 (with 95%CI of 1.03, 1.16 and 1.06, 1.15, respectively). One study (Balti et al., 2014) reported an OR of 1.62 (95%CI: 1.20, 2.04), which was originally a PM10 estimate pulled from a primary study that was converted into a PM2.5 estimate. Based on these findings, exposure to PM2.5 was significantly associated with the risk of developing T2DM.

3.1.3. p.m.10

Out of eight included studies, only four studies reported an association between PM10 exposure and T2DM (Yang et al., 2020; Liu et al., 2019a,b; Janghorbani et al., 2014; Wang et al., 2014). Two studies reported both HR and OR estimates (Yang et al., 2020 & Liu et al., 2019a, b) and two studies reported only RR estimates (Janghorbani et al., 2014 & Wang et al., 2014). Overall, every study except one (Janghorbani et al., 2014) has found a significant association between PM10 and T2DM.

Table 1
Characteristics of included studies in the umbrella review of outdoor air pollution exposure and the risk of T2DM.

Air pollutants	Authors (Year)	AMSTAR 2	Study Design	Objective(s)/Aim of the Study	No. of Primary Studies Included	Results [Effect Estimates (95% CI)]	Heterogeneity (I^2 value; *p*-value)	GRADE Rating
PM2.5	Balti et al. (2014)	High	SRMA	To evaluate and measure the relationship between exposure to major air contaminants and the risk of type 2 diabetes	10	**HR*** = 1.11 (95% CI: 1.03, 1.20) **OR*** = 1.62 (95% CI: 1.25, 2.10)	I^2 = 0.0%; P = 0.827 N/A	High
	Yang et al. (2020)	High	SRMA	To measure the relationship between air pollution and type 2 diabetes to determine whether those with diabetes are more susceptible to air pollution	86 [21 on T2DM incidence/25 on T2DM prevalence]	**HR** = 1.10 (95% CI: 1.04, 1.17) **OR** = 1.08 (95% CI: 1.04, 1.12)	I^2 = 74.4%; P = 0.000 I^2 = 84.3%; P = 0.000	High
	Janghorbani et al. (2014)	Moderate	SRMA	To determine the health impacts of air pollution exposure on the risk of diabetes and whether the associations differed depending on the kind of pollutant, study design, and geographic location	17 [11 on risk of diabetes-related morbidity (i.e., T2DM Incidence and Prevalence]	**RR** = 1.05 (95% CI: 0.99, 1.10)	I^2 = 43%; P = 0.151	Moderate
	Wang et al. (2014)	Moderate	SRMA	To evaluate how long-term exposure to air pollution affects the risk of type 2 diabetes.	10	**RR** = 1.39 (95% CI: 1.14, 1.68)	I^2 = 86.3%; P = 0.001	Moderate
	Eze et al. (2015)	Moderate	SRMA	To summarize the findings of research on the connection between air pollution and type 2 diabetes and to conduct a thorough review of epidemiological data about this relationship.	13 [7 on T2DM]	**RR** = 1.10 (95% CI: 1.02, 1.18)	I^2 = 0.0%; P = 0.473	Moderate
	He et al. (2017)	Moderate	MA	To evaluate the relationship between T2DM or GDM and exposure to atmospheric PM2.5 levels	11	**RR** = 1.25 (95%CI: 1.10, 1.43)	I^2 = 76.5%; P = 0.000	Moderate
	Liu et al. (2019)	Moderate	SRMA	To investigate the relationship between exposure to air pollution and the incidence and prevalence of type 2 diabetes.	30	**HR** = 1.10 (95% CI: 1.04, 1.16) **OR** = 1.09 (95% CI: 1.05, 1.13)	I^2 = 64%; $P < 0.001$ I^2 = 86%; $P < 0.001$	Moderate
	Chen et al. (2022a,b)	Critically Low	SRMA	To provide an overview of the data demonstrating the link between air pollution exposure and type 2 diabetes in developing nations	8	**OR** = 1.12 (95% CI: 1.07, 1.17)	I^2 = 76%; P = 0.0001	Very Low
PM10	Yang et al. (2020)	High	SRMA	To measure the link between air pollution and type 2 diabetes to determine whether those with diabetes are more susceptible to air pollution	86 [21 on T2DM incidence/25 on T2DM prevalence]	**HR** = 1.11 (95% CI: 1.00, 1.22) **OR** = 1.10 (95% CI: 1.03, 1.17)	I^2 = 70.6%; P = 0.005 I^2 = 89.5%; P = 0.000	High
	Janghorbani et al. (2014)	Moderate	SRMA	To determine the health impacts of pollution-related air exposure on the risk of diabetes and whether the associations differed depending on the kind of pollutant, study design, and geographic location	17 [11 on risk of diabetes-related morbidity (i.e., T2DM Incidence and Prevalence]	**RR** = 1.01 (95% CI:1.003, 1.013)	I^2 = 0.0%; P = 0.582	Moderate
	Wang et al. (2014)	Moderate	SRMA	To evaluate how prolonged exposure to air pollution affects the risk of type 2 diabetes.	10	**RR** = 1.34 (95% CI: 1.22, 1.47)	I^2 = 0%; $P < 0.001$	Moderate
	Liu et al. (2019)	Moderate	SRMA	To investigate the relationship between exposure to air pollutants and the incidence and prevalence of type 2 diabetes.	30	**HR** = 1.05 (95% CI: 0.98, 1.13) **OR** = 1.12 (95% CI: 1.06, 1.19)	I^2 = 48%; P = 0.11 I^2 = 89%; $P < 0.001$	Moderate
NO2	Balti et al. (2014)	High	SRMA	To evaluate and measure the relationship between exposure to major air contaminants and the risk of type 2 diabetes	10	**HR*** = 1.13 (95% CI: 1.04, 1.22) **OR*** = 1.16 (95% CI: 1.00, 1.35)	I^2 = 36.4%; P = 0.208 I^2 = 23.2%; P = 0.254	High
	Yang et al. (2020)	High	SRMA	to measure the link between air pollution and type 2 diabetes to determine whether those with diabetes are more susceptible to air pollution	86 [21 on T2DM incidence/25 on T2DM prevalence]	**HR** = 1.01 (95% CI: 0.99, 1.02) **OR** = 1.07 (95% CI: 1.04, 1.11)	I^2 = 56.1%; P = 0.034 I^2 = 91.1%; P = 0.000	High

(*continued on next page*)

Table 1 (*continued*)

Air pollutants	Authors (Year)	AMSTAR 2	Study Design	Objective(s)/Aim of the Study	No. of Primary Studies Included	Results [Effect Estimates (95% CI)]	Heterogeneity (I^2 value; *p*-value)	GRADE Rating
	Janghorbani et al. (2014)	Moderate	SRMA	To determine the health impacts of air pollution exposure on the risk of diabetes and whether the associations differed depending on the kind of pollutant, study design, and geographic location	17 [11 on risk of diabetes-related morbidity (i.e., T2DM Incidence and Prevalence]	**RR** = 1.04 (95% CI: 1.01, 1.08)	I^2 = 71.9%; P = 0.001	Moderate
	Wang et al. (2014)	Moderate	SRMA	To evaluate how long-term exposure to air pollution affects the risk of type 2 diabetes.	10	**RR** = 1.11 (95% CI: 1.07, 1.16)	I^2 = 43.6%; P < 0.001	Moderate
	Eze et al. (2015)	Moderate	SRMA	To summarize the findings of research on the connection between air pollution and type 2 diabetes and to conduct a thorough review of epidemiological data about this relationship.	13 [7 on T2DM]	**RR** = 1.08 (95% CI: 1.00, 1.17)	I^2 = 58.4%; P = 0.025	Moderate
	Liu et al. (2019)	Moderate	SRMA	To investigate the relationship between exposed to air pollutants and the incidence and prevalence of type 2 diabetes.	30	**HR** = 1.02 (95% CI: 0.99, 1.05) **OR** = 1.05 (95% CI: 1.03, 1.08)	I^2 = 94%; P < 0.001 I^2 = 77%; P < 0.001	Moderate
O3	Janghorbani et al. (2014)	Moderate	SRMA	To determine the health impacts of air pollution exposure on the risk of diabetes and whether the associations differed depending on the kind of pollutant, study design, and geographic location	17 [11 on risk of diabetes-related morbidity (i.e., T2DM Incidence and Prevalence]	**RR** = 1.07 (95% CI: 1.05, 1.09)	I^2 = 0.0%; P = 0.961	Moderate
SO2	Janghorbani et al. (2014)	Moderate	SRMA	To determine the health impacts of air pollution exposure on the risk of diabetes and whether the associations differed depending on the kind of pollutant, study design, and geographic location	17 [11 on risk of diabetes-related morbidity (i.e., T2DM Incidence and Prevalence]	**RR** = 1.05 (95% CI:1.012, 1.079)	I^2 = 0.0%; P = 0.613	Moderate
SO4	Janghorbani et al. (2014)	Moderate	SRMA	To determine the health impacts of air pollution exposure on the risk of diabetes and whether the associations differed depending on the kind of pollutant, study design, and geographic location geographic area ad type of pollutant	17 [11 on risk of diabetes-related morbidity (i.e., T2DM Incidence and Prevalence]	**RR** = 1.04 (95% CI:1.012, 1.079)	I^2 = 0.0%; P = 0.606	Moderate

Note: SRMA = systematic review and meta-analysis; MA = meta-analysis; N/A = not applicable; * The PM10 and NOx estimates in Balti et al. study (2014) were converted into PM2.5 and NO2 estimates, respectively.

Likewise, PM2.5, PM10 was found to be substantially linked to a higher risk of type 2 diabetes. [Incidence: Liu et al. (2019a,b), HR = 1.07 (95%CI: 1.01, 1.13); Prevalence: Yang et al. (2020), OR = 1.10 (95%CI: 1.03, 1.17); Liu et al. (2019a,b), OR = 1.11 (95%CI: 1.06, 1.16)].

3.1.4. NO2

For NO2 exposure, six studies reported the association between NO2 exposure and T2DM (Balti et al., 2014; Yang et al., 2020; Liu et al., 2019a,b; Janghorbani et al., 2014; Wang et al., 2014; Eze et al., 2015a, b). Three studies reported both HR and OR estimates (Balti et al., 2014; Yang et al., 2020; Liu et al., 2019a,b) and the other three studies (Janghorbani et al., 2014; Wang et al., 2014; Eze et al., 2015a,b) reported only RR estimates. NO2 and type 2 diabetes have only been found to be significantly correlated in four studies.

In conclusion, exposure to NO2 was substantially linked to a higher risk of T2DM [Incidence: Balti et al. (2014), HR = 1.13 (95%CI: 1.04, 1.22); Eze et al. (2015a,b), RR = 1.07 (95%CI: 1.02, 1.12); Prevalence: Yang et al. (2020), OR = 1.07 (95%CI: 1.04, 1.11); Liu et al. (2019a,b), OR = 1.06 (95%CI: 1.04, 1.08)].

3.1.5. Other air pollutants

Only one study (Janghorbani et al., 2014) had reported the relation among another air pollutants (i.e., O3, SO2, and SO4) and T2DM. Similar to the others, T2DM was significantly associated with O3, SO2, and SO4, but SO2 and SO4 exposure had a weaker association with their estimates were borderline statistically insignificant [O3, RR = 1.07 (95%CI: 1.05, 1.09); SO2, RR = 1.05 (95% CI:1.012, 1.079); SO4, RR = 1.04 (95% CI:1.012, 1.079)].

3.2. Systematic review and meta-analysis

Database searching turned up a total of 1524 records and 1460 records were excluded due to being duplicated or irrelevant to the current study. 37 research have been excluded because they did not fit the requirements for inclusion. In the end, 27 studies were identified as eligible for the analysis. Additionally, 21 records were identified through manual searching of the included articles' citation lists and two were excluded due to the studies not meeting the inclusion criteria. Finally, 46 studies were included in the final analysis (Fig. 2).

For an air pollution exposure, PM2.5, PM10, and NO2 exposure were the most common types of exposure reported in these studies. Nevertheless, multiple studies have also reported the association with the other main air pollutants (e.g., PM1, NOx, SO2, and O3). The included studies comprised a diverse range of countries from different regions, such as the USA, China, Canada, Denmark, Sweden, Taiwan, Iran, Germany, Italy, Malaysia, Indonesia, and the Netherlands. Most studies had either cohort or cross-sectional designs as their methodology. Exposure assessment was also varied across studies with the tools such as land-use regression, satellite-based estimates, or data from environmental agencies, were mostly used in these studies. The duration of exposure also varied considerably throughout the studies, spanning from a few months to more than three decades. The participant demographics in these studies exhibited a wide range of characteristics with multiple studies only included a certain group of participants such as elderly population, female population, or only African American women population, etc. T2DM cases were diagnosed based on methods such as fasting plasma glucose tests, self-reported physician diagnosis, hospital

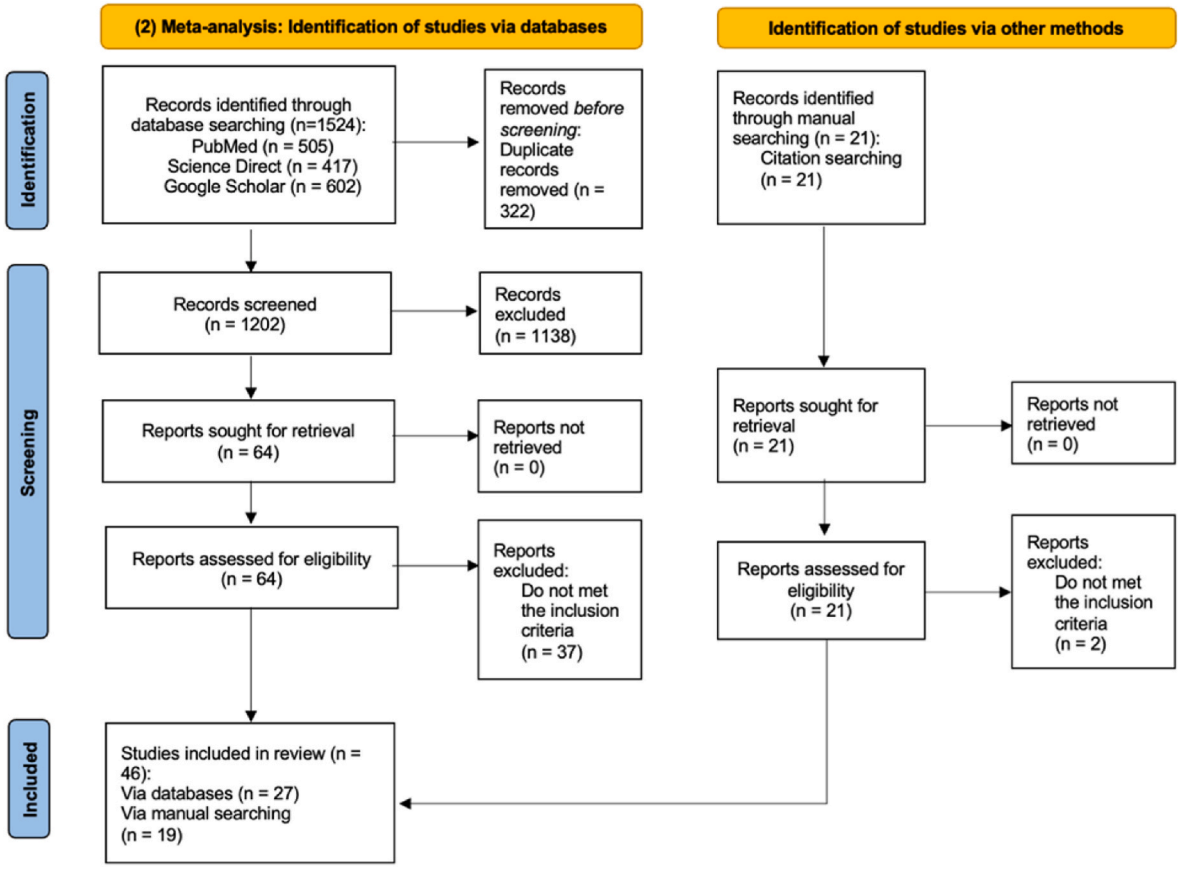

Fig. 2. PRISMA flow diagram of the systematic review and meta-analysis.

administrative records, specific T2DM registries, and/or anti-diabetic medication records. Additionally, some studies employed certain biomarker tests, such as HbA1c, for the purpose of diagnosis. The Supplementary contains the concluded, comprehensive summary of the included investigations. Table S.2.

3.2.1. *Quality assessment*

The studies' quality was evaluated separately for cohort and cross-sectional studies using the Newcastle-Ottawa Scale (NOS). For cohort studies, the maximum score the study could receive was 9 and for cross-sectional studies, the maximum score was 10. The detailed quality assessment of the included cohort and cross-sectional studies using the NOS were included in the Supplementary Table S3 and S.4, respectively. Most included studies obtained the high score of 8 and 9 with only one study having the lowest score of 5 (Jabbari et al., 2020). The included studies have demonstrated a consistently high level of methodological quality, especially in the areas of participant selection and outcome assessment. The high occurrence of scores 8 and above in cross-sectional studies, as well as the presence of maximum or near-maximum scores in cohort studies, highlighted the effectiveness of addressing important methodological issues in these studies, which further strengthened the reliability of these studies' results. Nevertheless, the regularity of high rankings did not diminish the significance of each study. The wide range of scores, particularly for studies with lower NOS scores, emphasizes the importance of conducting thorough assessments to gain a comprehensive understanding of their significance and constraints within the wider research framework. Overall, the studies are supported by high performance in important areas such as selection and outcome. However, occasional limitations in comparability, especially in cross-sectional designs, reminded us of the inherent complexity involved in such investigations.

3.2.2. *Meta-analysis results*

The association between different air pollutants and the risk of T2DM is depicted in Figs. 3 and 4, using odds ratios (ORs) and their 95% CI as a metric to quantify the impact. Out of 46 included studies, 45 were included in the final analysis. One study was excluded in the final analysis due to having a NOS score of 5 (low study quality).

3.2.2.1. p.m.2.5. Out of 45 studies, 29 studies were included in the final analysis (Bo et al., 2021; Bowe et al., 2018; Cai et al., 2023; Chen et al., 2013; Chilian-Herrera et al., 2021; Clark et al., 2017; Coogan et al., 2012, 2016b; Dzhambov and Dimitrova, 2016; Hansen et al., 2016; Honda et al., 2017; Lao et al., 2019; Lin et al., 2021; Liu et al., 2019a, 2022, 2023; Orioli et al., 2018; Park et al., 2015; Paul et al., 2020; Puett et al., 2011; Qiu et al., 2018; Renzi et al., 2018; Strak et al., 2017; Weinmayr et al., 2015; Wu et al., 2022; Yang et al., 2018b; Yitshak Sade et al., 2023; Zhou et al., 2022) . Most included studies had cohort study design, conducted in the USA or China, had a combination of methods for DM diagnosis (e.g., self-reported, blood-tests, administrative records, T2DM registries, intakes of anti-diabetic drugs records, etc.). The average age participants for the included primary studies were 54 years. The included primary studies had an average of 5.4 years for exposure and 9.14 years of follow-up. The average air pollution concentration level was 25.41 $\mu g/m^3$. The included OR estimates and 95%CIs were vastly different from each other with some had found an OR estimate as high as 1.650 (Wu et al., 2022), whereas some reported ORs that were more closely to 1 (Lin et al., 2021; Renzi et al., 2018). One study Dzambov and Dimitrova, 2016) also reported a very wide range of 95% CI, indicating the less precision in study results, which resulted in the study having less weight in the final analysis as shown in Fig. 3. Overall, the pooled result showed a significant association between PM2.5 exposure and increasing risk of T2DM (OR = 1.12, 95%CI: 1.09, 1.5)

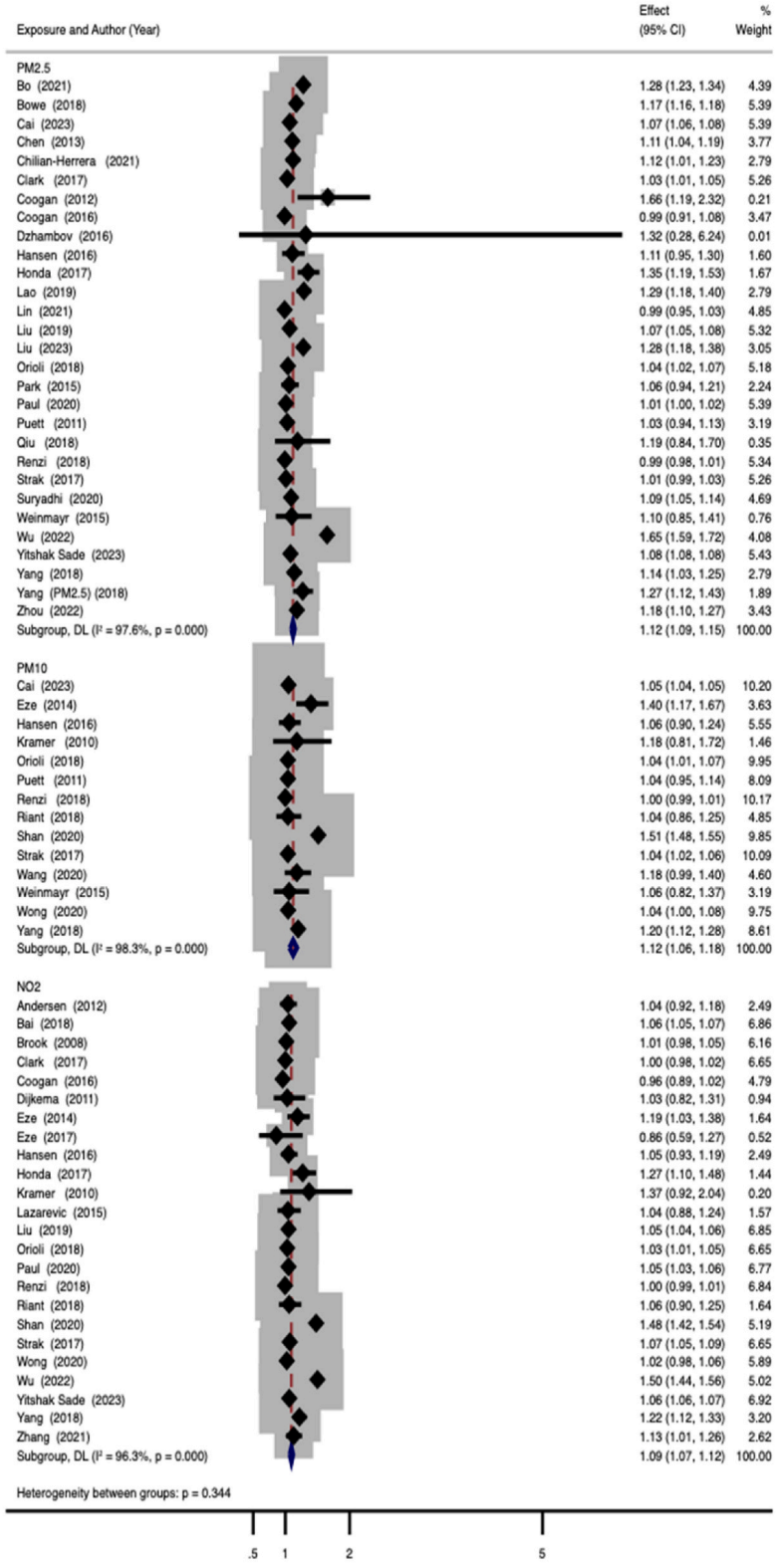

Fig. 3. Meta-analysis of main air pollutants exposure (PM2.5, PM10, and NO2) and the risk of T2DM.

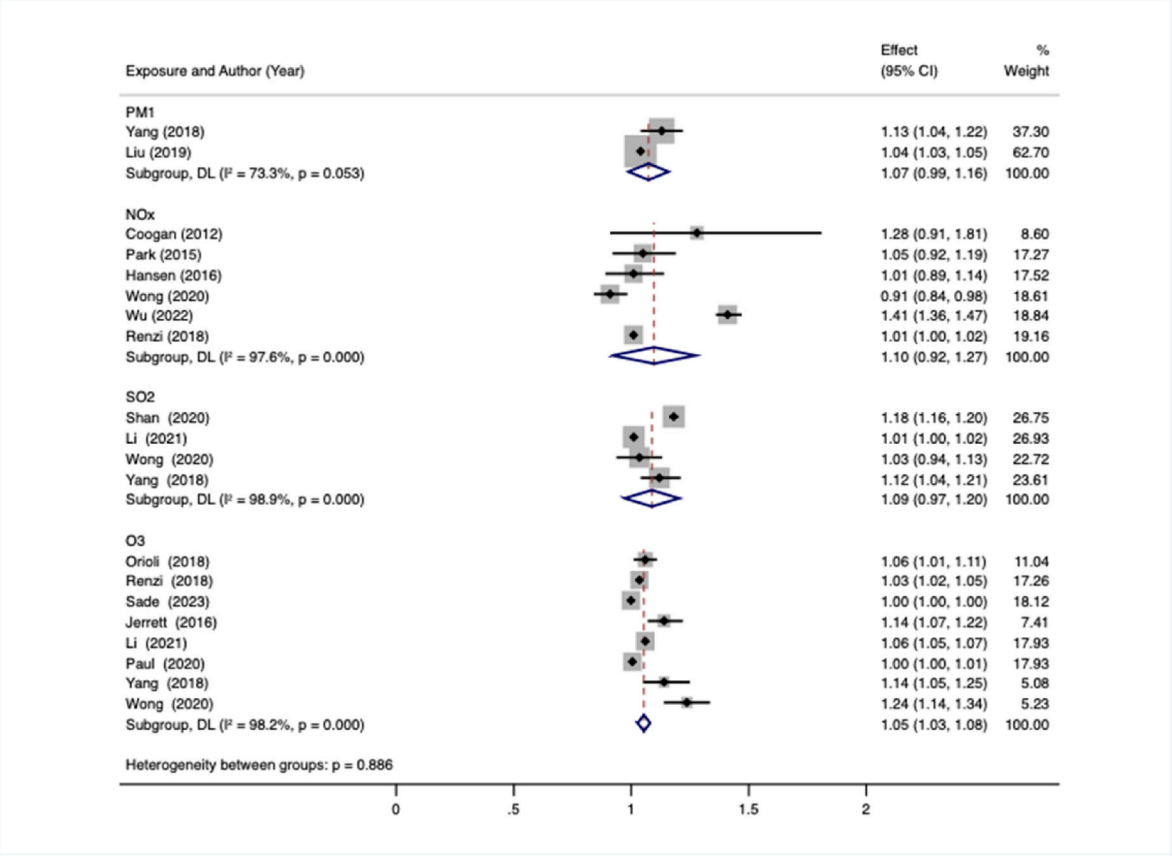

Fig. 4. Meta-analysis of other main air pollutants and the risk of T2DM.

with a high I^2 value of 97.6% and *p*-value of 0.0 indicating a high heterogeneity between studies.

3.2.2.2. p.m.10. For PM10 exposure, 14 studies were included in the final analysis (Cai et al., 2023; Eze et al., 2014; Hansen et al., 2016; Krämer et al., 2010; Orioli et al., 2018; Puett et al., 2011; Renzi et al., 2018; Riant et al., 2018; Shan et al., 2020; Strak et al., 2017; Wang et al., 2020, 2021; Weinmayr et al., 2015; Yang et al., 2018a). Most of the included studies had either cross-sectional or cohort study design, conducted in Europe, and used a combination of methods for DM diagnosis. Overall, the included studies had an average age of 53 years for participants, 6.1 years of exposure, 9.84 years of follow-up, and air pollution concentration level of 51.68 μg/m^3. All of the PM10 estimates had an OR estimate more than 1 with one had an elevated OR as high as 1.51 (Shan et al., 2020). The pooled result indicated a significant association between PM10 and an elevated risk of T2DM (OR = 1.12, 95% CI 1.06, 1.18). Similarly, to the PM2.5 pooled result, the PM10 pooled estimate had a high heterogeneity between studies with an I^2 value of more than 90% and *p*-value less than 0.05, indicating the significant variances between studies (Fig. 3).

3.2.2.3. NO2. The final analysis comprised 24 studies in total (Andersen et al., 2012; Bai et al., 2018; Brook et al., 2008; Clark et al., 2017; Coogan et al., 2016a;Dijkema et al., 2011; Eze et al., 2014; Eze et al., 2017; Hansen et al., 2016; Honda et al., 2017; Krämer et al., 2010; Lazarevic et al., 2015; Liu et al., 2019a; Orioli et al., 2018; Paul et al., 2020; Renzi et al., 2018; Riant et al., 2018; Shan et al., 2020; Strak et al., 2017; Wang et al., 2020; Wu et al., 2022; Yitshak Sade et al., 2023; Yang et al., 2018b; Zhang et al., 2021). Most of the included studies were cohort studies, but others were cross-sectional, case-control, or cohort studies. Mostly conducted in Europe, used a combination of methods for DM diagnosis (with mostly used administrative records as one of the methods), had an average age of 55.8 years for participants, average of 6.78 years for duration of exposure, average of 10.7 years for follow-up, and an average air pollution concentration level of 25.92 μg/m^3/15.56 ppb. A pooled NO2 result showed a significant but weakly association between NO2 exposure and T2DM (OR = 1.09, 95% CI: 1.07, 1.12) and had an I^2 value of 96.3% and *p*-value of 0.00, indicating a significant heterogeneity between studies.

3.2.2.4. Other pollutants (PM1, NOx, SO2 and O3). PM1, NOx, and SO2 and O3 exposure were other common air pollutants reported in these studies and usually were reported in a combination with PM2.5, PM10, or NO2. Most of these studies had a cohort study design, conducted in the USA, Europe, or China, and had an average age of participants more than 38 years old. Out of 45 included studies, 2 studies reported PM1 exposure estimates, 6 reported NOx estimates, 4 studies reported SO2 estimates and 8 studies reported O3 estimates. Only a pooled result from O3 exposure indicated a significant association between the air pollutant exposure and the risk of T2DM (OR = 1.05, 95% CI: 1.03, 1.08). Similarly, to the other air pollutants, the O3 pooled estimate had a high I^2 value (>90%) and a p-value of 0.00, indicating a high heterogeneity between studies [Fig. 4].

3.2.3. Publication bias

Publication bias test was performed only for PM2.5, PM10, and NO2 exposures. The test was not performed for the other air pollutants due to the small number of included studies (less than ten studies). According to the funnel plots and Egger's test, there was no obvious evidence of publication bias among the included studies (Egger's test: PM2.5, $p = 0.611$; PM10, $p = 0.343$; NO2, $p = 0.449$; the funnel plots are included in the Supplementary Figure S1).

3.2.4. Subgroup analyses

The subgroup analyses were performed for PM2.5, PM10, and NO2 exposure. The detailed association between air pollutant exposure (PM2.5, PM10, and NO2) and the risk of developing T2DM by subgroups is presented in Table 2. The subgroup analyses consisted of pooled models (fixed-effect vs random-effect models), study design, area of exposure (regions), average age of participants, DM diagnosis, average duration of exposure (for cross-sectional studies) or follow-up (for cohort studies) and average air pollution concentration level.

3.2.4.1. p.m.2.5. Based on the fixed-effects model, the pooled OR estimate was closely to 1 and was borderline insignificant (OR = 1.078; 95% CI: 1.076, 1.08) compared to the random-effect model' OR estimate (OR = 1.12, 95%CI: 1.09, 1.15), this difference emphasized that there was a possible significance variance between studies (heterogeneity) that needed to be addressed, thus making sub-group analysis an important analysis to use to determine these variances.

Based on the subgroup analysis, study design, DM diagnosis, years of follow-up (average), and air pollution concentration levels showed a significant heterogeneity between groups [p-value of less than 0.05; study design, $p = 0.002$; DM diagnosis, $p = 0.000$; years of follow-up, $p = 0.002$; air pollution concentration levels, $p = 0.000$] indicating that the high heterogeneity between studies may be due to the variances in these subgroups.

For the study design, both cohort and cross-sectional study design had significance results with cohort study design have a higher OR estimate [OR = 1.15 (95%CI: 1.11, 1.18) for cohort studies compared to OR = 1.07, (95%CI: 1.04, 1.10) for cross-sectional study] and was slightly higher than the pooled random-effect model estimate [OR = 1.12 (95%CI: 1.09, 1.15)]. These indicated that there was a possible bias due to study design as the results showed that the participants who were exposed to PM2.5 in cohort studies was more at risk of developing T2DM than participants in cross-sectional studies.

For DM diagnosis, the differences in reporting DM cases especially from the administrative records and combination of more than one method (I^2 value of more than 90%), was possibly one of reason for high heterogeneity between studies as the OR estimates from self-reported and administration records [OR = 1.07 (95%CI: 1.04, 1.10); OR = 1.04 (95%CI: 1.00, 1.09), respectively] showed a less significant risk of T2DM compared to the use of combination methods and blood test [OR = 1.19 (95%CI:1.10,1.28); OR = 1.17 (95%CI: 1.16, 1.18), respectively].

For duration of follow-up subgroup analyses, all categories had shown significant results with high heterogeneity (I^2 value more than 80%), indicating that high heterogeneity between studies may be due to the duration of follow-up. The results also indicated that with more years of follow-up, the risk of T2DM was also increasing but after 10 years of follow-up, the risk of T2DM seems to slightly be reducing [less than 4 years of follow-up, OR = 1.05, 95%CI: 1.01,1.09; 5–10 years follow-up, OR = 1.19, 95%CI: 1.10, 1.28; more than 10 years of follow-up, OR = 1.15, 95%CI: 1.08, 1.22)].

Lastly, the air pollution concentration levels also showed significant and similar patterns to the years of follow-up results [air concentration levels between 10 and 20 μg/m^3, OR = 1.12, 95%CI: 1.08, 1.17; 21–40 μg/m^3, OR = 1.16, 95%CI: 1.07, 1.25; 41–60 μg/m^3, OR = 1.23, 95%CI: 1.16, 1.30; >60 μg/m^3, OR = 1.08, 95%CI: 1.03, 1.14)], indicating a possible dose-response relationship between increasing air pollution exposure concentration level and the risk of T2DM, which was further explored in the dose-relationship section of this study. The significant high heterogeneity between groups also indicated that high heterogeneity between studies may be due to these variances.

For the other subgroups, while the heterogeneity between groups did not reveal a significant variance, the significant association within some of these subgroups was also worth mentioning. For example, all of the regions conducting the studies reported a significant association between PM2.5 exposure and the risk of T2DM, with North American had the lowest OR estimate (OR = 1.08, 95%CI: 1.04, 1.12), indicating that being exposed to PM2.5 in North America was less likely to increase the risk of developing T2DM than being exposed to PM2.5 in Asia or Europe region. And for the duration of the exposure subgroup, the results indicated that being exposed to PM2.5 for less than 4 years (OR = 1.08, 95%CI: 1.04, 1.11) was less likely to increase the risk of T2DM.

3.2.4.2. p.m.10. For PM10 exposure, a significant high heterogeneity between groups was only found in ages of participants (average) and DM diagnosis subgroups [ages of participants, $p = 0.000$; DM diagnosis, $p = 0.000$]. Among ages of participants subgroup, aged between 45 and 50 years and aged 56 years or older were found to be significant associated with the higher risk of T2DM [45–50 years, OR = 1.20 (95%CI: 1.12, 1.27); 56 years or older, OR = 1.04, (95%CI: 1.01, 1.07)] with an exception of aged less than 45 subgroup, which also found a significant association but only had one study included, which indicated that more studies was needed to further confirmed this relationship. Significant high heterogeneity between groups, especially from the unclear age group, indicating that age subgroup was possibly the reason for high heterogeneity between studies for PM10 exposure.

For DM diagnosis, the method of blood test showed a significantly higher result compared to self-reported DM [OR = 1.20, 95%CI: 1.12, 1.28); OR = 1.05 (95%CI: 1.04, 1.05), respectively]. These results indicated that being diagnosed by blood test significantly increased the risk of T2DM from PM10 exposure. Also, high heterogeneity from the combination of diagnosis methods was also possibly the reason for high heterogeneity between studies for PM10 exposure.

For the other subgroups, no significant high heterogeneity was found among these subgroups. But similar to PM2.5, being exposed to PM10 for less than 4 years in a cross-sectional study design had a lower risk of T2DM compared to the pooled random-effect model estimate [exposed less than 4 years, OR = 1.06 (95%CI: 1.03, 1.08), compared to the random-effects model OR = 1.12 (95%CI: 1.06, 1.18)]. In contrast to PM2.5 exposure, the higher PM10 exposure concentration levels were, the risk of T2DM was exponentially increased [less than 10 μg/m^3, OR = 1.04 (95%CI: 1.01, 1.07); 41–60 μg/m^3, OR = 1.05 (95%CI: 1.04, 1.05); more than 60 μg/m^3, OR = 1.31 (95%CI: 1.05, 1.56)] and the results were significant.

3.2.4.3. NO2. For NO2 exposure, a significant high heterogeneity between groups was found among study design ($p = 0.001$), ages ($p = 0.001$), DM diagnosis ($p = 0.000$), and years of follow-up ($p = 0.001$), which indicated that high heterogeneity between studies may be explained by these subgroups' variances.

For NO2 exposure, had cohort study design and being exposed to NO2 in Asia significantly increased the risk of T2DM [cohort study design, OR = 1.11 (95%CI: 1.08, 1.15); Asian, OR = 1.18 (95%CI: 1.02, 1.34); compared to the random-effect estimate, OR = 1.09 (95%CI: 1.07, 1.12)], while being exposed to NO2 in North America slightly reduced the risk of T2DM [OR = 1.04 (95%CI: 1.02, 1.06)].

For ages of participants, being aged between 45 and 55 years old was significantly associated with an increased risk of T2DM [45–50 years, OR = 1.22 (95%CI: 1.12, 1.33); 51–55 years, OR = 1.16 (95%CI: 1.08, 1.24) compared to aged 56 years or older, OR = 1.04 (95%CI: 1.02, 1.06)].

For years of follow-up, participants who have been follow-up for more than 10 years were significantly associated with an increased risk of T2DM (OR = 1.17, 95%CI: 1.11, 1.22).

Lastly, for the air pollution concentration levels (using μg/m^3 unit), similar pattern was observed in NO2 exposure, but the results were not significant [Only being exposure to NO2 concentration levels of 21–40 μg/m^3 was significantly associated with T2DM (OR = 1.06, 95%CI: 1.03, 1.08)]. For air concentration levels (using ppb units), only being exposed to NO2 of 15–20 ppb showed a significantly reduced risk of T2DM [OR

Table 2
The association between air pollutants exposure (PM2.5, PM10, NO2) and the risk of T2DM by subgroups.

	PM2.5		PM10		NO2	
	N	OR (95%CI) [Heterogeneity: I^2; *p*-value]	N	OR (95%CI) [Heterogeneity: I^2; *p*-value]	N	OR (95%CI) [Heterogeneity: I^2; *p*-value]
Models:						
Fixed-effect model	29	**1.078 (1.076, 1.08)** [I^2 = 97.6%; p = 0.000]	14	**1.05 (1.045, 1.054)** [I^2 = 98.3%; p = 0.000]	24	**1.055 (1.052, 1.058)** [I^2 = 96.3%; p = 0.000]
Random-effects model	29	**1.12 (1.09, 1.15)** [I^2 = 97.6%; p = 0.000]	14	**1.12 (1.06, 1.18)** [I^2 = 98.3%; p = 0.000]	24	**1.09 (1.07, 1.12)** [I^2 = 96.3%; p = 0.000]
Study design:						
Cohort	19	**1.15 (1.11, 1.18)** [I^2 = 98.3%; p = 0.000]	7	1.15 (0.89, 1.40) [I^2 = 99.2%; p = 0.000]	14	**1.11 (1.08, 1.15)** [I^2 = 65.9%; p = 0.003]
Cross-sectional	10	**1.07 (1.04, 1.10)** [I^2 = 85.3%; p = 0.000]	7	**1.06 (1.03, 1.08)** [I^2 = 73.4%; p = 0.000]	9	**1.07 (1.03, 1.10)** [I^2 = 97.8%; p = 0.000]
Case-control	N/A	N/A	N/A	N/A	1	1.01 (0.98, 1.05) [N/A]
Regions of Exposure:						
Asia	11	**1.14 (1.10, 1.19)** [I^2 = 91.5%; p = 0.000]	5	1.20 (0.99, 1.41) [I^2 = 99.4%; p = 0.000]	7	**1.18 (1.02, 1.34)** [I^2 = 98.2%; p = 0.000]
Europe	11	**1.15 (1.04, 1.27)** [I^2 = 98.4%; p = 0.000]	8	**1.03 (1.00, 1.07)** [I^2 = 72.2%; p = 0.003]	11	**1.11 (1.04, 1.18)** [I^2 = 96.5%; p = 0.000]
North America	7	**1.08 (1.04, 1.12)** [I^2 = 98.2%; p = 0.000]	1	1.04 (0.94, 1.13) [N/A]	5	**1.04 (1.02, 1.06)** [I^2 = 89.4%; p = 0.000]
Australia	N/A	N/A	N/A	N/A	1	1.04 (0.86, 1.22) [N/A]
Ages of Participants (Average):						
Less than 45 years	7	**1.12 (1.02, 1.23)** [I^2 = 93.3%; p = 0.000]	1	**1.51 (1.48, 1.55)** [N/A]	2	1.22 (0.71, 1.73) [I^2 = 99.3%; p = 0.000]
45–50 years	1	**1.14 (1.03, 1.25)** [N/A]	2	**1.20 (1.12, 1.27)** [I^2 = 0.0%; p = 0.859]	1	**1.22 (1.12, 1.33)** [N/A]
51–55 years	4	1.22 (0.89, 1.55) [I^2 = 99.2%; p = 0.000]	4	1.15 (0.98, 1.32) [I^2 = 50.0%; p = 0.112]	7	**1.16 (1.08, 1.24)** [I^2 = 97.1%; p = 0.000]
56 years or older	12	**1.11 (1.07, 1.15)** [I^2 = 97.0%; p = 0.000]	3	**1.04 (1.01, 1.07)** [I^2 = 0.0%; p = 0.990]	10	**1.04 (1.02, 1.06)** [I^2 = 84.7%; p = 0.000]
Unclear	5	1.05 (1.00, 1.11) [I^2 = 95.9%; p = 0.000]	4	1.03 (1.00, 1.06) [I^2 = 95.1%; p = 0.000]	4	1.03 (0.98, 1.08) [I^2 = 92.2%; p = 0.000]
DM Diagnosis:						
Self-reported	8	**1.07 (1.04, 1.10)** [I^2 = 66.2%; p = 0.004]	3	**1.05 (1.04, 1.05)** [I^2 = 0.0%; p = 0.694]	5	1.01 (0.97, 1.06) [I^2 = 28.1%; p = 0.234]
Blood test	2	**1.17 (1.16, 1.18)** [I^2 = 0.0%; p = 0.594]	1	**1.20 (1.12, 1.28)** [N/A]	1	**1.22 (1.12, 1.33)** [N/A]
Administrative records/ registries/ICD-10	7	1.04 (1.00, 1.09) [I^2 = 98.2%; p = 0.000]	2	1.00 (0.99, 1.01) [I^2 = 0.0%; p = 0.490]	8	**1.03 (1.01, 1.06)** [I^2 = 95.1%; p = 0.000]
Combination*	12	**1.19 (1.10, 1.28)** [I^2 = 97.6%; p = 0.000]	8	1.16 (0.98, 1.34) [I^2 = 98.7%; p = 0.000]	10	**1.18 (1.09, 1.28)** [I^2 = 97.9%; p = 0.000]
Duration of Exposure (Average):						
Less than 4 years	8	**1.08 (1.04, 1.11)** [I^2 = 85.6%; p = 0.000]	4	**1.06 (1.03, 1.08)** [I^2 = 79.8%; p = 0.002]	6	**1.08 (1.03, 1.12)** [I^2 = 73.8%; p = 0.002]
5–10 years	2	1.04 (0.92, 1.17) [I^2 = 79.0%; p = 0.029]	2	1.20 (0.85, 1.55) [I^2 = 86.9%; p = 0.006]	2	1.08 (0.93, 1.24) [I^2 = 69.8%; p = 0.069]
More than 10 years	N/A	N/A	1	1.04 (0.84, 1.23) [N/A]	1	1.06 (0.88, 1.24) [N/A]
Follow-up (Average):						
Less than 4 years	2	**1.05 (1.01, 1.09)** [I^2 = 88.2%; p = 0.004]	1	1.18 (0.97, 1.38)	2	1.03 (0.98, 1.07) [I^2 = 94.6%; p = 0.000]
5–10 years	11	**1.19 (1.10, 1.28)** [I^2 = 97.8%; p = 0.000]	2	1.00 (0.99, 1.01)	5	1.02 (0.98, 1.06) [I^2 = %; p = 0.003]
More than 10 years	6	**1.15 (1.08, 1.22)** [I^2 = 99.0%; p = 0.000]	4	1.20 (0.87, 1.53)	8	**1.17 (1.11, 1.22)** [I^2 = 98.4%; p = 0.000]
Air Pollution Concentration Levels (Average; μg/m^3):						
Less than 10 μg/m^3	3	1.04 (1.00, 1.07) [I^2 = 85.3%; p = 0.001]	N/A	N/A	N/A	N/A
10–20 μg/m^3	13	**1.12 (1.08, 1.17)** [I^2 = 98.5%; p = 0.000]	1	**1.04 (1.01, 1.07)** [N/A]	6	1.12 (0.96, 1.27) [I^2 = 97.7%; p = 0.000]
21–40 μg/m^3	7	**1.16 (1.07, 1.25)** [I^2 = 93.5%; p = 0.000]	7	1.03 (0.99, 1.07) [I^2 = 72.4%; p = 0.001]	9	**1.06 (1.03, 1.08)** [I^2 = 82.8%; p = 0.000]
41–60 μg/m^3	4	**1.23 (1.16, 1.30)** [I^2 = 20%; p = 0.287]	3	**1.05 (1.04, 1.05)** [I^2 = 0.0%; p = 0.808]	3	1.27 (0.85, 1.68) [I^2 = 99.3%; p = 0.000]
More than 60 μg/m^3	2	**1.08 (1.03, 1.14)** [I^2 = 38.0%; p = 0.204]	3	**1.31 (1.05, 1.56)** [I^2 = 96.5%; p = 0.000]	N/A	N/A
Air Pollution Concentration Levels (Average; ppb):						
Less than 5 ppb	N/A	N/A	N/A	N/A	N/A	N/A
5–10 ppb	N/A	N/A	N/A	N/A	1	1.04 (0.86, 1.22) [N/A]

(*continued on next page*)

Table 2 (*continued*)

	PM2.5		PM10		NO2	
	N	OR (95%CI) [Heterogeneity: I^2; *p*-value]	N	OR (95%CI) [Heterogeneity: I^2; *p*-value]	N	OR (95%CI) [Heterogeneity: I^2; *p*-value]
11–15 ppb	N/A	N/A	N/A	N/A	1	**1.27 (1.08, 1.46)** [N/A]
15–20 ppb	N/A	N/A	N/A	N/A	4	**1.04 (1.01, 1.06)** [I^2 = 86.3%; p = 0.000]

Abbreviations: N, number of included studies; ORs, Odd Ratios; 95%CI, 95% Confidence Intervals; DM, Diabetes Mellitus; ICD-10, International Classification of Diseases 10th Revision; N/A, not available/applicable; * DM diagnosis was a combination of two or three of these methods: self-reported DM diagnosis, blood-test, administrative records, T2DM registries and/or intakes of anti-diabetic medication records.
Note: The significant associations were highlighted in bold.

= 1.04, (95%CI: 1.01, 1.06)]. While the result from concentration of 11–15 ppb was significant, there was only one study included, so more studies were probably needed to confirm this relationship.

3.2.5. *Dose-response analyses*

For all three air pollutants, only PM10 was found to have a significant dose-response relationship with a p-value of 0.000 and F-statistic of 116.44 (degree of freedom: 2, 11). An adjusted r-squared value of 0.9467 indicated a high degree of model fit and both coefficients of the restricted cubic spline were also found to be statistically significant (Supplementary Table S4). Fig. 5 presented the dose-relationship between main air pollutants (i.e., PM2.5, PM10, and NO2) concentration level and the risk of T2DM. Based on the predicted value, the graph indicated that the risk of T2DM seems to be lower as the PM10 concentration level increased until the concentration level reached around 32 μg/m^3, which was where the risk of T2DM started to increase exponentially. This result further emphasized a significant non-linear relationship between PM10 exposure and the risk of T2DM.

4. Discussion

The study had found that outdoor exposure to air pollutants (such as PM2.5, PM10, and NO2) was significantly associated with the risk of developing T2DM with high heterogeneity between studies for all air pollutants exposure groups. The pooled estimates, especially PM10 and NO2, in this study were consistent with the results reported in earlier studies (Eze et al., 2015a,b; Liu et al., 2019a,b; Yang et al., 2020). For instance, an exposure to PM10 was associated with a 12% increased risk of T2DM, which was closed to the results from previous umbrella studies [e.g., Yang et al. (2020); OR = 1.10 (95% CI: 1.03, 1.17) and Liu et al. (2019a,b); OR = 1.12 (95%CI: 1.06, 1.19)]. Additionally, the subgroup analysis of PM2.5, PM10, and NO2 has further revealed a significant complex relationship that may be influenced by multiple factors including age, regions of exposure, air pollution concentration levels, types of study design and DM diagnosis methods.

Despite the finding of significant association between main outdoor air pollutants and T2DM, high heterogeneity was observed across

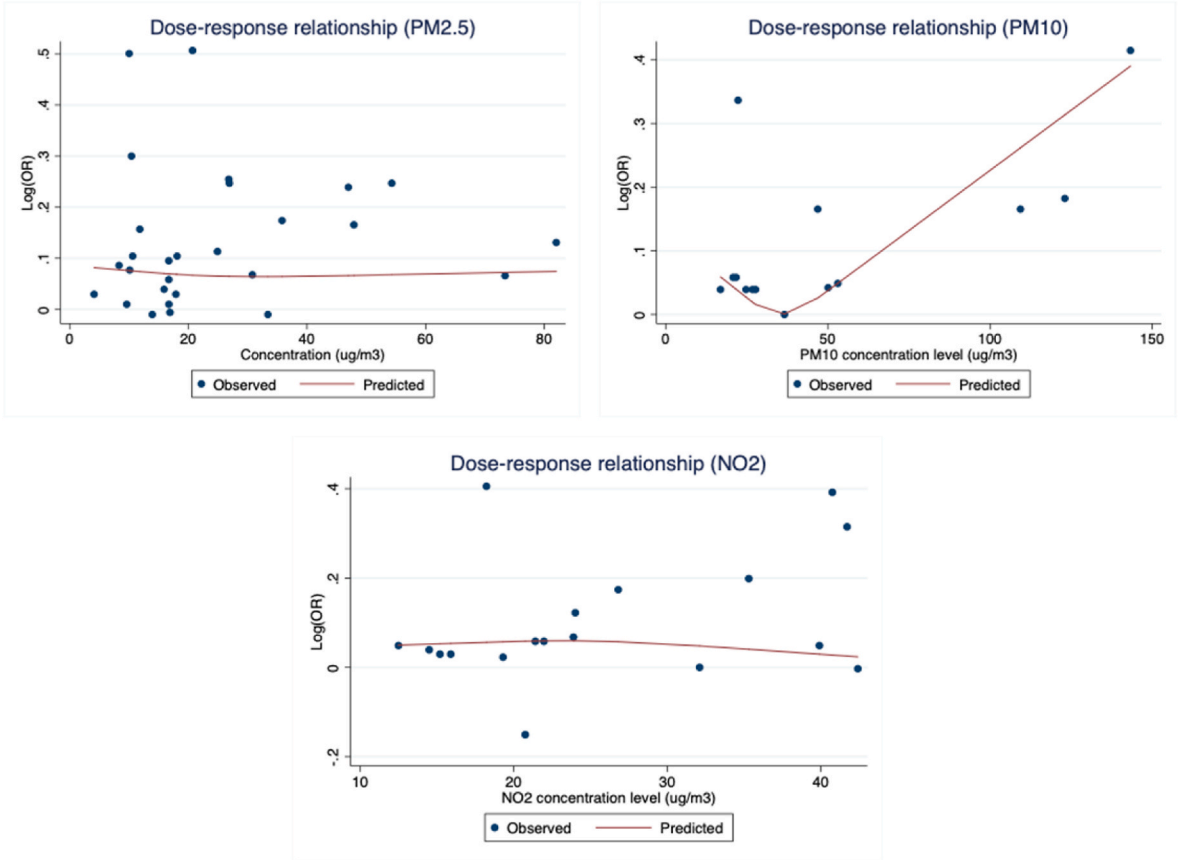

Fig. 5. Dose-response relationship between PM10, PM2.5, and NO2 and T2DM.

studies, especially in cohort designs, where PM2.5 and PM10 showed I^2 values exceeding 98%. This substantial variability mirrored findings from previous reviews but provides a more nuanced understanding of the potential sources of this heterogeneity (Yang et al., 2020). Our subgroup analyses revealed that heterogeneity remained high even when stratified by study design, geographical regions, and age groups. This finding suggested that variability in factors such as controlling confounders, exposure assessment methods, and population susceptibilities may play a crucial role in this. This finding emphasized the need for standardized methodologies in future research, particularly in terms of adjusting for confounders such as socioeconomic status, smoking, and pre-existing health conditions (Eze et al., 2015a,b; He et al., 2017).

Furthermore, our analysis revealed that for adults aged 45–55 years, the risk of T2DM was higher compared to the elderly who aged more than 65 years. This significant difference was consistent across all exposure groups. The increased susceptibility among middle-aged adults may be attributed to age-related factors such as occupational exposures, lifestyle choices, or other environmental interactions (Nishida and Yatera, 2022; Simoni et al., 2015). The adults were more likely to be exposed to outdoor air pollution due to commuting or outdoor jobs, than elderly who were more likely to stay at home or taking more precaution measures for their health due to comorbidity conditions or old ages (Simoni et al., 2003).

In terms of geographical variation, our study found a significantly higher risk of T2DM in Asia and Europe, particularly for PM2.5 and NO2 exposure. This may be due to the increase of industrialization, urbanization and fossil fuel and biomass burnings in Asia (Jion et al., 2023), and the high energy consumption and road transport emissions in Europe (The Council of the European Union, 2024), which might explain the higher risk of T2DM in these regions. By contrast, North America had a lower air pollution level, which may be attributed to the stricter air quality regulations in the region (United States Environmental Protection Agency,). Interestingly, the decreasing air concentration levels over the years in Europe and lower concentration levels in the North America region may contributed to the lower risk of T2DM. It was possible that for every decrease in air pollution concentration levels, especially PM2.5, contributed to the lower risk of T2DM over the years (Bo et al., 2021).

The types of study design were another important factor that may influence the result of this study. Cohort studies showed a higher risk of T2DM than cross-sectional studies, potentially because of the longer duration of air pollution exposure captured in cohort designs (e.g., PM2.5, OR = 1.15 (95%CI: 1.11, 1.18) for cohort study design, and 1.07 (95%CI: 1.04, 1.10) for cross-sectional study design). Also, the prolonged exposure to air pollution concentration level such as PM2.5 was found to increase the risk of developing T2DM. These results were seen with higher concentration levels of PM2.5 (>41 μg/m^3) and follow-up periods of over five years. Additionally, there was a huge variation in measurement uses in the included primary studies, which might also influence the air pollutants concentration level. This indicated there need to be a more standardized approach in measuring air pollution exposure, which could further reduce the bias due to this variability.

Moreover, the method of T2DM diagnosis significantly influenced our estimates. Studies using blood tests or a combination of diagnostic methods found higher risks compared to self-reported diagnoses (e.g., PM2.5: self-reported OR = 1.07 (95%CI: 1.04, 1.10) and blood test OR = 1.17 (95%CI: 1.16, 1.18)). This suggests that more accurate diagnostic methods may capture more T2DM cases into the studies, resulting in higher estimated risks. Interestingly, the estimates from blood test and combination methods were closer to the pooled estimate result (e.g., PM2.5, OR = 1.12 (95%CI: 1.09, 1.15)), suggesting that the use of blood test and combination methods may be more appropriate method for reporting T2DM than only using self-reported diagnosis method alone.

A key novel contribution of this study is the identification of a significant dose-response relationship between PM10 exposure and T2DM risk, which contrasts with the absence of such a relationship for PM2.5. Previous meta-analyses have not consistently reported a dose-response relationship for PM10 (Balti et al., 2014), and this finding suggested that PM10 may exerted stronger metabolic effects than previously recognized. The diverse composition of PM10, which often included toxic coarse particles from road dust, industrial activities, and traffic emissions which differed from the fine particles' composition in PM2.5, may significantly attributed to this relationship (Chen et al., 2022a,b). The absence of a dose-response relationship for PM2.5, despite its known harmful effects, raised questions regarding its specific role in metabolic health, particularly in comparison to PM10. This distinction composition between PM2.5 and PM10 was an important factor to note and could have further implications for public health policies that have previously focused on regulating PM2.5 levels.

The analysis revealed that for every PM10 concentration levels increased (after 32 μg/m^3), the risk of T2DM was increasing exponentially. This indicated that for every increase in PM10 concentration level, the risk of T2DM also increased and the result was similar to the previous study (Wang et al., 2020). For PM2.5 and NO2 exposure, although the dose-response results were not significant, similar patterns to those seen in previous studies (Liu et al., 2023; Eze et al., 2014) were observed. This suggests the possibility of a non-linear relationship for these pollutants that warrants further investigation. The absence of a distinct dose-response relationship may be attributed to reasons such as differences in levels of exposure, different susceptibilities among populations, and discrepancies in measurement techniques. Furthermore, prior research indicated that PM2.5 has a far higher surface area to volume ratio than PM10, which may allow it to absorb more dangerous substances. Furthermore, PM2.5 may remain in the air for long periods of time due to its reduced weight, which facilitates its entry into human lungs and blood vessels for systemic effects. Consequently, it is widely accepted that exposure to PM2.5 might result in a higher disease load and more noticeable health impacts than PM10 (Wang et al., 2021). However, the current study's findings showed a significant dose-response relationship between PM10 and TDM risk, especially at higher concentrations (>32 μg/m^3). This relationship was unique when compared to other pollutants such as PM2.5, implying that PM10 may have a stronger role in metabolic health (Cox, 2016). There was more than one linear association between particulate matter exposure levels and health risks. Very slight changes in exposure levels could cause significant changes in insulin resistance (IR)-related factors at lower doses, such as those found in a typical ambient environment (0.2 mg/day). At higher exposure levels, such as environments with active smoking (up to 200 mg/day), the dose-response curve plateaued. As a result, there might be differences between the effect outcomes in this study that were standardized by exposure concentration and the actual circumstances. The seemingly more pronounced risk alterations linked to PM10 levels at comparatively low exposure levels may be explained by this discrepancy.

Another mechanism that was important to note was a multifaceted interplay of systemic inflammation, oxidative stress, and metabolic dysregulation. Inflammatory cytokines, such as TNF-α and IL-6, impair insulin signaling through stress kinase pathways, while ROS-induced oxidative stress damages pancreatic β-cells and reduces insulin production. Adipose tissue inflammation further exacerbated glucose dysregulation by altering adipokine profiles. These combined effects created a metabolic environment conducive to the development and progression of Type 2 diabetes. PM10 inhalation activated immune responses through pattern recognition receptors (PRRs), such as Toll-like receptor 4 (TLR4), on immune cells, initiating the release of pro-inflammatory cytokines, including Interleukin-6 (IL-6) and Tumor Necrosis Factor-alpha (TNF-α) (Rajagopalan and Brook, 2012). These cytokines interfered with insulin signaling via molecular mechanisms that involved stress kinase pathways, such as c-Jun N-terminal kinase (JNK) and IκB kinase (IKK). Activation of these kinases led to the phosphorylation of insulin receptor substrate-1 (IRS-1) at inhibitory serine residues, impairing the insulin receptor's ability to propagate its signal. This

disruption of signaling cascades reduces glucose uptake, particularly in skeletal muscle tissue, by inhibiting the translocation of glucose transporter type 4 (GLUT4) to the cell membrane (Liu et al., 2022). Furthermore, chronic exposure to inflammatory cytokines compromised insulin receptor autophosphorylation, reduced tyrosine phosphorylation of IRS proteins, diminished phosphatidylinositol 3-kinase (PI3K) activity, and decreased Akt phosphorylation, further exacerbating insulin resistance (Sun et al., 2022).

PM10 exposure also triggered oxidative stress, a key contributor to β-cell dysfunction and insulin deficiency. Reactive oxygen species (ROS), such as superoxide and hydrogen peroxide, were predominantly generated through mitochondrial pathways. Ren et al. (2023) reported that PM10 exposure increased ROS production by 40–60% in experimental conditions, disrupting mitochondrial electron transport chain activity. This oxidative stress damaged pancreatic β-cells, leading to mitochondrial DNA damage and reduced ATP production, which impaired glucose-stimulated insulin secretion (Liu et al., 2016). The effects of oxidative stress extended to the endoplasmic reticulum (ER), where it activated the unfolded protein response (UPR). UPR pathways, including PERK-eIF2α and IRE1-XBP1, were dysregulated under sustained oxidative stress, impairing insulin synthesis and protein folding in β-cells (Zorena et al., 2022). Chronic ROS exposure also activated inflammatory signaling cascades within β-cells, ultimately inducing apoptosis and reducing insulin secretory capacity by up to 30% following prolonged PM10 exposure (Liu et al., 2016).

PM10 exposure adversely affected adipose tissue function through systemic and local inflammatory responses. Circulating PM10 particles infiltrated adipose tissue, activating resident macrophages and increasing the production of pro-inflammatory cytokines. Della Guardia and Shin (2022) observed a 40% rise in inflammatory markers, including IL-6 and TNF-α, within adipose tissue following chronic PM10 exposure. This local inflammation altered adipokine production, characterized by increased leptin and decreased adiponectin levels. Reduced adiponectin impairs insulin sensitivity by diminishing AMP-activated protein kinase (AMPK) activity, which was critical for glucose uptake and fatty acid oxidation (Della Guardia and Shin, 2022). Conversely, elevated leptin levels exacerbated central leptin resistance, which indirectly worsened systemic insulin resistance.

Lastly, our study explored the association between other, less frequently studied pollutants (i.e., PM1, O3, SO2, NOx) and T2DM risk. These pollutants have received less attention in the literature, and this study contributes new data on their potential metabolic impacts. However, we recognize that there are limitations in this study. For example, factors like gender and comorbid conditions were not fully explored, despite evidence suggesting that females may be more susceptible to air pollution-induced T2DM in some regions (Lao et al., 2019; Liu et al., 2023). Additionally, the limited number of primary studies on pollutants like PM10 and NO2 makes interpreting subgroup results more challenging. The persistent high heterogeneity observed after subgroup analysis further indicates the need for standardized air pollution exposure methods, as inconsistencies in measurement likely contributed to the variability in findings. Finally, given that T2DM is a chronic disease, it may take years to develop, limiting the ability of some studies to fully capture long-term impacts. Further research is necessary to examine the severity of T2DM outcomes, particularly as prolonged exposure to pollutants may increase mortality risks (Wu et al., 2022).

Theses umbrella review and meta-analysis helped providing important new insights into the relationship between outdoor air pollution and Type 2 Diabetes Mellitus (T2DM), expanding from the findings of previous systematic reviews and meta-analyses This study strength lied in a more detailed analysis of the heterogeneity between studies and the dose-response relationship of an outdoor air pollution exposure such as PM10 on the risk of T2DM, which has not been thoroughly explored in prior studies. Similar to prior studies, we found significant associations between exposure to major air pollutants and the risk of developing T2DM. However, our study had further explored the heterogeneity between studies across different study designs, exposure regions, and population characteristics, which has not been fully explored in previous studies. This study also highlighted the importance of tailoring public health interventions to specific regions. Stronger associations between PM10 and T2DM in Asia suggest the need for targeted pollution reduction policies in industrialized areas. Methodologically, this study's use of both umbrella review and meta-analysis approaches strengthened the robustness of the findings. While previous studies primarily used meta-analysis, our dual approach allowed for a critical evaluation of existing reviews alongside a synthesis of primary studies. By addressing limitations in prior reviews and incorporating the latest data, this study offered a more comprehensive understanding of air pollution's impact on T2DM.

5. Conclusion

From both umbrella review and meta-analysis of this study, an outdoor exposure to PM2.5, PM10, NO2, and O3 was found to be significantly associated with the risk of T2DM. Additionally, the subgroup analyses of PM2.5, PM10, and NO2 exposures revealed a complex relationship between outdoor air pollution exposure and the likelihood of developing T2DM that might be influenced or confounded by these factors. The pooled estimates results, especially for PM10 and NO2 were also close to the previous umbrella studies' results, which further supported and strengthened existing evidence of the risk of T2DM due to an outdoor air pollution exposure despite the evidence of high heterogeneity between studies.

This study highlighted the significance of air pollution exposure as a public health matter, specifically regarding chronic diseases such as T2DM. This emphasized the need for a more specific and comprehensive approach to identify the fundamental mechanisms of these relationships to help lawmakers implement more efficient public health policies and interventions. Moreover, the diversity in the effects of air pollutants on different demographic groups and geographical areas may require location-specific approaches. The results of this study provided a strong incentive for policymakers and health practitioners to incorporate environmental factors into their strategies to address the increasing occurrence of T2DM and air pollution exposure.

CRediT authorship contribution statement

Nichapa Parasin: Writing – original draft, Methodology, Investigation, Conceptualization. **Teerachai Amnuaylojaroen:** Writing – original draft, Software, Formal analysis, Conceptualization. **Surasak Saokaew:** Validation, Supervision, Methodology, Conceptualization. **Nuttawut Sittichai:** Methodology, Investigation, Data curation. **Natcha Tabkhan:** Writing – review & editing, Visualization, Data curation, Conceptualization. **Piyameth Dilokthornsakul:** Writing – review & editing, Visualization, Validation, Funding acquisition.

Declaration of competing interest

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envres.2025.120885.

Data availability

Data will be made available on request.

References

Andersen, Z.J., Raaschou-Nielsen, O., Ketzel, M., Jensen, S.S., Hvidberg, M., Loft, S., Tjønneland, A., Overvad, K., Sørensen, M., 2012. Diabetes incidence and long-term exposure to air pollution: a cohort study. Diabetes Care 35 (1), 92–98. https://doi.org/10.2337/dc11-1155.

Aromataris, E., Fernandez, R., Godfrey, C.M., Holly, C., Khalil, H., Tungpunkom, P., 2014. Methodology for JBI umbrella reviews. Faculty of Science, Medicine and Health - Papers: part A 3344. https://ro.uow.edu.au/smhpapers/3344.

Bai, L., Chen, H., Hatzopoulou, M., Jerrett, M., Kwong, J.C., Burnett, R.T., van Donkelaar, A., Copes, R., Martin, R.V., Van Ryswyk, K., Lu, H., Kopp, A., Weichenthal, S., 2018. Exposure to ambient ultrafine particles and nitrogen dioxide and incident hypertension and diabetes. Epidemiology 29 (3), 323–332. https://doi.org/10.1097/EDE.0000000000000798.

Balti, E.V., Echouffo-Tcheugui, J.B., Yako, Y.Y., Kengne, A.P., 2014. Air pollution and risk of type 2 diabetes mellitus: a systematic review and meta-analysis. Diabetes Res. Clin. Pract. 106 (2), 161–172. https://doi.org/10.1016/j.diabres.2014.08.010.

Bo, Y., Chang, L.Y., Guo, C., Lin, C., Lau, A.K.H., Tam, T., Yeoh, E.K., Lao, X.Q., 2021. Associations of reduced ambient PM2.5 level with lower plasma glucose concentration and decreased risk of type 2 diabetes in adults: a longitudinal cohort study. Am. J. Epidemiol. 190 (10), 2148–2157. https://doi.org/10.1093/aje/kwab159.

Bowe, B., Xie, Y., Li, T., Yan, Y., Xian, H., Al-Aly, Z., 2018. The 2016 global and national burden of diabetes mellitus attributable to $PM_{2.5}$ air pollution. Lancet Planet. Health 2 (7), e301–e312. https://doi.org/10.1016/S2542-5196(18)30140-2.

Brook, R.D., Jerrett, M., Brook, J.R., Bard, R.L., Finkelstein, M.M., 2008. The relationship between diabetes mellitus and traffic-related air pollution. J. Occup. Environ. Med. 50 (1), 32–38. https://doi.org/10.1097/JOM.0b013e31815dba70.

Cai, H., Du, Z., Lin, X., Lawrence, W.R., Hopke, P.K., Rich, D.Q., Lin, S., Xiao, J., Deng, X., Qu, Y., Lin, Z., Wang, X., Ju, X., Chen, S., Zhang, Y., Wu, W., Wang, Y., Gu, J., Hao, Y., Zhang, W., 2023. Interactions between long-term ambient particle exposures and lifestyle on the prevalence of hypertension and diabetes: insight from a large community-based survey. J. Epidemiol. Community 77 (7), 440–446. https://doi.org/10.1136/jech-2023-220480.

Campbell, M., McKenzie, J.E., Sowden, A., Katikireddi, S.V., Brennan, S.E., Ellis, S., Hartmann-Boyce, J., Ryan, R., Shepperd, S., Thomas, J., Welch, V., Thomson, H., 2020. Synthesis without meta-analysis (SWIM) in systematic reviews: reporting guideline. *BMJ*, l6890. https://doi.org/10.1136/bmj.l6890.

Chen, H., Burnett, R.T., Kwong, J.C., Villeneuve, P.J., Goldberg, M.S., Brook, R.D., van Donkelaar, A., Jerrett, M., Martin, R.V., Brook, J.R., Copes, R., 2013. Risk of incident diabetes in relation to long-term exposure to fine particulate matter in Ontario, Canada. Environ. Health Perspect. 121 (7), 804–810. https://doi.org/10.1289/ehp.1205958.

Chen, Z.H., Zhao, Z., Deng, C.W., Li, N.S., 2022a. Association between air pollution and type 2 diabetes mellitus in developing countries: a systematic review and meta-analysis. Chinese medical sciences journal = Chung-kuo i hsueh k'o hsueh tsa chih 37 (3), 218–227. https://doi.org/10.24920/004065.

Chen, Z., Salam, M.T., Toledo-Corral, C., Watanabe, R.M., Xiang, A.H., 2022b. Impacts of air pollution on type 2 diabetes: a systematic review and meta-analysis. Environ. Health 21 (1), 42.

Chilian-Herrera, O.L., Tamayo-Ortiz, M., Texcalac-Sangrador, J.L., Rothenberg, S.J., López-Ridaura, R., Romero-Martínez, M., Wright, R.O., Just, A.C., Kloog, I., Bautista-Arredondo, L.F., Téllez-Rojo, M.M., 2021. $PM_{2.5}$ exposure as a risk factor for type 2 diabetes mellitus in the Mexico City metropolitan area. BMC public health, 21(1) 2087. https://doi.org/10.1186/s12889-021-12112-w.

Clark, C., Sbihi, H., Tamburic, L., Brauer, M., Frank, L.D., Davies, H.W., 2017. Association of long-term exposure to transportation noise and traffic-related air pollution with the incidence of diabetes: a prospective cohort study. Environmental health perspectives, 125(8) 87025. https://doi.org/10.1289/EHP1279.

Coogan, P.F., White, L.F., Jerrett, M., Brook, R.D., Su, J.G., Seto, E., Burnett, R., Palmer, J.R., Rosenberg, L., 2012. Air pollution and incidence of hypertension and diabetes mellitus in black women living in Los Angeles. Circulation 125 (6), 767–772. https://doi.org/10.1161/CIRCULATIONAHA.111.052753.

Coogan, P.F., White, L.F., Yu, J., Burnett, R.T., Marshall, J.D., Seto, E., Brook, R.D., Palmer, J.R., Rosenberg, L., Jerrett, M., 2016a. Long term exposure to NO2 and diabetes incidence in the Black Women's Health Study. Environ. Res. 148, 360–366. https://doi.org/10.1016/j.envres.2016.04.021.

Coogan, P.F., White, L.F., Yu, J., Burnett, R.T., Seto, E., Brook, R.D., Palmer, J.R., Rosenberg, L., Jerrett, M., 2016b. PM2.5 and diabetes and hypertension incidence in the black women's health study. Epidemiology 27 (2), 202–210. https://doi.org/10.1097/EDE.0000000000000418.

Cox Jr., T.L.A., 2016. Rethinking the meaning of concentration-response functions and the estimated burden of adverse health effects attributed to exposure concentrations. Risk Anal. 36 (9), 1770–1779. https://doi.org/10.1111/risa.12670.

Deeks, J.J., Higgins, J., Altman, D.G., 2023. Chapter 10: analysing data and undertaking meta-analyses. In: Higgins, J., Thomas, J., Chandler, J., Cumpston, M., Li, T., Page, M., Welch, V. (Eds.), Cochrane Handbook for Systematic Reviews of Interventions. Cochrane Training. Sterne 2011 version 6.4.

Della Guardia, L., Shin, A.C., 2022. White and brown adipose tissue functionality is impaired by fine particulate matter (PM2.5) exposure. J. Mol. Med. (Berl.) 100 (5), 665–676. https://doi.org/10.1007/s00109-022-02183-6.

Dijkema, M.B., Mallant, S.F., Gehring, U., van den Hurk, K., Alssema, M., van Strien, R, T., Fischer, P.H., Nijpels, G., Stehouwer, C.D., Hoek, G., Dekker, J.M., Brunekreef, B., 2011. Long-term exposure to traffic-related air pollution and type 2 diabetes prevalence in a cross-sectional screening-study in The Netherlands. *Environmental health*. a global access science source 10 (76). https://doi.org/10.1186/1476-069X-10-76.

Dzhambov, A.M., Dimitrova, D.D., 2016. Exposures to road traffic, noise, and air pollution as risk factors for type 2 diabetes: a feasibility study in Bulgaria. Noise Health 18 (82), 133–142. https://doi.org/10.4103/1463-1741.181996.

Eze, I.C., Schaffner, E., Fischer, E., Schikowski, T., Adam, M., Imboden, M., Tsai, M., Carballo, D., von Eckardstein, A., Künzli, N., Schindler, C., Probst-Hensch, N., 2014. Long-term air pollution exposure and diabetes in a population-based Swiss cohort. Environ. Int. 70, 95–105. https://doi.org/10.1016/j.envint.2014.05.014.

Eze, I.C., Hemkens, L.G., Bucher, H.C., Hoffmann, B., Schindler, C., Künzli, N., Schikowski, T., Probst-Hensch, N.M., 2015a. Association between ambient air pollution and diabetes mellitus in Europe and North America: systematic review and meta-analysis. Environ. Health Perspect. 123 (5), 381–389. https://doi.org/10.1289/ehp.1307823.

Eze, I.C., Schaffner, E., Foraster, M., Imboden, M., von Eckardstein, A., Kunzli, N., et al., 2015b. Long-term exposure to ambient air pollution and metabolic syndrome in adults. PLoS One 10 (6), e0130337.

Eze, I.C., Foraster, M., Schaffner, E., Vienneau, D., Héritier, H., Rudzik, F., Thiesse, L., Pieren, R., Imboden, M., von Eckardstein, A., Schindler, C., Brink, M., Cajochen, C., Wunderli, J.M., Röösli, M., Probst-Hensch, N., 2017. Long-term exposure to transportation noise and air pollution in relation to incident diabetes in the SAPALDIA study. Int. J. Epidemiol. 46 (4), 1115–1125. https://doi.org/10.1093/ije/dyx020.

Hansen, A.B., Ravnskjær, L., Loft, S., Andersen, K.K., Bräuner, E.V., Baastrup, R., Yao, C., Ketzel, M., Becker, T., Brandt, J., Hertel, O., Andersen, Z.J., 2016. Long-term exposure to fine particulate matter and incidence of diabetes in the Danish Nurse Cohort. Environ. Int. 91, 243–250. https://doi.org/10.1016/j.envint.2016.02.036.

Harrell, F.E., 2010. In: Regression Modeling Strategies: with Applications to Linear Models, Logistic and Ordinal Regression, and Survival Analysis, first ed. Springer New York https://link.springer.com/book/10.1007/978-1-4757-3462-1.

He, D., Wu, S., Zhao, H., Qiu, H., Fu, Y., Li, X., He, Y., 2017. Association between particulate matter 2.5 and diabetes mellitus: a meta-analysis of cohort studies. Journal of diabetes investigation 8 (5), 687–696. https://doi.org/10.1111/jdi.12631.

Honda, T., Pun, V.C., Manjourides, J., Suh, H., 2017. Associations between long-term exposure to air pollution, glycosylated hemoglobin and diabetes. Int. J. Hyg Environ. Health 220 (7), 1124–1132. https://doi.org/10.1016/j.ijheh.2017.06.004.

International Diabetes Federation, 2021. IDF Diabetes Atlas 2021.

Jabbari, F., Mohseni Bandpei, A., Daneshpour, M.S., Shahsavani, A., Nazari, Hashemi, S, S., Faraji Sabokbar, H., Momenan, A.A., Azizi, F., 2020. Role of air pollution and rs10830963 polymorphism on the incidence of type 2 diabetes: Tehran cardiometabolic genetic study. J. Diabetes Res. 2020, 2928618.

Janghorbani, M., Momeni, F., Mansourian, M., 2014. Systematic review and metaanalysis of air pollution exposure and risk of diabetes. Eur. J. Epidemiol. 29 (4), 231–242. https://doi.org/10.1007/s10654-014-9907-2.

Jion, M.M.M.F., Jannat, J.N., Mia, M.Y., Ali, M.A., Islam, M.S., Ibrahim, S.M., Pal, S.C., Islam, A., Sarker, A., Malafaia, G., Bilal, M., Islam, A.R.M.T., 2023. A critical review and prospect of NO_2 and SO_2 pollution over Asia: hotspots, trends, and sources. Sci. Total Environ. 876, 162851.

Krämer, U., Herder, C., Sugiri, D., Strassburger, K., Schikowski, T., Ranft, U., Rathmann, W., 2010. Traffic-related air pollution and incident type 2 diabetes: results from the SALIA cohort study. Environ. Health Perspect. 118 (9), 1273–1279. https://doi.org/10.1289/ehp.0901689.

Langendam, M.W., Akl, E.A., Dahm, P., Glasziou, P., Guyatt, G., Schünemann, H.J., 2013. Assessing and presenting summaries of evidence in cochrane reviews. *Systematic reviews, 2*(1). https://doi.org/10.1186/2046-4053-2-81.

Lao, X.Q., Guo, C., Chang, L.Y., Bo, Y., Zhang, Z., Chuang, Y.C., Jiang, W.K., Lin, C., Tam, T., Lau, A.K.H., Lin, C.Y., Chan, T.C., 2019. Long-term exposure to ambient fine particulate matter (PM2.5) and incident type 2 diabetes: a longitudinal cohort study. Diabetologia 62 (5), 759–769. https://doi.org/10.1007/s00125-019-4825-1.

Lazarevic, N., Dobson, A.J., Barnett, A.G., Knibbs, L.D., 2015. Long-term ambient air pollution exposure and self-reported morbidity in the Australian Longitudinal Study on Women's Health: a cross-sectional study. BMJ Open 5 (10), e008714. https://doi.org/10.1136/bmjopen-2015-008714.

Lin, J., Zheng, H., Xia, P., Cheng, X., Wu, W., Li, Y., Ma, C., Zhu, G., Xu, T., Zheng, Y., Qiu, L., Chen, L., 2021. Long-term ambient $PM_{2.5}$ exposure associated with cardiovascular risk factors in Chinese less educated population. BMC public health, 21(1) 2241. https://doi.org/10.1186/s12889-021-12163-z.

Liu, C., Yang, C., Zhao, Y., Ma, Z., Bi, J., Liu, Y., Meng, X., Wang, Y., Cai, J., Chen, R., Kan, H., 2016. Associations between long-term exposure to ambient particulate air pollution and type 2 diabetes prevalence, blood glucose, and glycosylated hemoglobin levels in China. Environ. Int. 92–93, 416–421. https://doi.org/10.1016/j.envint.2016.03.028.

Liu, F., Chen, G., Huo, W., Wang, C., Liu, S., Li, N., Mao, S., Hou, Y., Lu, Y., Xiang, H., 2019a. Associations between long-term exposure to ambient air pollution and risk of

type 2 diabetes mellitus: a systematic review and meta-analysis. Environ. Pollut. 252 (Pt B), 1235–1245. https://doi.org/10.1016/j.envpol.2019.06.033.
Liu, F., Guo, Y., Liu, Y., Chen, G., Wang, Y., Xue, X., Liu, S., Huo, W., Mao, Z., Hou, Y., Lu, Y., Wang, C., Xiang, H., Li, S., 2019b. Associations of long-term exposure to PM_1, $PM_{2.5}$, NO_2 with type 2 diabetes mellitus prevalence and fasting blood glucose levels in Chinese rural populations. Environ. Int. 133, 105213. https://doi.org/10.1016/j.envint.2019.105213. Pt B.
Liu, C., Chan, K.H., Lv, J., Lam, H., Newell, K., Meng, X., Liu, Y., Chen, R., Kartsonaki, C., Wright, N., Du, H., 2022. Long-term exposure to ambient fine particulate matter and incidence of major cardiovascular diseases: a prospective study of 0.5 million adults in China. Environ. Sci. Technol. 56 (18), 13200–13211.
Liu, C., Cao, G., Li, J., Lian, S., Zhao, K., Zhong, Y., Xu, J., Chen, Y., Bai, J., Feng, H., He, G., Dong, X., Yang, P., Zeng, F., Lin, Z., Zhu, S., Zhong, X., Ma, W., Liu, T., 2023. Effect of long-term exposure to PM2.5 on the risk of type 2 diabetes and arthritis in type 2 diabetes patients: evidence from a national cohort in China. Environ. Int. 171 (107741). https://doi.org/10.1016/j.envint.2023.107741.
Lusa, L., Ahlin, Č., 2020. Restricted cubic splines for modelling periodic data. PLoS One 15 (10), e0241364. https://doi.org/10.1371/journal.pone.0241364.
Manisalidis, I., Stavropoulou, E., Stavropoulos, A., Bezirtzoglou, E., 2020. Environmental and health impacts of air pollution: a review. Front. Public Health 8. https://doi.org/10.3389/fpubh.2020.00014.
McMaster University, 2021. GRADEpro. McMaster University. Version 3.6.1. www.gradepro.org.
National Institute of Environmental Health Sciences, 2001. Air Pollution and Your Health. National Institutes of Health. https://www.niehs.nih.gov/health/topics/agents/air-pollution/index.cfm.
Nishida, C., Yatera, K., 2022. The impact of ambient environmental and occupational pollution on respiratory diseases. Int. J. Environ. Res. Publ. Health 19 (5), 2788. https://doi.org/10.3390/ijerph19052788.
Orioli, R., Cremona, G., Ciancarella, L., Solimini, A.G., 2018. Association between PM10, PM2.5, NO2, O3 and self-reported diabetes in Italy: a cross-sectional, ecological study. PLoS One 13 (1), e0191112. https://doi.org/10.1371/journal.pone.0191112.
Page, M.J., McKenzie, J.E., Bossuyt, P.M., Boutron, I., Hoffmann, T.C., Mulrow, C.D., Shamseer, L., Tetzlaff, J.M., Akl, E.A., Brennan, S.E., Chou, R., Glanville, J., Grimshaw, J.M., Hróbjartsson, A., Lalu, M.M., Li, T., Loder, E.W., Mayo-Wilson, E., McDonald, S., McGuinness, L.A., Moher, D., 2021. The PRISMA 2020 statement: an updated guideline for reporting systematic reviews. Br. Med. J. 372, n71. https://doi.org/10.1136/bmj.n71.
Park, S.K., Adar, S.D., O'Neill, M.S., Auchincloss, A.H., Szpiro, A., Bertoni, A.G., Navas-Acien, A., Kaufman, J.D., Diez-Roux, A.V., 2015. Long-term exposure to air pollution and type 2 diabetes mellitus in a multiethnic cohort. Am. J. Epidemiol. 181 (5), 327–336. https://doi.org/10.1093/aje/kwu280.
Paul, L.A., Burnett, R.T., Kwong, J.C., Hystad, P., van Donkelaar, A., Bai, L., Goldberg, M. S., Lavigne, E., Copes, R., Martin, R.V., Kopp, A., Chen, H., 2020. The impact of air pollution on the incidence of diabetes and survival among prevalent diabetes cases. Environ. Int. 134 (105333). https://doi.org/10.1016/j.envint.2019.105333.
Puett, R.C., Hart, J.E., Schwartz, J., Hu, F.B., Liese, A.D., Laden, F., 2011. Are particulate matter exposures associated with risk of type 2 diabetes? Environ. Health Perspect. 119 (3), 384–389. https://doi.org/10.1289/ehp.1002344.
Qiu, H., Schooling, C.M., Sun, S., Tsang, H., Yang, Y., Lee, R.S., Wong, C.M., Tian, L., 2018. Long-term exposure to fine particulate matter air pollution and type 2 diabetes mellitus in elderly: a cohort study in Hong Kong. Environ. Int. 113, 350–356. https://doi.org/10.1016/j.envint.2018.01.008.
Rajagopalan, S., Brook, R.D., 2012. Air pollution and type 2 diabetes: mechanistic insights. Diabetes 61 (12), 3037–3045. https://doi.org/10.2337/db12-0190.
Ren, Z., Yuan, J., Luo, Y., Wang, J., Li, Y., 2023. Association of air pollution and fine particulate matter (PM2.5) exposure with gestational diabetes: a systematic review and meta-analysis. Ann. Transl. Med. 11 (1), 23. https://doi.org/10.21037/atm-22-6306.
Renzi, M., Cerza, F., Gariazzo, C., Agabiti, N., Cascini, S., Di Domenicantonio, R., Davoli, M., Forastiere, F., Cesaroni, G., 2018. Air pollution and occurrence of type 2 diabetes in a large cohort study. Environ. Int. 112, 68–76. https://doi.org/10.1016/j.envint.2017.12.007.
Riant, M., Meirhaeghe, A., Giovannelli, J., Occelli, F., Havet, A., Cuny, D., Amouyel, P., Dauchet, L., 2018. Associations between long-term exposure to air pollution, glycosylated hemoglobin, fasting blood glucose and diabetes mellitus in northern France. Environ. Int. 120, 121–129. https://doi.org/10.1016/j.envint.2018.07.034.
Rothstein, H.R., Sutton, A.J., Borenstein, M., 2005. Publication bias in meta-analysis. In wiley eBooks. https://doi.org/10.1002/0470870168.
Saokaew, S., Kanchanasurakit, S., Thawichai, K., Duangprom, P., Wannasri, M., Khankham, S., Kositamongkol, C., Chaiyakunapruk, N., Phisalprapa, P., 2021. Association of non-alcoholic fatty liver disease and all-cause mortality in hospitalized cardiovascular disease patients: a systematic review and meta-analysis. Medicine 100 (5), e24557. https://doi.org/10.1097/MD.0000000000024557.
Shan, A., Zhang, Y., Zhang, L.W., Chen, X., Li, X., Wu, H., Yan, M., Li, Y., Xian, P., Ma, Z., Li, C., Guo, P., Dong, G.H., Liu, Y.M., Chen, J., Wang, T., Zhao, B.X., Tang, N.J., 2020. Associations between the incidence and mortality rates of type 2 diabetes mellitus and long-term exposure to ambient air pollution: a 12-year cohort study in northern China. Environ. Res. 186 (109551). https://doi.org/10.1016/j.envres.2020.109551.
Shea, B., Reeves, B.C., Wells, G., Thuku, M., Hamel, C., Moran, J., et al., 2017. Amstar 2: a critical appraisal tool for systematic reviews that include randomised or non-randomised studies of healthcare interventions, or both. BMJ 358, j4008. https://doi.org/10.1136/bmj.j4008.
Shi, L., Lin, L., 2019. The trim-and-fill method for publication bias: practical guidelines and recommendations based on a large database of meta-analyses. Medicine 98 (23), e15987. https://doi.org/10.1097/MD.0000000000015987.
Simoni, M., Jaakkola, M.S., Carrozzi, L., Baldacci, S., Di Pede, F., Viegi, G., 2003. Indoor air pollution and respiratory health in the elderly. Eur. Respir. J. Suppl. 40. https://doi.org/10.1183/09031936.03.00403603, 15s–20s.
Simoni, M., Baldacci, S., Maio, S., Cerrai, S., Sarno, G., Viegi, G., 2015. Adverse effects of outdoor pollution in the elderly. J. Thorac. Dis. 7 (1), 34–45. https://doi.org/10.3978/j.issn.2072-1439.2014.12.10.
StataCorp, 2015. Stata Statistical Software. [Computer software]. StataCorp LLC. Version 14.0.
Strak, M., Janssen, N., Beelen, R., Schmitz, O., Vaartjes, I., Karssenberg, D., van den Brink, C., Bots, M.L., Dijst, M., Brunekreef, B., Hoek, G., 2017. Long-term exposure to particulate matter, NO_2 and the oxidative potential of particulates and diabetes prevalence in a large national health survey. Environ. Int. 108, 228–236. https://doi.org/10.1016/j.envint.2017.08.017.
Sun, J., Fang, R., Wang, H., Xu, D.X., Yang, J., Huang, X., Cozzolino, D., Fang, M., Huang, Y., 2022. A review of environmental metabolism disrupting chemicals and effect biomarkers associating disease risks: where exposomics meets metabolomics. Environ. Int. 158 (106941). https://doi.org/10.1016/j.envint.2021.106941.
United States Environmental Protection Agency (n.d.). Reducing Emissions from Hazardous Air Pollutants. https://www.epa.gov/haps/reducing-emissions-hazardous-air-pollutants#:~:text=Since%201990%2C%20EPA%20has%20issued, aerospace%20manufacturers%2C%20and%20steel%20mills.
Wang, B., Xu, D., Jing, Z., Liu, D., Yan, S., Wang, Y., 2014. Effect of long-term exposure to air pollution on type 2 diabetes mellitus risk: a systemic review and meta-analysis of cohort studies. Eur. J. Endocrinol. 171 (5), R173–R182. https://doi.org/10.1530/EJE-14-0365.
Wang, M., Jin, Y., Dai, T., Yu, C., Zheng, S., Nie, Y., Bai, Y., 2020. Association between ambient particulate matter (PM10) and incidence of diabetes in northwest of China: a prospective cohort study. Ecotoxicol. Environ. Saf. 202 (110880). https://doi.org/10.1016/j.ecoenv.2020.110880.
Wang, M., Zhou, T., Song, Y., Li, X., Ma, H., Hu, Y., Heianza, Y., Qi, L., 2021. Joint exposure to various ambient air pollutants and incident heart failure: a prospective analysis in UK Biobank. Eur. Heart J. 42 (16), 1582–1591. https://doi.org/10.1093/eurheartj/ehaa1031.
Weinmayr, G., Hennig, F., Fuks, K., Nonnemacher, M., Jakobs, H., Möhlenkamp, S., Erbel, R., Jöckel, K.H., Hoffmann, B., Moebus, S., Heinz Nixdorf Recall Investigator Group, 2015. Long-term exposure to fine particulate matter and incidence of type 2 diabetes mellitus in a cohort study: effects of total and traffic-specific air pollution. Environmental health. a global access science source 14 (53). https://doi.org/10.1186/s12940-015-0031-x.
Wells, G.A., Wells, G., Shea, B., Shea, B., O'Connell, D., Peterson, J., Welch Losos, M., Tugwell, P., Ga, S.W., Zello, G.A., Petersen, J.A., 2014. The Newcastle-Ottawa Scale (NOS) for Assessing the Quality of Nonrandomised Studies in Meta-Analyses.
Wu, Y., Zhang, S., Qian, S.E., Cai, M., Li, H., Wang, C., Zou, H., Chen, L., Vaughn, M.G., McMillin, S.E., Lin, H., 2022. Ambient air pollution associated with incidence and dynamic progression of type 2 diabetes: a trajectory analysis of a population-based cohort. BMC Med. 20 (1), 375. https://doi.org/10.1186/s12916-022-02573-0.
Yang, B.Y., Qian, Z.M., Li, S., Chen, G., Bloom, M.S., Elliott, M., Syberg, K.W., Heinrich, J., Markevych, I., Wang, S.Q., Chen, D., Ma, H., Chen, D.H., Liu, Y., Komppula, M., Leskinen, A., Liu, K.K., Zeng, X.W., Hu, L.W., Guo, Y., Dong, G.H., 2018a. Ambient air pollution in relation to diabetes and glucose-homoeostasis markers in China: a cross-sectional study with findings from the 33 Communities Chinese Health Study. The Lancet. Planetary health 2 (2), e64–e73. https://doi.org/10.1016/S2542-5196(18)30001-9.
Yang, Y., Guo, Y., Qian, Z.M., Ruan, Z., Zheng, Y., Woodward, A., Ai, S., Howard, S.W., Vaughn, M.G., Ma, W., Wu, F., Lin, H., 2018b. Ambient fine particulate pollution associated with diabetes mellitus among the elderly aged 50 years and older in China. Environ. Pollut. 243 (Pt B), 815–823.
Yang, B.Y., Fan, S., Thiering, E., Seissler, J., Nowak, D., Dong, G.H., Heinrich, J., 2020. Ambient air pollution and diabetes: a systematic review and meta-analysis. Environ. Res. 180 (108817). https://doi.org/10.1016/j.envres.2019.108817.
Yitshak Sade, M., Shi, L., Colicino, E., Amini, H., Schwartz, J.D., Di, Q., Wright, R.O., 2023. Long-term air pollution exposure and diabetes risk in American older adults: a national secondary data-based cohort study. Environ. Pollut. 320 (121056). https://doi.org/10.1016/j.envpol.2023.121056.
Zhang, Q., Liu, C., Wang, Y., Gong, J., Wang, G., Ge, W., Chen, R., Meng, X., Zhao, Y., Kan, H., 2021. Associations of long-term exposure to ambient nitrogen dioxide with indicators of diabetes and dyslipidemia in China: a nationwide analysis. Chemosphere 269 (128724). https://doi.org/10.1016/j.chemosphere.2020.128724.
Zhou, P., Mo, S., Peng, M., Yang, Z., Wang, F., Hu, K., Zhang, Y., 2022. Long-term exposure to $PM_{2.5}$ constituents in relation to glucose levels and diabetes in middle-aged and older Chinese. Ecotoxicol. Environ. Saf. 245, 114096. https://doi.org/10.1016/j.ecoenv.2022.114096.
Zorena, K., Jaskulak, M., Michalska, M., Mrugacz, M., Vandenbulcke, F., 2022. Air pollution, oxidative stress, and the risk of development of type 1 diabetes. Antioxidants (Basel), 11(10) 1908. https://doi.org/10.3390/antiox11101908.