Journal of Epidemiology and Public Health (2025), 10(03): 399-408
Masters Program in Public Health, Universitas Sebelas Maret

Meta-Analysis

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The Impact of Long-Term Particulate Matter with Diameter Micrometers ≤2.5 Exposure on Type 2 Diabetes Risk: A Meta-Analysis

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Received: Juny, 04 2025; Accepted: July 10, 2025; Available online: July 16, 2025

ABSTRACT

Background: The rising global prevalence of type 2 diabetes (T2D) has prompted investigations into environmental risk factors beyond traditional lifestyle causes. Air pollution, particularly fine particulate matter ($PM_{2.5}$) has emerged as a potential contributor to diabetes. This systematic review synthesizes evidence from large scale cohort studies to evaluate the association between long term air pollution exposure and T2D risk.
Subjects and Method: We analyzed prospective cohort studies (2020–2025) assessing air pollution and incident T2D, sourced from PubMed, Web of Science and EMBASE using the search terms: ("air pollution" OR "PM2.5") AND ("type 2 diabetes" OR "T2D") AND ("cohort" OR "longitudinal"). We using PECO framework (Populatio= adults population; Exposure=High Exposure of PM2.5; Comparison=Low Exposure of PM2.5; Outcomes=T2D). The independent variable was Exposure of PM2.5 and dependent variable was T2D. Primary studies included were cohort manuscript published in english with year of publication between 2020 until 2025, reported adjusted hazard ratios (aHR), and the subjects was adults without diabetes at baseline. Studies were evaluated using the CASP Scale, and data were analyzed using RevMan 13.
Results: This study includes 9 papers, using Cohort study designs, from China, United Kingdom, South Korea and United States. $PM_{2.5}$ exposure consistently increased T2D risk with stronger effects at lower exposure levels (aHR = 1.24; 95% Cl = 1.07 to 1.42; p=0.004).
Conclusion: Long-term $PM_{2.5}$ exposure significantly increases T2D risk and progression.

Keywords: Air Pollution, Type 2 Diabetes, Diabetes Mellitus, $PM_{2.5}$

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Cite this as:
Anggraeni T, Sarwoko, Herbasuki, Sunaryanti SSH (2025). The Impact of Long-Term Particulate Matter with Diameter Micrometers ≤2.5 Exposure on Type 2 Diabetes Risk: A Meta-Analysis. J Epidemiol Public Health. 10(3): 399-408. https://doi.org/10.26911/jepublichealth.2025.10.03.10.

BACKGROUND

Type 2 diabetes (T2D) has emerged as a global public health crisis, with prevalence rates escalating dramatically over the past few decades. According to the International Diabetes Federation (IDF), approximately 537 million adults were living with diabetes in 2021, a number projected to rise to 783 million by 2045 (International Diabetes Federation, 2021). T2D accounts for over 90% of all diabetes cases, driven largely by urbanization, sedentary lifestyles, and poor

dietary habits. The disease imposes a substantial economic burden, with global healthcare expenditures related to diabetes estimated at $966 billion in 2021, representing 9% of total health spending worldwide (International Diabetes Federation, 2021).

The World Health Organization (WHO) reports that diabetes was the ninth leading cause of death in 2019, with an estimated 1.5 million deaths directly attributed to the disease (World Health Organization, 2023). Furthermore, individuals with T2D experience reduced quality of life and productivity, exacerbating social and healthcare disparities, particularly in low and middle income countries where access to treatment remains limited (L et al., 2024).

Emerging evidence suggests that environmental factors, particularly long-term exposure to air pollution, may play a critical role in the development of T2D. Air pollutants such as particulate matter ($PM_{2.5}$ and PM_{10}), nitrogen dioxide (NO_2), and ozone (O_3) have been implicated in disrupting insulin signaling, promoting systemic inflammation, and inducing oxidative stress key pathways in diabetes pathogenesis (Eze et al., 2015).

A large scale cohort study by Brook et al. (2013) found that long-term $PM_{2.5}$ exposure was associated with a higher incidence of T2D, even after adjusting for lifestyle factors. Similarly, a meta analysis by He et al. (2017) reported that a 10 $\mu g/m^3$ increase in $PM_{2.5}$ was linked to a 25% increased risk of T2D. Additionally, NO_2 exposure has been shown to impair glucose metabolism, further supporting the role of air pollution in diabetes etiology (Balti et al., 2014).

Given the rising dual burden of urbanization and diabetes, there is an urgent need for a specific systematic review and meta analysis to synthesize current evidence, identify research gaps, and inform public health policies aimed at mitigating air pollution related diabetes risk. Such a review could guide regulatory actions, urban planning, and targeted interventions for vulnerable populations. Therefore, the aim of this study was to evaluate the association between long-term air pollution particularly $PM_{2.5}$ exposure and T2D risk.

SUBJECTS AND METHOD

1. Study Design

This meta analysis examines the association between air pollution exposure and type 2 diabetes risk through analysis of cohort studies published from 2020 to 2025. This research uses the PRISMA diagram. The article search was carried out based on the PECO eligibility criteria, namely Population = adults population; Exposure = High Exposure of PM2.5; Comparison = Low Exposure of PM2.5; Outcomes = T2D. We identified relevant studies through comprehensive searches of PubMed, Web of Science, and EMBASE using the search terms: ("air pollution" OR "PM2.5") AND ("type 2 diabetes" OR "T2D") AND ("cohort" OR "longitudinal"). The review focuses specifically on large scale population studies with robust exposure assessment methodologies.

2. Step of Meta Analysis

1) Create Research Questions using the PECO format which involves defining Population, Exposure, Comparison, and Outcome.
2) Search electronic databases (e.g., PubMed, Science Direct, Scopus) and non electronic sources for primary study articles.
3) Conduct a screening process to establish criteria of inclusion and exclusion criteria, followed by critical assesment.
4) Gather data from primary studies and compile effect estimates using RevMan application.

5) Analyze the findings and formulate the conclusions based on interpreted findings.

3. Inclusion Criteria

Studies were included if they employed prospective cohort design, published in year between 2020 until 2025, measured long-term exposure to specific air pollutants (PM2.5), reported adjusted hazard ratios for type 2 diabetes incidence, included adult populations without baseline diabetes, provided clear exposure assessment methodology, and were published in peer reviewed journals in English.

4. Exclusion Criteria

We excluded studies that used cross-sectional or case control designs, lacked individual level exposure assessment, focused only on occupational exposures, did not adjust for key confounders (such as age, sex, BMI, smoking), reported only unadjusted effect estimates, were conference abstracts or non peer reviewed publications, and had follow up periods shorter than 3 years.

5. Operational Definition of Variables

Type 2 Diabetes: Type 2 diabetes is a chronic metabolic disorder characterized by insulin resistance (reduced cellular response to insulin) and impaired insulin secretion by pancreatic β-cells.

Exposure to air pollution: exposure to air pollution refers to the inhalation or contact with harmful airborne pollutants, including: Particulate matter with Diameter Micrometers ≤2.5 ($PM_{2.5}$).

6. Study Instruments

The article search process adheres to the PRISMA flow diagram for systematic reviews. Furthermore, the quality of the research was evaluated utilizing the Cohort Study Checklist released by the Critical Appraisal Skills Program (CASP) in 2018.

7. Data Analysis

The data analysis involves rigorous data collection, synthesis, and analysis to draw evidence based conclusions. The research in this study followed the PRISMA flowchart to collect articles and used software Review Manager version 5.3 to analyzed effect size of findings.

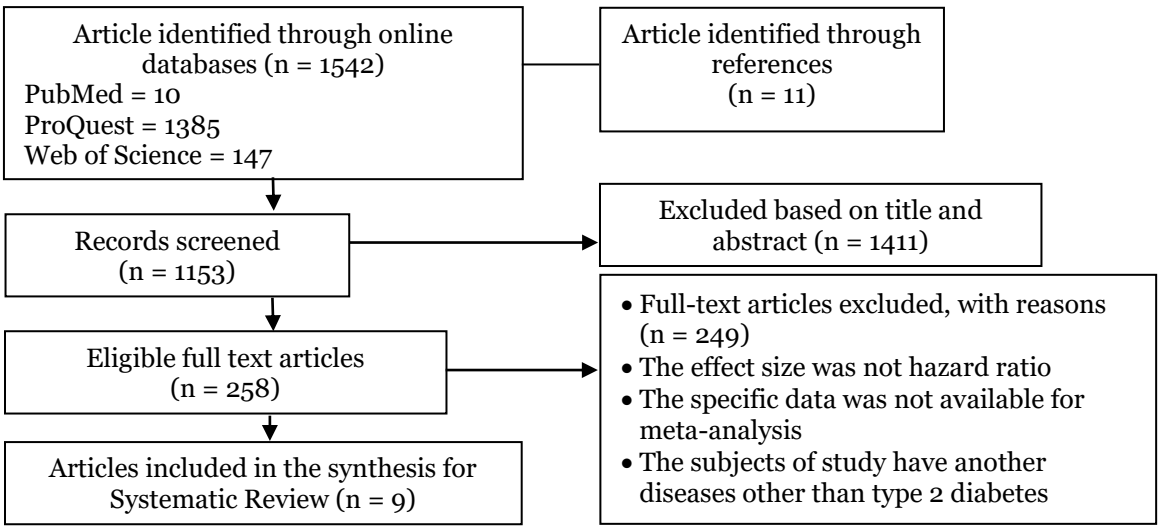

Figure 1. PRISMA Flowchart

RESULTS

Figure 1 illustrates the results of the article search, which showed 1,542 articles that were first found in the databases that were used. There were 1,153 articles left after 1,411 articles was eliminated based on title and abstract. Nine publications out of 258 articles that satisfied quantitative requirements were subjected to a meta analysis after 249 articles were excluded with reasons (Figure 1).

The primary studies which were predominantly large scale cohort studies conducted in the China (1 study), United Kingdom (6 studies), South Korea (1 study), and the United States (1 study) (Figure 2), with sample sizes ranging from 14,667 to 264,859,458 participants. Most studies utilized data from the UK Biobank, assessing long-term exposure to air pollutants of $PM_{2.5}$ and their association with incident T2D (Table 2).

Figure 2. Map of the distribution of articles included in the mata-analysis

Table 1. Critical Appraisal Checklist using CASP of the Impact of Long-Term Particulate Matter with Diameter Micrometers ≤2.5 Exposure on Type 2 Diabetes Risk

Primary Study	Criteria												
Author (Year)	1	2	3	4	5	6	7	8	9	10	11	12	Total
Fan et al., (2024)	2	2	2	2	2	2	2	2	2	2	2	2	24
Fan et al. (2025)	2	2	2	2	2	2	2	2	2	2	2	2	24
Hu et al. (2023)	2	2	2	2	2	2	2	2	2	2	2	2	24
Hu et al. (2025)	2	2	2	2	2	2	2	2	2	2	2	2	24
Li et al. (2022)	2	2	2	2	2	2	2	2	2	2	2	2	24
Li et al. (2022)	2	2	2	2	2	2	2	2	2	2	2	2	24
Shin & Kim (2023)	2	2	2	2	2	2	2	2	2	2	2	2	24
Wu et al. (2022)	2	2	2	2	2	2	2	2	2	2	2	2	24
Yitshak et al., (2023)	2	2	2	2	2	2	2	2	2	2	2	2	24

Description of the question criteria:

1 = Does the cohort study address the clinical problem clearly?

2 = Were the cohorts (study subjects in both exposed and non-exposed groups) selected in

the right way?
3 = Are social isolation and loneliness accurately measured to minimize bias?
4 = Were the outcomes (T2D) accurately measured to minimize bias?
5 = Did the researcher identify all important confounding factors? Does the researcher account for confounding factors in the design and/or analysis?
6 = Does the research subject complete the research time in full? Were the research subjects followed up for a sufficiently long time?
7 = Are the results of this study reported in the aHR?
8 = Are the results precise?
9 = Can the results be trusted?
10 = Are the results applicable to the local (local) population?
11 = Are the results of this study compatible with the available evidence?
12 = Does the implications of this research suitable for practice?

Answer score description:

0 = No
1 = Can't tell
2 = Yes

Table 2. Primary Studies Included of Meta-analysis of the Impact of Long-Term Particulate Matter with Diameter Micrometers ≤2.5 Exposure on Type 2 Diabetes Risk

Author (year)	Study Design	Country	Sample Size	P	E	C	O
Fan et al. (2024)	Cohort	United Kingdom	78,230	Participants aged 40-70 years without baseline diabetes	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk
Fan et al. (2025)	Cohort	United Kingdom	77,278	Adults from the UK Biobank cohort were enrolled	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk
Hu et al. (2023)	Cohort	United Kingdom	390,834	Participants in UK Biobank	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk
Hu et al. (2025)	Cohort	China	1,300,000	Using 7–year cohort data of Chinese over 40	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk
Li et al. (2022)	Cohort	United Kingdom	359,153	Participants without diabetes at baseline were included	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk
Li et al. (2022)	Cohort	United Kingdom	156,490	Participants free of diabetes mellitus	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk

Author (year)	Study Design	Country	Sample Size	P	E	C	O
Shin and Kim (2023)	Cohort	South Korea	14,667	Adults aged 40-69 years	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk
Wu et al. (2022)	Cohort	United Kingdom	398,993	Participants free of diabetes and diabetes-related events at recruitment	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk
Yitshak et al. (2023)	Cohort	United States	264,859, 458	Participants aged 65 years and olderin	High Exposure of PM2.5	Low Exposure of PM2.5	PM2.5 increased the T2D risk

Table 3. Adjusted Hazard Ratio (aHR) effect of the Impact of Long-Term Particulate Matter with Diameter Micrometers ≤2.5 Exposure on Type 2 Diabetes Risk

Author (year)	aHR	CI 95%	
		Lower limit	Upper Limit
Fan et al. (2024)	1.09	1.09	1.02
Fan et al. (2025)	1.09	1.03	1.16
Hu et al. (2023)	1.05	1.01	1.1
Hu et al. (2025)	1.13	1.03	1.14
Li et al. (2022)	1.19	1.14	1.24
Li et al. (2022)	1.69	1.42	2.02
Shin and Kim (2023)	1.49	1.11	2
Wu et al. (2022)	1.63	1.59	1.67
Yitshak et al. (2023)	1.074	1.08	1.089

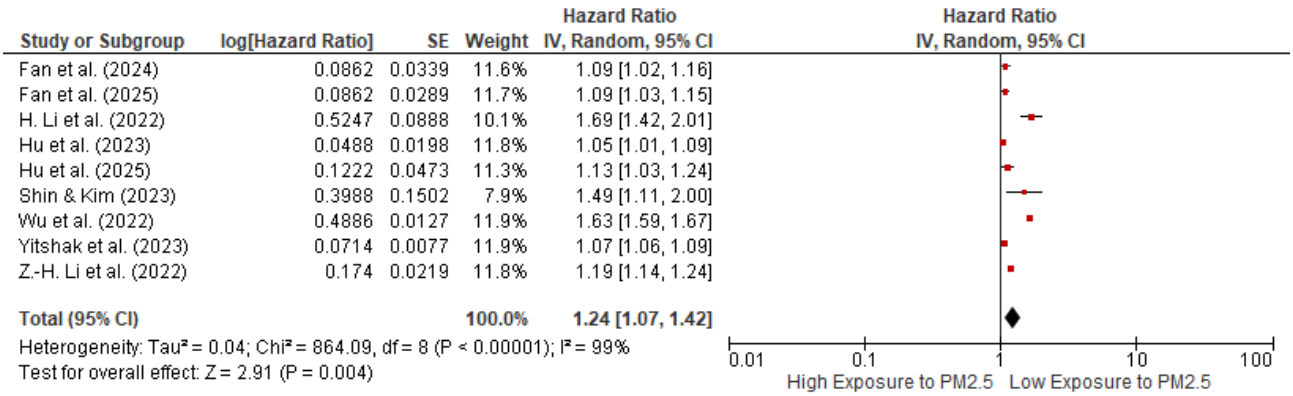

Figure 3. Forest Plot of the Impact of Long-Term Particulate Matter with Diameter Micrometers ≤2.5 Exposure on Type 2 Diabetes Risk

The results of this study showed that high exposure of $PM_{2.5}$ had a 1.20 times risk of developing T2D compared to people who had low exposure of $PM_{2.5}$ (aHR= 1.24; 95% Cl= 1.07 to 1.42; p= 0.004). High heterogeneity is observed among the studies (I^2 = 98%, p <0.001), so the Random Effect Model (REM) is used (Figure 3).

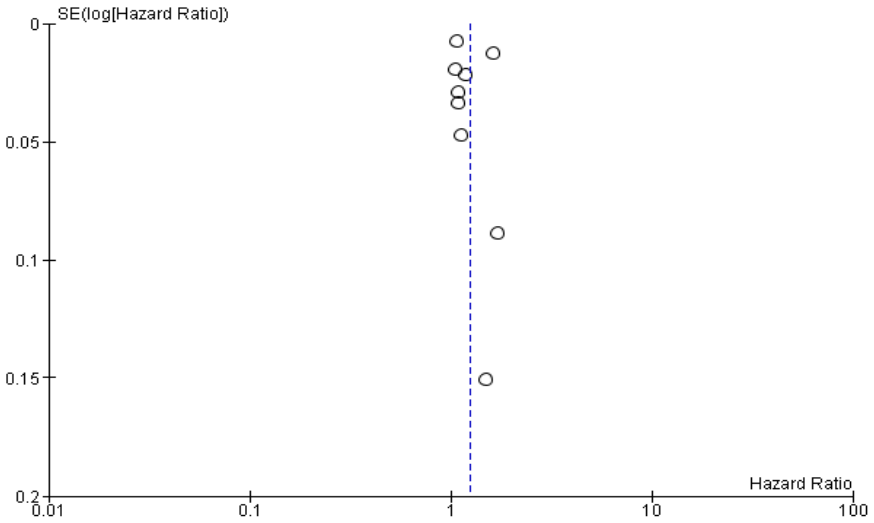

Figure 4. Funnel Plot of the Impact of Long-Term Particulate Matter with Diameter Micrometers ≤2.5 Exposure on Type 2 Diabetes Ris

Funnel Plot of the influence of high exposure of PM2.5 on Incidence of T2D, the plots on the right and left sides are asymmetrical to each other and do not form an inverted funnel. The left plot has a standard error between >0 to <0.05 and the right plot has a standard error between >0 to <0.15. This identifies that in this study there is a real bias (underestimate).

DISCUSSON

There is already meta analysis conducted on air pollution and type 2 diabetes in 2019 (Liu et al., 2019). However this study was conducted with article from before 2020. This current review offer the more update evidence with the addition of discussion about the modification factors. When we breathe in polluted air especially fine particles like PM2.5 these pollutants don't just stay in our lungs. They enter the bloodstream, where they activate immune cells and trigger a chronic low grade inflammatory response. This inflammation is problematic because it disrupts insulin signaling. Normally, insulin helps cells take up glucose, but when inflammatory molecules like TNF-α and IL-6 are constantly circulating, cells become resistant to insulin (Rajagopalan and Brook, 2012).

Air pollution is packed with reactive oxygen species unstable molecules that damage cells. PM2.5 exposure increases oxidative stress, particularly in liver and fat tissue which leads worsens insulin resistance and pancreatic β-cells which leads to impairs insulin production. Over time, this oxidative damage weakens mitochondria (the cell's energy factories), making it harder for the body to regulate blood sugar properly (Mukai et al., 2022).

NO_2 and NO_x (common in traffic pollution) don't just irritate the lungs they also damage blood vessels. This endothelial dysfunction reduces blood flow to insulin sensitive tissues like muscles, further contributing to glucose intolerance (Cersosimo and De Fronzo, 2006). When we combine these effectschronic inflammation, oxidative stress, mitochondrial damage, and poor blood flow the body's ability to manage glucose deteriorates. Over years of exposure this leads higher insulin resistance, declining β-cell function, and increased risk of full-blown type 2 diabetes (Alderete et al., 2017).

While the current body of research provides compelling evidence linking air pollution to type 2 diabetes, several important limitations should be noted when interpreting these findings. First, we must acknowledge that the vast majority of these studies come from high income countries particularly the United Kingdom's extensive UK Biobank research. This heavy reliance on data from wealthy nations leaves critical gaps in our understanding of how these relationships might play out in low and middle income countries, where pollution exposures are often higher and healthcare systems less robust.

The single Chinese study in our review begins to address this gap, but more diverse geographical representation is clearly needed. Second, despite researchers' best efforts to control for confounding variables, the potential for residual confounding remains a persistent concern. While studies adjusted for obvious factors like age, smoking status, and physical activity, there may be other unmeasured lifestyle or environmental variables influencing these relationships. For instance, neighborhood characteristics or access to green spaces factors known to affect both pollution exposure and diabetes risk were not consistently accounted for across studies. Third, we've established clear epidemiological associations, we're still lacking sufficient mechanistic studies to explain exactly how air pollution triggers or exacerbates diabetes at the biological level. The current evidence points to plausible pathways like systemic inflammation and oxidative stress, but more research is needed to confirm these mechanisms and identify potential points for intervention. Finally, and perhaps most importantly for public health applications, we have limited data on optimal intervention strategies. While some studies suggest dietary modifications like increased antioxidant intake may help, we lack clinical trial evidence to confirm these observational findings. Similarly, we need more research on whether improving air quality through policy changes or personal interventions (like air purifiers) can effectively reduce diabetes risk in exposed populations. These limitations don't invalidate the strong associations we've observed, but they do highlight important directions for future research particularly the need for more diverse population studies, deeper mechanistic investigations, and intervention trials to translate these findings into actionable public health strategies.

This comprehensive body of research provides strong, consistent evidence that long term exposure to air pollution particularly PM2.5 and NO2 significantly increases the risk of developing type 2 diabetes and its complications. The findings underscore the importance of considering environmental factors in diabetes prevention strategies and highlight the need for stricter air quality regulations to protect public health. Future research should focus on identifying the most effective interventions to mitigate these risks, particularly in high exposure populations.

AUTHOR CONTRIBUTION

Titik Angraeni is the main researcher who has a topic, seeks, collects, review and analyze research data.

FINANCIAL SUPPORT AND SPONSORSHIP

Nil.

ACKNOWLEDGEMENT

The researchers would like to thank all those who have helped in the preparation of this article and also to the database providers including PubMed, Web of Science, and EMBASE.

CONFLICT OF INTEREST

The authors declare that the study was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

REFERENCE

Alderete TL, Habre R, Toledo Corral CM, Berhane K, Chen Z, Lurmann FW, Weigensberg MJ, et al (2017). Longitudinal Associations Between Ambient Air Pollution With Insulin Sensitivity, β-Cell Function, and Adiposity in Los Angeles Latino Children. Diabetes. 66(7): 1789–1796. doi: 10.2337/db16-1416.

Ananthesh L, Cutinha RM, Sahoo SS, Dsouza JD, Shetty S, Gururaj C, Kellarai A (2024). Effect of healthcare expenditure on the health related quality of life among diabetic patients of South India: A cross sectional study. Clin Epidemiol Glob Health. 25: 101460. doi: 10.1016/j.cegh.2023.1-01460.

Balti EV, Echouffo Tcheugui JB, Yako YY, Kengne AP (2014). Air pollution and risk of type 2 diabetes mellitus: A systematic review and meta analysis. Diabetes Res Clin Pract. 106(2): 161–172. doi:10.1016/j.diabres.2014.08.

Brook RD, Cakmak S, Turner MC, Brook JR, Crouse DL, Peters PA, van Donkelaar A, et al (2013). Long Term Fine Particulate Matter Exposure and Mortality From Diabetes in Canada. Diabetes Care. 36(10): 3313–3320. doi:10.2337-/dc12-2189.

Cersosimo E, DeFronzo RA (2006). Insulin resistance and endothelial dysfunction: the road map to cardiovascular diseases. Diabetes Metab Res Rev. 22(6): 423–436. doi: 10.1002/dmrr.6-34.

Eze IC, Hemkens LG, Bucher HC, Hoffmann B, Schindler C, Künzli N, Schikowski T, Probst Hensch NM (2015). Association between Ambient Air Pollution and Diabetes Mellitus in Europe and North America: Systematic Review and Meta Analysis. Environ Health Perspect. 123(5): 381–389. doi: 10.12-89/ehp.1307823.

Fan C, Wang W, Xiong W, Li Z, Ling L (2025). Beverage consumption modifies the risk of type 2 diabetes associated with ambient air pollution exposure. Ecotoxicol Environ Saf. 290: 11-7739. doi: 10.1016/j.ecoenv.2025.1177-39.

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

Hu M, Hao X, Zhang Y, Sun X, Zhang M, Zhao J, Wang Q (2025). Long term exposure to particulate air pollution associated with the progression of type 2 diabetes mellitus in China: effect size and urban rural disparities. BMC Public Health. 25(1): 1565. doi: 10.11-86/s12889-025-22394-z.

Hu X, Yang T, Xu Z, Jin J, Wang J, Rao S, Li G, et al (2023). Mediation of metabolic syndrome in the association between long-term co exposure to road traffic noise, air pollution and incident type 2 diabetes. Ecotoxicol Environ Saf. 258: 114992. doi: 10.1016/j.ecoenv.2023.1-14992.

International Diabetes Federation. (2021). IDF Diabetes Atlas, 10th Edition. Available from: https://www.diabetes-atlas.org.

Li H, Cai M, Li H, Qian ZM, Stamatakis K, McMillin SE, Zhang Z, et al (2022). Is dietary intake of antioxidant vitamins

associated with reduced adverse effects of air pollution on diabetes? Findings from a large cohort study. Ecotoxicol Environ Saf. 246: 114182. doi: 10.1016/j.ecoenv.2022.114182.

Li ZH, Zhong WF, Zhang XR, Chung VC, Song WQ, Chen Q, Wang XM, et al. (2022). Association of physical activity and air pollution exposure with the risk of type 2 diabetes: a large population based prospective cohort study. Environ Health. 21(1): 106. doi: 10.11-86/s12940-022-00922-3.

Liu F, Chen G, Huo W, Wang C, Liu S, Li N, Mao S, et al (2019). Associations between long term exposure to ambient air pollution and risk of type 2 diabetes mellitus: A systematic review and meta analysis. Environ Pollut. 252: 1235–1245. doi: 10.1016/j.envpol-.2019.06.033.

Mukai E, Fujimoto S, Inagaki N. (2022). Role of Reactive Oxygen Species in Glucose Metabolism Disorder in Diabetic Pancreatic β-Cells. Biomolecules. 12(9): 1228. doi: 10.3390-/biom12091228.

Rajagopalan S, Brook RD. (2012). Air Pollution and Type 2 Diabetes. Diabetes. 61(12): 3037–3045. doi: 10.2337/db12-0190.

Shin MK, Kim KN (2023). Association between long term air pollution exposure and development of diabetes among community dwelling adults: Modification of the associations by dietary nutrients. Environ Int. 174: 107-908. doi: 10.1016/j.envint.2023.-107908.

World Health Organization (2023). Diabetes Fact Sheet. Available from: https://-www.who.int/news-room/fact sheets/-detail/diabetes.

Wu Y, Zhang S, Qian SE, Cai M, Li H, Wang C, Zou H, et al (2022). Ambient air pollution associated with incidence and dynamic progression of type 2 diabetes: a trajectory analysis of a population based cohort. BMC Med. 20(1): 375. doi: 10.1186/s12916-022-02573-0.