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Air pollution, traffic noise, greenness, and temperature and the risk of incident type 2 diabetes

Results from the KORA cohort study

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Introduction: Type 2 diabetes (T2D) is a major public health concern, and various environmental factors have been associated with the development of this disease. This study aimed to investigate the longitudinal effects of multiple environmental exposures on the risk of incident T2D in a German population-based cohort.

Methods: We used data from the KORA cohort study (Augsburg, Germany) and assessed exposure to air pollutants, traffic noise, greenness, and temperature at the participants' residencies. Cox proportional hazard models were used to analyze the associations with incident T2D, adjusting for potential confounders.

Results: Of 7736 participants included in the analyses, 10.5% developed T2D during follow-up (mean: 15.0 years). We found weak or no association between environmental factors and the risk of T2D, with sex and education level significantly modifying the effects of air pollutants.

Conclusion: Our study contributes to the growing body of literature investigating the impact of environmental factors on T2D risks and suggests that the impact of environmental factors may be small.

Keywords: Air pollution; Greenness; Traffic noise; Temperature; Type 2 diabetes; Environmental epidemiology; Population-based cohort

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The data are subject to national data protection laws. Therefore, data cannot be made freely available in a public repository. However, data can be requested through an individual project agreement with KORA. To obtain permission to use KORA data under the terms of a project agreement, please use the digital tool KORA.PASST (https://www.helmholtz-munich.de/en/epi/cohort/kora).

M.B. did the data analyses and wrote the manuscript. M.B., A.S., L.S., B.T., K.W., and A.P., have verified the underlying data and reviewed the manuscript. A.S., K.W., and A.P. were involved in the study design, results interpretation, and review of the manuscript. A.P. provided oversight on the KORA study design. K.W. was involved in the acquisition of the KORA data, the modeling of air pollution concentrations as well as the assignment of all exposure data. All authors read and approved the final manuscript.

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Introduction

Type 2 diabetes (T2D) is projected to become the 7th highest cause of mortality by 2030.[1,2] Alongside well-known risk factors such as obesity, physical inactivity, and genetic predisposition, several environmental factors have been linked to T2D risk in previous studies.[3,4]

Four reviews[5–8] compiled data from epidemiological studies and suggest a positive association between long-term exposure to particulate matter (PM) and nitrogen dioxide (NO_2), and prevalence and incidence of T2D. Similarly, meta-analyses suggested a positive correlation between exposure to traffic noise, particularly during nighttime, and a higher risk of T2D incidence.[9,10] Moreover, access to green spaces, as measured by either the proportion of greenness or the proximity to green areas, has shown beneficial effects on reducing the likelihood of developing T2D.[11] The association of temperature with T2D risk and fasting plasma glucose levels is, however, complex, with some studies suggesting increased risk with higher temperatures,[12] while others indicated U-shaped relationships involving both high and low temperatures.[13]

Despite these findings, studies assessing multiple environmental exposures are still scarce, and there is a lack of longitudinal investigations examining the link with T2D incidence. In this article, we

What the study adds

This study provides critical insights into the link between environmental exposures and T2D. It emphasizes the significance of exposure to $PM_{2.5}$, highlights the need for cautious interpretation due to uncertainties, and underscores the sex-specific variations in pollutant effects on T2D risk. These findings contribute to our understanding of T2D risk factors.

aimed to investigate the longitudinal effects of various exposures on T2D incidence in a German population-based cohort.

Methods

Study population

We used data from the third (KORA S3, 1994–1995, n = 4856) and fourth (KORA S4, 1999–2001, n = 4260) survey of the KORA (Cooperative Health Research in the Region of Augsburg) study[14] and follow-up information until 2016. For our analysis, we excluded participants who requested data withdrawal (N = 80), with a diabetes diagnosis at baseline (N = 381), lacked follow-up data (N = 521), with unknown residential address (N = 270), or had missing values in the main model's covariates (N = 128) (Figure S1, http://links.lww.com/EE/A267).

Incident T2D assessment

T2D incidence was assessed through self-reported, clinically diagnosed diabetes assessed through follow-up questionnaires that were validated by physicians, medical chart review, or self-reported use of glucose-lowering medication.[15,16] Self-reported dates of diagnosis, were verified through medical records or physician contact. (Text S1, http://links.lww.com/EE/A267).

Exposure assessment

Annual mean concentrations of air pollutants, including nitrogen oxides (NO_2, NO_x), ozone (O_3), PM in different size classes (PM_{10} [≤10 µm], $PM_{2.5}$ [≤2.5 µm], $PM_{2.5absorbance}$ [$PM_{2.5abs}$] as an indicator for soot, PM_{coarse} [2.5–10 µm]), and particle number concentration (PNC) as an indicator for ultrafine particles were estimated using land-use regression models for 2014–2015.[17] Traffic noise exposure was estimated for 2011 using the noise and air pollution information system for Augsburg city and georeferenced pictures for rural areas.[18] Greenness within 300 m and 1000 m buffers was assessed using normalized difference vegetation index (NDVI) from Landsat 5 Thematic Mapper satellite images for 1994–1995 (S3) and 1999–2001 (S4).[19] Annual mean and standard deviation (SD) air temperature were extracted from temperature maps developed within a multi-stage modeling approach for 2000.[20] All exposures were assigned to the participants' residential baseline addresses.

Statistical methods

We used the Cox proportional hazards model to analyze the association between environmental factors and incident T2D, with follow-up time as the underlying timescale and an indicator variable for subcohort (S3/S4). Participants were censored at the time of the event, withdrawal request, death, emigration, loss to follow-up, or the end of the study period, whichever came first.

Three models with varying degrees of confounder adjustment were specified a priori based on previous research[21] and data availability. The minimum model included age, sex, and subcohort indicator, the main model was further adjusted for other baseline characteristics (body mass index [BMI], smoking behavior, alcohol consumption, education level, physical activity, and dietary score[22]), and the extended model additionally included clinical information (cardiovascular diseases (CVD), waist-hip ratio, and cholesterol level) (Text S2, http://links.lww.com/EE/A267).

In premodels, we tested nonlinearity but observed no considerable deviance from linearity. Therefore, exposure variables were included as linear terms (Figure S2, http://links.lww.com/EE/A267).

Effect modification was assessed by including an interaction term between each exposure variable and potential effect modifiers (sex, age, obesity, educational level, and physical activity) (Figure S3, http://links.lww.com/EE/A267). Results are presented as hazard ratios (HRs) per interquartile range (IQR) increase in exposure variables with 95% confidence intervals (CIs).

We used R 3.6.1 with "survival," "mgcv," and "raster" packages for all statistical analyses.

Sensitivity analysis

To evaluate the robustness of our findings, we used an alternative statistical model (Poisson regression) and applied the main model separately for the two subcohorts.

Results

Altogether, 7864 (86.3%) participants out of 9116 participants were included in our study (Figure S1, http://links.lww.com/EE/A267). Of these, 10.5% developed T2D until the end of follow-up (Table 1). At baseline, the mean age was 49.2 years, almost half of the participants were male (49.2%), and the mean BMI was 27.0 kg/m². About a quarter of the participants reported being active smokers, and 47.0% reported being physically active (Table 1).

Annual average concentrations of NO_2, PM_{10}, and $PM_{2.5}$ at participants' residences were below the European air quality standard values but exceeded the current WHO guideline values. The mean levels of traffic noise were above the European-recommended maximum values (Table 2).

Although some of the environmental exposures showed positive effect estimates (e.g. $PM_{2.5}$ showing the strongest effect estimate) confidence limits were large as estimated with the main model (Figure 1). The incorporation of additional covariates in the models had little to moderate impact on the HRs and 95% CIs compared to the minimum adjusted model. Sex modified the effect of NO_x, O_3, PM_{10}, and PNC on T2D incidence, with O_3 being more pronounced in females and the others in males (Figure S3, http://links.lww.com/EE/A267). The effect of $PM_{2.5abs}$ was modified by educational level.

Poisson models provided similar associations (Figure S4, http://links.lww.com/EE/A267) as the main Cox model. Also, stratified analyses by subcohort showed comparable results (Figure S5, http://links.lww.com/EE/A267) to the main pooled model.

Discussion

Our longitudinal analysis showed weak or no associations between the various environmental factors and T2D incidence, after adjusting for confounding factors. These results were consistent across different analytical approaches and stratified analyses of the two subcohorts. Considering the lack of significant associations in the single-exposure models, we refrained from conducting multi-exposure models.

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Table 1.

Baseline characteristics of study participants

Variable	KORA S3 + S4 (n = 7864)[a]		KORA S3 (n = 4042)[a]	KORA S4 (n = 3822)[a]
	Missing n (%)	Mean ± SD/ n (%)	Mean ± SD/ n (%)	Mean ± SD/ n (%)
Incident T2D during follow-up	0 (0.0)	829 (10.5)	486 (12.0)	343 (9.0)
Age (years)	0 (0.0)	49.2 ± 13.8	49.6 ± 13.9	48.8 ± 13.7
Sex (male)	0 (0.0)	3,873 (49.2)	2020 (50.0)	1852 (48.5)
BMI (kg/m²)	71 (0.9)	27.0 ± 4.5	27.0 ± 4.4	27.0 ± 4.6
Waist-hip ratio	55 (0.7)	0.9 ± 0.1	0.9 ± 0.1	0.9 ± 0.1
Education level	8 (0.1)			
Low		1148 (14.6)	676 (16.7)	472 (12.4)
Middle		5671 (72.2)	2895 (71.6)	2776 (72.8)
High		1037 (13.2)	470 (11.6)	567 (14.9)
Smoking status	5 (0.1)			
Current smoker		2012 (25.6)	1013 (25.1)	999 (26.2)
Ex-smoker		2486 (31.6)	1243 (30.8)	1243 (32.6)
Never smoker		3361 (42.8)	1786 (44.2)	1575 (41.3)
Alcohol intake (g/day)	17 (0.2)	16.3 ± 22.1	16.8 ± 22.8	15.9 ± 21.3
Physical activity (active)	18 (0.2)	3687 (47.0)	1784 (44.1)	1903 (50.0)
Dietary score	21 (0.3)	15.3 ± 3.6	15.3 ± 3.6	15.2 ± 3.6
Cardiovascular disease (yes)	17 (0.2)	3057 (39.0)	1674 (41.4)	1383 (36.3)
Total cholesterol (mg/dL)	139 (1.8)	229.2 ± 43.7	230.8 ± 44.0	227.4 ± 43.3

[a]n refers to the sample size before excluding participants with missing confounders used in the main model.

BMI indicates body mass index; KORA, Cooperative Health Research in the Region of Augsburg; S3, Third cross-sectional health survey of the KORA cohort; S4, Fourth cross-sectional health survey of the KORA cohort; SD, standard deviation; T2D, type 2 diabetes.

Table 2.

Descriptive statistics of annual average air pollutant concentrations, traffic noise, NDVI, and air temperature at residences

Variable		KORA S3 + S4 (n = 7864)[a]	KORA S3 (n = 4042)[a]	KORA S4 (n = 3822)[a]
		Mean ± SD	Mean ± SD/ n (%)	Mean ± SD/ n (%)
Air pollutant	NO_2 (μg/m³)	14.2 ± 4.4	14.1 ± 4.3	14.5 ± 4.5
	NO_x (μg/m³)	22.1 ± 7.1	22.0 ± 6.9	22.2 ± 7.4
	O_3 (μg/m³)	38.8 ± 2.4	38.8 ± 2.4	38.9 ± 2.5
	PM_{10} (μg/m³)	16.6 ± 1.5	16.5 ± 1.5	16.7 ± 1.5
	$PM_{2.5}$ (μg/m³)	11.8 ± 1.0	11.8 ± 1.0	11.8 ± 1.0
	$PM_{2.5, abs}$ (10^{-5}/m)	1.2 ± 0.2	1.2 ± 0.2	1.2 ± 0.2
	PM_{coarse} (μg/m³)	4.9 ± 1.0	4.8 ± 1.0	5.0 ± 1.0
	PNC (10^3/cm³)	7.3 ± 1.8	7.2 ± 1.8	7.3 ± 1.8
Noise	Daily average traffic noise (dB)	54.7 ± 6.6	54.6 ± 6.6	54.9 ± 6.6
	Nighttime average traffic noise (dB)	45.7 ± 6.4	45.6 ± 6.3	45.8 ± 6.4
Greenness	NDVI in 300 m buffer	0.5 ± 0.1	0.4 ± 0.1	0.5 ± 0.1
	NDVI in 1000 m buffer	0.5 ± 0.1	0.5 ± 0.1	0.5 ± 0.1
Temperature	Annual mean temperature (°C)	10.6 ± 0.4	10.6 ± 0.4	10.6 ± 0.4
	Annual SD temperature (°C)	6.1 ± 0.2	6.0 ± 0.2	6.1 ± 0.2

The EU air quality standard values are 40 μg/m³ for PM_{10} and NO_2, and 25 μg/m³ for $PM_{2.5}$. The WHO air quality guideline values are 10 μg/m³ for NO_2, 15 μg/m³ for PM_{10}, and 5 μg/m³ for $PM_{2.5}$. The WHO air quality guideline value for O_3 is 60 μg/m³, which is calculated based on the peak season. The EU-recommended maximum value for traffic noise during night is 40 dB in residential areas.

[a]n refers to the sample size before excluding participants with missing confounders used in the main model.

NDVI values below 0 were excluded since they represent water or bare rocks.

KORA indicates Cooperative Health Research in the Region of Augsburg; NDVI, normalized difference vegetation index; NO_2, nitrogen dioxide; NO_x, nitrogen oxide; O_3, ozone; PM_{10}, particulate matter with an aerodynamic diameter ≤10 μm; $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤2.5 μm; $PM_{2.5,abs}$, $PM_{2.5}$ absorbance; PM_{coarse}, particulate matter with an aerodynamic diameter of 2.5–10 μm; PNC, particle number concentration; S3, Third cross-sectional health survey of the KORA cohort; S4, Fourth cross-sectional health survey of the KORA cohort; SD, standard deviation.

Systematic reviews and meta-analyses have reported both positive and null associations between environmental exposures and T2D risk, indicating the complexity of these relationships.[3,11,23] They indicated robust results for T2D prevalence and heterogeneity between studies for incident T2D. A meta-analysis of cohort studies found higher risks of T2D associated with exposure to $PM_{2.5}$ (risk ratio [RR]: 1.39 [1.14–1.68]), PM_{10} (RR: 1.34 [1.22–1.47]), and NO_2 (RR: 1.11 [1.07–1.16]),[5] while another meta-analysis only reported an association for $PM_{2.5}$.[3] Our study found positive but nonsignificant associations for $PM_{2.5}$ (HR: 1.07 [0.97–1.17]) and weak associations for other air pollutants. Air pollution may contribute to insulin resistance and chronic inflammation, which are key mechanisms in the development of T2D.[24]

Similarly, a higher diabetes risk has been associated with higher levels of traffic noise.[9,23] However, this was not confirmed in a meta-analysis of four studies (pooled odds ratio [OR]: 1.49 [0.78–2.82]).[11] In our study, we observed weak and nonsignificant associations for traffic noise during the day (HR: 1.02 [0.93–1.11]) or at night (HR: 1.02 [0.94–1.11]). Noise-induced sleep disturbance and chronic stress may contribute to insulin resistance and an increased risk of diabetes.[25]

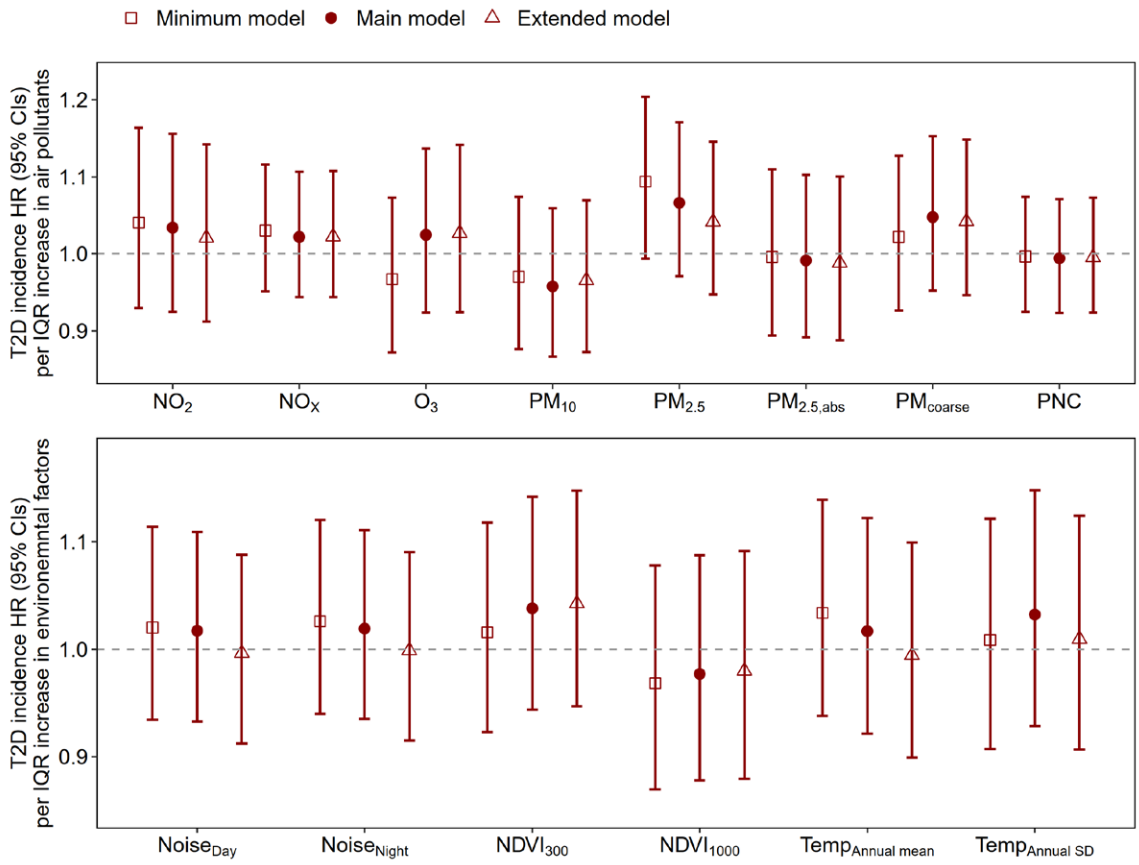

Figure 1. Hazard ratios and 95% CIs for the associations between environmental factors, and the risk of incident T2D. Minimum model included age, sex, and subcohort indicator. Main model was further adjusted for BMI, smoking status, alcohol consumption, education level, physical activity, and dietary score. Extended model additionally included cardiovascular diseases, waist-hip ratio, and total cholesterol level. HRs are expressed per IQR increase for each exposure variable. The IQRs were as follows: NO_2: 7.0 μg/m³, NO_X: 8.1 μg/m³, O_3: 3.6 μg/m³, PM_{10}: 2.2 μg/m³, $PM_{2.5}$: 1.3 μg/m³, $PM_{2.5,abs}$: 0.3 10^{-5}/m, PM_{coarse}: 1.4 μg/m³, PNC: 1.9 10^3/cm³, $Noise_{Day}$: 8.2 dB, $Noise_{Night}$: 7.9 dB, $NDVI_{300}$:0.12, $NDVI_{1000}$: 0.14, $Temp_{annual\ mean}$: 0.6 °C, $Temp_{annual\ SD}$: 0.2 °C. NDVI values below 0 were excluded since they represent water or bare rocks. Note: Minimum and main models were based on data for 7696 participants. In extended models we excluded 141 participants with missing clinical information, resulting in 7555 participants. BMI indicates body mass index; CI, confidence interval; HR, hazard ratio; IQR, interquartile range; NDVI, normalized difference vegetation index; NO_2, nitrogen dioxide; NO_x, nitrogen oxide; O_3, ozone; PM_{10}, particulate matter with an aerodynamic diameter ≤10 μm; $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤2.5 μm; $PM_{2.5,abs}$, $PM_{2.5}$ absorbance; PM_{coarse}, particulate matter with an aerodynamic diameter of 2.5–10 μm; PNC, particle number concentration; SD, standard deviation; T2D, type 2 diabetes; Temp, temperature.

Systematic reviews reported that green spaces may have a protective effect on T2D.[11,26,27] A meta-analysis provided further evidence that exposure to greenspace is associated with wide-ranging health benefits, including a significant reduction in T2D incidence (pooled OR: 0.72 [0.61–0.85]).[28] However, we observed only weak associations. Green spaces play a crucial role in promoting physical activity and protecting against air and noise pollution, all of which may contribute to mitigating chronic inflammation processes.[29]

Studies on the effect of air temperature on T2D risk are limited. In a study, diabetes incidence increased by 0.31 [0.19–0.43] per 1 °C increase in annual mean temperature.[30] In this study, we observed a weak effect of annual mean air temperature increase (HR: 1.02 [0.92–1.12]). In previous research, both higher temperatures[31] and lower exposure to cold[32] were associated with increased insulin resistance. However, our study found no evidence of this effect.

As previously reported,[33–36] our study found that sex and education level significantly modified air pollutant effects. However, our results showed opposite directions, possibly due to population variations, exposure misclassification, or chance findings. Lifestyle and physiological differences between sexes or education levels may contribute to these variations.[34,37]

Strengths and limitations

Our study's strengths include the use of two datasets from a large and well-characterized population-based prospective cohort, comprehensive exposure assessment, adjustment for important confounders, and application of various analytical approaches. However, limitations such as single-time point exposure assessment, potential exposure misclassification, not accounting for residential mobility from baseline to follow-up, and lack of information on other lifestyle and clinical factors should be acknowledged. Potentially, measurement error may have resulted in underestimation of the underlying associations and insufficient statistical power may be responsible for the wide CI. Our analyses were also restricted to a specific population and geographic region.

Conclusions

Although we observed weaker associations than previous studies indicated, our study contributes to the literature on environmental factors on incident T2D. Future research should continue exploring the role of the environment in T2D development.

Conflicts of interest statement

The authors declare that they have no conflicts of interest with regard to the content of this report

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