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Longitudinal associations of long-term exposure to ambient air pollution, residential greenness, and air temperature with type 2 diabetes subphenotypes: Results from the KORA cohort study

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ABSTRACT

Objective: To examine the association between environmental exposures and specific type 2 diabetes (T2D) subphenotypes.

Research design and methods: We categorized T2D participants from the KORA F4 (2006–2008) and FF4 (2013–2014) study waves into three phenotypes using k-means clustering: Cluster A (insulin deficiency); Cluster B (age-related diabetes); and Cluster C (higher insulin resistance). The annual averages of fine particulate matter ($PM_{2.5}$) and $PM_{2.5}$ absorbance ($PM_{2.5}$abs), annual air temperature mean (T_m) and standard deviations (T_{sd}), and greenness (NDVI), were assessed at participants' residences. Covariate-adjusted mixed multinomial logistic regression models were fitted to examine the effects of environmental exposures on diabetes subphenotypes. We also calculated joint odds ratios (ORs) to estimate the additive effects of exposure mixtures.

Results: The longitudinal analysis showed that interquartile range (IQR) increases in $PM_{2.5}$ (OR = 1.29, 95 % confidence interval [CI]: 1.01, 1.64) and $PM_{2.5}$abs (OR = 1.30, 95 % CI: 1.01, 1.67) were associated with higher odds of being in T2D Cluster C, compared to normoglycemic individuals. Furthermore, we found that IQR increases in $PM_{2.5}$ and T_{sd}, alongside with decreases in NDVI and T_m increased the odds of being in Cluster B (joint OR = 1.41, 95 % CI: 1.03, 1.93) and Cluster C (joint OR = 1.55, 95 % CI: 1.02, 2.36), while the combination of $PM_{2.5}$abs with other exposures increased the odds of Cluster C (joint OR = 1.54, 95 % CI: 1.01, 2.33).

Conclusions: Our study contributes to an enhanced understanding of the associations between environmental exposures and diabetes, indicating increased risks for age-related and insulin-resistant diabetes.

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1. Background

Diabetes presents a major public health challenge, accounting for 3.4 million deaths in 2024 (IDF, 2025). It can be generally classified into type 1 diabetes, type 2 diabetes (T2D), gestational diabetes, and specific types of diabetes, with T2D accounting for 90–95 % of all diabetes cases (ElSayed et al., 2023). T2D is a complex and heterogeneous disease influenced by multiple factors, including genetics and environmental factors. Treating T2D participants as a single homogeneous group has been shown to be insufficient in identifying patients who need more intensive treatment and in predicting related complications (Herder and Roden, 2022).

Based on six variables (glutamate decarboxylase antibodies, age at diagnosis, body mass index [BMI], hemoglobin A1c [HbA1c], and estimates of β-cell function and insulin resistance from the Homeostatic Model Assessment 2 [HOMA2-B and HOMA2-IR]), Ahlqvist et al. (Ahlqvist et al., 2018) identified five diabetes clusters and showed that patients in different clusters showed differences in disease progression, treatment, and development of diabetic complications. This approach may allow for tailored and targeted early treatment, thereby representing a first step towards precision medicine in diabetes. Several subsequent studies have replicated these clusters in different populations (Schrader et al., 2022; Zou et al., 2019; Dennis et al., 2019). However, in our previous study,researchers found that Ahlqvist's clusters did not apply to participants of the Cooperative Health Research in the Region of Augsburg (KORA) study (Dong et al., 2025). Instead, three de novo diabetes clusters (phenotypes) were reported: Cluster A, in which individuals were characterized by insulin deficiency; Cluster B, which was age-related diabetes; and Cluster C, marked by insulin resistance. In the current study, we further described participants' transitions between different T2D clusters over time.

Air pollution has been identified as one of the major environmental problems worldwide. Current systematic reviews and *meta*-analyses have shown that air pollution, including particulate matter (PM) with an aerodynamic diameter $\leq$ 2.5 μm ($PM_{2.5}$) (Eze et al., 2015; Liu, et al., 1987; Yang et al., 2020; Wang, 2014; Yang et al., 2020; Balti, 2014), PM $\leq$ 10 μm (PM_{10}) (Liu, et al., 1987; Yang et al., 2020; Wang, 2014; Yang et al., 2020), and nitrogen dioxide (NO_2) (Eze et al., 2015; Liu, et al., 1987; Yang et al., 2020; Wang, 2014; Balti, 2014), increases T2D risk. For example, a recent *meta*-analysis showed significant associations between T2D incidence and $PM_{2.5}$ (11 studies; hazard ratio [HR]: 1.10, 95 % confidence interval [CI]; 1.04–1.17 per 10 $\mu g/m^3$ increment) as well as PM_{10} (6 studies; HR: 1.11; 95 % CI: 1.00–1.22) (Yang et al., 2020). In addition, extreme air temperatures, both heat and cold, have been associated with increased diabetes risk (Song, 2021). Conversely, observational studies have suggested that exposure to green spaces may reduce T2D risk (Sørensen et al., 2022; Rodriguez-Loureiro, 2022; Yu, 2023). However, none of these studies has addressed the effects of environmental exposures on the risk of specific T2D subphenotypes.

Several studies reported that air pollution and a lack of green space were independently associated with a higher risk of T2D (Sørensen et al., 2022; Li et al., 2021; Clark et al., 2017). Researchers even found that the cumulative risk of these exposures may be higher than the risk estimates of any single exposure alone (Sørensen et al., 2022). Therefore, there is a need for more robust evidence on the combined effects of these environmental risk factors to design efficient prevention strategies that comprehensively address the different factors.

Additionally, research investigating the relationships between environmental exposures and different diabetes subgroups could help identify vulnerable populations that are particularly affected by different environmental exposures. This understanding may not only help elucidate the mechanisms by which environmental exposures contribute to diabetes but may also help reduce the incidence of diabetes and related mortality burden.

Therefore, based on data from two KORA study waves, we aimed to: 1) assign participants from the first follow-up examination (KORA F4) to different diabetes clusters (phenotypes), using centroids from the second follow-up examination (KORA FF4), and describe the cluster's transformation from F4 to FF4; 2) examine the cross-sectional and, in particular. the longitudinal associations between individual environmental exposures (air pollution, green space, and air temperature) and diabetes subphenotypes; 3) investigate the independent and combined effects of environmental exposures on T2D subphenotypes.

2. Methods

2.1. Study population

We used data from the prospective population-based KORA study, which was conducted in the city of Augsburg (Germany) and two adjacent counties, Augsburg and Aichach-Friedberg (Holle et al., 2005). In 1999–2001, 4,261 participants aged 25–74 years with German nationality were recruited for the fourth cross-sectional health survey of the KORA cohort (KORA S4), which used a randomized two-stage cluster sample with equal strata by sex and age from the target population of all German residents in the study regions (Laxy, et al., 2016; Rathmann, 2003). Two follow-up studies were carried out: the KORA F4 was conducted in 2006–2008 and consisted of 3,080 participants; the KORA FF4 was conducted in 2013–2014 and included 2,279 participants. At baseline and follow-up surveys, computer-assisted personal interviews, self-administered questionnaires, and standardized physical examinations were performed for participants, and blood samples were collected. The population selection process for the main analysis is shown in Fig. S1.

The KORA study was approved by the Ethics Committee of the Bavarian Medical Association (Bayerische Landesärztekammer). All participants provided written informed consent.

2.2. Study procedures

2.2.1. Outcome definition

To determine the prevalence of previously known T2D, participants were asked whether they suffered from diabetes. These self-reports were validated by the responsible physician, either from the medical chart review or based on the current use of glucose-lowering medication (Huth et al., 2019). Participants without known diabetes were assigned to receive a standard 75-g oral glucose tolerance test (OGTT). Blood samples were drawn without stasis after an overnight fast of at least eight hours and two hours after glucose solution intake. We divided participants into normoglycemia (i.e., fasting glucose $<$ 6.1 mmol/l and 2-h-glucose $<$ 7.8 mmol/l), prediabetes (fasting glucose $\geq$ 6.1 mmol/l but $<$ 7.0 mmol/l, and 2-h-glucose $<$ 7.8 mmol/l [isolated impaired fasting glucose (IFG)] or fasting glucose $<$ 6.1 mmol/l and 2-h-glucose $\geq$ 7.8 mmol/l but $<$ 11.1 mmol/l [isolated impaired glucose tolerance (IGT)], or both [IFG and IGT]), and newly diagnosed diabetes (fasting glucose $\geq$ 7.0 mmol/l or 2-h-glucose $\geq$ 11.1 mmol/l), according to the 1999/2006 WHO criteria (Alberti and Zimmet, 1998; WHO, 2006). We assumed newly diagnosed and known diabetes patients for whom the diabetes type could not be validated and for whom there was no conflicting information to have T2D. Our study included both known and newly diagnosed diabetes.

We divided T2D participants from KORA FF4 into three subphenotypes using BMI, age at examination, HbA1c, HOMA2-B, HOMA2-IR, and high-sensitivity C-reactive protein (hs-CRP), as reported in the study by Dong et al. (Dong et al., 2025). Briefly, we centered the above variables to a mean value of 0 and a standard deviation (SD) of 1 separately in males and females, and then applied k-means clustering to divide the participants into three different clusters: Cluster A, in which individuals were characterized by insulin deficiency (low HOMA2-B); Cluster B, characterized by older participants with high HbA1c; and Cluster C, characterized by insulin resistance (high HOMA2-IR).

2.2.2. Environmental exposure assessment

We assigned annual averages of ambient air pollution, greenness, and both the mean and SD of air temperature to the study participants, based on their home addresses. Briefly, residential annual average concentrations of ultrafine particles (PM $\leq$ 100 nm in aerodynamic diameter, represented by particle number concentration [PNC]), $PM_{2.5}$, between 2.5 and 10 μm (PM_{coarse}) and PM_{10}, $PM_{2.5}$ absorbance ($PM_{2.5}$abs, a proxy of elemental carbon related to traffic exhaust), NO_2, and nitrogen oxides (NO_x), were estimated by land-use regression models for 2014–2015 (Wolf et al., 2017). Exposure to residential greenness was assessed with the normalized difference vegetation index (NDVI), an indicator of vegetation density. For the present study, we used the NDVI map from Landsat 7 Enhanced Thematic Mapper (ETM) in 300 m, 500 m, and 1000 m buffers around the home addresses. We calculated and assigned the mean of the years 2006 and 2008 for F4 participants, and years 2013 and 2015 for FF4 participants, respectively. To examine the long-term effects of air temperature and its variability, our study calculated the annual average (T_m) and SD (T_{sd}) of air temperature using highly resolved estimations from a multi-stage regression-based model (Nikolaou et al., 2023). More details can be found in the Supplementary Materials.

2.2.3. Covariates

Socio-demographic variables, lifestyle characteristics, clinical treatments, and parental history of diabetes were assessed in standardized interviews by trained staff. We included age, sex, family status (living alone, living with others), years of education (highest level of vocational training and school graduation), socioeconomic status (SES; quintiles of the Helmert Index (Helmert and Shea 1994), based on school education, professional status, family income), smoking status (never, former, and current smokers), alcohol consumption (never, moderate and high), physical activity (regularly more than 2 h a week, regularly approx. 1 h per week, irregularly about 1 h a week, and almost no or no physical activity), BMI (kg/m^2), waist circumference (cm), systolic blood pressure (mmHg), high-density lipoprotein (mg/dL), triglyceride (mg/dL), antidiabetic medication (yes, no), and parental history of diabetes (yes, no). Details of these variables can be found in the Supplementary Materials.

2.3. Statistical analysis

2.3.1. Descriptive analysis

We described participants' characteristics as mean and SD for continuous variables with normal distribution, median and interquartile range (IQR) for continuous variables without normal distribution, and frequency (percentage) for categorical variables. Environmental exposures were summarized as mean ± SD, median (IQR), and minimum to maximum. Spearman correlation coefficients were calculated to assess their correlations.

2.3.2. Cluster transformation from F4 to FF4

We calculated the Euclidean distance of F4 T2D participants to cluster centers derived from FF4 coordinates (Table S1), which were obtained from the study of Dong et al. (Dong et al., 2025); using scaled clustering variables. We then assigned these F4 participants to different clusters based on which cluster they were most similar to, with the closest distance. We described the transformation of participants between different clusters from F4 to FF4 using a Sankey plot.

2.3.3. Association between environmental exposures and T2D subphenotypes

2.3.3.1. Single-exposure model. We fitted multinomial logistic regression models to simultaneously examine the associations between environmental exposures and T2D subphenotypes (Cluster A, Cluster B, and Cluster C), compared with participants with normal glycemia. We first performed cross-sectional analyses to examine the associations between T2D subphenotypes and an IQR increase in each individual exposure using only KORA FF4 data. For the longitudinal analysis, our analyses included all participants with at least one measurement. We assumed that measurements from different visits in each individual might not be independent, so multinomial logistic models with random intercepts for participants were applied to examine associations of exposures with repeated T2D subphenotypes. To exclude any bias from prediabetes, we restricted the reference group to participants with normoglycemia. More detailed information on sample size is provided in Fig. S1.

Covariates were selected a priori based on the disjunctive cause criterion, which should be the cause of either the exposure or the outcome, or both, but not belong to the potential pathway linking the exposure to the outcome (VanderWeele, 2019). We adjusted for age, sex, and study visits (F4/FF4, for longitudinal analysis only) in the minimum models. For the main models, family status, education years, BMI, smoking status, alcohol consumption, and physical activity were additionally included. Extended models further controlled for waist circumference, systolic blood pressure, high-density lipoprotein, and triglycerides. Effects estimates are presented as odds ratios (ORs) with 95 % confidence intervals (CI) per IQR increase in environmental exposure.

2.3.3.2. Multi-exposure model. We applied multi-exposure models in the longitudinal analysis to assess the independent effects of the exposures on T2D subphenotypes. We estimated two-, three-, and four-exposure models for all exposure combinations with a Spearman correlation coefficient $\leq$ 0.7.

2.3.3.3. Additive effects between exposures. We calculated joint odds ratios (JORs) to estimate the combined additive effects of individual exposures using the cumulative risk index (CRI) method. Referring to the study by Voss et al. (Voss et al., 2021); the JOR in our cases was defined as the OR for an IQR increase in air pollution and temperature deviation alongside an IQR decrease in residential greenness and air temperature mean, compared to no increase in all exposures.

2.3.3.4. Sensitivity analysis. We conducted several sensitivity analyses to test the robustness of our results. First, we excluded normoglycemic participants with missing values for clustering variables (HbA1c, HOMA2-B, and HOMA2-IR) to improve their comparability with diabetes participants from different clusters. Second, we pooled the diabetes-free and prediabetic participants and repeated the analysis with the pooled participants as the reference group. Third, we adjusted for glucose-lowering medication intake in the main model. Fourth, to balance the distribution of confounders, we included the generalized propensity score (GPS) in the regression models and fitted models weighted by inverse probability weights (IPW). Fifth, to test the robustness of the results of the longitudinal analysis, we applied generalized estimating equations (GEE) with a multinomial link and a "compound symmetry" working correlation to assess the associations between exposure variables and T2D subphenotypes. Sixth, we restricted the analyses to participants who had repeated measurements to reduce selection bias. Seventh, to avoid the effects of extreme environmental exposures, we excluded subjects exposed to environmental exposures greater than 99 % of the exposure values. Finally, we considered the effects of a parental history of diabetes on our associations.

All statistical analyses were performed with R (version 3.6.2), and the significance level was set at a two-sided *p*-value of less than 0.05.

3. Results

3.1. Study population

We assigned T2D participants in F4 to specific clusters with clustering centrioles in FF4: 22 participants (6.9 %) were assigned to Cluster A, which showed relatively high BMI and HbA1c but low HOMA2-B, 199 participants (62.8 %) to Cluster B, which were older, and 96 participants (30.3 %) to Cluster C, which was characterized by high HOMA2-IR. From F4 to FF4, most of the participants in Cluster B and Cluster C remained in the same clusters (84 out of 199 and 24 out of 96, respectively). Moreover, prediabetes in F4 contributed a lot to samples of Cluster B and Cluster C in FF4 (57/182 and 32/91, respectively) (Fig. S2, Fig. S3 and Table S2).

Participants' characteristics stratified by diabetes subphenotypes are shown in Table 1. Briefly, we included 2,947 participants (496 prediabetes) in F4, with a mean age of 56.1 years (SD of 13.2), and 1,526 (51.8 %) were female. In FF4, 2,150 individuals (397 prediabetes) participated, with a mean age of 60.2 (12.2) years, and 1,100 (51.2 %) were female. Participants with diabetes had fewer education years, were more often current smokers, had a lower percentage of high-level alcohol consumption, and were less physically active.

3.2. Environmental exposures

Table 2 presents the descriptive statistics and Spearman correlation coefficients for environmental exposures. For both F4 and FF4 waves, the annual average concentrations of $PM_{2.5}$, PM_{10}, and NO_2 exceeded the recommended WHO air quality guidelines levels (5 μg/m^3 for $PM_{2.5}$, and 10 μg/m^3 for PM_{10} and NO_2) (World Health Organization, 2021). However, concentrations were within the current European limit values (annual averages of 25 μg/m^3 for $PM_{2.5}$ and 40 μg/m^3 for PM_{10} and NO_2, respectively) (Directive 2008/50/EC). Air pollutants showed strong positive correlations with each other ($r \geq 0.60$) but highly negative correlations with greenness ($r \leq -0.70$). Annual T_m and T_{sd} were negatively correlated with each other and weakly or moderately correlated with other exposures.

3.3. Cross-sectional analysis

Associations between environmental exposures and T2D subphenotypes, as identified through cross-sectional analysis, are presented in Table 3. We found that annual air temperature variables were associated with Cluster B; the ORs per an IQR increase in T_{sd} and a decrease in T_m were 1.32 (95 % CI: 1.05, 1.66) and 1.22 (95 % CI: 1.01, 1.47), respectively. Our results also showed higher odds of being in Cluster C associated with an IQR increase in PM_{10} (OR = 1.40, 95 % CI: 1.02, 1.91).

3.4. Longitudinal analysis

Fig. 1 and Table S3 present the results of the longitudinal analyses. An IQR increase in NO_2 (OR = 0.56, 95 % CI: 0.34, 0.93) was negatively associated with Cluster A. Marginal associations were found between $PM_{2.5}$ (OR = 1.19, 95 % CI: 0.99, 1.43) and T_{sd} (OR = 1.22, 95 % CI: 0.99, 1.49) and Cluster B. For Cluster C, we found that $PM_{2.5}$ (OR = 1.29, 95 % CI: 1.01, 1.64) and $PM_{2.5}$abs (OR = 1.30, 95 % CI: 1.01, 1.67) significantly increased the odds, while PM_{coarse} (OR = 1.22, 95 % CI: 0.98, 1.54), PM_{10} (OR = 1.25, 95 % CI: 0.99, 1.58) and NDVI (OR = 1.22, 95 % CI: 0.97, 1.55) marginally increased it.

3.5. Multi-pollutant models

The results of the multi-exposure models in the longitudinal analysis are shown in Fig. 1. We found that the associations of $PM_{2.5}$ and $PM_{2.5}$abs and Cluster C were independent of T_m, T_{sd}, and their

Table 1
Description of participants' characteristics in KORA F4 (2006–2008) and FF4 (2013–2014).

Variables	F4				FF4			
	Normoglycemia (N = 2134)	Cluster A (N = 22)	ClusterB (N = 199)	Cluster C (N = 96)	Normoglycemia (N = 1452)	Cluster A (N = 28)	Cluster B (N = 182)	Cluster C (N = 91)
Age (years)	52.8 (12.6)	55.3 (10.2)	69.2 (8.14)	65.6 (8.87)	56.8 (11.5)	56.6 (11.2)	72.7 (7.75)	67.1 (9.96)
Female	1164 (54.5 %)	13 (59.1 %)	77 (38.7 %)	39 (40.6 %)	804 (55.4 %)	12 (42.9 %)	71 (39.0 %)	40 (44.0 %)
Living with others	1616 (75.7 %)	12 (54.5 %)	152 (76.4 %)	76 (79.2 %)	1092 (75.2 %)	20 (71.4 %)	127 (69.8 %)	63 (69.2 %)
Education years	11.0 (10.0, 13.0)	10.0 (10.0, 12.0)	10.0 (10.0, 12.0)	10.0 (10.0, 12.0)	11.0 (10.0, 13.0)	10.5 (10.0, 12.0)	10.0 (10.0, 13.0)	10.0 (10.0, 12.0)
SES (scores)	14.0 (10.4, 18.0)	10.8 (7.25, 14.8)	11.0 (9.00, 15.0)	12.0 (9.00, 16.0)	15.0 (12.0, 19.0)	13.5 (9.75, 15.0)	13.0 (10.0, 18.0)	13.0 (10.5, 16.0)
BMI (kg/m^2)	26.6 (4.33)	33.6 (7.03)	29.5 (3.94)	34.2 (4.48)	26.6 (4.48)	36.7 (7.09)	28.8 (3.93)	34.0 (4.47)
Smoking status								
Former	439 (20.6 %)	5 (22.7 %)	22 (11.1 %)	9 (9.4 %)	249 (17.1 %)	4 (14.3 %)	11 (6.0 %)	8 (8.8 %)
Current	814 (38.1 %)	9 (40.9 %)	95 (47.7 %)	54 (56.3 %)	605 (41.7 %)	12 (42.9 %)	88 (48.4 %)	40 (44.0 %)
Alcohol consumption								
Moderate	1150 (53.9 %)	9 (40.9 %)	97 (48.7 %)	40 (41.7 %)	830 (57.2 %)	12 (42.9 %)	102 (56.0 %)	33 (36.3 %)
High	370 (17.3 %)	2 (9.1 %)	34 (17.1 %)	16 (16.7 %)	263 (18.1 %)	4 (14.3 %)	33 (18.1 %)	13 (14.3 %)
Physical activity								
Regularly approx.1 h/wk	671 (31.4 %)	8 (36.4 %)	44 (22.1 %)	25 (26.0 %)	499 (34.4 %)	5 (17.9 %)	35 (19.2 %)	18 (19.8 %)
Irregularly about 1 h/wk	292 (13.7 %)	5 (22.7 %)	21 (10.6 %)	8 (8.3 %)	191 (13.2 %)	6 (21.4 %)	29 (15.9 %)	17 (18.7 %)
Almost no or no	599 (28.1 %)	7 (31.8 %)	100 (50.3 %)	48 (50.0 %)	337 (23.2 %)	16 (57.1 %)	81 (44.5 %)	40 (44.0 %)
WC (cm)	90.5 (12.8)	109 (14.3)	102 (9.97)	112 (10.6)	93.0 (13.1)	119 (15.6)	103 (10.9)	113 (10.7)
SBP (mmHg)	119 (17.4)	127 (19.1)	134 (20.4)	132 (16.5)	116 (16.3)	133 (21.3)	127 (18.5)	126 (19.2)
HDL (mg/dL)	1.49 (0.372)	1.23 (0.291)	1.29 (0.295)	1.17 (0.284)	1.77 (0.483)	1.38 (0.430)	1.57 (0.416)	1.37 (0.377)
TG (mg/dL)	1.27 (0.855)	2.59 (2.81)	1.89 (1.55)	2.16 (1.28)	1.25 (0.749)	2.23 (1.06)	1.64 (0.926)	1.91 (0.965)
Glucose-lowering medication	0 (0 %)	11 (50.0 %)	103 (51.8 %)	38 (39.6 %)	0 (0 %)	17 (60.7 %)	105 (57.7 %)	38 (41.8 %)
Parental diabetes history	530 (24.8 %)	13 (59.1 %)	82 (41.2 %)	35 (36.5 %)	420 (28.9 %)	16 (57.1 %)	81 (44.5 %)	38 (41.8 %)

Descriptive statistics are presented by mean (SD) and median [Q1, Q3] for continuous variables with and without normal distribution, and n (%) for categorical variables.

Abbreviation: KORA: Cooperative Health Research in the Region of Augsburg. F4: first follow-up examination of the KORA fourth cross-sectional health survey. FF4: second follow-up examination of the KORA fourth cross-sectional health survey. SES: socioeconomic status. BMI: body mass index. WC: waist circumference. SBP: systolic blood pressure. HDL: high-density lipoprotein cholesterol. TG: triglyceride. Cluster A, insulin-deficient diabetes. Cluster B, age-related diabetes. Cluster C, insulin-resistant diabetes.

Table 2
Descriptive statistics and Spearman correlations of air pollution, air temperature, and greenness in longitudinal analysis.

Exposures	Mean ± SD	Median (IQR)	Min, Max	***Air pollution***							***Air temperature***		***Greenness: NDVI***		
				$PM_{2.5}$	$PM_{2.5}$abs	PM_{10}	PM_{coarse}	PNC	NO_2	NO_x	T_{sd}	T_m	300 m	500 m	1000 m
Air pollution															
$PM_{2.5}$ (μg/m^3)	11.7 ± 1.02	11.8(1.44)	8.2,14.1	1											
$PM_{2.5}$abs (10‘-5/m)	1.2 ± 0.17	1.2(0.26)	0.8,1.7	0.7	1										
PM_{10} (μg/m^3)	16.5 ± 1.45	16.2(2.07)	12.1,22.0	0.6	0.8	1									
PM_{coarse} (μg/m^3)	4.9 ± 1.10	4.9(1.51)	2.5,8.2	0.6	0.8	0.8	1								
PNC (10^3/cm^3)	7.1 ± 1.73	7.1(1.99)	2.9,14.1	0.7	0.8	0.8	0.8	1							
NO_2 (μg/m^3)	14.1 ± 4.42	13.7(6.97)	6.9,27.2	0.8	0.9	0.7	0.8	0.8	1						
NO_x (μg/m^3)	21.8 ± 7.21	22.8(9.30)	3.9,46.0	0.8	0.8	0.8	0.8	0.9	0.9	1					
Air temperature (°C)															
T_{sd}	6.9 ± 0.87	6.3(1.75)	5.8,8.1	0.3	0.4	0.2	0.4	0.3	0.4	0.3	1				
T_m	9.2 ± 0.76	9.3(1.40)	7.8,10.7	0.3	0.2	0.2	0.1	0.1	0.2	0.2	−0.6	1			
Greenness: NDVI															
300 m buffer	0.4 ± 0.08	0.4(0.12)	0.1,0.7	−0.7	−0.8	−0.7	−0.8	−0.8	−0.8	−0.8	−0.3	−0.2	1		
500 m buffer	0.4 ± 0.09	0.5(0.13)	0.2,0.7	−0.8	−0.8	−0.7	−0.8	−0.8	−0.9	−0.8	−0.3	−0.2	1	1	
1000 m buffer	0.5 ± 0.09	0.5(0.14)	0.3,0.7	−0.8	−0.9	−0.7	−0.8	−0.7	−0.9	−0.8	−0.4	−0.3	0.9	0.9	1

Abbreviation: SD: standard deviation. IQR: interquartile range. PM: particulate matter. $PM_{2.5}$: PM with an aerodynamic diameter less than 2•5 μm. $PM_{2•5}$abs: $PM_{2•5}$ absorbance. PM_{coarse}: PM with an aerodynamic diameter of 2•5–10 μm. PM_{10}: PM with an aerodynamic diameter less than 10 μm. PNC: particle number concentration. NO_2: nitrogen dioxide. NO_x: nitrogen oxides. T_{sd}: standard deviation of temperature. T_m: mean temperature. NDVI: normalized difference vegetation index.

combination, and the effect of PM_{10} was independent of T_m. However, some changes occurred when we included other exposures. The marginal association between $PM_{2.5}$ and Cluster B became significant when we adjusted for T_m or the combination of T_m and T_{sd}. T_m showed a positive association with Cluster A when we adjusted for T_{sd}, and its combination with NDVI, PM_{10}, $PM_{2.5}$abs, PM_{coarse}, or PNC, and a positive association with Cluster B when $PM_{2.5}$ was included. The marginal effect of T_{sd} on Cluster B disappeared when we adjusted for air pollution, except for PM_{10}, NDVI, and T_m. The results of the multi-pollutant model for other exposures are presented in Fig. S4.

3.6. Cumulative effects of exposure mixture

We applied AIC and BIC to select the exposures for calculating the JORs, which are presented in Tables S4-S6. The JORs per IQR increases in air pollution and T_{sd}, alongside an IQR decrease in T_m and NDVI, can be found in Fig. 2. In general, we found larger effects of co-exposures on diabetes subphenotypes (including Cluster B and Cluster C) than the single exposure. For example, positive associations were found for the combination of $PM_{2.5}$, T_{sd}, NDVI, and T_m with Cluster B and Cluster C; the JOR for Cluster B from this four-exposure model was 1.41 (95 % CI: 1.03, 1.93), whereas for Cluster C, the JOR was 1.55 (95 % CI: 1.02, 2.36). The results for $PM_{2.5}$abs and its co-exposure with other exposures were similar to those for $PM_{2.5}$. The JORs for Cluster B from two-, three-, and four-exposure models were 1.30 (95 % CI: 1.01, 1.67), 1.39 (95 % CI: 1.03, 1.88), and 1.33 (95 % CI: 0.98, 1.82), respectively. JORs for Cluster C from two-, three-, and four-exposure models were 1.39 (95 % CI: 0.99, 1.95), 1.52 (95 % CI: 1.02, 2.26), and 1.54 (95 % CI: 1.01, 2.33), respectively.

3.7. Sensitivity analysis

We conducted several sensitivity analyses to assess the robustness of our results, which can be found in Fig. S5. Generally, we found very similar associations between diabetes subphenotypes and air pollutants. However, some differences were observed for air temperature variables. The association between T_{sd} and diabetes subphenotypes tended to be close to zero when we excluded extreme exposure values. In contrast, the effect seemed to increase when we included only participants who participated both studies. For T_m, we found higher effects on Cluster B and Cluster C in the models when we excluded extreme exposure values.

4. Discussion

In the current study, we divided the T2D participants into three clusters: Cluster A, which was characterized by insulin deficiency; Cluster B, defined as age-related diabetes; and Cluster C, which was characterized by high levels of insulin resistance. Participants from Cluster B and Cluster C showed relatively stable trajectories; most of them remained in the same clusters at the follow-up. Our longitudinal analysis found that higher $PM_{2.5}$ and $PM_{2.5}$ absorbance were associated with increased Cluster C risk, which was independent of T_{sd} and T_m. In the joint analyses, we found positive effects of increased $PM_{2.5}$/$PM_{2.5}$abs and T_{sd} and decreased T_m and NDVI on Cluster B and Cluster C.

In contrast to previous studies that mainly focused on the prevalence or incidence of overall T2D, we classified T2D participants into three subphenotypes with different traits. Our analyses observed associations between increased exposure to $PM_{2.5}$ and the odds of being in T2D Cluster C (independent of other exposures) and Cluster B (after controlling for other exposures). To our knowledge, this is the first study to expand the effects of environmental exposures to the specific T2D subphenotypes, although their effects on T2D risk have been widely reported. Previous systematic reviews and *meta*-analyses have widely reported associations between $PM_{2.5}$ and the prevalence and incidence of T2D (Liu, et al., 1987; Yang et al., 2020; He et al., 2017). Additionally, a *meta*-analysis showed that black carbon was positively associated with

Table 3
Odds ratios (ORs) (together with 95% confidence intervals) of diabetes subphenotypes for an interquartile range (IQR) change of environmental exposures in the cross-sectional analysis (FF4).

Exposures	Models	Cluster A	Cluster B	Cluster C
Air pollution				
$PM_{2.5}$	Minimum	0.71(0.44,1.15)	1.22(0.96,1.56)	1.26(0.92,1.72)
	Main	0.80(0.48,1.34)	1.26(0.98,1.62)†	1.36(0.98,1.89)†
	Extended	0.81(0.48,1.36)	1.26(0.98,1.62)†	1.35(0.97,1.88)†
$PM_{2.5}$abs	Minimum	0.66(0.37,1.18)	1.12(0.87,1.45)	1.33(0.96,1.83)†
	Main	0.76(0.42,1.38)	1.17(0.90,1.52)	1.37(0.98,1.93)†
	Extended	0.78(0.42,1.45)	1.19(0.91,1.54)	1.43(1.01,2.01)*
PM_{coarse}	Minimum	0.70(0.42,1.16)	1.10(0.87,1.38)	1.10(0.82,1.47)
	Main	0.81(0.48,1.37)	1.15(0.91,1.46)	1.24(0.91,1.69)
	Extended	0.86(0.50,1.49)	1.18(0.92,1.50)	1.28(0.93,1.76)
PM_{10}	Minimum	0.71(0.40,1.27)	1.08(0.85,1.38)	1.32(0.98,1.78)†
	Main	0.83(0.47,1.48)	1.12(0.88,1.44)	1.40(1.02,1.91)*
	Extended	0.90(0.49,1.64)	1.15(0.90,1.48)	1.45(1.05,2.00)*
PNC	Minimum	0.73(0.47,1.13)	1.14(0.94,1.39)	1.13(0.88,1.44)
	Main	0.78(0.50,1.23)	1.17(0.96,1.43)	1.18(0.91,1.53)
	Extended	0.82(0.51,1.32)	1.19(0.97,1.45)†	1.23(0.94,1.61)
NO_2	Minimum	0.50(0.26,0.96)*	1.14(0.87,1.49)	1.14(0.81,1.60)
	Main	0.54(0.27,1.07)†	1.18(0.90,1.55)	1.19(0.83,1.72)
	Extended	0.56(0.27,1.14)	1.19(0.90,1.57)	1.25(0.86,1.81)
NO_x	Minimum	0.68(0.43,1.07)†	1.11(0.89,1.38)	1.14(0.87,1.51)
	Main	0.72(0.44,1.18)	1.15(0.92,1.44)	1.22(0.91,1.64)
	Extended	0.75(0.45,1.25)	1.17(0.93,1.46)	1.26(0.93,1.71)
Air temperature				
T_{sd}	Minimum	0.82(0.49,1.37)	1.25(1.00,1.57)†	1.06(0.80,1.42)
	Main	0.95(0.54,1.67)	1.32(1.05,1.66)*	1.19(0.87,1.63)
	Extended	1.02(0.56,1.83)	1.38(1.09,1.74)*	1.34(0.97,1.86)†
T_m	Minimum	0.99(0.66,1.50)	1.17(0.97,1.41)†	1.03(0.81,1.30)
	Main	1.12(0.71,1.77)	1.22(1.01,1.47)*	1.14(0.88,1.47)
	Extended	1.18(0.73,1.89)	1.26(1.04,1.53)*	1.23(0.95,1.60)
NDVI				
300 m	Minimum	0.72(0.41,1.25)	1.05(0.83,1.34)	1.25(0.92,1.70)
	Main	0.83(0.48,1.46)	1.09(0.85,1.40)	1.29(0.94,1.79)
	Extended	0.86(0.48,1.54)	1.11(0.87,1.43)	1.30(0.94,1.80)
500 m	Minimum	0.59(0.32,1.08)†	1.04(0.80,1.34)	1.20(0.87,1.66)
	Main	0.66(0.35,1.22)	1.07(0.82,1.39)	1.24(0.88,1.75)
	Extended	0.68(0.36,1.29)	1.08(0.83,1.41)	1.24(0.88,1.77)
1000 m	Minimum	0.53(0.29,0.99)*	1.08(0.83,1.39)	1.20(0.86,1.66)
	Main	0.55(0.28,1.07)†	1.12(0.86,1.46)	1.24(0.87,1.76)
	Extended	0.55(0.28,1.10)†	1.13(0.87,1.48)	1.25(0.88,1.79)

Note: We used normoglycemia as the reference group. The minimum model was adjusted for age and sex. The main model was adjusted for age, sex, family status, education years, body mass index, smoking status, alcohol consumption, and physical activity. The extended model was adjusted for covariates in the main model, plus waist circumference, systolic blood pressure, high-density lipoproteins cholesterol, and triglyceride. Effects were estimated for an IQR decrease in mean temperature and greenness, and an IQR increase in air pollution and temperature deviation.
Abbreviation: PM: particulate matter. $PM_{2.5}$: PM with an aerodynamic diameter less than 2•5 μm. $PM_{2•5}$abs: $PM_{2•5}$ absorbance. PM_{coarse}: PM with an aerodynamic diameter of 2•5–10 μm. PM_{10}: PM with an aerodynamic diameter less than 10 μm. PNC: particle number concentration. NO_2: nitrogen dioxide. NO_x: nitrogen oxides. T_{sd}: standard deviation of temperature. T_m: mean temperature. NDVI: normalized difference vegetation index. Cluster A, insulin-deficient diabetes. Cluster B, age-related diabetes. Cluster C, insulin-resistant diabetes.
† $P < 0.1$.* $P < 0.05$.** $P < 0.001$.

T2D incidence (Chen et al., 2024). Using $PM_{2.5}$abs as a surrogate marker for black carbon, we also found that it independently increased the odds of being in Cluster C. In our analyses, exposure to PM_{10} was associated with Cluster C after adjustment for mean air temperature. Previous *meta*-analyses have reported a positive association between PM_{10} and T2D prevalence (Liu, et al., 1987; Yang et al., 2020). However, the evidence for incidence is mixed, with one *meta*-analysis showing an association (Yang et al., 2020), whereas another did not (Liu, et al., 1987). For NO_2, we reported an adverse effect on Cluster A, which disappeared in the multi-pollutant model. However, previous *meta*-analyses have shown that NO_2 is associated with a higher T2D prevalence (Liu, et al., 1987; Yang et al., 2020). We also found no significant associations between PM_{coarse}, PNC, NO_x, and NDVI with T2D subphenotypes. Nevertheless, previous studies have shown that higher levels of PM_{coarse} (Li et al., 2022), UFP (Sørensen et al., 2022; Sørensen et al., 2022), and NO_x (Zheng et al., 2024; Renzi et al., 2018), but lower exposure to green space (Twohig-Bennett and Jones, 2018; den Braver et al., 2018) were all associated with increased risks of T2D.

In our participants, we found that a decrease in the annual average air temperature increased the odds of being in Clusters A and B after including other environmental exposures. Previous studies have reported mixed results regarding these associations: some showed a dip pattern in temperature-related T2D prevalence (Luo et al., 2021); while others found positive associations between average air temperature and T2D prevalence (Speakman and Heidari-Bakavoli, 2016; Valdés et al., 2019). Additionally, we found a positive association between temperature deviations and Cluster B, which became less significant in the multi-pollutant models. However, we could not find any existing studies in this area.

Our findings on the relationship between environmental exposures and specific subphenotypes add evidence to the associations between these risk factors and T2D and offer new insights into the potential biological mechanisms by which environmental exposures may influence insulin-resistant diabetes. Current evidence suggests that certain environmental exposures (e.g., PM_{10}) may have varying effects on the onset and progression of diabetes. Our analyses combined known and newly diagnosed participants because of the limited sample size. Further research should address the differential associations between the

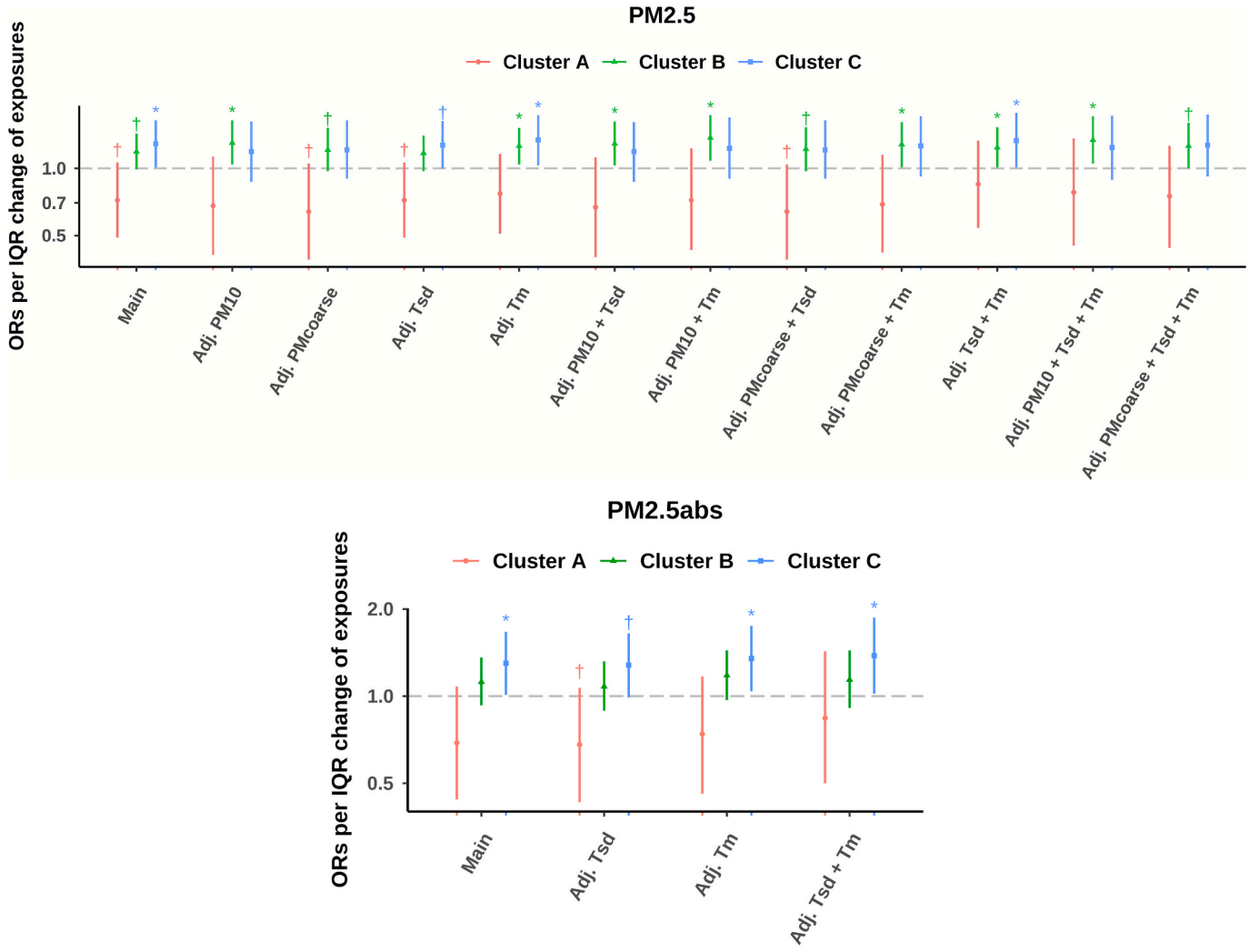

Fig. 1. Odds ratios (ORs) of diabetes subphenotypes associated with an interquartile range (IQR) change in air pollution, temperature, and NDVI from single, two-, three-, and four-exposure models in longitudinal analysis. Note: We used normoglycemia as the reference group. Models were adjusted for age, sex, visits, family status, education years, body mass index, smoking status, alcohol consumption, and physical activity. Effects were estimated for an IQR decrease in mean temperature and NDVI in a 300 m buffer, and an IQR increase in air pollution and temperature deviation. Abbreviation: PM: particulate matter. $PM_{2.5}$: PM with an aerodynamic diameter less than 2•5 μm. $PM_{2•5}$abs: $PM_{2•5}$ absorbance. PM_{coarse}: PM with an aerodynamic diameter of 2•5–10 μm. PM_{10}: PM with an aerodynamic diameter less than 10 μm. T_{sd}: standard deviation of temperature. T_m: mean temperature. NDVI: normalized difference vegetation index. Cluster A, insulin-deficient diabetes. Cluster B, age-related diabetes. Cluster C, insulin-resistant diabetes. † $P < 0.1$. * $P < 0.05$. ** $P < 0.001$.

environments and the prevalence and incidence of T2D subphenotypes. Furthermore, our analysis revealed that participants transitioned between different subphenotypes; future studies focusing on how environmental factors influence this transition could contribute to disease management and enhanced understanding of the underlying biological mechanisms.

One of the potential mechanisms linking air pollution to diabetes is insulin resistance. In our study, exposure to high levels of air pollution was mainly associated with increased odds of being in Cluster C, where participants had higher levels of HOMA-IR. Meta-analyses have reported consistent positive effects of PM_{10} and NO_2 on HOMA-IR (Gong et al., 2024; Dang et al., 2018). However, research on $PM_{2.5}$ yields mixed results: one study found only a weak positive effect (Gong et al., 2024), while another found no effect at all (Dang et al., 2018). Insulin resistance, a key factor in the pathogenesis of T2D, can be triggered by systemic inflammation and oxidative stress. It has been postulated that exposure to air pollution may accelerate this process. In studies conducted on mice, researchers reported that exposure to $PM_{2.5}$ induced insulin resistance, increased visceral adipose tissue, and caused immune response dysregulation and inflammation triggered by pulmonary oxidative stress (Sun et al., 2009; Haberzettl et al., 2016). Another animal experiment reported that exposure to PM2.5 impaired glucose and insulin tolerance, and regulated chromatin remodelers, while the cessation of exposure was associated with a reversal of insulin resistance and exposure-induced changes in the transcriptome (Rajagopalan et al., 2020). These findings may help to clarify the associations reported in our study.

Another potential pathway linking air pollution and diabetes is obesity. Epidemiologic evidence has shown that air pollution is associated with increased obesity risk (Lin et al., 2022). Cellular damage and/or cellular metabolism related to glucose utilization and low levels of outdoor physical activity in areas with high pollution levels may explain the relationship between air pollution and obesity. The cellular mechanisms through which environmental pollutants alter glucose metabolism include increased reactivity of lung tissue to pollutant stress and increased inflammatory signals. The combination of low-grade inflammation, oxidative stress, and mitochondrial imbalance reduces intracellular glucose transport in skeletal muscle (Merz and Thurmond, 2020); increases neo-glucogenesis in the liver by damaging the lipolysis and lipogenesis balance and promoting steatohepatitis (Liu et al., 2014;

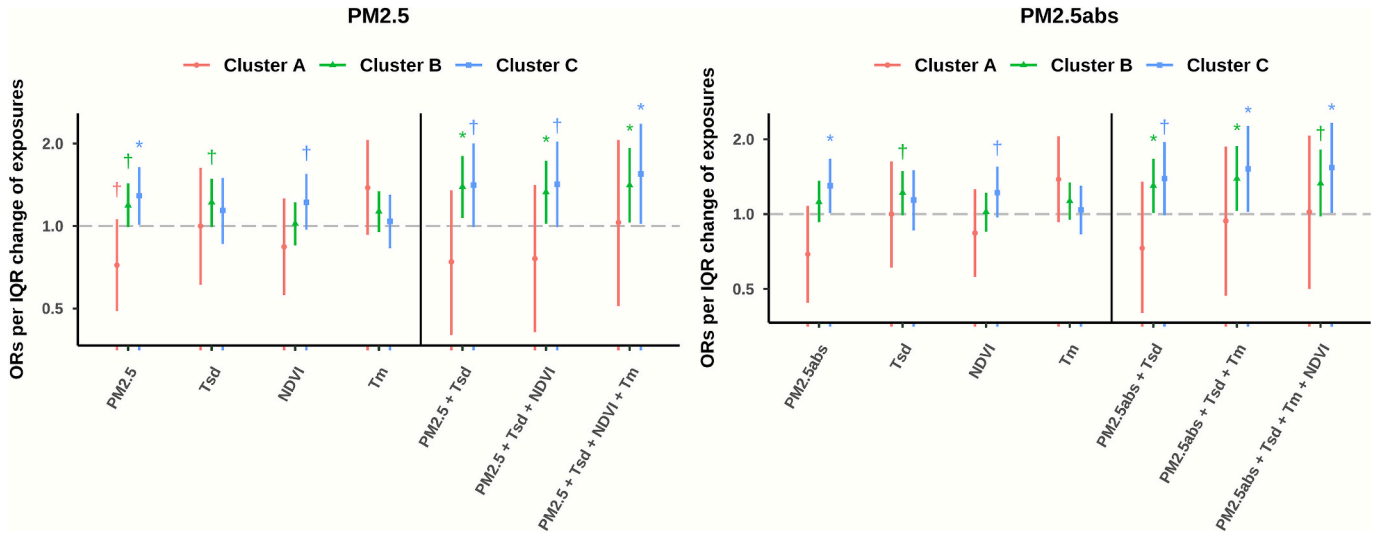

Fig. 2. Cumulative Risk Index of diabetes subphenotype associated with an interquartile range (IQR) change in air pollution, temperature, and NDVI from single- and multi-exposure models in the longitudinal analysis. Note: We used normoglycemia as the reference group. Models were adjusted for age, sex, visits, family status, education years, body mass index, smoking status, alcohol consumption, and physical activity. Effects were estimated for an IQR decrease in mean temperature and NDVI in a 300 m buffer, and an IQR increase in air pollution and temperature deviation. Abbreviation: PM: particulate matter. $PM_{2.5}$: PM with an aerodynamic diameter less than 2•5 μm. $PM_{2•5}$abs: $PM_{2•5}$ absorbance. T_{sd}: standard deviation of temperature. T_m: mean temperature. NDVI: normalized difference vegetation index. Cluster A, insulin-deficient diabetes. Cluster B, age-related diabetes. Cluster C, insulin-resistant diabetes. . † $P < 0.1$. * $P < 0.05$. ** $P < 0.001$.

Zheng et al., 2013); and reduces the protein expression that regulates adiposity metabolism in adipose tissue (Campolim et al., 2020). However, we failed to find an increased Cluster A risk with higher effects of air pollution, in which participants showed relatively high BMI and hs-CRP. Participants in Cluster A were characterized by higher insulin deficiency, which is thought to be genetically determined and may partly explain the non-significant effects. Our original clustering analysis also reported the highest polygenic risk scores in Cluster A participants (Dong et al., 2025).

We also found that $PM_{2.5}$, $T_{m,}$ and T_{sd} increased the odds of being in Cluster B, which was defined as age-related diabetes. Glycemia increases with age. Chia et al. (Chia et al., 2018) proposed a model of aging that concluded that the body composition changes intrinsically connected with aging contribute to excessive adipose accumulation, which causes insulin resistance. Researchers showed that aging resulted in the perturbation of the muscle Glycerophosphocholine phosphodiesterase 1-glycerophosphocholine (Gpcpd1-GPC) metabolic pathway in rodents and humans, which plays an important role in regulating glucose homeostasis (Cikes et al., 2024). The associations observed in our study suggest that non-optimal environmental exposures may accelerate age-related impaired glucose tolerance, but more evidence should be added.

Our multi-pollutant analyses revealed that the associations between $PM_{2.5}$, $PM_{2.5}$abs, and PM_{10} and Cluster C were independent of air temperature. However, the effects of $PM_{2.5}$ on Cluster B, mean air temperature on Clusters A and B, and T_{sd} on Cluster B were modified after adjustment for other exposures. To our knowledge, no studies have examined the independent effects of air pollution and temperature variables on diabetes risk. Several studies examined the mutual confounding of air pollution, greenness, and noise on diabetes risk, but the results were inconsistent (Sørensen et al., 2022; Clark et al., 2017; Klompmaker et al., 2019). Generally, most of the evidence supported the independent effects of air pollution and greenness on diabetes, although some heterogeneity was reported.

In our joint analyses, we found that increases in $PM_{2.5}$/$PM_{2.5}$abs in combination with T_{sd} increased the odds of being in Clusters B and C. Additionally, the effects of exposure mixtures were higher than those of single-exposure models. Several studies reported significant joint effects of environmental co-exposures. In a Danish study recruiting 1,922,545 participants, researchers reported significant effects of co-exposures to UFP, NO_2, noise, and greenness on T2D incidence, and the CRI was higher than the individual effects (Sørensen et al., 2022). Another cross-sectional study of 354,827 Dutch participants also supported that the JOR of the combination of NO_2, the intrinsic measures of PM, noise, and NDVI on diabetes prevalence was greater than the single-exposure OR (Klompmaker et al., 2019). In a prospective study with 390,834 participants in the UK Biobank, researchers found that exposure to 24-hour road traffic noise, $PM_{2.5}$, and NO_2, as well as their co-exposures, increased the risk of T2D incidence; however, the JOR for co-exposures was not greater than the individual OR (Hu et al., 2023).

The significant associations between co-exposures and T2D subphenotypes indicate the integrated effect of poor residential environments on human health, highlighting the importance of mitigating environmental exposures through comprehensive interventions. Furthermore, we found that the JORs from the multi-pollutant models were higher than the ORs from the single-exposure models, suggesting that considering environmental exposures separately may lead to underestimating the effects. This may be plausible as people are often co-exposed to air pollution, lack of green space, and air temperature, which may simultaneously affect health via subclinical inflammation, endothelial dysfunction, and reduced physical activity (Münzel et al., 2017; Tainio et al., 2021). However, caution should be taken when interpreting these results, as some deviation may be induced by multicollinearity between exposures in the CRI calculation. For example, the analysis included NDVI, which was highly correlated with other exposures in our data.

5. Strengths and limitations

Our study has several strengths. To our knowledge, this is the first study to examine the associations between long-term environmental exposures and T2D subphenotypes. We used data from the KORA study, a well-established prospective cohort study that collected various demographic, lifestyle, clinical, and environmental data. This allowed us to explore the temporal transmission of diabetes clusters through repeated assessments, to adjust for multiple confounders in our analyses, and to calculate both single and joint effects of co-occurring exposures using comprehensive environmental exposure data. The consistent results from several sensitivity analyses indicate the robustness of our

findings.

Several limitations, however, should also be mentioned. We assigned the environmental exposures to the participants' home addresses, which may lead to exposure misclassification. We do not know the extent to which the assigned values correspond to real personal exposures, as the duration of time spent at home was unavailable. This study only used air pollution data from 2014 to 2015 due to data availability constraints. The actual exposures at KORA F4, which occurred between 2006 and 2008, may differ. Nevertheless, several previous studies have shown that the spatial variation of air pollution remained stable over time (for ten years or longer) (Eeftens et al., 2011; de Hoogh et al., 2018; Gulliver et al., 2011). Moreover, researchers have demonstrated that back-extrapolation of air pollution data prior to 2014 did not alter the estimated health effects (Wolf et al., 2021). Despite previous *meta*-analyses demonstrating different effects of air pollution on T2D onset and progression, we pooled newly diagnosed and known diabetes for the clustering and association analyses due to the limited sample size for diabetes, differences in disease duration, medication, glucose control, susceptibility to adverse metabolic effects of environmental exposures, and the proportion of subphenotypes that may influence the results. Besides, the small sample size, especially in Cluster A, limited our statistical power, which should be regarded as rather exploratory. The participants in the KORA study were mainly of European descent, so the results may not be generalized to other ethnicities. Our analyses focused on four ubiquitous environmental exposures; however, further studies should also address other exposures, such as nighttime light, persistent organic pollutants, and metals. Finally, the observational study design limits the ability to infer causality.

6. Conclusions

The current study suggests that environmental exposures appear more relevant to the risk of insulin-resistant and age-related T2D than to insulin-deficient T2D. Our study highlights the need for a more accurate classification of T2D to fully disentangle diabetes risk factors. Furthermore, the results indicate both the independent and additive effects of environmental exposures on the diabetes subphenotypes, suggesting the importance of using not only isolation but also multi-exposure strategies in future research.

Contribution statement

Yue Xi and Annette Peters conceptualized the research question. Yue Xi drafted the analysis plan, performed the statistical analysis, and wrote the manuscript with guidance from Susanne Breitner-Busch and Annette Peters. Susanne Breitner-Busch and Annette Peters provided advice on statistical analysis and results visualization. Yue Xi and Qiuling Dong did the clustering analysis. Kathrin Wolf, Marco Dallavalle, Nikolaos Nikolaou, Josef Cyrys, Harald Grallert, Birgit Linkohr, Wolfgang Rathmann, Christian Herder, Lars Schwettmann, and Barbara Thorand contributed data. All authors were involved in the review, editing, and final approval of the manuscript. Annette Peters acquired the funding.

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The funding sources had no role in the collection, analysis, or interpretation of data, writing of the report, or in the decision to submit the paper for publication.

CRediT authorship contribution statement

Yue Xi: Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Conceptualization. **Susanne Breitner-Busch:** Writing – review & editing, Visualization, Methodology. **Qiuling Dong:** Writing – review & editing, Methodology. **Kathrin Wolf:** Writing – review & editing, Data curation. **Marco Dallavalle:** Writing – review & editing, Data curation. **Nikolaos Nikolaou:** Writing – review & editing, Data curation. **Josef Cyrys:** Writing – review & editing, Data curation. **Harald Grallert:** Writing – review & editing, Data curation. **Birgit Linkohr:** Writing – review & editing, Data curation. **Wolfgang Rathmann:** Writing – review & editing, Data curation. **Christian Herder:** Writing – review & editing, Data curation. **Lars Schwettmann:** Writing – review & editing, Data curation. **Barbara Thorand:** Writing – review & editing, Data curation. **Reiner Jumpertz von Schwartzenberg:** Writing – review & editing, Supervision. **Annette Peters:** Writing – review & editing, Visualization, Supervision, Methodology, Funding acquisition, Data curation, Conceptualization.

Declaration of competing interest

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

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2025.110043.

Data availability

Data will be made available on request.

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