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**Review Article**

# Type 2 diabetes and the urban exposome: role of air pollution, noise, and built environment in the risk of type 2 diabetes: systematic review and meta-analysis

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## Abstract

Air pollution, noise, and built environment are associated with the epidemics of type 2 diabetes (T2D). The extent to which these have independent and/or joint effects on T2D and whether some components of the urban exposome have stronger effects remains unclear. We conducted a systematic review of the associations of 11 environmental exposures of urban exposome with the risk of T2D. We searched PubMed and Scopus since 2005 until January 2025 for studies on association of T2D in adults with air pollution; particles with a diameter of less than 2.5 ($PM_{2.5}$) and 10 μm ($PM_{10}$), nitrogen dioxide ($NO_2$), ozone ($O_3$) and black carbon (BC), noise; traffic-, railway-, and aircraft noise, and built environment; greenness, walkability, and population density. We included 151 articles, one study referring to exposome approach. Air pollutants were associated with T2D risk in meta-analyses, BC showing strongest association, OR: 1.32, 95% CI: 1.15-1.50 (n = 8). Subgroup analyses and meta-regression for $PM_{2.5}$, $PM_{10}$, $NO_2$, and $O_3$ by study characteristics highlighted variations in risk estimates but didn't explain considerable heterogeneity. Traffic noise was associated with T2D (OR: 1.06, 95% CI: 1.03, 1.08, n = 11). In qualitative synthesis, living environment with higher walkability and greenness showed inverse association with T2D. Results indicate that air pollution and traffic noise are associated with increased risk of T2D. Greener and walkable living environment can potentially reduce risk of T2D. It remained unclear whether the effects were independent. Future studies should consider environmental joint exposures. Advancing use of exposome approach can help understand T2D risk comprehensively.

**Key words:** exposome; type 2 diabetes; air pollution; traffic noise; built environment; meta-analysis.

## Introduction

Despite existing research knowledge and the implementation of public health interventions addressing lifestyle factors associated with type 2 diabetes (T2D), such as smoking, unhealthy diet, and sedentary behavior,[1] the global prevalence of diabetes continues to increase.[2] It is forecasted that 853 million people will be living with diabetes by 2050, 90% of whom will consist of adults with T2D.[2] Urban living environments are increasingly recognized as risk factors for T2D, and urban design-based interventions have been proposed as a promising approach to public health.

Air pollution, in particular, has been a key focus in diabetes epidemiology, accounting for approximately 40% of studies on environmental determinants of diabetes.[3] It is estimated that approximately a fifth of the global burden of T2D is attributable to air pollution, 13.4% from ambient $PM_{2.5}$ and 6.5% from household air pollution.[4] Previous reviews have shown a relationship between air pollution and the risk of T2D but have focused on single pollutants.[5-9] Many studies consider mainly $PM_{2.5}$, and the evidence for less studied air pollutants such as Ozone ($O_3$) or Black Carbon (BC) is still scarce.[3] Environmental noise exposure refers to any unwanted noise created by human activities that are harmful to human health and quality of life.[10] Dzhambov et al. found that people exposed to high noise levels at home might be at higher risk (19% - 22%) for developing T2D.[11] Sakhvidi et al. reported an association between aircraft noise (OR: 1.17, 95% CI: 1.06, 1.29, n = 4) and road traffic noise (OR: 1.07, 95% CI: 1.02-, 1.12, n = 3) with T2D, but no association was observed for railway noise exposure.[12] The number of studies in these reviews is small, and the research evidence on the relationship between source-specific noise exposures and T2D is still limited.

Green space is a common measure of the built environment, which refers to any land covered with grass, trees, plants, or other vegetation.[13] Walkability is another common measure of the built environment. It can be composed of several indexes such as population density, street connectivity, or the number of walkable destinations such as access to parks, public transport, or food outlets.[14] The possible relationship between green space and T2D has been reviewed in a systematic review by De la Fuente et al.[15] who found seven studies assessing the risk of T2D in adult populations and a systematic review and meta-analysis of three studies by Sharifi et al.[13] Both supported the hypothesis that people exposed to more green spaces have a reduced risk of T2D.

There is robust evidence that urban environmental exposures influencing the risk of T2D in a population cannot be reduced to

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only one of its components. These exposures rarely exist in isolation and influence the risk of chronic diseases like T2D within the broader context as a complex system. The concept of exposome incorporates these characteristics. It was introduced in 2005 to synthesize ideas brought forward by scientific frameworks, such as The Human Genome Project,[16] The Social Determinants of Health,[17] and the Environmental Cause of Diseases.[18] Exposome refers to the totality of environmental exposures, a compilation of all physical, chemical, biological, and (psycho) social influences that "impact biology".[19] The concept of exposome has since played a key role in shaping a more holistic approach to environmental influences on health, enabling a more comprehensive understanding of disease etiology.[20] The holistic hypothesis posits that individual environmental factors are interconnected, and their effects on health can only be fully understood in the context of the whole system. Review articles analyzing the exposome approach in the context of cardiometabolic health in general have been published.[21-24] Yet, to date, there is a lack of systematic reviews and meta-analyses examining both the individual and combined effects of urban exposome components on T2D in the post-exposome era.

Adopting the holistic hypothesis, the aim of the current study was to systematically review and analyze existing research on the association between the physical environment constituting the urban exposome and its association with T2D. We identified and included three distinct environmental exposure groups that may influence the risk of T2D: air pollution, noise, and the built environment. We utilized the PECOS framework to create the search strategy and research question. It defines the Population, Exposure, Comparator, Outcomes, and Study Design as pillars of the review question and is increasingly used in the field of environmental health.[25] The PECOS framework for this review is available in Table S1. The defined PECOS research question is as follows: Among the general adult population (P) what is the effect of environmental exposures (air pollution, noise, and built environment) and their joint effect (E/C) on the risk of type 2 diabetes (O) in observational studies (S)?

## Methods

This systematic review and meta-analysis followed the Preferred Reporting Items for Systematic Review and Meta-Analysis (PRISMA) 2020 guidelines.[26] The protocol was registered to the International Prospective Register of Systematic Reviews (PROSPERO) with registration number CRD42021264893.[27]

### Search strategy

The search strategy (Table S2) was developed in cooperation with a health science librarian and conducted in two electronic databases, PubMed and Scopus. We used keywords (MeSH-terms), a set of synonyms for the keywords, and text words (titles and abstracts) combined and truncated where appropriate. In addition to selected electronic databases the reference lists of selected studies and relevant review articles were scanned for additional studies. Search results were transferred into the Covidence software[28] for the selection process. The selection of articles to be included was performed independently by two evaluators (MH, WS). Evaluators first screened titles and abstracts, and in the second stage, full texts were evaluated based on the pre-defined selection criteria. Discrepancies between the evaluators were resolved by discussion.

### Selection criteria

Original research articles published in English as full publications in peer-reviewed journals between January 2005 and January 2025 were included. Only studies in human populations examining the role of one or multiple environmental exposures as independent variables in relation to T2D were included. Adult populations, all nationalities, and ethnicities were included to provide a broad overview. Eligible studies had to report quantitative measures of the association between environmental exposure and T2D. Studies excluded in the full-text review ($n = 156$) are listed in Table S3.

### Definition of the exposure

Environmental exposures were defined as air pollution, noise, and the built environment. For air pollution particles with a diameter of less than 2.5 ($PM_{2.5}$) and 10 μm ($PM_{10}$), nitrogen dioxide ($NO_2$), ozone ($O_3$), and black carbon (BC) were included. For noise: traffic noise, aircraft noise, and railway noise were included, and for built environment: greenness, walkability, and population density.

### Definition of the outcome

Articles were included if T2D cases were identified through register-based, clinical, or self-reported diagnostics. Register-based diagnostics were derived from hospital and patient registers, national registers, or medication reimbursement registers. Clinical diagnostics for T2D was defined by clinical cut-off values, including measurements of fasting plasma glucose, glycated hemoglobin, and oral glucose tolerance test results collected during a clinical examination performed by health professionals. Self-reported diagnostics refer to T2D status obtained from surveys and questionnaires.

### Data extraction

For all records, the following characteristics were recorded: title, author(s), publication year, country of study, study design, number of participants, mean age, sex ratio, definition- and assessment method of environmental exposure(s), outcome(s), T2D definition and assessment method, statistical methods, covariates, effect size measure, results as a measure of association and statistical significance, and direction of the association. Information on whether an exposome method was used in each study was extracted as a categorical variable (yes/no). In case of missing or incomplete data from the selected study, an attempt to retrieve information was made by contacting the authors. If the studies reported the effect estimates for both continuous and categorical environmental exposure, the results from continuous exposure were extracted. For categorical exposures (tertiles, quartiles, quintiles) the results were recorded as high versus low; the highest- and the lowest study-specific category. For studies using time-series analysis, the most recent results were included.

### Statistical analyses

We extracted and pooled effect estimates from the single-pollutant models reported as main or fully adjusted model. Joint exposure models were extracted and reported separately to describe the combined effect of multiple environmental exposures on the risk of T2D. To be able to compare the effect estimates between the single-exposure and joint exposure models, we calculated the absolute risk differences between the fully adjusted single-exposure and joint exposure models. To allow a comparison between the studies, air pollution effect measures were pooled for a fixed increment of 10 $\mu g/m^3$, except for BC which was standardized to a 5 $\mu g/m^3$ increment due to lower exposure units used in included studies. Noise exposures were pooled for a fixed increment of 10 dB.

$NO_2$ and $O_3$ exposures that were expressed in parts per billion (ppb) were first converted to $\mu g/m^3$ using the general formulas described below.

$$Concentration\ (\mu g/m^3) = molecular\ weight \times concentration\ (ppb) \div 24.45$$

$$\text{Nitrogen dioxide } NO_2 = 46.01\ g/\text{mol} \times 1\ \text{ppb} \div 24.45 = 1.88\ \mu g/m^3$$

$$\text{Ozone } O_3 = 48\ g/\text{mol} \times 1\ \text{ppb} \div 24.45 = 1.96\ \mu g/m^3,$$

where the value of 24.45 is the volume (liters) of a mole (gram molecular weight) of a gas when the temperature is at 25°C and the pressure is at 1 atmosphere (1 atm = 1.01325 bar). 25°C and pressure of 1 atmosphere are what is normally assumed for the conversion factors.

The following formulas were then used for standardization and obtaining standard errors as described in Yang et al.[29] Similar standardization was conducted for noise exposure as a fixed increment per 10 dB.

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

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

$$SE_{standardized} = (CI - upper_{standardized}) - (CI - lower_{standardized})/3.92$$

Meta-analyses were performed separately for each exposure when available. When the same study population was represented in multiple articles, we included the one that had the most relevance to the meta-analysis in terms of the number of participants, exposure measurement, and/or year published. There is no guideline for the minimum number of studies needed for a meta-analysis. We considered four risk estimates for each exposure as a minimum to justify running a meta-analysis. We pooled the risk estimates reported as hazard ratio (HR), odds ratios (OR), or risk ratio (RR), and 95% confidence intervals (95% CI), based on guidelines indicating that HR, OR and RR may be combined when the outcome of interest is common, and the effect size is small.[8,30,31] The random effects model with the DerSimonian and Laird method (inverse-variance method) was used to incorporate an assumption that the different studies are estimating different but related effects.[32]

Subgroup analyses were used to consider possible modification of effects by study characteristics: study design (longitudinal and cross-sectional), geographic region (Europe, Asia, North America, South-America, Oceania, and Africa), T2D definition (self-reported and register or clinical measures), adjustment for relevant factors related to T2D (yes: 5 or more included versus no: less than 5), adjustment for other environmental risk factors; $PM_{2.5}$, $PM_{10}$, $NO_2$, $O_3$, BC, traffic-, railway- or aircraft noise, walkability, greenness or population density (yes or no), and risk of bias score (low, moderate or high). Meta-regression was performed to identify potential sources of heterogeneity within these study characteristics. Statistical analyses were conducted using IBM SPSS Statistics (29.0).

### Risk of bias

We assessed the risk of bias (ROB) of all included studies using a self-developed tool. We integrated relevant components from the Newcastle–Ottawa Scale (NOS)[33] and the WHO Risk of Bias Assessment Instrument for Systematic Reviews Informing the WHO Global Air Quality Guidelines.[34] From the NOS, we incorporated key concepts related to selection of participants, ascertainment of exposure and outcomes, and control for confounding. From the WHO instrument, we drew on its domain-based approach focusing on study design, exposure assessment methods, outcome validity, and adjustment for key confounders and co-exposures. The evaluation included four main domains: (1) Study design, (2) Exposure measurement, (3) Outcome measurement, (4) testing for confounding, and had six questions:

1. Is the study design longitudinal?
2. Was the exposure measured before the outcome?
3. Was the exposure measured/modeled from the participant's home address?
4. Was the outcome measured by clinical measure or using register-based data, or self-reported data combined with clinical and/or register data?
5. Did the study adjust for T2D risk factors and potential confounders related to environmental exposures (at least 5 out of 8 equals yes: age, sex, socioeconomic status [SES, at least one of the following: socioeconomic position or status, education, employment or income status, deprivation], body mass index (BMI), smoking, physical activity, family history of diabetes, measure of nutrition/diet)?
6. Was the model adjusted for one or more environmental risk factors (air pollution, noise, green space)?

Each question was answered by the reviewer either yes, no, or information not available, yes giving one point and other options zero points. The results from the six questions were then rated as high ROB (0 to 2 points), moderate ROB (3-4), or low ROB (5-6). The evaluation was conducted by one reviewer (MH). We did not exclude any studies based on their ROB assessment, but we utilized the results in sensitivity analyses to identify how different sources of heterogeneity may affect the meta-analysis results.

The potential presence of publication bias and its impact on the results of the meta-analyses were assessed using statistical tests such as funnel plots and Egger's test. The $I^2$ statistic ($I^2$) and Tau-squared ($\tau^2$) values were calculated as a measure of heterogeneity across studies. $\tau^2$ measures the variance among the studies, and $I^2$ describes the percentage of the total variability (from 0 to 100%) in effect estimates that is due to heterogeneity rather than sampling error. We used the following guide to interpret the $I^2$ values in the context of meta-analyses: 0% to 40%: might not be important, 30% to 60%: may represent moderate heterogeneity, 50% to 90%: may represent substantial heterogeneity, and 75% to 100%: considerable heterogeneity.[35]

## Results

The systematic literature searches yielded a total of 13 629 records. After removing the duplicate records and screening according to the set selection criteria, 151 articles were identified as eligible for this review. The process is described in Figure 1 as

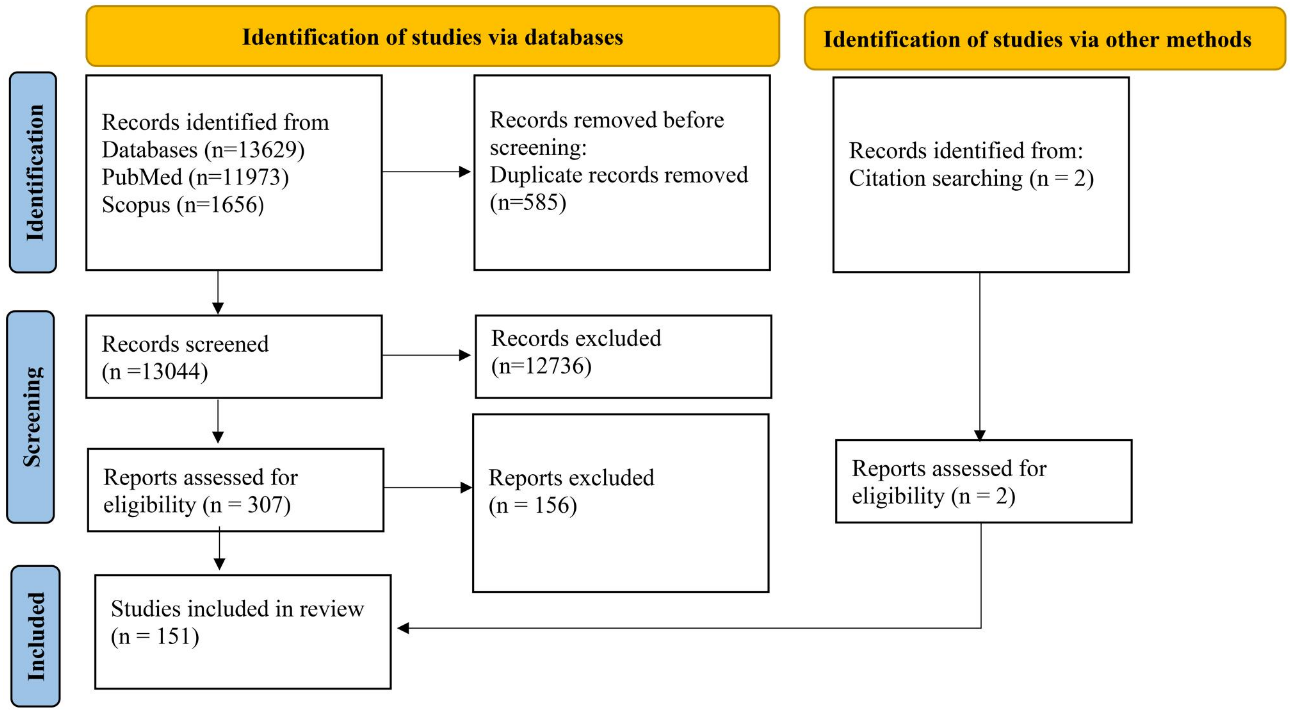


**Figure 1.** PRISMA flowchart of the selection of studies.

a PRISMA flow diagram. The included studies had different combinations of environmental exposures; 133 studies used air pollution as the main exposure, 20 used noise, and 39 built environment. The most studied pollutant was $PM_{2.5}$ (n = 90) and the least population density (n = 1) followed by aircraft noise (n = 5). Only one of the studies included by Ohanyan and colleagues, used an exposome method.[36] In the risk of bias (ROB) assessment, 37 studies were evaluated as having high ROB, 57 studies with moderate ROB, and 57 with low ROB. The ROB assessment for each included study is available in the supplementary materials Table S4.

## Air pollution

The relationship between exposure to air pollution and T2D was evaluated for $PM_{2.5}$, $PM_{10}$, $NO_2$, $O_3$, and BC. Study characteristics are described in Table 1. Separate meta-analyses were conducted for each pollutant, and the results are shown in Figures 2-8. Reasons for exclusion from meta-analyses are described in Table S5.

### *Particles with a diameter of less than 2.5 μm*

Altogether, 90 studies used $PM_{2.5}$ as the main exposure. In the meta-analysis, T2D was positively associated with $PM_{2.5}$ with an OR of 1.19 (95% CI: 1.16-1.22, n = 57, Figure 2). The meta-analysis showed considerable heterogeneity between the studies ($I^2$= 96.8%). The funnel plot analysis (Figure S1) showed studies concentrating on the right side of the funnel, and Egger's test indicated a risk of publication bias (*P*-value <0.001). The Trim-and-Fill analysis imputed 15 studies (Figure S2), and the overall effect estimate of observed and imputed studies decreased to OR: 1.14 (95% CI: 1.10-1.17). Standardization of two studies, Chen et al.[37] and Niedermayer et al.[38] increased their risk estimates substantially, from HR: 1.35 (95% CI: 0.83-2.18) to 2.58 (95% CI: −3.03 - 8.20) and from OR: 1.2, 95% CI: 0.99-1.46 to 3.68, 95% CI: −3.32-10.68, respectively. We performed a leave-one-out analysis, but excluding either one of these studies did not change the overall effect estimate from the meta-analysis.

Subgroup analysis highlighted some variation by study characteristics (Figure S7) but did not explain the considerable heterogeneity between the studies. Meta-regression analysis (Table S6) showed that studies with a moderate risk of bias had a smaller risk of T2D compared to studies with a low risk of bias (estimate: −0.10, 95% CI: −0.18, −0.02, *P*-value: 0.019). Studies with a high risk of bias also indicated a smaller risk of T2D compared to low risk of bias group, but the result was not significant (estimate: −0.12, 95% CI: −0.24, 0.01, *P*-value: 0.063). When comparing the study regions, the European and Asian region showed higher risk of T2D with estimates 0.12 (95% CI: 0.00-0.23, *P*-value: 0.054) and estimate: 0.09 (95% CI: 0.00-0.18, *P*-value: 0.054) respectively when compared with the North America as the reference region. Longitudinal studies had a higher risk estimate (0.10, 95% CI: 0.02-0.18, *P*-value: 0.020) when compared to cross-sectional studies.

### *Particles with a diameter of less than 2.5 μ and joint exposure*

Of the 90 studies considering the association between $PM_{2.5}$ and T2D, 26 evaluated joint exposure to air pollution, noise, or the built environment. Three studies found no association between $PM_{2.5}$ and T2D in either single-exposure or joint-exposure models adjusted for air pollution ($PM_{10}$, $NO_2$, or $O_3$).[39-41] In seven studies, adjustment for other air pollutants did not considerably change the association (risk differences between single- and joint exposure models ranged between −0.01 and 0.04).[42-48] In three studies, the adjustment for air pollution ($NO_2$, $PM_{10}$, or $O_3$) decreased the association between $PM_{2.5}$ and T2D to nonsignificant.[49-51] For Clark et al. the association was independent of covarying noise exposure (OR: 1.03, 95% CI: 1.02-1.05), but further adjustment for greenness and walkability attenuated the estimate to nonsignificant (OR: 1.01, 95% CI: 1.00 - 1.03).[52] Sorensen et al. reported that the association between $PM_{2.5}$ and T2D (HR: 1.05, 95% CI: 1.03-1.06) was reduced to unity or below in two-, three- and four-pollutant models when ultrafine particles, elemental carbon,

**Table 1.** Characteristics of included air pollution articles (n = 133). The table is organized in alphabetical ascending order of the first author.

| Author | Country | Study | N | Age | Baseline | Follow-up | Exposure | Units |
|---|---|---|---|---|---|---|---|---|
| Anderson et al., 2012[127] | Denmark | The Danish Diet, Cancer, and Health Cohort | 51 818 | 56.1 | 1993/1997 | 9.7 years | $NO_2$ | per IQR 4.9 μg/m$^3$ |
| Badpa et al., 2024[111] | Germany | Cooperative Health Research in the Region of Augsburg KORA-Study | 7736 | 49.2 | 1994-1995, 1999-2001 | 15.0 years | $PM_{2.5}$<br>$PM_{10}$<br>$NO_2$<br>$O_3$ | per IQR 1.3 μg/m$^3$<br>per IQR 2.2 μg/m$^3$<br>per IQR 7.0 μg/m$^3$<br>per IQR 3.6 μg/m$^3$ |
| Bai et al., 2018[71] | Canada | Ontario Population Health and Environment Cohort | 1 056 012 | 51.1 | 1996 | 17.0 years | $NO_2$ | per IQR 4.0 μg/m$^3$ |
| Su et al., 2023[128] | China | Urban and Rural Elderly Population study | 222 179 | 69.73 | 2015 | – | $PM_{2.5}$<br>$O_3$ | per 10 μg/m$^3$<br>per IQR |
| Bo et al., 2021[129] | Taiwan | MJ Health cohort study | 146 789 | 38.82 | 2001-2014 | 5.0 years | $PM_{2.5}$ | tertiles: −31.99 to −0.99; −0.99 to 0.27; 0.27–32.7 μg/m$^3$ |
| Bowe et al., 2018[130] | US | A cohort of US veterans | 1 729 108 | 61.2 | 2003 | 8.5 years | $PM_{2.5}$ | per 10 μg/m$^3$ |
| Brook et al., 2008[131] | Canada | Register-based cohort | 7634 | 49.85 | 1992-1999 | - | $NO_2$ | per 1 ppb |
| Cervantes-Martínez et al., 2022[55] | Mexico | The Mexican Teachers' Cohort[a] | 13 669 | 43 | 2008 | 11.5 years | $PM_{2.5}$<br>$NO_2$ | per 10 μg/m$^3$<br>per 10 ppb |
| Chen et al., 2013[132] | Canada | Canadian Community Health Surveys | 62 012 | 54.9 | 1996 | 8.32 years | $PM_{2.5}$ | per 10 μg/m$^3$ |
| Chen et al., 2022[133] | China | Wuhan Chronic Disease Cohort | 10 253 | – | 2019 | – | $PM_{2.5}$<br>$PM_{10}$<br>$NO_2$ | per 1 μg/m$^3$<br>per 1 μg/m$^3$<br>per 1 μg/m$^3$ |
| Chen et al., 2024[37] | China | Anhui Cohort Study of Older People Health | 2766 | 71.68 | 2001-2003 | 5.55 years | $PM_{2.5}$ | per IQR 3.16 μg/m$^3$ |
| Chilian-Herrera et al., 2021[134] | Mexico | National Health and Nutrition Survey Mexico | 2297 | 49.3 | 2012 | – | $PM_{2.5}$ | per 10 μg/m$^3$ |
| Clark et al., 2017[52] | Canada | Population Data BC | 380 738 | 58 | 1994-1998 | 4.0 years | $PM_{2.5}$<br>$NO_2$<br>BC | per IQR 1.6 μg/m$^3$<br>per IQR 8.4 μg/m$^3$<br>per IQR 0.9 $10^{-5/m}$ |
| Coogan et al., 2012[135] | US | Black Women's Health Study | 3992[a] | 39.35 | 1995 | 10.0 years | $PM_{2.5}$ | per 10 μg/m$^3$ |
| Coogan et al., 2016[136] | US | Black Women's Health Study | 43 003[a] | 38.7 | 1995 | 16.0 years | $PM_{2.5}$<br>$NO_2$ | per IQR 2.9 μg/m$^3$<br>per IQR 9.7 ppb |
| Cui et al., 2024[57] | China | Chronic disease surveillance project: middle-aged and elderly individuals in Anhui Province, China | 79 623 | 57.14 | 2017-2020 | – | $PM_{2.5}$<br>BC | per IQR 7.95 μg/m$^3$<br>per IQR 0.30 μg/m$^3$ |
| Dijkema et al., 2011[137] | Netherlands | Hoorn Screening Study for type 2 diabetes | 8018 | 58 | 1998-2000 | – | $NO_2$ | quartiles: 8.8 to 14.2; 14.2 to 15.2; 15.2 to 16.5; 16.5-36.0 μg/m$^3$ |
| Dimakakou et al., 2020[138] | UK | UK Biobank | 502 504 | NA | 2006-2010 | – | $PM_{2.5}$ | per 1 μg/m$^3$ |
| Dzhambov et al., 2016[139] | Bulgaria | Cross-sectional study in Plovdiv city, Bulgaria | 513 | 36.45 | 2014 | – | $PM_{2.5}$ | categories: 0.0 to 17.5; 17.5 to 20.3; 20.3 to 25.0; 25.0 to 40; 40.0 to 66.8 μg/m$^3$ |
| Dzhambov et al., 2025[69] | Bulgaria | Cross-sectional study in 5 Bulgarian cities | 4640 | 49.0 | 2023 | – | $NO_2$ | per 5 μg/m$^3$ |
| Elbarbary et al., 2020[140] | China | Study on global AGEing and adult health (SAGE) | 8179 | 62.9 | 2007-2010 | – | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$ |

(continued)

**Table 1.** (continued)

| Author | Country | Study | N | Age | Baseline | Follow-up | Exposure | Units |
|---|---|---|---|---|---|---|---|---|
| Eze et al., 2014[64] | Switzerland | Swiss Study on Air Pollution and Lung Disease in Adults | 6392 | 52 | 2002 | – | $PM_{10}$<br>$NO_2$ | per 10 $\mu g/m^3$<br>per 10 $\mu g/m^3$ |
| Eze et al., 2017[68] | Switzerland | Swiss Study on Air Pollution and Lung Disease in Adults. | 2631 | 59.2 | 2002 | 8.3 years | $NO_2$ | per IQR 15 $\mu g/m^3$ |
| Fan et al., 2024[141] | UK | UK Biobank | 78 230 | cat. 40-70 | 2010 | 12.19 years | $PM_{2.5}$<br>$PM_{10}$<br>$NO_2$ | per IQR 1.26 $\mu g/m^3$<br>per IQR 1.79 $\mu g/m^3$<br>per IQR 10.32 $\mu g/m^3$ |
| Fan et al., 2025[142] | UK | UK Biobank | 77 278 | cat. 40-70 | 2006-2010 | 12.19 years | $PM_{2.5}$<br>$PM_{10}$<br>$NO_2$ | per IQR 1.26 $\mu g/m^3$<br>per IQR 1.79 $\mu g/m^3$<br>per IQR 10.32 $\mu g/m^3$ |
| Guo et al., 2021[143] | Taiwan | MJ Health cohort study | 156 314 | 40.7 | 2001 | 5.2 years | $PM_{2.5}$ | per 10 $\mu g/m^3$ |
| Hansen et al., 2016[49] | Denmark | Danish Nurse Cohort | 24 174[a] | 54 | 1993 | 15.3 years | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per 10 $\mu g/ m^3$<br>per IQR 3.1 $\mu g/m^3$<br>per IQR 7.5 $\mu g/m^3$ |
| Hassanvand et al., 2018[144] | Iran | National surveillance of risk factors of noncommunicable diseases | 2903 | 55.31 | 2011 | – | $PM_{10}$ | Unclear |
| Hegelund et al., 2024[145] | Denmark | Danish nationwide sample | 3 111 988 | 51.4 | 2000 | 15.2 years | $PM_{2.5}$<br>$NO_2$ | per IQR 1.96 $\mu g/m^3$<br>per IQR 10.23 $\mu g/m^3$ |
| Hernandez et al., 2018[146] | US | Selected Metropolitan/ Micropolitan Area Risk Trends from Behavioral Risk Factor Surveillance System | 1 158 547 | – | 2002-2008 | – | $PM_{2.5}$<br>$O_3$ | per 10 $\mu g/m^3$<br>per 10 ppb |
| Honda et al., 2017[44] | US | National Social Life, Health, and Aging Project | 916 | 69.6 | 2005-2011 | – | $PM_{2.5}$<br>$NO_2$ | per IQR 3.9 $\mu g/m^3$<br>per IQR 8.6 ppb |
| Howell et al., 2019[74] | Canada | CANHEART-Cohort: The Cardiovascular Health in Ambulatory Care Research Team | 2 496 458 | 53.2 | 2008 | – | $NO_2$ | per 10 ppb |
| Hu et al., 2024[147] | China | Shanghai High-Risk Diabetic Screen Project | 9371<br>1128 | 52.92<br>51.13 | 2002-2013<br>2014-2018 | –<br>– | $PM_{2.5}$<br>$PM_{2.5}$ | per 10 $\mu g/m^3$<br>per 10 $\mu g/m^3$ |
| Hu et al., 2023[51] | UK | UK Biobank | 390 834 | 56.3 | 2006-2010 | 10.9 years | $PM_{2.5}$<br>$NO_2$ | per IQR 1.3 $\mu g/m^3$<br>per IQR 9.8 $\mu g/m^3$ |
| Huo et al., 2022[148] | China | Henan Rural Cohort Study | 11 640 | – | 2015-2017 | – | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per 1 $\mu g/m^3$<br>per 1 $\mu g/m^3$<br>per 1 $\mu g/m^3$ |
| Jabbari et al., 2020[149] | Iran | Tehran Cardiometabolic Genetic Study | 2428 | 45.4 | 2009 | 9.0 years | $PM_{10}$ | per 10 $\mu g/ m^3$ |
| Jerret et al., 2017[75] | US | Black Women's Health Study | 43 003[a] | – | 1995 | 8.0 years | $O_3$ | per 6.7 ppb |
| Kang et al., 2022[150] | China | Henan Rural Cohort study | 38 841 | 55.56 | 2015-2017 | – | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per 1 $\mu g/m^3$<br>per 1 $\mu g/m^3$<br>per 1 $\mu g/m^3$ |
| Kang et al., 2023[79] | China | Henan Rural Cohort Study | 38 442 | 55.56 | 2015-2017 | – | $PM_{2.5}$<br>BC | per 1 $\mu g/m^3$<br>per 1 $\mu g/m^3$ |
| Klompmaker et al., 2019[72] | Netherlands | Dutch Public Health Monitor | 354 827 | Cat. | 2012 | – | $PM_{2.5}$<br>$PM_{10}$<br>$NO_2$ | per IQR 0.83 $\mu g/m^3$<br>per IQR 1.24 $\mu g/m^3$<br>per IQR 7.85 $\mu g/m^3$ |
| Krämer et al., 2010[151] | Germany | SALIA- Study on the influence of Air pollution on Lung function, Inflammation and Aging | 1775[a] | 54.6 | 1985-1994 | 16.0 years | $PM_{10}$<br>$NO_2$ | Per IQR 10.1 $\mu g/m^3$<br>per IQR 24.9 $\mu g/m^3$ |

(continued)

**Table 1.** (continued)

| Author | Country | Study | N | Age | Baseline | Follow-up | Exposure | Units |
|---|---|---|---|---|---|---|---|---|
| Lao et al., 2019[152] | Taiwan | Taiwan MJ Cohort | 147 908 | 38.3 | 2001-2014 | 6.7 years | $PM_{2.5}$ | per quartiles: < 21.7; 21.7–<24.1; 24.1–<28.0; ≥ 28.0 $\mu g/m^3$ |
| Lee et al., 2021[153] | Japan | Center for Preventive Medicine, St. Luke's International HospitaL - database | 66 885 | 46 | 2005-2019 | – | $PM_{2.5}$ | per 1 $\mu g/m^3$ |
| Li et al., 2019[65] | China | Chronic Disease Surveillance System of Ningbo | 25 130 | 65.17 | 2008-2015 | – | $PM_{10}$<br>$O_3$ | per 10 $\mu g/m^3$<br>per 10 $\mu g/m^3$ |
| Li et al., 2021[76] | Taiwan | National Health Insurance Research Database | 6 426 802 | 39.84 | 2005 | 11.0 years | $O_3$ | per IQR 3.30 ppb |
| Li et al., 2022[56] | UK | UK Biobank | 263 733 | 56.48 | 2006-2010 | 11.94 years | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | quintiles (5) low to high<br>quintiles (5) low to high<br>quintiles (5) low to high |
| Li et al., 2024[154] | China | China-PAR sub-cohorts: China Multi-Center Collaborative Study of Cardiovascular Epidemiology; International Collaborative Study of Cardiovascular Disease in Asia; Community Intervention of Metabolic Syndrome in China Chinese Family Health Study | 71 689 | 51.28 | 2000 | 5.93 years | $PM_{2.5}$ | per 10$\mu g/m^3$ |
| Li et al., 2023[80] | China | China Multi-Ethnic Cohort study | 69 210 | 51.8 | 2018-2019 | – | $PM_{2.5}$<br>BC | per SD 20.5 $\mu g/m^3$<br>per SD 1.1 $\mu g/m^3$ |
| Li et al., 2024[48] | China | Prospective Cohort Study in China | 124 204 | 39 | 2005-2020 | 8.47 years | $PM_{2.5}$ | per 1 $\mu g/m^3$ |
| Li et al., , 2021[63] | UK | UK Biobank | 449 006 | 56.46 | 2006-2010 | 11.0 years | $PM_{2.5}$<br>$NO_2$ | per SD increase<br>per SD increase |
| Li et al., 2022[155] | UK | UK Biobank | 359 153 | 56.3 | 2006-2010 | 8.9 years | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per 10 $\mu g/m^3$<br>per 5$\mu g/m^3$<br>per 10 $\mu g/m^3$ |
| Li et al., 2019[156] | Taiwan | – | 505 151 | 42.6 | 2001 | 12.0 years | $PM_{2.5}$ | per 10 $\mu g/m^3$ |
| Liang et al., 2019[157] | China | Prediction for Atherosclerotic Cardiovascular Disease Risk in China (China PAR) | 88 397 | 51.7 | 1992-1994, 1998, 2000-2001, 2007-2008 | 2012-2015 | $PM_{2.5}$ | per 10 $\mu g/m^3$ |
| Liu et al., 2023[158] | China | China Health and Retirement Longitudinal Study (CHARLS) | 19 121 | 57.88 | 2011 | 8.0 years | $PM_{2.5}$ | per 10 $\mu g/m^3$ |
| Liu et al., 2022[43] | China | China Health and Retirement Longitudinal Study (CHARLS) | 9638<br>3510 | 60.3<br>59.3 | 2011<br>2015 | –<br>5.0 years | $PM_{10}$<br>$PM_{2.5}$<br>$NO^2$<br>$PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per 10 $\mu g/m^3$<br>per 10 $\mu g/m^3$<br>per 10 $\mu g/m^3$<br>per 10 $\mu g/m^3$<br>per 10 $\mu g/m^3$<br>per 10 $\mu g/m^3$ |
| Liu et al., 2022[77] | China | Henan Rural Cohort Study | 39 192 | 57.7 | 2015-2017 | – | $O_3$ | per IQR 4.04 $\mu g/m^3$ |
| Liu et al., 2016[159] | China | China Health and Retirement Longitudinal Study (CHARLS) | 11 847 | 59 | 2011-2012 | – | $PM_{2.5}$ | per IQR 41.1 $\mu g/m^3$ |

(continued)

**Table 1.** (continued)

| Author | Country | Study | N | Age | Baseline | Follow-up | Exposure | Units |
|---|---|---|---|---|---|---|---|---|
| Liu et al., 2019[160] | China | Henan Rural Cohort study | 39 191 | 55.6 | 2015-2017 | – | $PM_{2.5}$<br>$NO_2$ | per 1 μg/m$^3$<br>per 1 μg/m$^3$ |
| Liu et al., 2019[45] | China | Guangdong Gut Microbiome Project dataset | 6627 | 51.8 | 2015-2016 | – | $PM_{2.5}$ | per IQR 8.03 μg/m$^3$ |
| Lucht et al., 2020[161] | Germany | Heinz Nixdorf recall study | 2451 | 58.2 | 2000-2003 | 10.0 years | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per IQR 3.8 μg/m$^3$<br>per 1 μg/m$^3$<br>per 1 μg/m$^3$ |
| Ma et al., 2024[162] | China | Study in the national program Guangxi Zhuang Autonomous Region | 12 426 | 54.22 | 2018-2019 | – | $O_3$ | per IQR 1.18 μg/m$^3$ |
| Mandal et al., 2023[163] | India | Center for Cardiometabolic Risk Reduction in South Asia, *Chennai Region* | 5118 | 40.1 | 2010-2012 | 4.84 years | $PM_{2.5}$ | per 10 μg/m$^3$ |
| | | Center for Cardiometabolic Risk Reduction in South Asia, *Delhi Region* | 3675 | 44.6 | 2010-2012 | 4.84 years | $PM_{2.5}$ | per 10 μg/m$^3$ |
| McAlexander et al., 2022[164] | US | REGARDS: REasons for Geographic and Racial Differences in Stroke | 11 208 | 62.7 | 2003-2007 | 2013-2016 | $PM_{2.5}$ | per 5 μg/m$^3$ |
| Mei et al., 2023[165] | China | Community-based study in China | 4235 | 54.23 | 2018-2020 | – | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$<br>$O_3$ | per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$ |
| Niedermayer et al., 2024[38] | Germany | Cooperative Health Research in the Region of Augsburg (KORA) FIT-Study | 3034 | 63.2 | 2018-2019 | – | $NO_2$<br>$PM_{2.5}$<br>$PM_{10}$<br>$O_3$ | per IQR 6.3 μg/m$^3$<br>per IQR 1.4 μg/m$^3$<br>per IQR 2.0 μg/m$^3$<br>per IQR 3.5 μg/m$^3$ |
| O'Donovan et al., 2017[166] | UK | ADDITION-Leicester<br>Let's prevent<br>Walking away | 6171<br>3442<br>830 | 56.2<br>63.2<br>63.1 | 2004-2009<br>2009-2011<br>2010 | –<br>–<br>– | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$<br>$PM_{2.5}$<br>$PM_{10}$<br>$NO_2$<br>$PM_{2.5}$<br>$PM_{10}$<br>$NO_2$ | per 10 μg·m$^3$<br>per 10 μg·m$^3$<br>per 10 μg·m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$ |
| Ohanyan et al., 2022[36] | Netherlands | Population-based Occupational and Environmental Health Cohort Study (AMIGO) | 14 829 | 50.7 | 2011-2012 | – | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | Na<br>Na<br>Na |
| Orioli et al., 2018[46] | Italy | Italian National Institute of Statistics | 376 157 | 63.5 | 1999-2013 | – | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$<br>$O_3$ | per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$<br>per 10 μg/m$^3$ |
| Sung Kyun et al., 2015[167] | US | MESA: Multi-Ethnic Study of Atherosclerosis | 5839<br>5135 | 64.3<br>61.6 | 2000-2002<br>2000-2002 | –<br>9.0 years | $PM_{2.5}$<br>$PM_{2.5}$ | per IQR 2.43 μg/m$^3$<br>per IQR 2.43 μg/m$^3$ |
| Paul et al., 2020[50] | Canada | Ontario Population Health and Environment Cohort | 790 461 | 55.5 | 2001 | 15.0 years | $PM_{2.5}$<br>$NO_2$<br>$O_3$ | per IQR 3.5μg/m$^3$<br>per IQR 13.8ppb<br>per IQR 6.3ppb |
| Puett et al., 2011[41] | US | Nurses' Health Study & Health Professionals Follow-Up Study | 89 460 | 56 | 1989-2002 | 13.0 years | $PM_{10}$<br>$PM_{2.5}$ | per IQR 7 μg/m$^3$<br>per IQR 4 μg/m$^3$ |

(continued)

**Table 1.** (continued)

| Author | Country | Study | N | Age | Baseline | Follow-up | Exposure | Units |
|---|---|---|---|---|---|---|---|---|
| Qiu et al., 2018[168] | Hong Kong | Chinese Elderly Health Services cohort | 53 905 | 72.4 | 1998 | 9.8 years | $PM_{2.5}$ | per IQR 3.2 μg/m$^3$ |
| | | | 61 447 | 72 | 1998 | – | $PM_{2.5}$ | per IQR 3.2 μg/m$^3$ |
| Renzi et al., 2018[39] | Italy | Rome Longitudinal Study | 65 955 | – | 2008 | 6.0 years | $PM_{10}$ | per 10 μg/m$^3$ |
| | | | 106 387 | – | 2008 | – | $PM_{2.5}$ | per 5-μg/m$^3$ |
| | | | | | | | $NO_2$ | per 10-μg/m$^3$ |
| | | | | | | | $O_3$ | per 10-μg/m$^3$ |
| | | | | | | | $PM_{10}$ | per 10 μg/m$^3$ |
| | | | | | | | $PM_{2.5}$ | per 5-μg/m$^3$ |
| | | | | | | | $NO_2$ | per 10-μg/m$^3$ |
| | | | | | | | $O_3$ | per 10-μg/m$^3$ |
| Requia et al., 2017[169] | Canada | Canadian community health survey data | 5 570 326 | – | 2007-2014 | – | $PM_{2.5}$ | per 10 μg/m$^3$ |
| Riant et al., 2018[66] | France | ELISABET: Prevalence and underdiagnosis of airway obstruction among middle-aged adults in northern France | 2797 | 53 | 2011-2013 | – | $PM_{10}$ | per 2 μg/m$^3$ |
| | | | | | | | $NO_2$ | per 5-μg/m$^3$ |
| Sade et al., 2023[47] | US | Medicare enrollees 65-y and older in the fee-for-service program, part A and part B, in the US | 41 780 637 | 75.97 | 2000 | until 2016 | $PM_{2.5}$ | per 5 μg/m$^3$ |
| | | | | | | | $NO_2$ | per 5 ppb |
| | | | | | | | $O_3$ | per 5 ppb |
| Shan et al., 2020[67] | China | China Northern 4 Cities Cohort Study | 38 529 | 44.12 | 1998 | 12.0 years | $PM_{10}$ | per 10 μg/m$^3$ |
| | | | | | | | $NO_2$ | per 10 μg/m$^3$ |
| Shen et al., 2024[42] | China | National Free Preconception Health Examination Project in China | 20 076 032[a] | 27.04 | 2010-2015 | – | $PM_{2.5}$ | per IQR 27 μg/m$^3$ |
| Shin et al., 2023[170] | South Korea | Cardiovascular Disease Association Study | 14 667 | 58.6 | 2005-2011 | until 2016 | $PM_{2.5}$ | per 10 μg/m$^3$ |
| | | | | | | | $NO_2$ | per 10-ppb |
| Sohn et al., 2017[171] | South-Korea | Korea Community Health Survey | 52 127 | 46.7 | 2012 | – | $PM_{10}$ | per 1000 ppm |
| Sommar et al., 2023[172] | Sweden | The Västerbotten intervention programme | 33 766 | 40 | 1985-2014 | until 2015 | $PM_{10}$ | per 10 μg/m$^3$ |
| | | | | | | | $PM_{2.5}$ | per 5 μg/m$^3$ |
| Strak et al., 2017[40] | Netherlands | Dutch national health survey | 289 703 | Cat. ≥ 19 | 2012 | – | $PM_{10}$ | per IQR 1.20 μg/m$^3$ |
| | | | | | | | $PM_{2.5}$ | per IQR 0.81 μg/m$^3$ |
| | | | | | | | $NO_2$ | per IQR 7.76 μg/m$^3$ |
| Sun et al., 2025[58] | China | Participants with annual check-ups at 37 community hospitals in Tianjin Binhai New Area | 65 824 | 64.64 | 2014 | 8 years | $PM_{2.5}$ | per SD 15.03 μg/m$^3$ |
| | | | | | | | BC | per SD 0.464 μg/m$^3$ |
| Suryadhi et al., 2020[173] | Indonesia | Indonesia Basic Health Research | 64 7947 | 41.9 | 2013 | – | $PM_{2.5}$ | per 10 μg/m$^3$ |
| Sørensen et al., 2022[53] | Denmark | Danish Register data | 1 922 545 | 57.5 | 2005 | 11.2 years | $PM_{2.5}$ | per IQR 1.85 μg/m$^3$ |
| Sørensen et al., 2022[174] | Denmark | Danish National Health Survey | 234 018 | 52 | 2010, 2013 | until 2017 | $PM_{2.5}$ | per 5 μg/m$^3$ |
| | | | | | | | $NO_2$ | per 10 μg/m$^3$ |
| Sørensen et al., 2023[62] | Denmark | Danish Register data | 1 843 597 | 58.9 | 2005 | 9.5 years | $PM_{2.5}$ | per 5 μg/m$^3$ |
| | | | | | | | $NO_2$ | per 10 μg/m$^3$ |
| Sørensen et al., 2022[73] | Denmark | Danish Register data | 2 631 488 | 51.7 | 2005 | 13.0 years | $PM_{2.5}$ | per IQR 1.85 μg/m$^3$ |
| | | | | | | | $NO_2$ | per IQR 7.15 μg/m$^3$ |
| Tani et al., 2023[175] | Japan | Individuals enrolled in health checkups in Okayama, Japan | 75 553 | 69.9 | 2006-2008 | – | $PM_{2.5}$ | per IQR 2.1 μg/m$^3$ |
| Wang et al., 2020[176] | China | Jinchang Cohort | 19 884 | 48.18 | 2011 | 2.28 years | $PM_{10}$ | per 10 μg/m$^3$ |
| Wang et al., 2022[78] | China | China Health and Retirement Longitudinal Study (CHARLS) | 13 548 | 59 | 2011 | 7.0 years | $O_3$ | per 10 μg/m$^3$ |

(continued)

**Table 1.** (continued)

| Author | Country | Study | N | Age | Baseline | Follow-up | Exposure | Units |
|---|---|---|---|---|---|---|---|---|
| Wang et al., 2024[81] | China | Jinchang Cohort | 19 884 | 48.18 | 2011-2013 | 2.28 years | $PM_{2.5}$<br>BC | per 37.08 μg/$m^3$<br>per 1.48 μg/$m^3$ |
| Weinmayr et al., 2015[177] | Germany | Heinz Nixdorf Recall Study | 3607 | 59.65 | 2000-2003 | 5.1 years | $PM_{10}$<br>$PM_{2.5}$ | per IQR 3.78 μg/$m^3$<br>per IQR 2.29 μg/$m^3$ |
| Wong et al., 2020[178] | Malaysia | Malaysian National Health and Morbidity Surveys | 29 460 | – | 2015 | – | $PM_{10}$<br>$NO_2$<br>$O_3$ | per IQR 10.34 μg/$m^3$<br>per IQR 9.57 μg/$m^3$<br>per IQR 7.83 μg/$m^3$ |
| Wu et al., 2022[54] | UK | UK Biobank | 398 993 | 55.49 | 2006-2010 | 12.0 years | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per IQR 3.25 μg/$m^3$<br>per IQR 2.31 μg/$m^3$<br>per IQR 7.08 μg/$m^3$ |
| Wu et al., 2023[179] | UK | UK Biobank | 162 334 | 53.99 | 2006-2010 | 11.7 years | $PM_{2.5}$<br>$PM_{10}$<br>$NO_2$ | per IQR 1.29 μg/$m^3$<br>per IQR 1.77 μg/$m^3$<br>per IQR 10.20 μg/$m^3$ |
| Wu et al., 2024[180] | China | Cohort in Yinzhou District, Ningbo, China. | 24 147 | 62.9 | 2015-2018 | until 2021 | $PM_{2.5}$<br>$PM_{10}$<br>$NO_2$ | per IQR 5.64 μg/$m^3$<br>per IQR 7.91μg/$m^3$<br>per IQR 8.75 μg/$m^3$ |
| Yang et al., 2018[181] | China | 33 Communities Chinese Health Study | 15 477 | 45 | 2009 | – | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$<br>$O_3$ | per IQR 19 μg/$m^3$<br>per IQR 26 μg/$m^3$<br>per IQR 9 μg/$m^3$<br>per IQR 22 μg/$m^3$ |
| Yang et al., 2018[182] | China | Study on global AGEing and adult health (SAGE) | 11 504 | 62.7 | 2007-2010 | – | $PM_{2.5}$ | per 10 μg/$m^3$ |
| Ye et al., 2022[183] | China | China Health and Retirement Longitudinal Study (CHARLS) | 19 529 | 62.06 | 2018 | – | $PM_{2.5}$ | per IQR 16.2 μg/$m^3$ |
| Yu et al., 2021[184] | US | The Sacramento Area Latino Study on Aging | 1090 | 70.5 | 1998 | 10.0 years | $O_3$ | per 10-ppb |
| Xu et al., 2023[70] | UK | UK Biobank | 82 548 | 55.49 | 2006-2011 | 13.76 years | $PM_{2.5}$<br>$NO_2$ | per SD 1.07 μg/$m^3$<br>per SD 9.23 μg/$m^3$ |
| Zadeh et al., 2023[185] | Iran | Tehran Lipid and Glucose Study | 5024 | 40.6 | 2001 | 12.2 years | $PM_{10}$<br>$NO_2$<br>$O_3$ | per 10 μg/$m^3$<br>per 10 μg/$m^3$<br>per 10 μg/$m^3$ |
| Zhang et al., 2021[186] | China | China Health and Retirement Longitudinal Study (CHARLS) | 13 013 | 61.88 | 2015 | – | $NO_2$ | per IQR (12.39 μg/$m^3$) |
| Zhang et al., 2024[61] | China | China Health and Retirement Longitudinal Study (CHARLS) | 9242 | 59.0 | 2011-2012 | until 2018 | $PM_{2.5}$ | per 10 μg/$m^3$ |
| Zheng et al., 2024[59] | UK | UK Biobank | 162 579 | 55.7 | 2010 | 10.1 years | $PM_{2.5}$<br>$PM_{10}$<br>$NO_2$ | per IQR 2.249 μg/$m^3$<br>per IQR 3.163 μg/$m^3$<br>per IQR 7.353 μg/$m^3$ |
| Zhou et al., 2022[187] | China | China Health and Retirement Longitudinal Study (CHARLS) | 13 589 | 59.5 | 2011-2012 | – | $PM_{2.5}$<br>BC | per IQR 27.4 μg/$m^3$<br>per IQR 2.2 μg/$m^3$ |
| Zhou et al., 2024[60] | China | Sub-cohort of the China Multi-Ethnic Cohort | 17 566 | 51.4 | 2018 | 4.2 years | $PM_{2.5}$<br>$NO_2$<br>$O_3$<br>BC | per IQR 8.21 μg/$m^3$<br>per IQR 15.75 μg/$m^3$<br>per IQR 1.96 μg/$m^3$<br>per IQR 1.51μg/$m^3$ |
| Zou et al., 2023[188] | UK | UK Biobank | 372 530 | 55.7 | 2006-2010 | 12.6 years | $PM_{10}$<br>$PM_{2.5}$<br>$NO_2$ | per IQR 3.15 μg/$m^3$<br>per IQR 2.26 μg/$m^3$<br>per IQR 6.90 μg/$m^3$ |

[a]Only women in the study population.

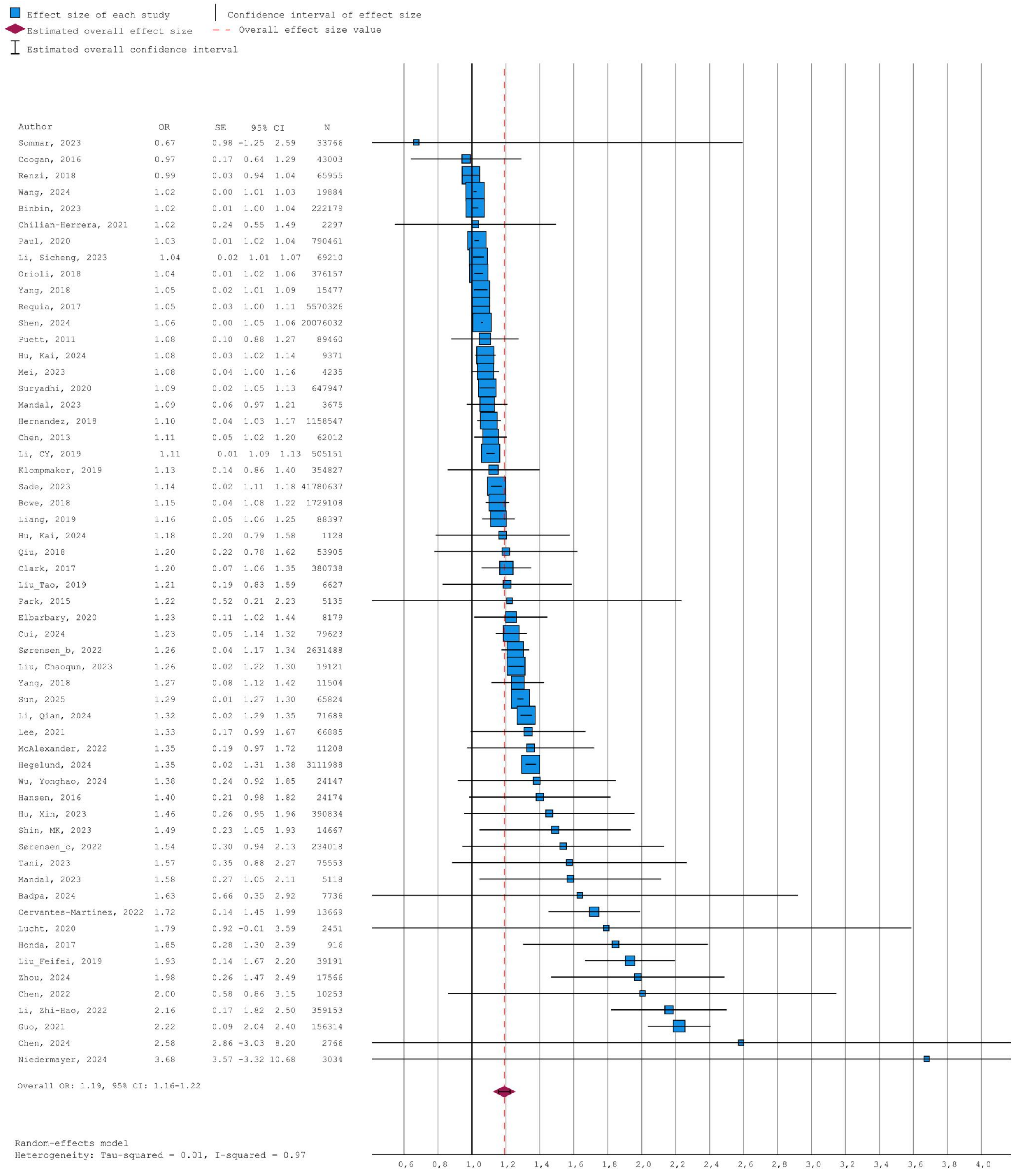


**Figure 2.** Results of the random effects meta-analysis of the association between PM2.5 per 10 μg/m3 and T2D (OR with 95% CI).

and/or $NO_2$ were included.[53] In three studies, the association between $PM_{2.5}$ and T2D changed considerably when adjusting for $NO_2$; Wu et al.[54] reported a 14.1% relative decrease in risk estimate, Cervantes-Martinez et al.[55] observed a 19.8% decrease, whereas Li et al.[56] found a 7.27% relative increase in the risk estimate.

Quantile g-computing (QGC) method was used in four studies.[57-60] Zheng et al. reported higher risk estimate for joint exposure model when using the QGC method for air pollutants $PM_{2.5}$, $PM_{10}$, $NO_2$, sulphur dioxide ($SO_2$), nitrogen oxides ($NO_x$), and benzene (OR: 1.16, 95% CI: 1.10-1.22) compared to the single-exposure model of $PM_{2.5}$ (OR: 1.08, 95% CI: 1.03-1.14).[59] In

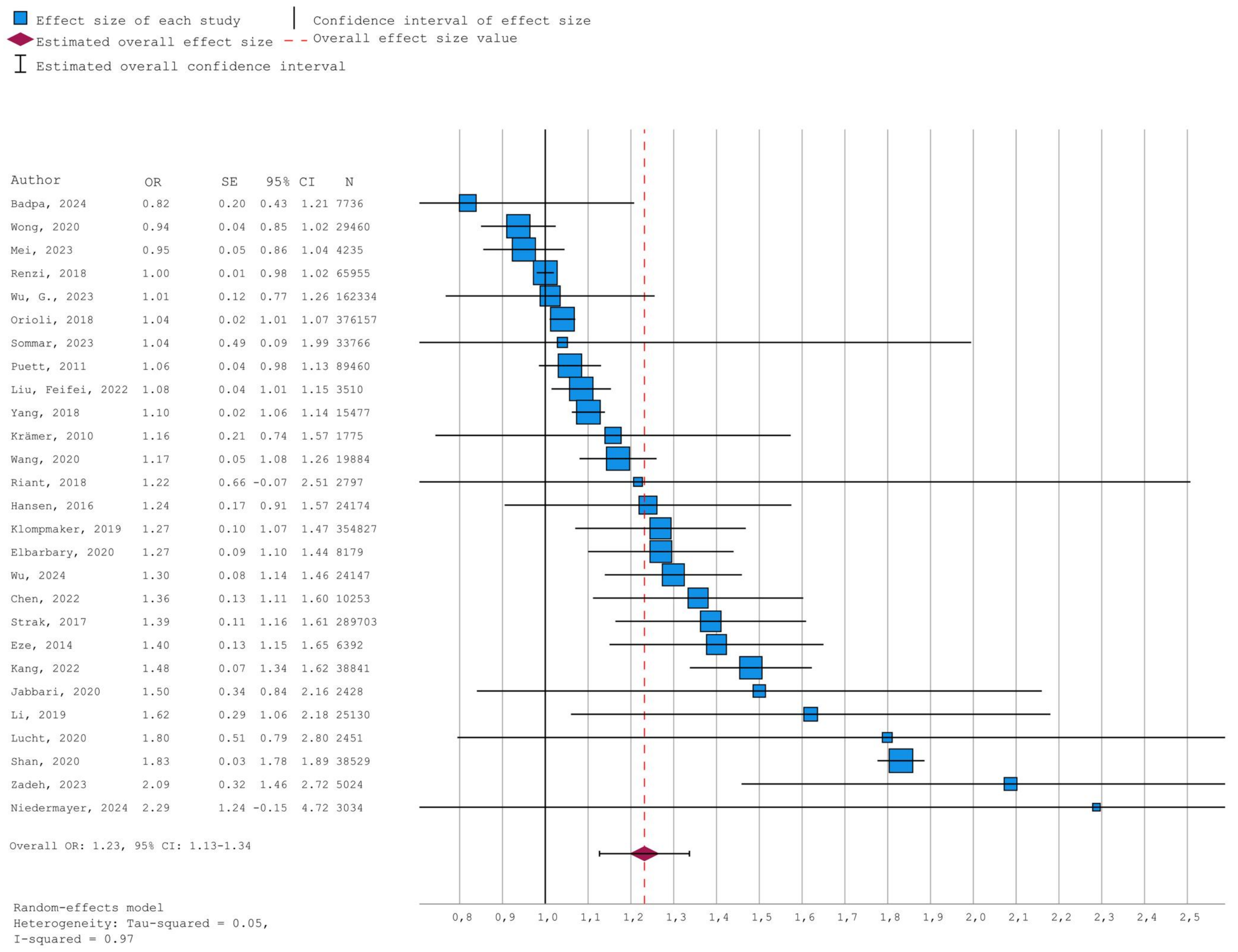


**Figure 3.** Results of the random effects meta-analysis of the association between PM10 per 10 μg/m3 and T2D (OR with 95% CI).

contrast, Zhou et al. reported a lower risk estimate from joint exposure of air pollutants $PM_{2.5}$ mass, $NO_2$, $O_3$, nitrate, ammonium, organic matter (OM), BC, chloride, and sulfate (HR: 1.48, 95% CI: 1.26-1.73) compared to the single-exposure model of $PM_{2.5}$ (HR: 1.75, 95% CI: 1.42-2.16).[60] Cui et al. also reported a lower risk estimate for joint exposure of six air pollutants ($PM_{2.5}$, BC, OM, ammonium, sulfate, and nitrate with OR: 1.06 (95% CI: 1.01-1.11) compared to the single-exposure model of $PM_{2.5}$ (OR: 1.18, 95% CI: 1.11-1.25).[57] Furthermore, the observed association of joint exposure using QGM was influenced by the stratification of green space exposure (measured as tree and grass cover). The risk of T2D was higher in the group exposed to low levels of green space (OR: 1.51, 95% CI: 1.38-1.64) compared to high exposure group, where a potential protective effect of green space was reported (OR: 0.85, 95% CI: 0.79-0.90).[57]

The potential modification effect of green space on the association between $PM_{2.5}$ and T2D was also assessed in three other studies. Zhang et al. found a 6% increase in the risk of T2D in a single-exposure model of $PM_{2.5}$ (HR: 1.06, 95% CI: 1.02-1.10). They further tested for the potential interactive effect of air pollution and greenness using the relative excess risk due to interaction (RERI) but did not detect a strong interaction effect between $PM_{2.5}$ and normalized difference vegetation index (NDVI) on diabetes, with RERI of −0.092 (95% CI: −0.551, 0.287).[61] Sørensen et al. assessed the effect modification by population density, road traffic noise, and surrounding green space, but no consistent indications of effect modification were found.[62] Sun et al. examined how green space (NDVI) influences the association between air pollutants and the risk of T2D. In subgroup analyses, participants with high $PM_{2.5}$ exposure had a greater risk of T2D in areas with low green space (HR: 2.39, 95% CI: 2.25–2.53) than those in areas with high green space (HR: 2.33, 95% CI: 2.18–2.48). The risk was considerably lower among participants with both low $PM_{2.5}$ exposure and low green space (HR: 1.13, 95% CI: 1.04–1.21). The low $PM_{2.5}$ with high green space was used as the reference group.[58] Hu et al. utilized the cumulative risk index (CRI) method and reported similar association between the single exposure model of $PM_{2.5}$ (HR: 1.05, 95% CI: 1.01, 1.10) and joint exposure of road traffic noise, $PM_{2.5}$, and $NO_2$ (HR: 1.06, 95% CI: 1.02-1.09).[51] Li et al. utilized air pollution score ($PM_{2.5}$, $PM_{2.5–10}$, $NO_2$, and $NO_x$) and found similar associations in the single-exposure model of $PM_{2.5}$ and the joint exposure of air pollutant score (HR: 1.04, 95% CI: 1.02-1.06).[63]

### *Particles with a diameter of less than 10 μm*

$PM_{10}$ was used as the main exposure in 40 studies, from which 27 were included in the meta-analysis. Every 10 μg/m$^3$ increase in $PM_{10}$ was associated with an increased risk of T2D OR: 1.23 (95% CI: 1.13-1.34, [$I^2$ = 97.1%, Figure 3]). Results from Egger's test suggest the presence of publication bias (P-value <0.001), and in funnel plot analysis, the studies were concentrated on the right side

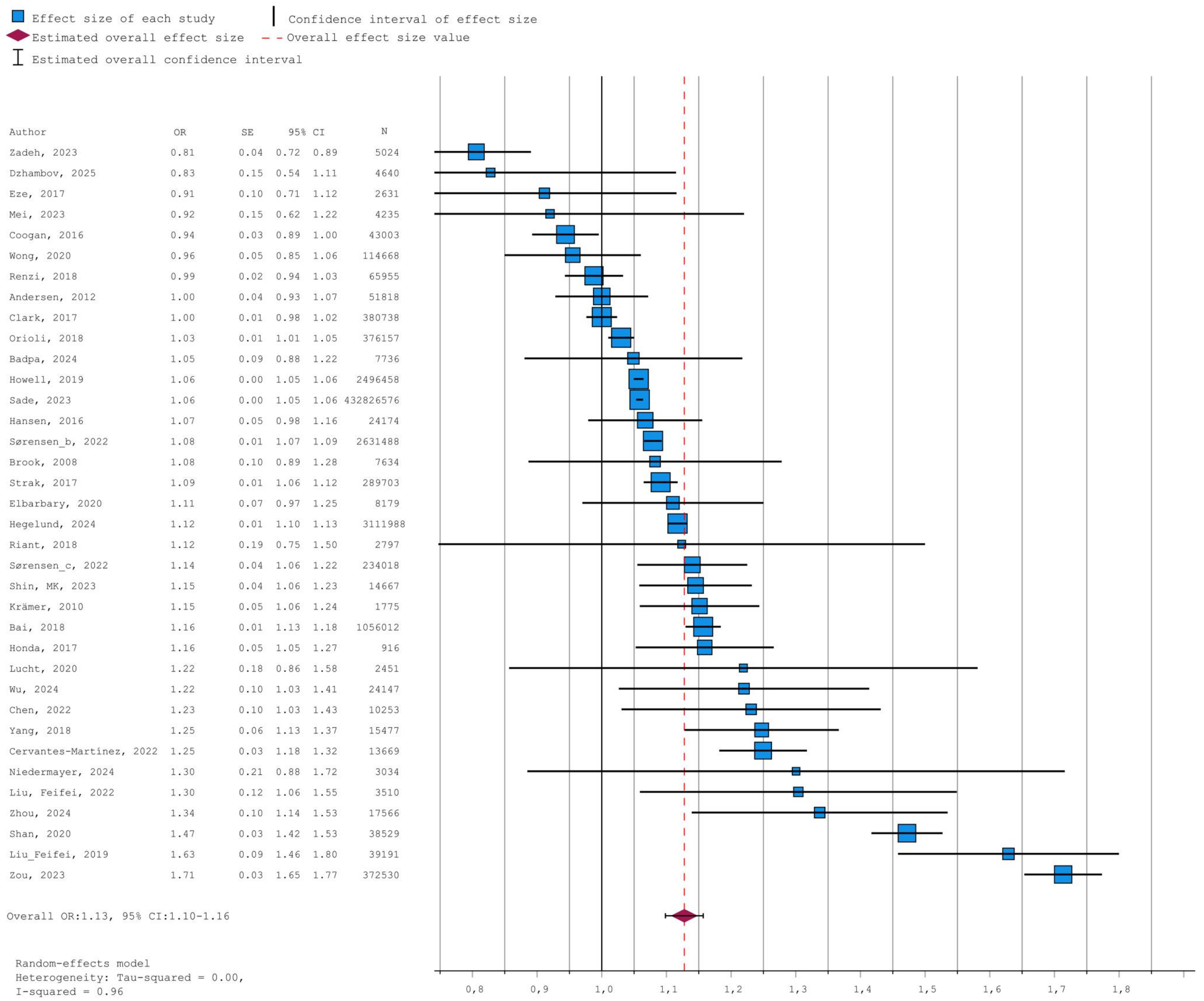


**Figure 4.** Results of the random effects meta-analysis of the association between NO2 per 10 μg/m3 and T2D (OR with 95% CI).

of the funnel (Figure S3). The Trim-and-Fill analysis imputed one study, but the observed plus imputed study effect estimate did not differ from the original effect estimate. In subgroup- or meta-regression analyses the study characteristics did not explain the considerable heterogeneity between the studies (Figure S7 and Table S6).

### *Particles with a diameter of less than 10 μm and joint exposure*

Of these 40 studies, 12 used multi-exposure models by adjusting for air pollution, walkability, railway-, or traffic noise.[39-41,43,46,49,54,56,64-67] Four studies did not find an association between $PM_{10}$ and T2D in eiher single-exposure or joint-exposure model adjusted for air pollution ($PM_{2.5}$, $NO_2$ or $O_3$).[39,41,49,66] In six studies, the risk estimate remained similar or showed slight attenuation after adjustment, with the difference between single- and multi-exposure models ranging from −0.01 to 0.05.[43,46,54,56,64,67] The most pronounced difference was reported by Li et al. where further adjustment of air pollutants ($SO_2$, $NO_2$, and $O_3$) substantially attenuated the association from RR: 1.62 (95% CI: 1.16-2.28) to RR: 1.10 (95% CI: 0.15-8.32).[65] Strak et al. reported a similar association when adjusting for $PM_{2.5}$ (OR: 1.05, 95% CI: 1.03-1.07), but adjusting for $NO_2$, the association attenuated and lost significance (from OR: 1.04, 95% CI: 1.02-1.06 to OR: 1.00, 95% CI: 0.97-1.02).[40] Zheng et al. utilized the QGC method for joint exposure of air pollutants (benzene, $NO_2$, $SO_2$, $PM_{10}$, and $PM_{2.5}$) and reported a higher risk estimate from the joint model (HR: 1.16, 95% CI: 1.10-1.22) compared to the single-exposure model of $PM_{10}$ (HR: 1.06, 95% CI: 1.01-1.120).[59]

**Nitrogen dioxide:** $NO_2$ was used as the main exposure in 59 studies, from which 36 were included in the meta-analysis. Every 10 μg/m$^3$ increase in $NO_2$ was significantly associated with an increased risk of T2D with an overall effect estimate OR: 1.13 (95% CI: 1.10-1.16, $I^2 = 96.5\%$, Figure 4). The funnel plot analysis appeared asymmetric with a scattered plot (Figure S4), and Egger's test was statistically significant ($P$-value <0.001), indicating a possible risk of bias. The Trim-and-Fill analysis did not impute any additional studies. Subgroup analyses (Figure S7) or meta-regression analyses (Table S6) per study characteristics were not able to explain the considerable heterogeneity between the studies. Only the study region, the Asian region, compared to North America, had a significant difference; studies conducted in the Asian region had a higher risk compared to studies conducted in North America (Estimate: 0.12, 95% CI: 0.004-0.05, $P$-value: 0.04).

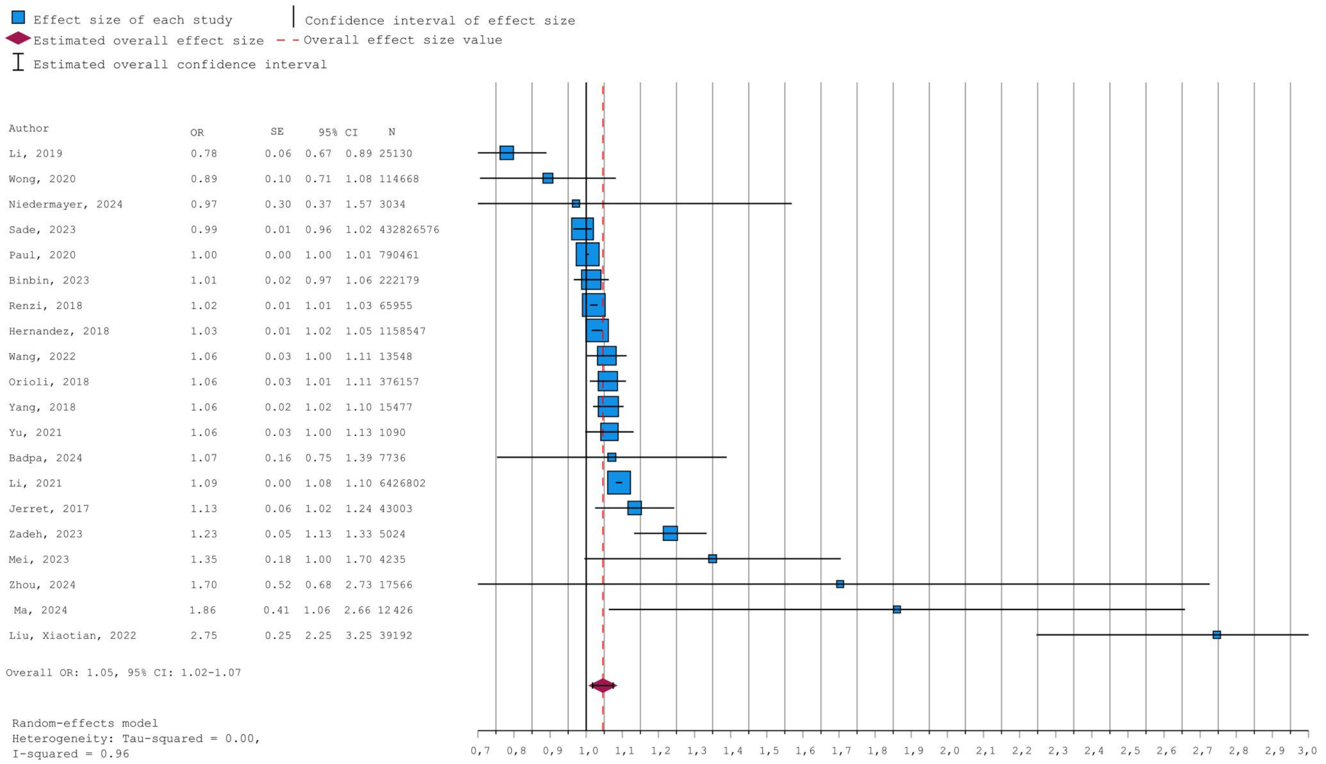


**Figure 5.** Results of the random effects meta-analysis of the association between O3 per 10 µg/m3 and T2D (OR with 95% CI).

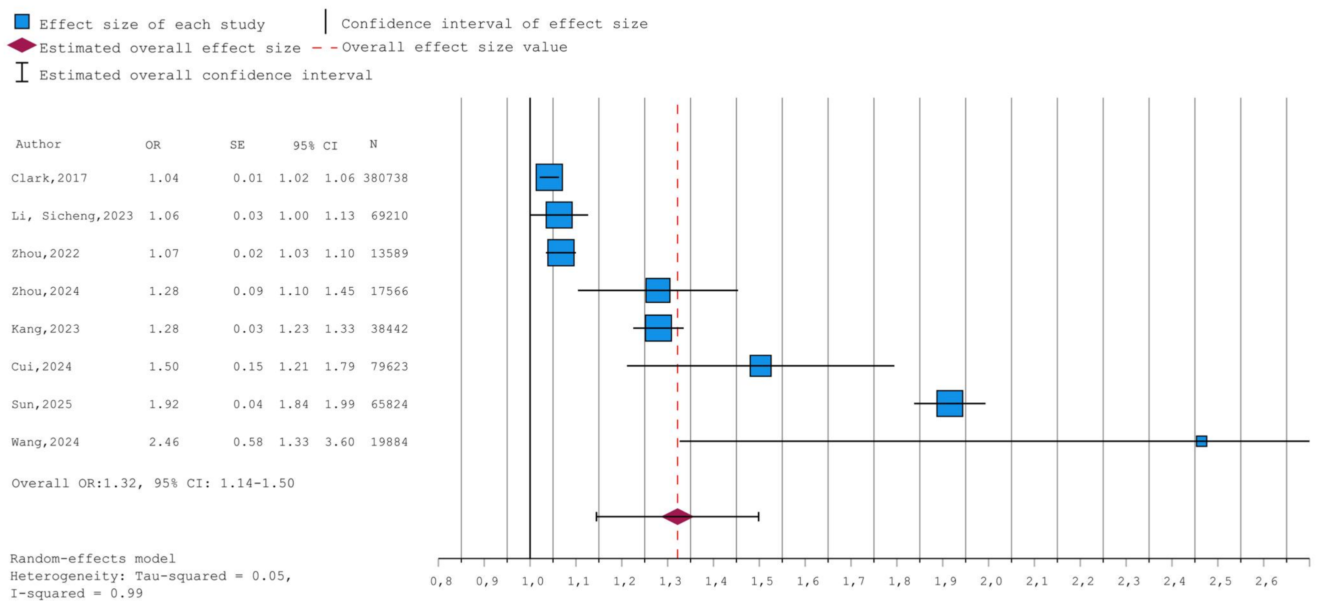


**Figure 6.** Results of the random effects meta-analysis of the association between T2D and BC per 5 µg/m3 (OR with 95% CI).

### *Nitrogen dioxide and joint exposure*

The joint exposure of environmental exposures was assessed in 23 of the 59 studies. Three studies found no association between $NO_2$ and T2D in either the single-exposure or joint-exposure model[49,68,69] and in three studies[56,64,70] association lost significance when adjusted for air pollution or noise exposures. The risk remained similar in six studies[39,40,46,47,50,71] and attenuated in three studies[44,54,55] where risk differences between single- and joint exposure models were between 0.10 and 0.32. The risk increased in one study, which showed the most pronounced difference when adjusting the single-exposure model for $PM_{10}$, the HR of T2D increased from 1.47 (95% CI: 1.42- 1.53) to HR: 2.23 (95% CI: 2.13-2.33). However, adjusting for $SO_2$ resulted in a more modest increase (HR: 1.61, 95%CI: 1.55-1.67).[67]

Three studies[51,53,72] utilized the CRI method to assess the joint exposure of environmental variables. Klompmaker et al. found the risk from the cumulative index ($NO_2$, traffic noise, NDVI) higher than the risk estimate of the single-exposure models of

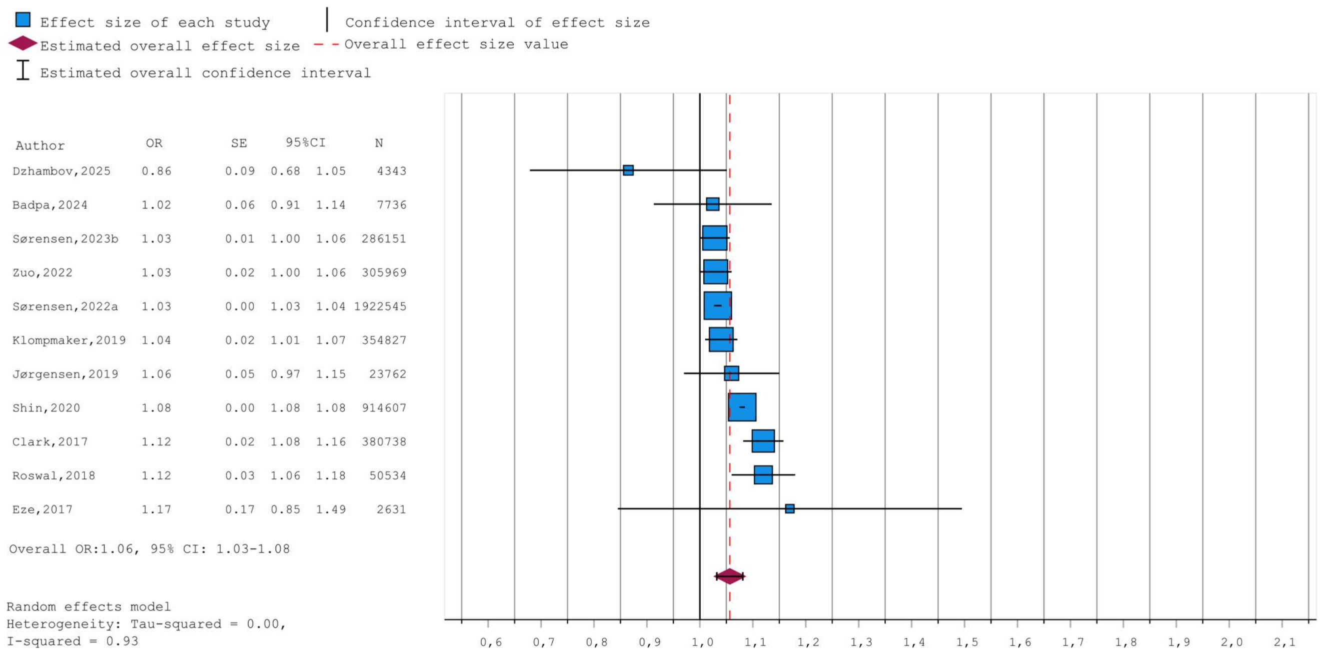


**Figure 7.** Results of the random effects meta-analyses of the association between traffic noise per 10 dB and T2D (OR with 95% CI).

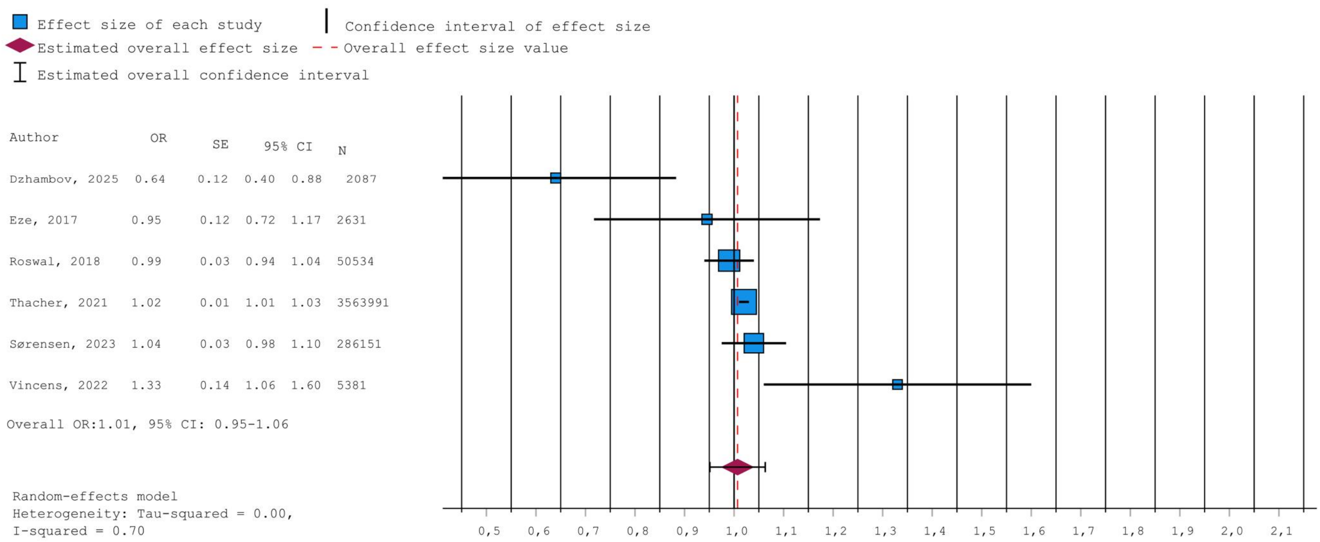


**Figure 8.** Results of the random effects meta-analyses of the association between railway noise per 10 dB and T2D (OR with 95% CI).

$NO_2$ exposure.[72] A similar result was reported by Sørensen et al. with single-exposure of $NO_2$ HR: 1.06 (95% CI: 1.05- 1.07) and CRI: of 1.13 (95% CI: 1.11-1.15) including total ultrafine particles (UFP), $NO_2$, noise, and green space.[73] Hu et al. reported similar association from the single exposure model (HR: 1.07, 95% CI: 1.02, 1.11) and the cumulative risk index of road traffic noise, $PM_{2.5}$, and $NO_2$ (HR: 1.06 (95% CI: 1.02-1.09).[51]

Zhou et al. used the QGC method and reported lower risk in the joint exposure model of air pollutants $PM_{2.5}$ mass, $NO_2$, $O_3$, OM, BC, nitrate, ammonium, chloride, and sulfate (HR: 1.48, 95% CI: 1.26—1.73) compared to single-exposure model of $NO_2$ (HR: 1.58, 95% CI: 1.25-1.99).[60] Whereas Zheng et al. reported a higher risk of T2D when using QGC method for joint exposure of air pollutants benzene, $NO_x$, $NO_2$, $SO_2$, $PM_{10}$, and $PM_{2.5}$ (HR: 1.16, 95% CI: 1.10-1.22) compared to the single-exposure model of $NO_2$ (HR: 1.07, 95% CI: 1.02-1.12).[59] Sørensen et al. assessed the effect modification by population density, road traffic noise, and surrounding green space, but no consistent indications of effect modification were found.[62] Howell et al. reported an increased risk of T2D when further adjusting $NO_2$ for walkability (from OR: 1.11, 95% CI: 1.10-1.13 to OR: 1.16, 95% CI: 1.14-1.17). Significant interaction was found between $NO_2$ and walkability, indicating that at low levels of $NO_2$, the likelihood of T2D was higher among those living in less walkable neighborhoods. However, the probability of T2D rose in highly walkable neighborhoods and became comparable across all levels of walkability.[74]

### *Ozone*

$O_3$ was used as the main exposure in 20 studies, all included in the meta-analysis. For every 10 µg/m$^3$ increase in $O_3$, the risk of

T2D was OR: 1.05 (95% CI: 1.02-1.08, $I^2$ = 96.6%, Figure 5). The funnel plot analysis appeared asymmetric (Figure S6), and Egger's test was statistically significant ($P$-value < 0.001). The Trim-and-fill analysis imputed 3 studies, but the estimate of the corrected combined effect size did not change from the original meta-analysis estimate. Subgroup analyses per study characteristics (Figure S7) showed some variation, but adjustment and ROB-score were the only significant covariates in meta-regression analyses (Table S6). Studies that adjusted for less than five out of eight T2D risk factors (age, sex, SES, BMI, smoking, physical activity, family history of diabetes, measure of nutrition/diet) showed lower risk compared to studies that did adjust at least for 5 of these covariates (estimate: −0.10, 95% CI: −0.16, −0.03, $P$-value: 0.006). Studies with high ROB showed a smaller risk compared to studies with low ROB (estimate: −0.12, 95% CI: −0.023, −0.01, $P$-value: 0.03). Other study characteristics did not explain the considerable heterogeneity that was observed between the studies.

### *Ozone and joint exposure*

Joint effects of $O_3$ and environmental exposures were assessed in eight studies.[39,50,60,65,75-78] The most pronounced difference was observed by Zhou et al. where the single exposure model of $O_3$ was not significant (HR: 1.11, 95% CI: 0.99-1.24) but when using the QGC method for joint exposure to air pollutants ($PM_{2.5}$ mass, $NO_2$, $O_3$, OM, BC, nitrate, ammonium, chloride, and sulfate) the risk of T2D increased to HR: 1.48 (95% CI: 1.26-1.73).[60] Li et al. found differing results when adjusting for co-pollutants. The association between $O_3$ and T2D in the single-pollutant model was protective (RR: 0.78, 95% CI: 0.68-0.90), but after adjustment for co-pollutants ($PM_{10}$, $NO_2$, $SO_2$), the association became nonsignificant (RR: 1.07, 95% CI: 0.35-6.81).[65]

Li et al. found a slightly stronger association when adjusting for $SO_2$ and $PM_{2.5}$ HR: 1.09 (95% CI: 1.09-1.10) or $SO_2$ and $PM_{10}$ HR: 1.08 (95% CI: 1.07-1.08) compared to the single-exposure model of $O_3$ HR: 1.06 (95% CI: 1.05-1.06).[76] In single-exposure models, Renzi et al. found a modest association between $O_3$ and T2D for incident cases (HR: 1.01, 95% CI: 1.00 - 1.02) but not for prevalent cases. (OR: 1.00, 95% CI: 0.99-1.01). The results stayed similar when adjusting for noise (day-evening-night level, Lden) and green space (NDVI) or in two-pollutant models adjusting for another pollutant ($PM_{10}$, $PM_{2.5}$, $NO_2$, $NO_x$).[39] Similarly, Paul et al. found a modest association from the single-exposure model (HR: 1.01, 95% CI: 1.00–1.01) which remained when adjusting for other air pollutants ($PM_{2.5}$ and $NO_2$).[50] Liu et al. reported that compared to the single-exposure model (OR: 1.50, 95% CI: 1.40-1.62) adjustment for $PM_{2.5}$ in a two-pollutant model resulted in a slightly higher risk estimate (OR: 1.52, 95% CI: 1.35-1.72), whereas adjustment for $PM_{10}$ (OR: 1.40, 95% CI: 1.25-1.57) or $NO_2$ (OR: 1.48, 95% CI: 1.31-1.68) yielded lower estimates.[77]

In a study by Jerret et al. adjustment for $PM_{2.5}$ and $NO_2$, the risk from the single-exposure model (HR: 1.18, 95% CI: 1.04-1.34) attenuated to a non-significant (HR: 1.13, 95% CI: 0.97-1.31). They also explored the possible modification effect but found no interaction between $PM_{2.5}$ and $O_3$, but borderline evidence of an interaction between $O_3$ and $NO_2$, where the HRs for $O_3$ levels were larger in areas of lower $NO_2$ (interaction $P$-value: 0.09).[75] For Wang et al. adjustment for $PM_{2.5}$ did not change the result of the single-exposure model (HR: 1.06, 95% CI: 1.00-1.11) but the effect estimate was slightly stronger in the high $PM_{2.5}$ level compared to the low $PM_{2.5}$ level (HR = 1.07, 95% CI: 1.01-1.12) with no between-group significance.[78]

### *Black carbon*

BC was used as the main exposure in eight studies. The meta-analysis, including all these studies, showed a significant association between T2D and BC per 5 μg/m$^3$ increase OR: 1.32 (95% CI: 1.15-1.50, $I^2$ = 98.6%, Figure 6). The funnel plot was asymmetric (Figure S6), and Egger's test was significant ($P$-value: <0.001). The Trim-and-fill analysis did not impute any studies. Subgroup- or meta-regression analyses were not conducted due to a small number of studies.

### *Black carbon and joint exposure*

Joint effects of BC and other environmental exposures were examined in seven studies.[52,57,58,60,79-81] Clark et al. was the only study using only adjustment, reporting that the association observed in the single-exposure model (OR: 1.03, 95% CI: 1.01-1.04) attenuated after adjusting for transportation noise, greenness, and walkability (OR: 1.00, 95% CI: 0.98-1.02).[52] Li et al. reported smaller risk estimate in the joint exposure model (OR: 1.04, 95% CI: 1.01-1.07) using weighted quantile sum (WQS) method for a score of $PM_{2.5}$, BC, nitrate, organic matter, soil particles, ammonium, sulfate compared to the single exposure of BC (OR: 1.07, 95% CI: 1.01-1.15).[80] Kang et al. utilized proportion and residual analyses to specify the most responsible constituents of $PM_{2.5}$ (BC, OM, ammonium, nitrate, inorganic sulfate, soil particles, and sea salt) showing that BC was the most responsible constituent, in which 1% increase in the proportion of BC corresponded with 1.51-fold risk (95% CI: 1.29-1.77) for T2D.[79]

The QGC method was used in four studies.[57,58,60,81] Cui et al. assessed the joint exposure of air pollutants ($PM_{2.5}$, BC, organic matter, ammonium, nitrate, sulfate) and the risk of diabetes was higher in single exposure models (for BC OR: 1.13, 95% CI: 1.07-1.20) compared to the joint exposure (OR 1.06, 95% CI: 1.01-1.11).[57] Sun et al. assessed the joint exposure of BC, OM, ammonium salt, nitrate, sulfate, and chloride which showed a stronger association (HR: 1.46, 95% CI: 1.43-1.49) compared to single-exposure model of BC (HR: 1.40, 95% CI: 1.38-1.42). In stratification analyses the participants with high exposure to BC had higher risk of T2D in areas with low green space (low NDVI) (HR: 2.18, 95% CI: 2.05-2.31) compared to areas with high NDVI (HR: 1.95; 95% CI: 1.83-2.08), using the low BC/high NDVI group as the reference group.[58] Wang et al. reported that in joint exposure model of BC, sulfate, nitrate, ammonium, and organic matter the risk of T2D was lower but more precise (HR: 1.27, 95% CI: 1.09-1.49) than in single-exposure model of BC (3.80, 95% CI: 1.83-7.16). Further adjustment of the joint exposure model by $NO_2$, $PM_{10}$, and $SO_2$ increased the risk to HR: 1.35 (95% CI: 1.15-1.60). Of the five constituents, BC had the greatest positive contribution (32.7%) to the mixing effect on the risk of T2D.[81] Zhou et al. reported a higher risk in the joint exposure model of air pollutants: $PM_{2.5}$ mass, $NO_2$, $O_3$, nitrate, ammonium, organic matter, BC, chloride, and sulfate (HR: 1.48, 95% CI: 1.26—1.73) compared to single-exposure model of BC (HR: 1.45, 95% CI: 1.18-1.78).[60]

## Noise exposure

The characteristics of the 20 studies that used noise as main exposure are shown in Table 2 Of those, 18 studied road traffic noise, 6 railway noise, and 5 aircraft noise exposure. Of the included studies, 16 were conducted in Europe, two in Canada, one in North America, and one in the Asian region. Regarding the study design, 13 studies used a longitudinal study design and seven cross-sectional design.

**Table 2.** Characteristics of included noise exposure articles (n = 20). The table is organized in alphabetical ascending order of the first author.

| Author, Year | Country | Study | N | Age | Baseline | Follow-up | Outcome | Exposure | Units |
|---|---|---|---|---|---|---|---|---|---|
| Badpa et al., 2024[111] | Germany | Cooperative Health Research in the Region of Augsburg (KORA-Study) | 7736 | 49.2 | 1994-1995, 1999-2001 | 15.0 years | Self-reported, Register | Traffic | per IQR 7.9 dB during night |
| Clark et al., 2017[52] | Canada | Population Data BC | 380 738 | 58.00 | 1994 | 4.0 years | Register | Traffic | Lden per 1 IQR (6.8dBa) |
| Dzhambov et al., 2016[139] | Bulgaria | Cross-sectional study in Plovdiv city, Bulgaria | 513 | 36.45 | 2014 | – | Self-reported | Traffic | Categories: Lden 71-80 dB with ref. category 51-70 dB |
| Dzhambov et al., 2025[69] | Bulgaria | Cross-sectional study in 5 Bulgarian cities | 4640 | 49 | 2023 | – | Self-reported | Traffic<br>Railway<br>Aircraft | per 5 dB<br>per 5 dB<br>per 5 dB |
| Eriksson et al., 2014[89] | Sweden | Stockholm Diabetes Prevention Program | 5156 | 47.00 | 1992 | 8.9 years | Self-reported, Clinical | Aircraft | Categories: ≥ 50 versus < 50 dB(A) |
| Eze et al., 2017[68] | Switzerland | Swiss study on Air Pollution and Respiratory Diseases in Adults | 2631 | 59.20 | 2002 | 8.3 years | Self-reported Clinical | Traffic,<br>Aircraft<br>Railway | Lden per 1 IQR (10 dB)<br>Lden per 1 IQR (12dB)<br>Lden per 1 IQR (11dB) |
| Hu et al., 2023[51] | UK | UK Biobank | 390 834 | 56.3 | 2006-2010 | 10.9 years | Register | Traffic | per IQR 3.5 |
| Jørgensen et al., 2019[88] | Denmark | The Danish Nurse Cohort | 23 762[a] | 54.00 | 1993 | 15.2 years | Register | Traffic | Lden per 10 dB increase |
| Klompmaker et al., 2019[72] | Netherlands | Dutch Public Health Monitor | 354 827 | Cat. | 2012 | – | Self-reported | Traffic | Lden Per 5 dB |
| Letellier et al., 2023[82] | USA | The Community of Mine Study | 573<br>316 | 58.7 | 2014-2017 | – | Clinical | Aircraft<br><br><br>Traffic | Static and dynamic exposure; ≥45 dB(A), ≥ median (0.10) and as continuous exposure<br>Static; ≥53 dB(A) census tract level, ≥55 dB(A) buffer around home, continuous, dynamic; ≥ median (0.13) and continuous exposure |
| Ohanyan et al., 2022[36] | Netherlands | UK Biobank | 14 410 | 50.70 | 2011-2012 | – | Self-reported | Traffic | Categories: Lden>55 dB versus <55dB |

(continued)

**Table 2.** (continued)

| Author, Year | Country | Study | N | Age | Baseline | Follow-up | Outcome | Exposure | Units |
|---|---|---|---|---|---|---|---|---|---|
| Roswal et al., 2018[83] | Denmark | Danish Diet, Cancer and Health Cohort | 50 534 | 56.00 | 1993 | 15.5 years | Register | Traffic<br>Railway | Lden per 10 dB<br>Lden per 10 dB |
| Shin et al., 2020[84] | Canada | Ontario Population Health and Environment Cohort | 914 607 | 55.30 | 2001 | 15.0 years | Register | Traffic | per 1 IQR (10 dB) |
| Sørensen et al., 2013[189] | Denmark | Danish Diet, Cancer and Health Cohort | 50 187 | 56.10 | 1993 | 9.6 years | Register | Traffic | per 10 dB |
| Sørensen et al., 2022[53] | Denmark | Danish National Register data | 1 922 545 | 57.5 | 2005 | 11.2 years | Register | Traffic | per IQR: 10.6 dB in most exposed facade and per IQR 9.5dB least exposed facade |
| Sørensen et al., 2023[85] | Denmark | Danish National Health Survey | 286 151 | 55.20 | 2010 | 6.2 years | Register | Traffic<br>Railway | per 10dB increase[a]<br>per 10 dB increase[a]<br>[a]Least and most exposed facades |
| Thacher et al., 2021[86] | Denmark | Danish National Register data | 3 563 991 | 50.75 | 2000 | 12.9 years | Register | Traffic<br>Railway<br>Aircraft | per 10 dB increase[a]<br>per 10 dB increase[a]<br>[a]Grouping: Lden max and Lden min Cat. (5): <45, 45-49 50-54, 55-59, ≥60 |
| Vincens et al., 2022[190] | Sweden | Swedish register data | 5381 | Cat. | 2017 | – | Register | Railway | per 10 dB |
| Yu et al., 2024[191] | China | Data from 480 community residents in China | 480 | 54 | 2017-2018 | – | Clinical | Traffic | Q1 (<51.5 dB), Q2 (51.5-<53.9 dB), Q3 (53.9-<58.0 dB), Q4 (≥58.0 dB) and as continuous exposure |
| Zuo et al., 2022[87] | UK | UK Biobank | 305 969 | 57.10 | 2006-2010 | 11.9 years | Register | Traffic | per 10 dB |

[a]Study population only women.

### *Traffic noise*

Traffic noise was used as the main exposure in 18 studies, of which 11 were included in the meta-analysis. We found a 6% increase (OR: 1.06, 95% CI: 1.03-1.08; $I^2 = 92.8\%$) in the risk of T2D associated with a 10 dB increase in exposure to traffic noise (Figure 7). The funnel plot analysis (Figure S8) and Egger's test (P-value > 0.001) indicated the potential presence of publication bias. The Trim-and-Fill method did not impute additional studies. Due to the similarity of study characteristics, we did not perform any further subgroup analysis.

### *Traffic noise and joint exposure*

Of these 18 studies, 13 assessed the joint exposure of the selected environmental exposures.[51-53,68,69,72,82-88] Nine studies accounted for air pollution, green space, walkability, railway- or aircraft noise as potential confounders.[52,68,69,82-87] In five of these, the risk estimates remained or slightly attenuated after adjusting for one or more co-environmental exposures (risk differences between single- and joint exposure models between 0.00 and 0.02).[52,83,84,86,87] Dzhambov et al. did not find an association between traffic noise and T2D in either single- or joint exposure models.[69] Letellier et al. did not report separately the results from single-exposure model, but in their two-exposure model adjusted for $NO_2$, they did not find a significant association between traffic noise and the risk of T2D (OR: 1.02, 95% CI: 0.84-1.24).[82] Eze et al. showed the most pronounced difference between single- and multi-exposure models. Specifically, adjusting the single-exposure model for green space, $NO_2$, aircraft- and railway noise increased the RR of T2D from 1.17 (95% CI: 0.88-1.53) to RR: 1.35 (95% CI: 1.02-1.78).[68] Sørensen et al. reported that the association observed in the single-exposure models of traffic noise exposure (least- and most-exposed facades) weakened after adjusting for railway noise and $PM_{2.5}$. In modification analyses, the CIs were overlapping, but the association of traffic noise exposure with T2D seemed strongest among people living in suburban areas (population density of 101–2000 persons per $km^2$).[85]

Three studies utilized a CRI method to assess the joint exposure of environmental variables.[51,53,72] In all three, the risk from the cumulative index was higher than the risk estimate of the single-exposure models of traffic noise exposure. Klompmaker et al. also tested for the potential interaction effect of green space but did not find a significant interaction between NDVI and road traffic noise on the risk of T2D. [72] Three studies further tested for the potential effect modification of air pollution on the association between road traffic noise and T2D but didn't find a difference in the associations.[68,85,88]

### *Railway noise*

The meta-analysis for railway noise exposure included all six extracted studies, all conducted in the European region. The results (Figure 8) did not show an association between exposure to railway noise and T2D (OR: 1.01, 95% CI: 0.95-1.06; $I^2 = 69.8\%$) with an indication of publication bias (Egger's test P-value: <0.001). The Trim-and-Fill method imputed one additional study and increased the estimated overall effect estimate (observed plus imputed) to OR: 1.03, 95% CI: 0.96-1.09 (Figure S9). Due to the small number of studies and the similarity of study characteristics, we did not perform any further subgroup analysis.

**Railway noise and joint exposure:** Joint effects of railway noise and other environmental exposures were examined in four studies.[68,83,85,86] Eze et al. found no evidence of association between railway noise and the risk of T2D either in single exposure model or joint-exposure model adjusting for green space, $NO_2$, aircraft noise, and road traffic noise.[68] Similarly, Roswall et al. observed no association in either single or two exposure models, the latter adjusted for road traffic noise. They further investigated the potential effect modification by road traffic noise exposure but found no interaction.[83] Sørensen et al. reported that the association observed in the single-exposure model weakened and lost significance after adjusting for road traffic noise and $PM_{2.5}$.[85] Thacher et al. also found that the association between railway noise and the risk of T2D was slightly attenuated when further adjusting for $PM_{2.5}$, green space, road-, and aircraft noise.[86]

### *Aircraft noise*

Five studies assessed aircraft noise exposure and the risk of T2D.[68,69,83,86,89] Only one of the studies found a significant association between aircraft noise and T2D, where independent of the other transportation noise sources and $NO_2$, the risk of incident diabetes was RR: 1.92, 95% CI: 1.04-3.55.[68] Meta-analysis was not performed due to the low number of studies that could be standardized for the analysis.

### *Aircraft noise and joint exposure*

Joint exposures were examined in three of the five studies. Eze et al. observed an increase in the multiexposure model adjusting for $NO_2$, road- and railway noise exposure (from RR: 1.92, 95% CI: 1.04-3.55 to RR: 1.96, 95% CI: 1.00-3.81). However, further adjustment for green space attenuated the results to non-significant (RR: 1.87, 95% CI: 0.96-3.62).[68] In the study by Thacher et al. further adjustment for green space, $PM_{2.5}$, road traffic- and railway noise slightly increased the risk of T2D, but being significant only in the category of 50–54 decibels (HR: 1.04, 95% CI: 1.01-1.07), under 45 decibels was used as the reference category.[86] Letellier et al. did not report results from the single-exposure model, but in their two-exposure model adjusted for $NO_2$, they did not find an association between aircraft noise exposure and the risk of T2D (OR: 1.58, 95% CI: 0.85-2.93).[82]

## Built environment

The characteristics and main results of the 39 studies exploring the built environment (green space, walkability, and population density) are presented in Table 3 Due to the high variation in exposure assessment methods, we provide here a narrative synthesis of the results without performing meta-analyses for the association of the built environment exposures with T2D.

### *Green space*

A total of 30 studies used green space as the main exposure. The studies were conducted in various geographic locations utilizing different assessment methods of exposure to green spaces. The most commonly used measure of green space was NDVI, which indicates the amount of green vegetation in the environment. NDVI was measured with buffers varying from 100 meters to 3 kilometers. Out of 19 cross-sectional studies, 13 reported an inverse association between exposure to green space and T2D[36,72,90-100] and five did not find a significant association.[38,69,101-103] Plans et al. found a significant association for women only, while the OR for a model including both women and men was 1.44 CI: 0.82-2.52 in high (quartile 1) versus low (quartile 4) green space density.[104]

Eight studies with a longitudinal study design reported a lower risk of T2D with higher greenness.[52,58,61,105-109] Yu et al. showed the strongest association, where IQR increase in the cumulative average of NDVI in the 250-meter buffer was associated with a 44% (HR: 0.56, CI: 0.51, 0.61) reduction in risk of T2D in China. Results remained similar with 500 and 1000 m buffers.

**Table 3.** Characteristics of included built environment articles (n = 38). The table is organized in alphabetical ascending order of the first author.

| Author, Year | Country | Study | N | Age | Baseline | Follow-up | Exposure | Confounders | Results |
|---|---|---|---|---|---|---|---|---|---|
| Albers et al., 2024[110] | Netherlands | The Maastricht Study | 6695 | 60.0 | 2010-2020 | 6.2 years | Walkability | Age, sex | Walkability index 0-100 (1650 m radius) per IQR , 52.23-22.87 HR: 1.23, 95% CI: 0.95-1.58 for incident diabetes. |
| Anza-Ramirez et al., 2022[102] | Latin America | SALURBAL project (Salud Urbana en America Latina/ Urban Health in Latin America) | 122 211 | 42.6 | 2002-2013 | – | Green space | Age, sex, education, population educational attainment at sub-city level % of urban area, country, sub-city intersection density and population density, city isolation- and fragmentation | Per 1 SD (0.2) of sub-city greenness (median NDVI) OR: 0.98, 95% CI: 0.94; 1.02. |
| | | | | | | – | Population density | Age, sex, education, population educational attainment at sub-city level % of urban area, country, sub-city intersection density, greenness, city isolation- and fragmentation | Per 1 SD of sub-city population density (4.876/km2) OR: 0.96, 95% CI: 0.92-1.00. |
| Astell-Burt et al., 2014[101] | Australia | 45 and Up Study | 267 072 | cat | 2006-2009 | – | Green space | Age, sex, couple status, ancestry, country of birth, language spoken at home, weight, risk of psychological distress, smoking, hypertension, diet, active lifestyle, employment status, annual income, education | Green space categories (4) in 21-40% OR: 0.99, 95% CI: 0.96-1.03. 41-60%: OR 0.90; 95% CI 0.85-0.96. 61-80%: OR: 91, 95% CI: 0.84-0.99. 81%+: OR: 0.94, 95% CI: 0.85-1.03. |
| Badpa et al., 2024[111] | Germany | KORA-study: Cooperative Health Research in the Region Augsburg | 7736 | 49.2 | KORA S3: 1994-1995 KORA S4: 1999-2001 | until 2016 | Green space | Age, sex, sub-cohort indicator, BMI, smoking status, alcohol consumption, education level, physical activity, and dietary score. | Green space (NDVI 1000m buffer) per IQR 0.14 HR: 0.98, 95% CI: 0.88-1.09. |
| Bodicoat et al., 2014[91] | UK | ADDITION-Leicester, Let's Prevent Diabetes, Walking Away from Diabetes | 10 476 | 59 | 2004-2011 | – | Green space | Age, sex, urban/rural location, area level social deprivation | Green space (3km buffer) quartiles (4) highest vs lowest quartile OR: 0.53, 95% CI: 0.35-0.82. |
| Booth et al., 2019[117] | Canada | National register data, Canada | 958 567 | 48.5 | 2002 | 9.2 years | Walkability | Age, CVD, hypertension, SES, Ethnicity, immigration status, city/town of residency | Walkability per categories, low versus highest category HR: 0.85, 95% CI: 0.78-0.93. |
| Booth et al., 2013[113] | Canada | National register data, Canada | 1,239,262 | 45 | 2005 | 5.0 years | Walkability | Age, sex, income (area level poverty used as a surrogate) | Most walkable quintile (5) versus least walkable quintile (1) RR: 1.32, 95% CI: 1.26-1.38 for men and RR: 1.24, 95% CI: 1.18-1.31 for women. |
| Clark et al., 2017[52] | Canada | Population Data British Columbia | 380 738 | 58.0 | 1994 | 4.0 years | Green space | Age, gender, and area-level household income | NDVI 100m buffer per IQR 0.12, OR: 0.90, 95% CI: 0.87-0.92. |
| | | | | | | | Walkability | Age, gender, and area-level household income | Neighborhood walkability index per IQR 4.3, OR: 1.01, 95% CI: 0.98-1.04. |

(continued)

**Table 3.** (continued)

| Author, Year | Country | Study | N | Age | Baseline | Follow-up | Exposure | Confounders | Results |
|---|---|---|---|---|---|---|---|---|---|
| Dalton et al., 2016[105] | UK | European Prospective Investigation of Cancer Norfolk | 23 865 | 59.1 | 1993-1997 | until 2007 | Green space | Sex, age, BMI, parental DM, SES | Greenspace 800m buffer per quantiles (4) ref 1. least green versus 4 most green HR: 0.81, 95% CI: 0.65-0.99. |
| Dendup et al., 2019[90] | Australia | 45 and Up Study | 46 786 | cat | 2006-2009 | – | Green space | Age, sex, household income, education, economic status, couple status | Per 1% increase in total green space OR: 0.993, 95% CI: 0.988 to 0.998. |
| | | | 43 137 | cat | 2006-2009 | 2012-2015 | Green space | See above | Per 1% increase in total green space OR: 0.99, 95% CI: 0.99-1.00. |
| Doubleday et al., 2022[106] | US | MESA: Multi-Ethnic Study of Atherosclerosis | 5574 | – | 2000 | 15.8 years | Green space | Age, sex, ethnicity, education, income, employment status, neighborhood deprivation, neighborhood social cohesion-, walkability- and safety, urbanicity, site, family history of DM, BMI, physical activity, chronic stress, smoking, drinking | Green space per IQR (55) increase annual median NDVI with 1 km buffer HR: 0.79, 95% CI: 0.63-0.99. |
| Dzhambov et al., 2025[69] | Bulgaria | Cross-sectional study in 5 Bulgarian cities | 4640 | 49.0 | 2023 | – | Green space | Age, sex, ethnicity, education, income adequacy, employment, city, and urbanicity | NDVI (300m buffer) per 0.1 increase; categories (4) 1:ref. 0.19-0.32: 1.00 2: 0.33-0.37 OR: 0.89, 95% CI: 0.64-1.26 3: 0.38-0.43 OR: 1.32, 95% CI:0.95, 1.85 4: 0.42-0.75 OR: 0.84, 95% CI: 0.59, 1.20. |
| | | | 4369 | | | | Walkability | Age, sex, ethnicity, education, income adequacy, employment, city, and urbanicity | Walkability (300m buffer) OR: 1.04, 95% CI: 0.87-1.23. |
| Fan et al., 2019[92] | China | Cross-sectional study in Kashgar city, China | 4670 | 47.2 | 2016 | – | Green space | Age, sex, education, marital status, physical activity | Green space (NDVI 1 km buffer) per one IQR (0.6) increase: OR: 0.92, 95% CI: 0.86, −0.99. |
| Frank et al., 2022[115] | Canada | My Health My Community | 22 418 | 45.58 | 2013-2014 | – | Walkability | Age, gender, income. ethnicity, regional accessibility, years in the neighborhood | Walkability per quintiles (5) ref. car dependent (Q1) versus walkable category (Q5) OR: 0.62, 95% CI: 0.45-0.85. |
| Glazier et al., 2014[116] | Canada | Canada census survey, national health survey, and diabetes database | 2 446 029 | Between 30-64 | 2003-2009 | – | Walkability | Age, sex | Walkability (800m buffer) per quintiles (5) Q1:Q5 RR: 1.33, 95% CI: 1.33-1.33. |
| Howell et al., 2019[74] | Canada | Cardiovascular Health in Ambulatory Care Research Team CANHEART-Study | 2,496,458 | 53 | | – | Walkability | Age, sex, ethnicity, immigration history, neighborhood COPD, comorbidity burden | Walkability per quintiles (5) lowest vs. highest walkability OR: 1.25, 95% CI: 1.22. |
| Hu et al., 2023[98] | China | Chinese Longitudinal Healthy Longevity Survey | 3924 | 84.6 | 2017-2018 | – | Green space | marital status, education level, household income level, smoking status, and drinking status. | NDVI (500m buffer) per quartiles (4) Q1: ≤0.14, Q2: >0.14-0.17), Q3: >0.17-0.21, Q4: >0.21. OR 0.55, 95% CI: 0.43-0.71. |

(continued)

**Table 3.** (continued)

| Author, Year | Country | Study | N | Age | Baseline | Follow-up | Exposure | Confounders | Results |
|---|---|---|---|---|---|---|---|---|---|
| Hua et al., 2024[121] | US | The New York University Women's Health Study | 11 307[a] | 50.4 | 1985-1991 | 25.6 years | Walkability | Age, race, education level, smoking, alcohol use and parity. Model 3 further included neighborhood poverty level, moving | Walkability as the neighborhood walkability score (residential density, destination accessibility, street connectivity, and rail transit density) per SD 0.9 HR: 0.89, 95% CI 0.86-0.92. |
| Ihlebæk et al., 2018[103] | Norway | Oslo Health Study (HUBRO) | 8638 | Cat. 1. 29-39; 2. 40-59; 3. 60 | 2000-2001 | – | Green space | Age, ethnicity, education, civil status, smoking, PA, occupation, negative life events, social support, stability in neighborhood, income, % living in owned house, education | Green space per quintiles (5) least (1) versus highest quintile (5) for women OR: 1.91, 95% CI: 0.49-7.43 for men OR: 0.96, 95% CI: 0.27-3.44. |
| Jian et al., 2024[99] | China | Population-based survey: members of Third Division of the Xinjiang Production and Construction Corps | 9723 | 18y and older: 7,718 ≤ 50y (79.4%) & 2,005 >50y (20.6%) | 2016 | – | Green space | Age, sex, education level, marital status | NDVI (500m buffer) per IQR (value na) OR: 0.85, 95% CI: 0.74-0.97. |
| Kartschmit et al., 2020[118] | Germany | Heinz Nixdorf Recall Study, Dortmund Health Study, KORA, CARLA, Study of Health in Pomerania | 16,008 | 58.7 | 1997-2006 | – | Walkability | Sex, age, education, cohort, BMI | Walkability (impedance) per 1 SD (291.3) RR: 1.05, 95% CI: 0.99-1.11. |
| | | | 12 105 | 55.95 | 1997-2006 | 9.2 years | Walkability | Sex, age, education, cohort, BMI | Walkability (impedance) per 1 SD (286.8) RR: 1.01, 95% CI: 0.95-1.08. |
| Khan et al., 2021[93] | Bangladesh | Bangladesh Demographic and Health Survey | 2367.00 | 49.3 | 2011 | – | Green space | Age, sex, education, working status, marital status | Per 1 SD increase in green space exposure (EVI) OR: 0.81, 95% CI: 0.69-0.94. |
| Klompmaker et al., 2019[72] | Netherlands | Dutch Public Health Monitor 2012 | 354 827 | NA | 2012 | – | Green space | Age, sex, marital status, region of origin, education, work, income, smoking, alcohol consumption, PA, BMI, neighborhood SES | Green space (NDVI 300 m buffer) per IQR 0.13 OR: 0.91, 95% CI: 0.89-0.93. |
| Li et al., 2021[94] | China | Henan Rural Cohort Study | 39 019 | 55.58 | 2015-2017 | – | Green space | Age, sex, BMI, income, PA, education, marital status, smoking, drinking, diet, family history of DM | Green space (NDVI 500 m buffer) per IQR OR: 0.87, 95% CI: 0.83-0.90. |
| Makhlouf et al., 2023[119] | US | Population Level Analysis and Community Estimates Data US, American Community Survey Data | 315 221 353 | NI | 2021 | – | Walkability | Age, sex, race, social vulnerability index | Walkability across quartiles Q1 (least walkable) through Q4 (most walkable) multivariable linear regression model: $\beta = -0.243$ (0.002) p-value=<0.001. |

(continued)

**Table 3.** (continued)

| Author, Year | Country | Study | N | Age | Baseline | Follow-up | Exposure | Confounders | Results |
|---|---|---|---|---|---|---|---|---|---|
| Makramet al., 2025[100] | US | Houston Methodist Learning Health System Outpatient Registry | 1 077 181 | 52.0 | 2016-2022 | – | Green space | Age, sex, race/ethnicity, area deprivation index, WalkScore | Green space per NatureScore™ (0-100) Nature Adequate OR: 1.00 (0.98-1.02), nature Rich OR: 0.98 (0.96-1.00), Nature Utopia 0.92 (0.90-0.94). |
| Müller et al., 2018[95] | Germany | Dortmund Health Study | 1312 | 52.6 | 2003-2004 | – | Green space | Age, sex, education, income, living with/without a partner, migration background, unemployment rate | Proportion of green space Ref T1 (5.13-23.16) to T2 (23.37-30.95) OR:1.89, 95% CI: 1.07-3.33. |
| Müller-Riemenschneider et al., 2013[112] | Australia | Western Australian Health and Wellbeing Surveillance System | 5970 | cat | 2003-2006 | – | Walkability | Age, sex education, marital status, household income, diet, PA, sedentary behavior | Walkability per less walkable neighborhood (ref.) versus high walkable neighborhoods OR: 1.08, 95% CI: 0.72-1.62. |
| Niedermayer et al., 2024[38] | Germany | KORA-FIT (Part of the Cooperative Health Research in the Region of Augsburg, KORA) | 3034 | 63.2 | 2018-2019 | – | Green space | Age, sex, alcohol, smoking, physical activity, education | Green space (NDVI 500 m buffer) per IQR: 0.1, OR: 0.90, 95% CI: 0.74-1.09. |
| Ohanyan et al., 2022[36] | Netherlands | AMIGO: Population-based Occupational and Environmental Health Cohort Study | 14 829 | 50.7 | 2011-2012 | – | Green space | Age, sex, duration of living at the current address, participants-, mother's, and father's country of birth, civil state, education, employment, smoking | Green space (NDVI 100 m buffer) B-estimate: 0.0663 SE:0.0429. |
| Plans et al., 2022[104] | Spain | Heart Healthy Hoods cohort study | 1625 | 56 | 2017 | – | Green space | Age, sex, migration status, SES, population density | Green space density (500m buffer) per quantiles (4) ref. Q1 high versus Q4 low OR: 1.44, 95% CI: 0.82-2.52. |
| Sun et al., 2025[58] | China | Prospective cohort study in Tianjin, China | 65 824 | 64.64 | 2014 | until 2021 | Green space | Age, gender, BMI, exercise frequency, smoking status, and alcohol frequency | NDVI (500m buffer) per SD 0.045 HR: 0.90, 95% CI: 0.88-0.92. |
| Sundquist et al., 2015[120] | Sweden | National register and healthcare data, Sweden | 512,061 | 49.0 | 2006 | 2007-2010 | Walkability | Age, sex, household income, education | Walkability per 10 quintiles ref. Q10 (5.27) to Q1 (−3.44) OR: 1.16, 95% CI: 1.00-1.34. |
| Sørensen et al., 2022[53] | Denmark | National Register Data | 1 922 545 | 57.5 | 2005 | 11.2 years | Green space | Age, sex, calendar-year, civil status, individual and family income, country of origin, occupational status, education, neighborhood-level % of population with: low income, only basic education, unemployed, manual labor, non-Western background, criminal record, sole-providers, live in social housing. | NonGreen150m: % of areas within 150 m not classified as agricultural areas, household gardens, recreational areas, forests, open nature areas, HR: 1.05, 95% CI: 1.04-1.05. NonGreen1000m: % of areas with 1000 m buffer that are not publicly accessible green areas, HR: 1.03, 95% CI: 1.02-1.04. |

(continued)

**Table 3.** (continued)

| Author, Year | Country | Study | N | Age | Baseline | Follow-up | Exposure | Confounders | Results |
|---|---|---|---|---|---|---|---|---|---|
| Tsai et al., 2020[107] | Taiwan | National Health Insurance Research Database | 429 504 | 42.0 | 2001 | 11.0 years | Green space | Age, sex, SES, insurance amount, occupational type, comorbidities | Green space per IQR (0.11 465) OR: 0.80, 95% CI: 0.71-0.90. |
| Yang et al., 2019[97] | China | The 33 Communities Chinese Health Study | 15 477 | 45.0 | 2009 | – | Green space | Age, sex, ethnicity, education, family income | Green space (NDVI 500 m buffer) per 0.1-unit increase OR: 0.88, 95% CI: 0.82-0.94. |
| Yang et al., 2023[108] | UK | UK Biobank | 379 238 | 56.4 | 2006-2020 | 12.4 years | Green space | Age, sex, ethnicity, assessment center, deprivation, education, economic status, smoking, alcohol intake, diet, sedentary time, family history of DM | Green space (300m buffer) per 10 unit increase in the percentage of green space HR: 0.98, 95% CI: 0.98-0.99. |
| Yu et al., 2022[109] | China | Yinzhou cohort | 22 535 | 61.47 | 2015-2018 | 3.8 years | Green space | Age, sex, marital status, education, income, BMI, smoking, alcohol consumption, PDI, PA, history of hypertension- and dyslipidemia, $PM_{2.5}$ | Green space (NDVI 250 m buffer) HR: 0.56, 95% CI: 0.51-0.61. |
| Yu et al., 2023[96] | China | Fujian Behavior and Disease Surveillance Cohort | 50 593 | 53.8 | 2018 | – | Green space | Age, sex, marital status, education, occupation, smoking, drinking status, sleep quality, diet, temperature, humidity | Green space (NDVI 500m buffer) per 0.1-unit increase OR: 0.81, 95% CI: 0.79-0.83. |
| Zhang et al., 2024[61] | China | China Health and Retirement Longitudinal Study (CHARLS) | 9242 | 59.0 | 2011-2012 | Follow-ups 2013, 2015, 2018. | Green space | Age, gender, education level, marriage, residence, region, cash at home, smoking status, drinking status, BMI, sleep duration, social activity, health status in youth, hypertension and dyslipidemia | NDVI high-level (≥ 0.2726) compared with low-level group (< 0.2726) HR: 0.80, 95% CI: 0.69-0.93. |

PA: physical activity, DM: diabetes mellitus, SES: socioeconomic status. cat: categories.
[a]Only women in the study population.

Furthermore, living in the highest quartile of cumulative average NDVI within a 250 m buffer was associated with a 57% (HR = 0.43, 95% CI: 0.36, 0.52) reduction in diabetes risk compared with the lowest quartile.[109] Dendup et al. studied prevalent and incident cases of diabetes separately and found an association only in categories ≥30% compared with 0%-4% total green space (OR: 0.70, CI: 0.51-0.96) in Australia.[90]

Albers et al. examined the standardized proportion of greenspace within a 1650 m radius in the Netherlands and found non-significant trends towards decreased risk for both prevalent and incident T2D.[110] Badpa et al. studied the association between NDVI and T2D in Germany. Their result with a wider buffer size (1000 m per IQR 0.14) did not reach significance but was in the expected direction, showing inverse association with the risk of T2D: HR: 0.98, 95% CI: 0.88- 1.09. Using the smaller buffer size (300 m per IQR 0.12), the risk changed in an unexpected direction, but remained statistically non-significant (HR: 1.04, 95% CI: 0.94-1.14).[111] Sørensen et al. explored the association of green space and T2D using variables for a non-green living environment. NonGreen150m, which measured the percentage of areas within 150 meters of the residential address, not classified as agricultural areas, household gardens, recreational areas, forests and open nature areas, was associated with higher risk of T2D (HR: 1.05, 95% CI: 1.04-1.05. NonGreen1000m measured the percentage of areas within 1000 meters of the residential address that are not publicly accessible green areas (ie, not classified as recreational areas, forests and open nature areas) and was also associated with a higher risk of T2D (HR 1.03, 95% CI: 1.02-1.04).[53]

### *Green space and joint exposure*

The joint environmental exposure was considered in nine articles.[36,52,53,58,61,72,98,102,108] The protective association of residential greenness remained in the study conducted by Clark et al. where the strongest association was found in a model further adjusting for traffic noise, $PM_{2.5}$, and walkability (OR: 0.89, 95% CI: 0.86-0.92).[52] For Anza-Ramirez et al. further adjustment for all built environment exposures (sub-city intersection- and population density, city isolation- and fragmentation) did not change the result for green space exposure (NDVI) and the risk of T2D considerably (from OR: 0.97, 95% CI: 0.93-1.01 to OR: 0.98, 95% CI: 0.94-1.02).[102]

The mediating role of air pollution was assessed in three studies.[72,98,108] Hu et al. reported that the estimated associations between green space (NDVI) and diabetes were mediated by $PM_{2.5}$ (5.0%, 95% CI: 0.6%-12%), $NO_2$ (41.0%, 95% CI: 6.4%- 76.0%), and $O_3$ (10.7%, 95% CI: 3.7%-23.0%).[98] Klompmaker et al. reported that the proportion mediated by $NO_2$ depended on the buffer size of green space; NDVI with a 300 m buffer proportion mediated by $NO_2$ was 0.20 (95% CI: 0.08-0.33) and with a larger buffer, 1000 m: 0.34, 95% CI: 0.15-0.53.[72] Yang et al. found evidence of the mediating role of $PM_{2.5}$ in the estimated effect between green space and T2D, with a mediation proportion of 37.0%. When exploring the modification effect, they found no evidence for $PM_{2.5}$, but for $NO_2$ they observed a protective effect of green space in low levels of $NO_2$ (HR: 0.97, 95% CI: 0.96-0.99) but not in higher quartiles (*P*-value for interaction: 0.098). Sun et al.[58] categorised air pollutants ($PM_{2.5}$, BC, OM, ammonium salt, nitrate, sulfate, and chloride) and NDVI into high and low groups based on their medians. Using low pollutant concentration and high NDVI as the reference, all the other combinations showed a higher risk of diabetes, indicating that green space can offer a degree of protective effect when exposed to pollutants and NDVI concurrently.[108]

Two studies utilized a CRI method to assess the joint exposure of environmental variables.[53,72] Sorensen et al. used two green space variables (NonGreen 150 m buffer and NonGreen 1000 m buffer) that were associated with the risk of T2D in single- and two-pollutant models (adjusting for both NonGreen exposures). In a multi-pollutant model including ultrafine particles, $NO_2$, road traffic, NonGreen150m and NonGreen1000m, the risk was slightly attenuated but remained associated with T2D (NonGreen150m HR: 1.04, 95% CI: 1.03-1.04) and NonGreen1000m HR: 1.02, 95% CI: 1.01-1.03). To quantify the cumulative burden of these environmental exposures the CRI method was used, increasing the risk of T2D to HR: 1.12 (95% CI: 1.11-1.13).[53] Similarly, in the study by Klompmaker et al. exposure to green space (NDVI 300 m) was associated with T2D in both single- and two-pollutant models (adjusting for traffic noise). When using the CRI method for the joint exposure of $NO_2$, NDVI, traffic noise, and oxidative potential metric with dithiothreitol assay, the combined exposure was larger than in the single exposure models.[72] Ohanyan et al. utilized a multi-exposure Random Forest analysis (RF) where green space (NDVI 1 km) reached statistical significance, whereas it was not significantly associated with T2D in the single-exposure model for either NDVI 100 m or NDVI 1 km.[36]

### *Walkability*

Altogether 13 studies explored walkability and its association with T2D.[52,69,110,112-121] Of the longitudinal studies, two from Canada found that high walkability was associated with lower T2D incidence among the Canadian adult population.[113,114] Hua et al. studied walkability and risk of T2D in the New York University Women's Health Study and found that women living in the most walkable neighborhood had 25%-33% reduced risk of diabetes.[121] A study conducted in the Netherlands by Albers et al. used a walkability index (0-100 per IQR 52.23-22.87) with both prevalent and incident T2D and reported evidence for an inverse association only with the prevalent cases of T2D.[110] Kartschmit et al.[118] and Clark et al.[52] did not find a significant association with walkability and T2D in German or Canadian adult populations. Four cross-sectional studies reported an inverse association between high walkability and T2D.[74,113,115,119] Sundquist et al.[120] and Müller-Riemenschneider et al.[112] also observed an inverse association between high walkability and T2D in the crude models, but adjusting for individual-level factors diminished the association. Dzhambov et al. didn't find an association between walkability and T2D in their study in five Bulgarian cities (OR: 1.04, 95% CI: 0.87-1.23).[69]

### *Walkability and joint exposure*

Of these 13 studies, three considered joint environmental exposure. For Dzhambov et al., similar to their single-exposure model, there was no association between walkability and risk of T2D when adjusted for environmental co-exposures (OR: 1.12, 95% CI: 0.91-1.38).[69] The protective association of higher walkability and T2D became significant in the study conducted by Clark et al. when further adjusting for traffic noise, $PM_{2.5}$, and greenness (from OR: 1.01, 95% CI: 0.98-1.04 to OR: 0.95, 95% CI: 0.91-0.99).[52] Howell et al. adjusted for $NO_2$, which changed the risk of T2D in the lowest quintile of walkability (versus the highest) from OR: 1.16, 95% CI: 1.13-1.19 to OR: 1.25, 95% CI: 1.22-1.29. Further interaction analysis identified significant interaction effects between walkability and $NO_2$, indicating that at low levels of $NO_2$, the likelihood of diabetes was higher among those living in less walkable neighborhoods. When the levels of $NO_2$ increased, the probability of diabetes rose in highly walkable neighborhoods and became comparable across all levels of walkability.[74]

### *Population density*

One study by Anza-Ramirez et al. studied the role of population density on the risk of T2D. They analysed data from 122 211 individuals from 10 Latin American countries. In a single exposure model (adjusted for age, sex, education, population educational attainment at sub-city level, percentage of urban area, and country as a fixed effect) sub-city population density showed no clear association with T2D, as the OR per 1 SD increase (4.876/km$^2$) was near null (OR: 0.99, 95% CI: 0.94-1.03). When further adjusting for all built environment exposures (sub-city intersection density and greenness, city isolation- and fragmentation) the risk of T2D slightly attenuated to OR: 0.96, 95% CI: 0.92-1.00.[102]

### *Heterogeneity analysis*

To understand the reasons for high heterogeneity, subgroup analyses and univariate meta-regression analyses were used to explore the influence of specific study characteristics on observed effect estimates. The study characteristics considered were study design, geographic region, outcome measurement method, adjustment for relevant factors related to T2D, adjustment for other environmental risk factors, and risk of bias score. These additional analyses were not able to explain the high heterogeneity between studies (Figure S7 and Table S6).

## Discussion

This systematic review and meta-analysis included 151 studies related to the Urban Exposome of T2D. The research knowledge of these studies was synthesized with narrative syntheses per exposure group (air pollution, noise, and built environment) and, when feasible, meta-analysed with possible subgroup analyses to evaluate whether the associations varied by individual study characteristics.

The results of the meta-analyses suggested a positive association between air pollutants $PM_{2.5}$, $PM_{10}$, $NO_2$, $O_3$, BC, and T2D. Our results for $PM_{2.5}$, $PM_{10}$, and $NO_2$ were similar to previous systematic reviews and meta-analyses.[5,7,8] For instance, compared with the results of Yang et al. for air pollution and risk of T2D, our meta-analyses showed stronger associations. For $PM_{2.5}$, they reported the HR of 1.10 (95% CI: 1.04-1.17) and for incident cases OR: 1.08 (95% CI: 1.04-1.12) compared to our result of OR 1.19 (95% CI: 1.16-1.22), which combines both the incident and prevalent cases. For $PM_{10}$, they found HR of 1.11 (95% CI: 1.00-1.22) and OR: 1.10 (95% CI: 1.03-1.17), whereas our result was OR: 1.23 (95% CI: 1.13-1.34). For $NO_2$ the HR was 1.01 (95% CI: 0.99-1.02) and OR: 1.07 (95% CI: 1.04-1.11) compared to ours OR: 1.13 (95% CI: 1.10-1.16). Similar to our results, they found a high between-study heterogeneity for the meta-analyses.[5] The result for a positive association between ozone exposure and risk of T2D was in line with a recent systematic review and meta-analysis by Yu et al. which included fewer studies but had a similar effect size of 1.06 (95% CI: 1.02–1.11, n = 5) to ours OR: 1.05 (95% CI: 1.02-1.08, n = 20).[9] We were not able to identify prior meta-analyses on BC and the risk of T2D. Therefore, our result on BC and risk of T2D (OR: 1.32, CI: 1.15-1.50, n = 8) brings new knowledge to this field of research. Of the air pollutants included in this review, BC showed the strongest association with T2D, while being the least studied air pollutant. $PM_{2.5}$ was the most studied exposure, considered as the main exposure in 90 studies, while showing the highest risk of publication bias. The high number of imputed studies (n = 15) in Trim-and-Fill analysis and a decrease in the effect estimate (from OR: 1.19, 95% CI: 1.16-1.22 to OR: 1.14, 95% CI: 1.10-1.17) suggest that the publication bias might have led to an overestimation of the effect size in the observed studies for PM2.5. Utilizing air pollution scores with methods such as Weighted Quantile Sum (WQS) regression or Quantile g-computation (QGC) method can help to understand the cumulative burden of air pollution, providing more insights into the association with T2D instead of focusing on $PM_{2.5}$ or other air pollutants alone.

Differences in the overall effect sizes of the observed associations per sub-groups were small but can still provide important information to highlight which factors might contribute to a higher risk of T2D. From meta-regression analyses, we found that a study region was a significant covariate for $PM_{2.5}$ in both European and Asian regions, showing a higher risk of T2D compared to North America. Also, $NO_2$ studies conducted in the Asian region showed a higher risk of T2D compared to studies conducted in North America. However, the study regions included in this review did not cover any African countries, and only two studies were from South America and four from Australia. Furthermore, only one of the studies assessing noise exposure was conducted in Asia. Between 2021 and 2045, the most significant relative increase in the prevalence of diabetes is expected to occur in middle-income countries (21.1%) compared to high- (12.2%) and low-income countries (11.9%).[4] Together, these findings call for more research focused on low- and middle-income countries.

Results from the road traffic- and railway noise meta-analyses were similar to the systematic review and meta-analysis from 2018 by Sakhvidi et al. where traffic noise was positively associated with T2D, and no evidence for association was observed for railway noise exposure.[12] More research is needed to assess the role of noise exposure, especially the role of aircraft noise, which had conflicting results in narrative synthesis. The qualitative synthesis of 38 studies on built environment exposures (green space, walkability, and population density) indicated that the living environment with higher walkability and greenness has an inverse association with T2D. We identified only one study that directly examined the relationship between population density and the risk of T2D. However, population density is frequently included in walkability indexes and was studied in that context in four of the included studies.[74,113,116,117] Our narrative review was in line with previous reviews; Sharifi et al. reported from a meta-analysis that more access to green space was associated with lower odds of diabetes OR: 0.79 (95% CI: 0.67-0.90).[13] Similarly, De la Fuente et al. reviewed studies on green space exposure between 2009 and 2020 and found evidence supporting the protective role of green spaces in the urban context against T2D and other chronic health conditions, such as obesity and sedentary behaviors.[15]

In this review, 55 studies assessed the joint exposure of environmental exposures (air pollution, noise, or built environment) on the risk of T2D. The noise exposure studies were more consistent in considering other environmental co-exposures than the built environment or air pollution studies, the latter often considering only the other air pollutants. The joint exposures were commonly considered as confounders, but in recent years, the use of more complex statistical methods has increased. Three studies[51,53,72] utilized the Cumulative Risk Index (CRI) method to understand the cumulative burden of environmental exposures on the risk of T2D, and five studies[57-60,81] used Quantile g-computation (QGC) method to assess the joint effect of mixtures of air pollutants. One study utilized the penalised regression Least Absolute Shrinkage and Selection Operator (LASSO), Random Forest (RF), and Artificial Neural Networks (ANN) approaches to study the risk of T2D comprehensively.[36] As a whole, the difference to single exposure models was modest but signaled slightly smaller effect sizes in joint exposure models. This could potentially lead to overestimation in effect sizes in single-pollutant models, especially when considering the co-exposures only as confounding factors.

Only one of the included studies utilized the exposome approach, indicating that it is not yet widely understood in environmental epidemiology to assess the risk of T2D.[36] The exposome approach was developed to address more accurate and comprehensive environmental exposure data, including the selection, harmonization, description, and analysis of a large set of exposures, making it complex in many respects. This might explain its still scarce use in assessing the risks of T2D. Longitudinal studies that combine data from different sources (biological samples, physical examinations, questionnaires, national registers, and geospatial models) can provide adequate resources for successful exposome studies. In order to analyse various exposures simultaneously, statistical methods that consider the potential correlation or interaction between exposures are needed, such as the CRI and QGS methods. Within the exposome framework, advanced methods have been developed to study the individual and joint effects of multiple environmental exposures and have been reviewed extensively elsewhere.[122-126]

### Strengths and limitations

This is the largest systematic review and meta-analysis to date to assess the relationship between T2D and several different exposures of the Urban Exposome while simultaneously mapping the use of the exposome approach. This work provides a comprehensive synthesis of evidence, and by pooling the results into various meta-analyses, we were able to provide precise effect estimates for various environmental exposures related to the risk of T2D. By combining different study designs, settings, and populations, we were able to have greater generalizability of findings.

Our review also has some limitations. We were able to include studies only in English, and therefore, possible studies that would have met the inclusion criteria from other languages are missing. Not all studies distinguished the type of diabetes, especially in many register-based studies there was no distinction between type 1 and type 2 diabetes. However, approximately 90% of diabetes diagnoses among adult populations are T2D, and therefore this is unlikely to affect the results substantially. In future studies, it is highly important to distinguish the types of diabetes to understand the specific risk factors of each type. We did not use a validated tool for ROB assessment, which could be a possible limitation for our study. We decided to use the self-developed tool due to the variety of study designs and settings included in this review. The developed tool gives an overview of the possible sources of bias, and we did not exclude any studies based on it, but utilized a ROB score to understand the differences between the studies and ROB domains.

In our review, we have utilized a broad scope of observational studies to gain an extensive understanding of the role of environmental exposures on the risk of T2D. This resulted in many study designs with different exposure measures, which can create a potential risk of bias. Publication bias affected the investigated associations, and considerable heterogeneity was present in most of the meta-analytic estimates, partly preventing us from drawing very firm conclusions. However, we did various sensitivity analyses to strengthen the generalizability of our findings and to assess whether individual study characteristics affected the overall estimates. While we recognize these challenges that prevent us from making conclusions that the results would be causal, this review can still provide a better understanding of the current state of research and the possible role of environmental exposures in the risk of T2D. Future studies should utilize the more comprehensive approaches to understand both the harmful and beneficial exposures in the urban exposome and not solely focus on specific exposures such as $PM_{2.5}$. Translating research information to policymakers allows them to design policies on those conditions that can be modified. When successful, healthcare workers can implement new health interventions or programs to decrease the risks of adverse living environment.

## Conclusion

We conclude that exposure to air pollution ($PM_{2.5}$, $PM_{10}$, $NO_2$, $O_3$, and BC) and road traffic noise are associated with an increased risk of T2D. Furthermore, a greener and more walkable living environment can potentially reduce the risk of T2D. The knowledge of their joint effect and the mechanism of action in the population remains unclear. Future studies should consider joint exposures, as well as the standardization of the exposure and outcome assessment methods. The exposome approach was used only in one research article in the reviewed research. Advancing the use of the exposome approach and terminology can help in understanding the T2D risk comprehensively by enabling a holistic assessment of cumulative environmental exposures.

## Author contributions

Miia Halonen(Conceptualization [Equal], Data curation [Equal], Formal analysis [Lead], Methodology [Lead], Project administration [Lead], Validation [Equal], Visualization [Lead], Writing—original draft [Lead], Writing—review & editing [Lead]), Wnurinham Silva(Conceptualization [Equal], Data curation [Equal], Investigation [Equal], Writing—review & editing [Equal]), Susanna Pätsi (Methodology [Supporting], Validation [Equal], Writing—review & editing [Equal]), Jouko Miettunen(Methodology [Supporting], Validation [Equal], Writing—review & editing [Equal]), and Sylvain Sebert(Conceptualization [Equal], Funding acquisition [Lead], Supervision [Equal], Validation [Equal], Writing—review & editing [Equal]), Justiina Ronkainen(Conceptualization [Equal], Methodology [Supporting], Supervision [Equal], Validation [Equal], Writing—review & editing [Equal])

## Supplementary material

Supplementary material is available at *Exposome* online.

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## Conflicts of 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.

## Data availability

Data will be made available on reasonable request.

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