



Full length article

Association between transportation noise exposure and type 2 diabetes risk in a French prospective cohort: the E3N-generations cohort

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ABSTRACT

Background: It has been suggested that exposure to transportation noise is associated with an increased risk of type 2 diabetes (T2D), but only a few prospective cohort studies have investigated this hypothesis for railway and aircraft noise. In the BROUHAHA study, we examined this association using data from the E3N-Generations cohort of French women.

Methods: We included 18,926 women residing in the Île-de-France or Auvergne-Rhône-Alpes regions (France) between 2000 and 2014. Annual average L_{den} (day-evening-night level) and L_n (night level) values were estimated for road traffic, railway, and aircraft noise using strategic noise maps. Incident T2D cases were identified through follow-up questionnaires and validated using drug reimbursement insurance databases. Cox proportional hazards models were used to calculate adjusted hazard ratios (HRs) and 95 % confidence intervals (CI).

Results: A 10 dB(A) increase in road traffic noise exposure was associated with a moderate increase in T2D risk (L_{den} : HR = 1.08, 95 %CI: 1.00, 1.18; L_n : HR = 1.12, 95 %CI: 1.01, 1.25). Adjustment for NO_2 and $PM_{2.5}$ slightly increased the estimated HRs. No association was observed between railway or aircraft noise and T2D incidence.

Conclusion: In this prospective cohort study of French women, exposure to road traffic noise was associated with a moderate increase in T2D risk, independently of NO_2 and $PM_{2.5}$ exposure. This association was slightly stronger for night-time noise exposure than for day-evening-night exposure.

1. Introduction

The World Health Organization (WHO) Environmental Noise Guidelines, highlight the evidence on the health effects of transportation noise (World Health Organization, 2018). These guidelines are based on epidemiological studies demonstrating associations between exposure to transportation noise and annoyance (Guski et al., 2017; Lefèvre et al., 2020), sleep disturbance (Basner and McGuire, 2018; Brink et al., 2019; Nassur et al., 2019), cardiovascular diseases (Kourieh et al., 2022;

Münzel et al., 2021; Van Kempen et al., 2018), and impairment in cognitive performance in children, particularly in reading comprehension and long-term memory (Clark et al., 2020).

According to the European Environment Agency (2025), approximately 112 million Europeans (20 % of the population) are exposed to transportation noise levels exceeding the threshold set by the Environmental Noise Directive: 55 dB(A) for the day-evening-night period (L_{den} indicator) and 50 dB(A) for the night-time period (L_n indicator). Road traffic noise is the main contributor, with 92 million people exposed

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above 55 dB(A) for L_{den} and 58 million above 50 dB(A) for L_n . Railway noise affects 18 million people (4 %) for L_{den} and 13 million (3 %) for L_n , while aircraft noise affects 2.6 million (0.5 %) for L_{den} and fewer than 1 million (0.2 %) for L_n (European Environment Agency, 2025).

In France, the situation is more pronounced, with 36 % of the population exposed to transportation noise above 55 dB(A) for L_{den} and 21 % above 50 dB(A) for L_n (European Environment Agency, 2025).

The WHO (2018) has identified source-specific threshold levels of noise exposure associated with adverse health outcomes: 53 dB(A) for road traffic, 54 dB(A) for railway, and 45 dB(A) for aircraft noise (L_{den}), and 45, 44, and 40 dB(A) respectively for night-time noise (L_n). Exceeding these levels has been linked to non-auditory effects, including sleep disturbance and metabolic disorders, including type 2 diabetes (T2D) (World Health Organization, 2018).

Diabetes is a major public health concern, ranking among the ten leading causes of death in adults aged 20 to 79 years (World Health Organization, 2020). According to the International Diabetes Federation (IDF) Atlas (2025), diabetes caused 3.4 million deaths worldwide, including 33,903 in France in 2024. Globally, 11.1 % of the adult (≈ 589 million) and 6.5 % (≈ 4.1 million) in France were living with diabetes in 2024, accounting for an estimated global health expenditure of US\$ 1.015 trillion (International Diabetes Federation, 2025). These figures are projected to rise, reaching 13 % globally and 7.8 % in France by 2050, with global expenditures exceeding US\$ 1.043 trillion (International Diabetes Federation, 2025). T2D accounts for more than 90 % of all diabetes cases (International Diabetes Federation, 2025).

An association between transportation noise exposure and T2D is biologically plausible. Chronic noise exposure can disrupt sleep and activate physiological stress pathways (Sharma et al., 2022), triggering the hypothalamic–pituitary–adrenal axis and releasing stress hormones such as cortisol (Wagner et al., 2010), adrenaline, and dopamine (Dzhambov, 2015; Hammoudi et al., 2013). These hormonal responses can reduce insulin sensitivity (Dzhambov, 2015; Münzel et al., 2021), contributing to hyperglycemia and metabolic and endocrine dysregulation, hallmarks of T2D (Pyko et al., 2019; Zuo et al., 2022). Epidemiological studies further support including elevated blood pressure and glucose levels (Grundy et al., 2004; Li et al., 2021).

Few studies worldwide have examined the effects of environmental noise exposure on the risk of T2D, and the strength and statistical significance of the reported associations vary across studies (Hu et al., 2023; Münzel et al., 2018; Sørensen et al., 2023; Zare Sakhvidi et al., 2018; Zuo et al., 2022).

A 2018 meta-analysis (Zare Sakhvidi et al., 2018) including five prospective cohorts (Clark et al., 2017; Eriksson et al., 2014; Eze et al., 2017; Ohlwein et al., 2017; Sørensen et al., 2013), two cross-sectional studies (Dzhambov and Dimitrova, 2016; Rhee et al., 2008), and two case-control studies (Song, 2010; Spankovich et al., 2017), reported increased T2D risk with long-term transportation noise exposure, especially for road traffic noise (RR = 1.07; 95 % CI: 1.02, 1.12) and aircraft noise (RR = 1.17; 95 % CI: 1.06, 1.29) per 10 dB(A) increase. Another review (Van Kempen et al., 2018), analyzing two cross-sectional studies (Selander et al., 2009; Van Poll et al., 2014) and two cohort studies (Eriksson et al., 2014; Sørensen et al., 2013), found similar results for road traffic noise in one cohort study (RR = 1.08; 95 % CI = 1.02–1.14), but no significant associations for railway or aircraft noise. Both summary reviews did not differentiate between study designs (e.g., cross-sectional, case-control or cohort), even though cohort studies provide stronger evidence for causal inference (Mann, 2003). They also highlighted methodological limitations, including small samples, short follow-up, limited exposure characterization, and lack of distinction between diabetes types.

In 2022, a meta-analysis (Liu et al., 2023) including 4,994,171 participants and 417,332 T2D cases examined ten prospective cohort studies conducted in Denmark (Jørgensen et al., 2019; Roswall et al., 2018; Sørensen et al., 2013; Thacher et al., 2021), Sweden (Eriksson et al., 2014), Canada (Clark et al., 2017; Shin et al., 2020), Switzerland

(Eze et al., 2017), Germany (Ohlwein et al., 2019) and Greece (Dimakopoulou et al., 2017). Seven studies examined road traffic noise (Dimakopoulou et al., 2017; Eze et al., 2017; Jørgensen et al., 2019; Ohlwein et al., 2019; Shin et al., 2020; Sørensen et al., 2013; Thacher et al., 2021), two railway noise (Roswall et al., 2018; Thacher et al., 2021), three aircraft noise (Dimakopoulou et al., 2017; Eriksson et al., 2014; Thacher et al., 2021) and one mixed noise (Clark et al., 2017). Four road traffic noise studies (Eze et al., 2017; Shin et al., 2020; Sørensen et al., 2013; Thacher et al., 2021), one railway noise study (Thacher et al., 2021), and the mixed noise study reported significant positive association with risk T2D risk. The meta-analysis found increased T2D risk for road traffic noise (RR = 1.06; 95 % CI: 1.03, 1.09 per 10 dB(A) increase in L_{den}) (Liu et al., 2023), with smaller relative risks for railway noise (RR = 1.02 (95 % CI: 1.01, 1.03) and aircraft noise, (RR = 1.01 (95 % CI: 1.00, 1.01)). Similar findings were reported for road traffic noise in a 2023 meta-analysis (RR = 1.07; 95 % CI: 1.05, 1.10 per 10 dB(A) increase in L_{den}) (Wu et al., 2023) and in a large Danish cohort (HR = 1.06; 95 % CI: 1.02, 1.11 per 10 dB(A) increase in L_{den} at the least exposed façade and HR = 1.03 (1.00, 1.06) at the most exposed façade) (Sørensen et al., 2023).

Overall, evidence on the role of transportation noise exposure in T2D development is growing, but few prospective cohort studies have examined railway and aircraft noise. This study aimed to assess the association between transportation noise exposure and T2D risk in a cohort of women from the Île-de-France and Auvergne-Rhône-Alpes regions (France).

2. Methods

2.1. Study population

The present study, named BROUHAHA (Association between noise exposure and risk of cardio-metabolic diseases (T2D, hypertension or cardiovascular diseases)) was based on the initial E3N prospective cohort of French women, which is part of a larger familial cohort called E3N-Generations.

The E3N study is an ongoing prospective cohort study that included 98,995 women aged 40 to 65 at enrollment in 1990, all affiliated with MGEN, a French national health insurance provider mainly covering teachers. Affiliation with MGEN was an inclusion criterion for participation in E3N, ensuring a well-defined and homogeneous source population (Clavel-Chapelon et al., 1997). Since 1990, follow-up questionnaires have been sent out approximately every 2 to 3 years, collecting information on women's health status, medical history and events, and lifestyle factors. The average response rate per questionnaire was 83 %, and the rate of loss to follow-up since 1990 has been <3 %.

Since January 2004, for each cohort member, the health insurance provider has supplied data including drug reimbursements and medical consultation claims. The French National Commission on Information Technology and Liberties (CNIL) approved the study protocol for Data Protection and Privacy, and all participants signed an informed consent form at inclusion.

Due to the availability of noise maps needed to estimate women's noise exposure, our study population was limited to E3N women living in the Auvergne-Rhône-Alpes (AuRA) and Île-de-France (IdF) regions (France) over the period 2000–2014.

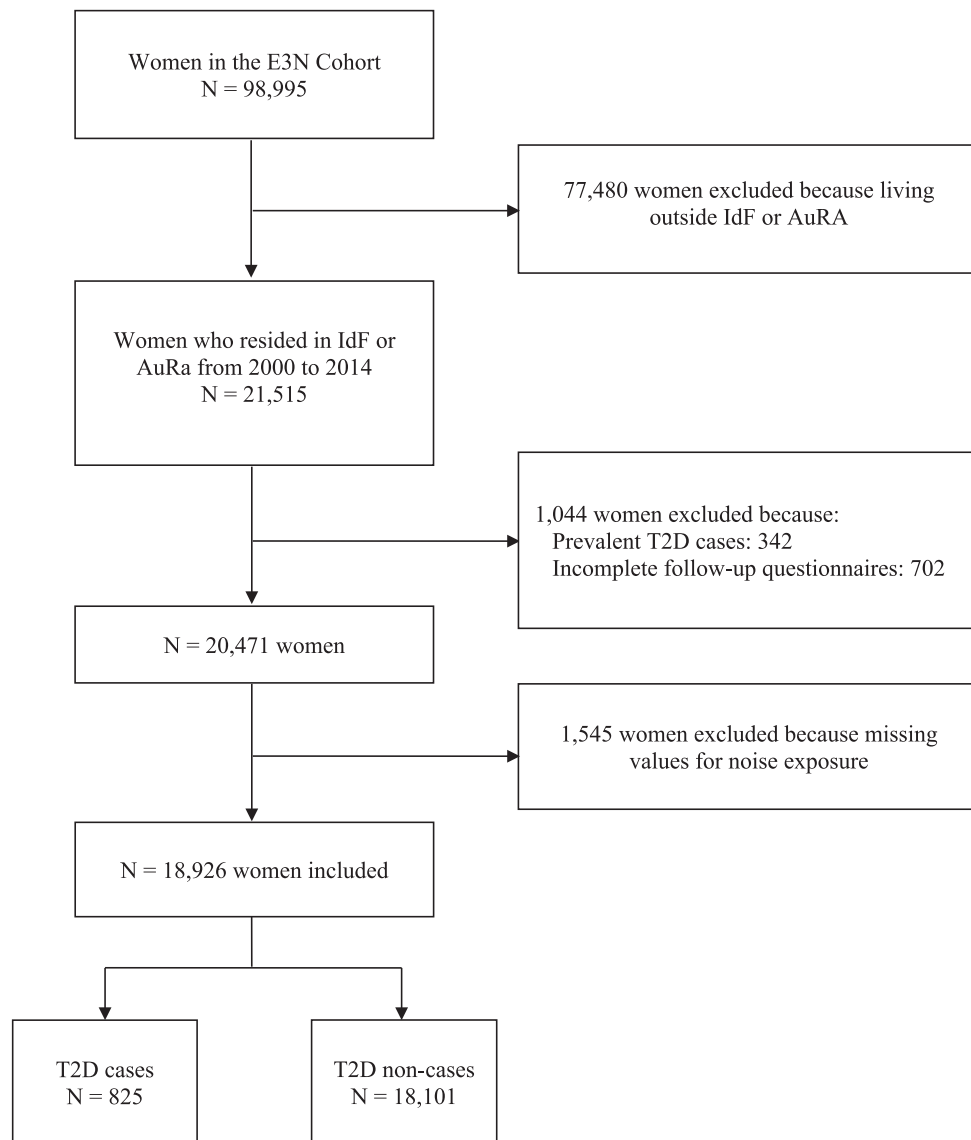
2.2. Transportation noise exposure assessment

The noise exposure of participants was estimated in three steps.

2.2.1. Geocoding of participants' addresses from 2000 to 2014

Geocoding of all participants' residential addresses was necessary to assess their exposure to transportation noise based on noise maps.

Addresses were obtained from the E3N follow-up questionnaires completed between 2000 and 2014 and geocoded by a trained



Abbreviations: T2D, Type 2 Diabetes; IdF, Île-de-France; AuRA, Auvergne Rhône-Alpes

Fig. 1. Flowchart of the inclusion of E3N participants in the study.

technician using BD Adresse® for ArcGIS (Faure et al., 2017). Approximately 89 % of the addresses were geocoded at the exact address level (i.e., building or house number), 10 % at the street or named place level, and the remaining 1 % at the city hall of the municipality of residence. For participants whose street number was missing but the street name was available, the address was positioned at the midpoint of the corresponding street segment. When only the municipality name was available, the address was geocoded at the town hall of the municipality of residence. This approach ensured that all participants were assigned a geographic coordinate with the highest possible spatial accuracy given the available address information.

2.2.2. Estimation of annual noise exposure, source by source, over the period 2000–2014

The exposure of participants to transportation noise (road traffic noise, railway noise and aircraft noise) was estimated by the two French noise observatories, Bruitparif and Acouité based on strategic noise maps (SNM) in accordance with European Directive 2002/49/EC. The SNMs provide modelled estimates of transportation noise levels derived

from average traffic and topographical data and represent annual average noise levels (Bruitparif, 2022). Participants' exposure to transportation noise was assessed at a height of 2 m from the most noise exposed façade of each residence.

For each participant, annual values of the L_{den} (Level day-evening-night) and L_n (Level night) noise indicators were calculated for each source of transportation noise (road traffic, railway and aircraft) over the study period (2000–2014), taking into account any change of address during the year.

These noise indicators correspond to those harmonized at the European level by the European Commission (Directive 2002/49/CE) and by the WHO for assessing the health effects of environmental noise. L_{den} is a noise level descriptor based on the equivalent continuous sound level over a 24-hour period, with penalties of +5 dB(A) for the evening (18.00–22.00) and +10 dB(A) for the night (22.00–6.00) in France. The L_n indicator represents the equivalent continuous sound level for the night period (22.00–6.00). These two indicators are used by the European Union for environmental noise management and are the most commonly employed in epidemiological studies on noise and health.

Noise indicators values were estimated at a spatial resolution of 5 m in IdF and 10 m in AuRA, expressed in dB(A) for all three noise sources, except for aircraft noise in AuRA, where values were available in 5 dB(A) intervals.

The SNMs have been produced since 2007 and updated every five years (European Directive 2002/49/EC) (European Commission, 2002). However, due to heterogeneity in earlier data, the first generation of maps used in this study was that produced in 2017 for IdF and 2018 for AuRA, corresponding to representative traffic data for 2012–2016 and 2012–2017, respectively. The 2017 SNMs were therefore used to assess participant's noise exposure for the year 2014.

Finally, noise exposure levels were back-extrapolated from 2013 to 2000 by applying a retrospective annual increase of 0.2 dB(A) for the

L_{den} (formula 3).

Multi-exposure according to the L_n indicator was not assessed in this study, as it did not offer additional spatial variability compared with L_{den} , and was therefore considered redundant for exposure modeling purposes.

Unlike the strategic noise maps publicly available, which typically apply a lower modeling limit of 55 dB(A) for L_{den} and 50 dB(A) for L_n , the noise maps used in this study provided continuous values without a minimum threshold, including low exposure levels. For consistency with other epidemiological studies (Sørensen et al., 2023; Thacher et al., 2021) and to account for background ambient noise, all values below 35 dB(A) were set to 35 dB(A) in the statistical analyses.

Formula 1

$$L_{den(aircraft\ equivalent\ road)} = \begin{cases} \frac{\left(3.1162 + \left(0.00098496 L_{den(air)}^2 \mp 0.139098 L_{den(air)} - 8.05911384\right)^{0.5}\right)}{0.0684}; & \text{for } L_{den(air)} > 45 \\ \frac{\left(2.05538 + \left(0.0008208 L_{den(air)}^2 \mp 0.1159152 L_{den(air)} - 5.9361076556\right)^{0.5}\right)}{0.057}; & \text{for } L_{den(air)} \leq 45 \end{cases}$$

L_{den} and L_n indicators related to road traffic noise and aircraft noise from Paris-Orly airport and 0.1 dB(A) per year for the L_{den} and L_n indicators related to aircraft noise from Paris-Charles-de-Gaulle and Paris-Le Bourget airports. For railway noise, exposure levels were assumed to

Formula 2

$$L_{den(rail\ equivalent\ road)} = \begin{cases} \frac{\left(3.1162 + \left(0.0038988 L_{den(rail)}^2 - 0.281175984 L_{den(rail)} + 4.13372212\right)^{0.5}\right)}{0.0684} & L_{den(rail)} > 54 \\ L_{den(rail)} & L_{den(rail)} \leq 54 \end{cases}$$

remain constant over this period. These annual variation coefficients (0.2 dB(A) and 0.1 dB(A)) were derived from analyses of 10-year observational data collected by noise monitoring stations in the Paris region (<https://rumeur.bruitparif.fr>) and in major cities of the AuRA region (<https://www.acoucite.org/>). They were further validated using traffic data from 2010 to 2012, incorporated into the 2012 round of SNMs.

2.2.3. Estimation of multiple annual exposure to noise, over the period 2000–2014

For each year of the study period, multiple exposure to transportation noise, that is, the combined exposure to road traffic, railway and aircraft noise, was assessed. Multi-exposure to noise is a complex topic, and there is currently no scientific consensus on the most appropriate assessment method.

We applied the ORHANE multi-exposure method (Observatoire Régional Harmonisé Auvergne-Rhône-Alpes des Nuisances Environnementales; CEREMA, 2022) based on the 2018 WHO environmental noise guidelines for the European region and the principle of equivalent annoyance. This approach relies on formulas (1) and (2) for calculating the road traffic equivalent L_{den} , as defined in Annex III of Directive 2002/49/EC (European Commission, 2002). For each participant and each year of the study period, this method provided a multi-exposure value to transportation noise, expressed as the road traffic equivalent

Formula 3

$$L_{den(multiexposition)} = 10 \log \left(10^{\frac{L_{den(rail\ equivalent\ road)}}{10}} + 10^{\frac{L_{den(aircraft\ equivalent\ road)}}{10}} + 10^{\frac{L_{den(road)}}{10}} \right)$$

2.3. Incidence of T2D

We identified cohort participants with T2D using the procedure described in detail elsewhere (Laouali et al., 2019). Briefly, the E3N cohort questionnaires collected information on the diagnosis of T2D.

From 2000 to 2003, a woman was considered a validated case of T2D if she reported at least one of the following: fasting plasma glucose ≥ 7.0 mmol/L or random glucose ≥ 11.1 mmol/L at diagnosis; use of a glucose-lowering medication; or most recent values of fasting glucose concentrations ≥ 7.0 mmol/L or HbA1c ≥ 53 mmol/mol (7.0 %) in the diabetes-specific questionnaire (Laouali et al., 2019; Vaduva et al., 2023).

From 2004 to 2014, a woman was considered a validated case of T2D if she reported and received at least two reimbursements for antidiabetic medication during the year, according to the drug reimbursement insurance database. The date of diagnosis was defined as the date of the first reimbursement (Laouali et al., 2019).

2.4. Statistical analyses

We considered several covariates based on current literature indicating associations with both noise exposure and T2D, and available in the follow-up questionnaires of E3N women cohort. The following covariates were time-dependent (assessed at baseline, and in 2002, 2005, 2008, 2011, and 2014): region of residence (two categories: IdF, AuRA); body mass index (BMI, kg/m^2 ; three categories (<25 , $25\text{--}29$, ≥ 30 kg/m^2); smoking status (never, former, current); physical activity (metabolic equivalent task-hour per week (MET-h/w) in quartiles); postmenopausal hormone therapy (MHT, two categories: never (no MHT use at any time), ever (MHT use reported at least once during follow-up) and air pollution (NO_2 or $\text{PM}_{2.5}$). Physical activity data were available only in 2002, 2008, and 2014.

Air pollution was included as an adjustment variable because transportation is a source of both noise and air pollution (Eminson et al., 2023; Héritier et al., 2019) and several studies have shown an association between exposure to air pollution and an increased risk of T2D (Liu et al., 2019). Annual exposures to nitrogen dioxide (NO_2 , $\mu\text{g/m}^3$) and fine particulate matter ($\text{PM}_{2.5}$, $\mu\text{g/m}^3$) were estimated for each participant's residential address over the study period using a land use regression model (LUR) and a chemistry transport model (CHIMERE), and categorized into quartiles. Detailed methods are described elsewhere (Amadou et al., 2020; Coudon et al., 2019).

Education level (three categories: secondary, 1–2-year university degree, ≥ 3 -year university degree) and family history of T2D (three categories: no family history, one relative, both parents) were assessed in 1990 and 2005, respectively. Adherence to a western dietary pattern (derived from principal component analysis and categorized in quartiles) was obtained from the 2005 dietary questionnaire. Alcohol consumption (three categories (0, 1–9, ≥ 10 g of ethanol/day) was collected in 1997.

Participant characteristics were described for the overall population and separately for T2D cases and non-cases, using means and standard deviations (SD) for continuous variables, and frequencies (%) for categorical variables. Differences between cases and non-cases were tested using Pearson's chi-squared test for categorical variables and student's *t*-test for continuous variables, after verifying the normality of the distributions.

We examined correlations between exposure levels to each noise source (for both L_{den} and L_n indicators) and air pollution (NO_2 or $\text{PM}_{2.5}$), using Pearson correlation coefficients.

To investigate the association between transportation noise exposure and T2D incidence, we estimated hazard ratios (HRs) with 95 % Confidence Intervals (CIs) per 10 dB(A) increase in road traffic noise, railway noise, aircraft noise, and multi-source noise, using Cox proportional hazards models with age as the time scale. Noise exposure was treated as time-dependent (assessed annually between 2000 and 2014). Because test for linearity of transportation noise exposure variables were not statistically significant (see Supplemental Material, Table S1), exposures were modeled as continuous variables.

For covariates with <5 % missing data, values were imputed using the median (for continuous variables) or the mode (for categorical variables). A separate "missing" category was created for covariates with >5 % missing data (smoking, alcohol consumption, BMI, MHT).

Four models were developed:

- Model 1: adjusted for age (as the time scale) and region of residence;
- Model 2: additionally adjusted for family history of diabetes, education level, smoking status, alcohol consumption, total physical activity, BMI, MHT, and western dietary pattern score;
- Model 3a: further adjusted for NO_2 exposure;
- and Model 3b: further adjusted for $\text{PM}_{2.5}$ exposure.

We also assessed potential effect modification by quartiles of NO_2 (and $\text{PM}_{2.5}$ respectively) by including an interaction term between noise

exposure and NO_2 exposure in model 3a (and $\text{PM}_{2.5}$ exposure in model 3b).

Because exposure estimates were retrospectively reconstructed and subject to uncertainty, residential mobility could introduce exposure misclassification. We therefore conducted sensitivity analyses, assessing the association between transportation noise exposure and T2D incidence among women who relocated at least once during follow-up and among those who never moved (see Supplemental material, Table S3).

All statistical analyses were performed using SAS software, version 9.4 (SAS Institute, Cary, NC, USA).

3. Results

Participants included in this study represents 19 % of the E3N cohort (Table S2). Fig. 1 shows the flowchart for inclusion. Of the 98,995 women initially enrolled in the E3N cohort, 77,480 were excluded because they resided outside the IdF or AuRA regions during the study period (2000–2014). Among the 21,515 women who lived in IdF or AuRA, we excluded prevalent T2D cases ($n = 342$) and women who did not complete one or more of the 2000, 2002, 2005, 2008, 2011 or 2014 questionnaires collecting information on health status and lifestyle factors ($n = 702$) and women with missing data on residential address history ($n = 1545$). Thus, 18,926 were finally included in the analyses. Among them, the overall incidence of T2D was 4.4 % during a median follow-up of 12.8 ± 3.2 years.

Characteristics of the study population overall and by T2D status (non-cases vs. cases) are presented in Table 1. The mean age at baseline was 59.3 ± 6.3 years in the overall population. Compared with non-diabetic women, those with T2D were older ($p < 0.0001$), less educated ($p < 0.0001$), and exposed to higher levels of NO_2 pollution ($p = 0.04$). The proportions of obese women ($p < 0.0001$), women who had never used MHT ($p = 0.01$) and women with a family history of diabetes ($p < 0.0001$) were also higher among diabetic participants. In total, 14,655 women (77 %) never moved between 2000 and 2014, among whom 640 developed T2D ($p = 0.92$).

We observed strong correlations between L_{den} and L_n for each noise source (road traffic, railway and aircraft), with Pearson correlation coefficients (ρ) ranging from 0.80 to 0.98. Similarly, multi-exposure L_{den} was highly correlated with both road traffic noise L_{den} and L_n ($\rho = 0.94$ and 0.95 respectively) (Fig. 2). Moderate correlations were found between road traffic noise L_{den} and NO_2 ($\rho = 0.56$) or $\text{PM}_{2.5}$ ($\rho = 0.51$) and between L_n and NO_2 ($\rho = 0.55$) or $\text{PM}_{2.5}$ ($\rho = 0.50$).

Associations between road traffic noise, railway noise, aircraft noise, or multi-exposure noise and the risk of T2D are shown in Table 2, according to the four models. We found a trend toward a positive association between exposure to road traffic noise, expressed as L_{den} or L_n , and incident T2D in model 1 (adjusted for age and region of residence), and a positive association in model 2 (further adjusted for family history of diabetes, education level, smoking status, alcohol consumption, total physical activity, BMI, postmenopausal hormone therapy and western dietary pattern score (Fig. 3). Exposure to railway noise, aircraft noise, or multi-exposure noise, expressed as L_{den} or L_n , was not associated with incident T2D in models 1 or 2.

In model 3a (further adjusted for NO_2), exposure to road traffic noise, expressed as L_{den} or L_n was associated with a higher risk of T2D (HR = 1.14; 95 % CI: 1.04, 1.26 and 1.21; 95 % CI: 1.07, 1.36, respectively, per 10-dB(A) increase in L_{den} or L_n). The HRs were slightly attenuated in model 3b (adjusted for $\text{PM}_{2.5}$ instead of NO_2), with HRs of 1.10 (1.04, 1.26) and 1.15 (1.03, 1.28), respectively, per 10-dB(A) increase in L_{den} or L_n (Fig. 4).

Multi-exposure noise, expressed as L_{den} , was associated with incident T2D in model 3a adjusted for NO_2 (HR = 1.13; 95 % CI: 1.01, 1.27 per 10-dB(A) increase in L_{den}) but not in model 3b adjusted for $\text{PM}_{2.5}$ (HR = 1.07; 95 % CI: 0.96, 1.20 per 10-dB(A) increase in L_{den}) (Fig. 4).

In models 3a and 3b, significant interactions were observed between road traffic noise (L_{den}) and air pollution ($p_{\text{interaction}}$ value = 0.02 for

Table 1
Characteristics of the study population at baseline (2000) and during the period 2000–2014.

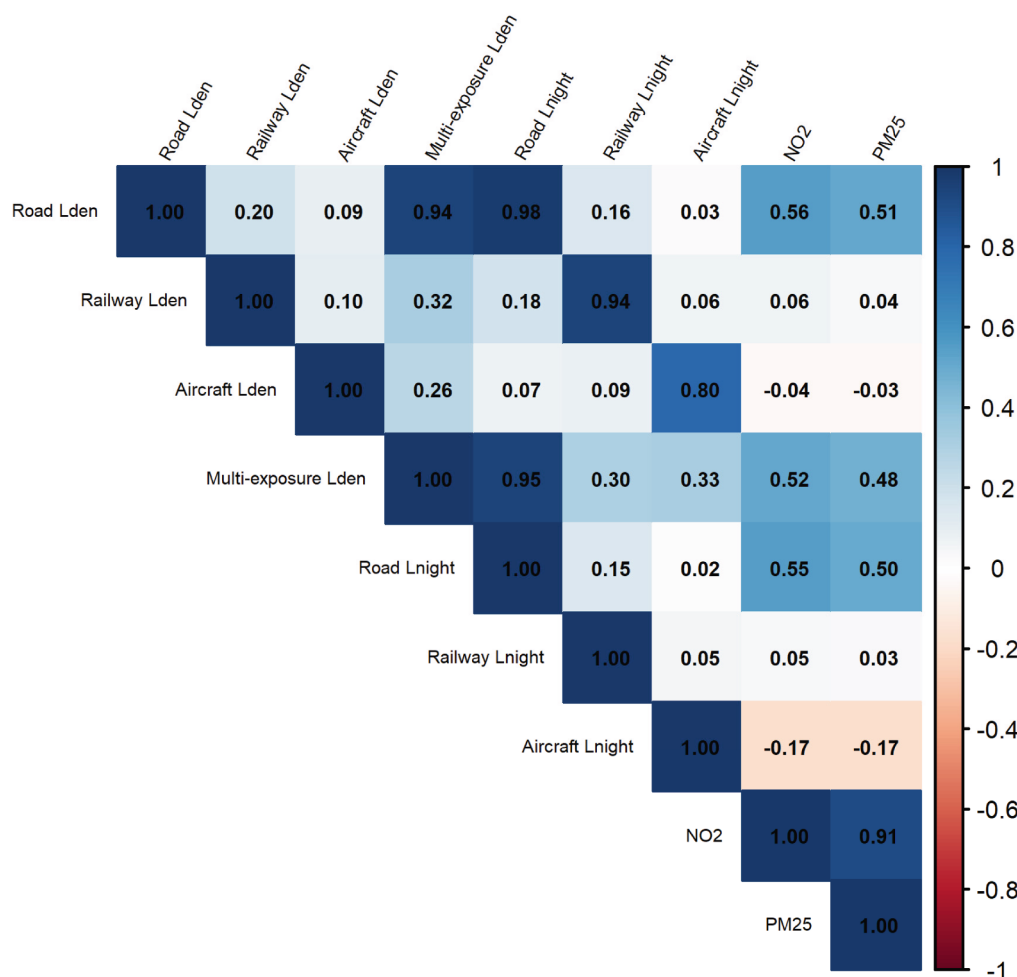
Characteristics ^a	All N = 18,926	T2D non-cases N = 18,101	T2D cases N = 825	p-value ^b
Follow-up (years), means \pm SD	12.8 \pm 3.2	13.1 \pm 3.0	7.5 \pm 4.0	<0.0001
Age (years), means \pm SD	59.3 \pm 6.3	59.2 \pm 6.3	60.7 \pm 6.6	<0.0001
Body Mass Index (kg/m ²), n (%)				<0.0001
<25	12,598 (66.6)	12,322 (68.1)	276 (33.5)	
25–29	3,852 (20.4)	3,580 (19.8)	272 (33.0)	
≥ 30	884 (4.7)	688 (3.8)	196 (23.8)	
Missing	1,592 (8.3)	1,511 (8.3)	81 (9.7)	
Smoking status, n (%)				0.78
Never	8,831 (46.7)	8,456 (46.7)	375 (45.5)	
Former	6,744 (35.6)	6,457 (35.7)	287 (34.7)	
Current	1,938 (10.2)	1,849 (10.2)	89 (10.7)	
Missing	1,413 (7.5)	1,339 (7.4)	74 (9.0)	
Alcohol consumption in 1997 (g/day), n (%)				0.09
0	7,694 (40.7)	7,343 (40.6)	351 (42.6)	
1–9	4,549 (24.0)	4,379 (24.2)	170 (20.6)	
>10	5,508 (29.1)	5,272 (29.1)	236 (28.6)	
Missing	1,175 (6.2)	1,107 (6.1)	68 (8.2)	
Education in 1990, n (%)				<0.0001
Secondary	1,739 (9.2)	1,607 (8.9)	132 (16.0)	
1-to-2-year university degree	7,764 (41.0)	7,407 (40.9)	357 (43.3)	
≥ 3 -year university degree	8,743 (46.2)	8,428 (46.6)	315 (38.2)	
Missing	680 (3.6)	659 (3.6)	21 (2.5)	
Total physical activity in 2002 (METs-h/week), Quartiles n (%)	74.5 (46.6)	74.5 (46.5)	75.3 (50.1)	0.65 0.56
Q1: < 41.8	4,293 (22.7)	4,101 (22.7)	192 (23.3)	
Q2: 41.8–66	4,249 (22.5)	4,085 (22.6)	164 (19.9)	
Q3: 66–97.2	4,337 (22.8)	4,158 (23.0)	179 (21.6)	
Q4: >97.2	4,296 (22.7)	4,119 (22.8)	177 (21.5)	
Missing	1,751 (9.3)	1,638 (8.9)	113 (13.6)	
Postmenopausal hormone therapy, n (%)				0.006
Never	7,185 (38.0)	6,835 (37.8)	350 (42.4)	
Ever	11,358 (60.0)	10,901 (60.2)	457 (55.4)	
Missing	383 (2.0)	365 (2.0)	18 (2.2)	
Family history of diabetes in 2005, n (%)				<0.0001
No family history	16,533 (87.4)	15,900 (87.8)	633 (76.7)	
One relative	1,486 (7.9)	1,360 (7.5)	126 (15.3)	
Both parents	59 (0.2)	53 (0.3)	6 (0.7)	
Missing	848 (4.5)	788 (4.4)	60 (7.3)	
Western dietary pattern score in 2005, Quartiles n (%)				0.13
Q1	3,759 (19.9)	3,627 (20.1)	132 (16.0)	
Q2	3,760 (19.9)	3,604 (19.9)	156 (18.9)	
Q3	3,760 (19.9)	3,617 (20.0)	143 (17.3)	
Q4	3,760 (19.9)	3,590 (19.8)	170 (20.6)	
Missing	3,887 (20.4)	3,663 (20.2)	224 (27.2)	
Region of residence, n (%)				0.24
Auvergne-Rhône-Alpes	8,523 (45.0)	8,135 (44.9)	388 (47.0)	
Île-de-France	10,403 (55.0)	9,966 (55.1)	437 (53.0)	
Exposure in terms of L _{den} (dB(A)) in exposed women (levels above 0 dB(A))				
Road traffic noise (n = 16,747), mean \pm SD (min–max)	61.3 \pm 7.3 (22.1–78.8)	61.3 \pm 7.3 (22.1–78.8)	61.5 \pm 7.4 (44.9–76.1)	0.47
Railway noise (n = 10,762), mean \pm SD (min–max)	43.2 \pm 9.6 (2.8–82.4)	43.3 \pm 9.6 (2.8–82.4)	42.8 \pm 9.3 (2.8–73.1)	0.35
Aircraft noise (n = 1,456), mean \pm SD (min–max)	51.4 \pm 5.0 (40.0–67.7)	51.3 \pm 5.0 (40.0–67.7)	51.3 \pm 4.9 (42.0–63.4)	0.98
Exposure in terms of L _n (dB(A)) in exposed women (levels above 0 dB(A))				
Road traffic noise (n = 16,740), mean \pm SD (min–max)	52.2 \pm 6.9 (12.7–69.5)	52.2 \pm 6.9 (12.7–69.5)	52.4 \pm 6.9 (35.4–69.0)	0.47
Railway noise (n = 7,110), mean \pm SD (min–max)	41.9 \pm 7.8 (30.0–76.0)	41.9 \pm 7.8 (30.0–76.0)	41.1 \pm 7.5 (30.2–66.6)	0.06
Aircraft noise (n = 690) ^c , mean \pm SD (min–max)	46.6 \pm 3.2 (41.4–56.6)	46.6 \pm 3.2 (41.4–56.6)	46.7 \pm 2.8 (41.1–55.7)	0.87
Multi-exposure in terms of L _{den} (dB(A)), mean \pm SD (min–max)	60.5 \pm 8.6 (45.0–85.6)	60.5 \pm 8.6 (45.0–85.6)	60.3 \pm 8.7 (46.9–77.5)	0.58
NO ₂ (µg/m ³), min–max Quartiles n (%)	16.2–52.4	16.2–52.4	16.3–52.4	
Q1: <27.4	4,730 (25.0)	4,505 (24.9)	225 (27.3)	0.12
Q2: 27.4–33.9	4,702 (24.8)	4,504 (24.9)	198 (24.0)	
Q3: 33.9–43.7	4,406 (23.3)	4,201 (23.2)	205 (24.8)	
Q4: >43.7	5,088 (26.9)	4,891 (27.0)	197 (23.9)	
PM _{2.5} (µg/m ³), min–max Quartiles n (%)	5.8–25.1	5.8–25.1	6.4–25.1	
Q1: <12.2	4,694 (24.8)	4,482 (24.8)	212 (25.7)	0.49
Q2: 12.2–15.6	4,749 (25.1)	4,542 (25.0)	207 (25.1)	
Q3: 15.6–20.3	4,646 (24.5)	4,433 (24.5)	213 (25.8)	
Q4: >20.3	4,837 (25.6)	4,644 (25.7)	193 (23.4)	

Abbreviations: T2D, Type 2 Diabetes; L_{den}, level day-evening-night; L_n, level night; NO₂, nitrogen dioxide; PM_{2.5}, particulate matter with aerodynamic diameter less than or equal to 2.5 µm.

^a Some characteristics are from baseline (2000 questionnaire), while others (Alcohol consumption, education, Total physical activity, Family history of diabetes and Western dietary pattern score) were taken from the closest available questionnaire when not available at baseline.

^b p-values were obtained using the Pearson chi² test for categorical variables and the student's t-test for continuous variables.

^c Exposed to aircraft noise; L_n was not assessed for AuRA (n = 8523) because no aircraft L_n data were available for the 2018 SNMs.



Abbreviations: L_{den}, level day-evening-night; L_n, level night; NO₂, nitrogen dioxide; PM_{2.5}, particulate matter with aerodynamic diameter less than or equal to 2.5 µm

Fig. 2. Correlation matrix between transportation noise and air pollutants (annual mean values) at baseline (2000). Abbreviations: L_{den}, level day-evening-night; L_n, level night; NO₂, nitrogen dioxide; PM_{2.5}, particulate matter with aerodynamic diameter less than or equal to 2.5 µm.

NO₂ and 0.007 for PM_{2.5}, respectively). A 10-dB(A) increase in road traffic noise (L_{den}) was associated with a higher risk of T2D in the highest quartile of NO₂ exposure (HR = 1.62; 95 % CI: 1.12, 2.19) and in the highest quartile of PM_{2.5} exposure (HR = 1.60; 95 % CI: 1.20, 2.14) but not in the 1st, 2nd or 3rd quartiles (Table 3).

Sensitivity analyses using fully adjusted models 3a and 3b showed that road traffic noise (L_{den} or L_n) and multi-exposure noise (L_{den}) were associated with incident T2D among women who never moved during the follow-up period, but not among those who moved at least once, in model 3a adjusted for NO₂ (Table S3). HRs were slightly attenuated and not significant in model 3b adjusted for PM_{2.5}. However, the number of women who never moved was more than three times higher than that of women who moved at least once.

4. Discussion

This study evaluated the association between exposure to road traffic, railway, aircraft, and multi-source transportation noise and the incidence of T2D in a cohort of women living in IdF and AuRA, the two most densely populated regions of France (Insee, 2024). An increase in road traffic noise was associated with a higher incidence of T2D, even after adjusting for air pollution (NO₂ or PM_{2.5}), whereas no significant

association was observed for railway noise or aircraft noise.

Our findings for road traffic noise are consistent with those of recent meta-analyses (Liu et al., 2023; Wu et al., 2023) and the large Danish cohort study by Sørensen and al (Sørensen et al., 2023), which reported a positive association with T2D. Our higher estimates could be explained by the fact that our cohort included only women. In Sørensen et al. (2023), the highest HR for road traffic noise (L_{den} at the most exposed façade) was observed among women compared with men, suggesting that they may be more sensitive to noise exposure (Sørensen et al., 2023).

Only two cohort studies have examined the night-time indicator (L_n) in relation to T2D. One large Canadian study reported a significant association (Shin et al., 2020), while a smaller German study found a positive but non-significant trend (Ohlwein et al., 2019). In our study, associations tended to be stronger for L_n than for L_{den}, suggesting that sleep disturbance may contribute to the metabolic effects of noise exposure (Pyko et al., 2019; Zuo et al., 2022).

We found no significant association for railway or aircraft noise. This aligns with most previous cohort studies, except for Thacher et al. (2021), who observed a weak but significant effect of railway or aircraft noise in a very large Danish population. Differences in sample size and lower exposure levels in our population may partly explain the lack of

Table 2

Hazard Ratios (95 % CI) estimated by Cox models for the association between transportation noise levels in terms of L_{den} or L_n and the risk of T2D in the E3N cohort (N = 18,926; 2000–2014).

Exposure to noise	Model 1 HR (95 % CI)	Model 2 HR (95 % CI)	Model 3a HR (95 % CI)	Model 3b HR (95 % CI)
L_{den} (per a 10 dB(A) increase)				
Road traffic noise	0.99 (0.92, 1.06)	1.08 (1.00, 1.18)	1.14 (1.04, 1.26)	1.10 (1.01, 1.20)
Railway noise	0.95 (0.86, 1.04)	0.94 (0.85, 1.04)	0.94 (0.85, 1.04)	0.94 (0.85, 1.04)
Aircraft noise	1.02 (0.86, 1.21)	0.96 (0.78, 1.17)	0.92 (0.75, 1.13)	0.94 (0.77, 1.16)
Multi-exposure noise	0.96 (0.87, 1.05)	1.06 (0.95, 1.18)	1.13 (1.01, 1.27)	1.07 (0.96, 1.20)
L_n (per a 10 dB(A) increase)				
Road traffic noise	1.00 (0.91, 1.09)	1.12 (1.01, 1.25)	1.21 (1.07, 1.36)	1.15 (1.03, 1.28)
Railway noise	0.90 (0.78, 1.03)	0.89 (0.76, 1.03)	0.88 (0.76, 1.03)	0.88 (0.76, 1.03)
Aircraft noise	1.12 (0.79, 1.58)	1.00 (0.67, 1.49)	0.97 (0.64, 1.47)	0.98 (0.65, 1.48)

Abbreviations: L_{den} , level day-evening-night; L_n , level night; HR, hazard ratio.

M1: Adjustment for age (as the time-scale) and region of residence.

M2: M1 + family history of diabetes, education level smoking status, alcohol consumption, total physical activity, BMI, postmenopausal hormone therapy, and western dietary pattern score.

M3a: M2 + NO₂ pollution

M3b: M2 + PM_{2.5} pollution

association. In our cohort, a smaller proportion of participants exceeded WHO threshold levels for railway and aircraft noise than for road traffic noise. Average railway noise levels were below WHO guideline thresholds, while exposure to aircraft noise concerned fewer than 10 % of participants.

Among the 18,926 participants, 57 % (n = 10,762) and 38 % (n = 7110) were exposed to railway noise in terms of L_{den} and L_n respectively, including 465 and 319 T2D cases. For aircraft noise, data were missing for 45 % of participants, and only 8 % (n = 1456) and 4 % (n = 690) were exposed to L_{den} and L_n , respectively, including 60 and 28 T2D cases.

In our cohort, exposure to road traffic noise was the most common, with 77 % and 76 % of participants exceeding the WHO thresholds for L_{den} and L_n , respectively. In contrast, only a small proportion of women were exposed to railway (9 % and 13 %) or aircraft noise (7 % and 4 %) levels above the recommended limits, reflecting the predominance of road traffic noise in our study areas. This imbalance in exposure distribution may partly explain why associations were observed for road traffic noise but not for railway or aircraft noise, as statistical power was lower for these latter sources.

Regarding multi-exposure to transportation noise, we observed a significant association in the model adjusted for NO₂ (model 3a) but not for PM_{2.5} (model 3b). This is consistent with Clark and al. (2017) in Canada, who also reported a positive association between combined noise exposure and T2D, mainly driven by road traffic noise (Clark et al., 2017). In our study, multi-exposure was strongly correlated with road traffic noise (Pearson correlation coefficient (ρ) = 0.94), supporting this interpretation.

We observed that in the full model adjusted for NO₂ (model 3a; HR = 1.14; 95 % CI: 1.01, 1.27), the HR was higher than in model 2 (HR = 1.08; 95 % CI: 1.00, 1.18), even though the confidence intervals overlapped. This result could be due to the fact that our population was limited to participants living in two highly urbanized French regions (Île-de-France and Auvergne Rhône-Alpes), both strongly exposed to air pollution (min–max NO₂ = 16.3–52.4 µg/m³, median = 33.9), whereas according to the WHO, the recommended limit value not to be exceeded is 10 µg/m³ (World Health Organization Regional Office for Europe, 2013).

In our study, the association with road traffic noise in terms of L_n was stronger than in terms of L_{den} (HR = 1.21; 95 % CI: 1.07, 1.36 vs HR = 1.14; 95 % CI: 1.04, 1.26 when considering NO₂) suggesting that sleep disturbance could be a key mechanism underlying the harmful effects of noise on T2D. Exposure to transportation noise has been associated with sleep disturbances (Sharma et al., 2022), which induce persistent

abnormalities in glucose regulation and may promote the development of T2D (Pyko et al., 2019; Zuo et al., 2022).

When our results were restricted to participants exposed to the highest quartiles of NO₂ and PM_{2.5}, we found an association ($p_{interaction}$ value = 0.02 and 0.007 respectively), but not in the 1st, 2nd and 3rd quartiles, indicating that the association between road traffic noise (L_{den}) and incidence of T2D may be limited to populations with high exposure to air pollution (NO₂ and PM_{2.5}). Jorgenson et al. (2019) also analyzed this association by NO₂ and PM_{2.5} exposure subgroup and observed no statistically significant difference between strata, with interaction p-values > 0.29. However, the number of incident cases in their subgroups was not homogeneous. Moreover, the level of air pollution exposure in their study population was much lower than that in ours (low: <7 µg/m³, high: >16) vs (quartile 1: <27 µg/m³, quartile 4: >44) for NO₂ and (low: <18 µg/m³, high: >22) vs (quartile 1: <12 µg/m³, quartile 4: >20) for PM_{2.5}) (Jorgenson et al., 2019).

This study presents some limitations. Transportation noise exposures were estimated using modeled data, which may introduce some degree of exposure misclassification. However, since the noise model is unlikely to differ between T2D cases and non-cases, such misclassification would likely be non-differential. In most cases, this would bias relative risk estimates toward the null value (Sørensen et al., 2013). Although the noise maps used in this study provided continuous values, we set a lower limit of 35 dB(A) for all indicators. This approach is commonly used in environmental noise epidemiology but may slightly underestimate exposure contrasts in very quiet residential areas. However, such areas are relatively uncommon in our study regions, which are among the most urbanized in France.

In addition, no information was available on the bedroom orientation; neighborhood noise; individual noise protection measures (e.g., use of earplugs), or the average amount of time participants spent at home. For instance, Oftedal et al. (2015) observed a trend toward stronger associations between noise exposure and obesity markers among participants whose bedroom facade faced the road, compared with those whose bedrooms were oriented away from traffic (Oftedal et al., 2015).

Another limitation is the relatively low proportion of participants exposed to railway and aircraft noise in our study. While 88 % of participants were exposed to road traffic noise (based on both L_{den} and L_n), only 43 % and 38 % were exposed to railway noise in terms of L_{den} and L_n respectively. Exposure to aircraft noise was particularly low, with only 8 % of participants exposed in term of L_{den} and 4 % in term of L_n . The limited number of participants exposed to specific noise sources, particularly aircraft noise, along with the relatively small number of

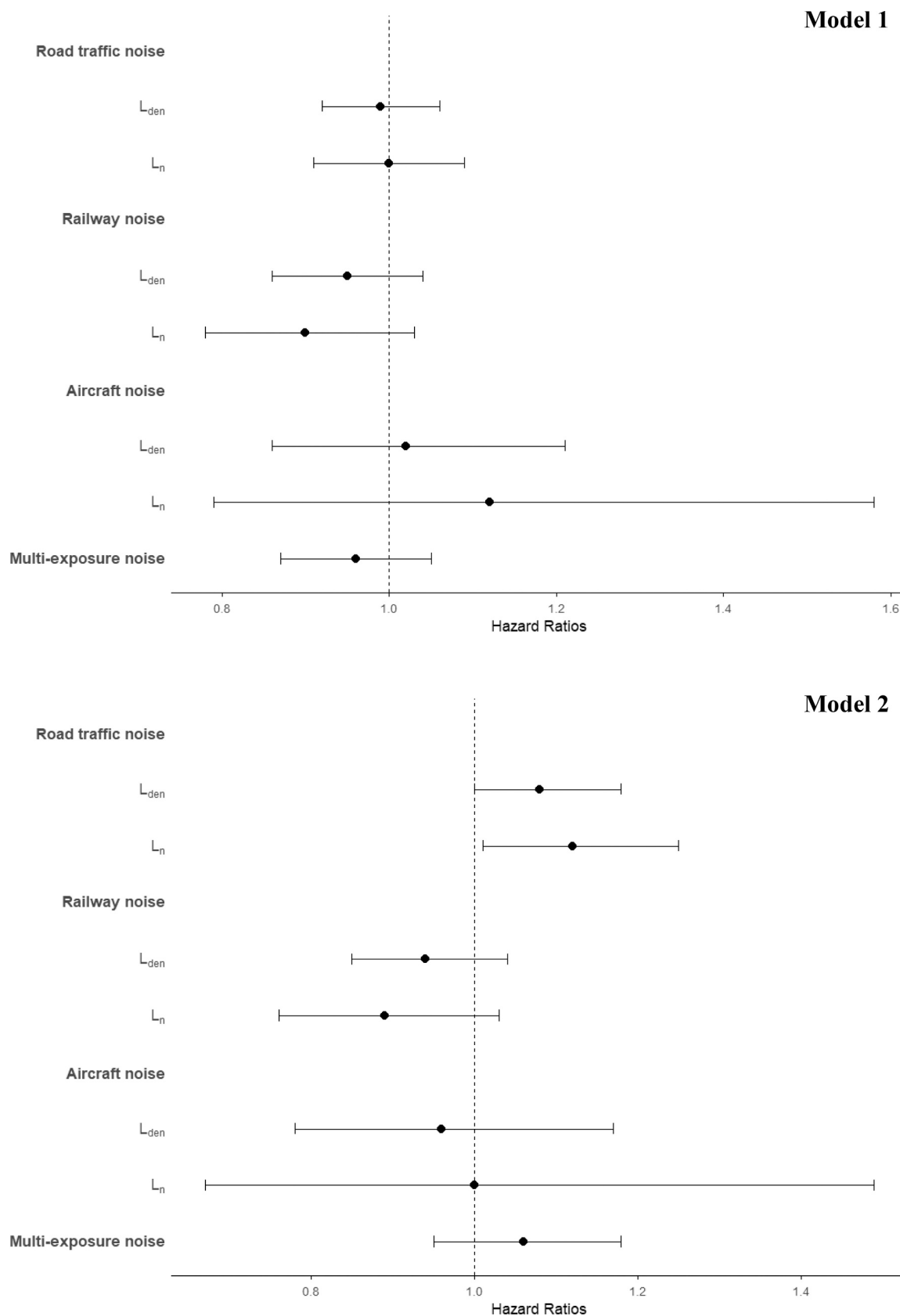


Fig. 3. Hazards Ratios (95 % confidence intervals) for the association between transportation noise exposure (L_{den} and L_n) and the risk of T2D in the E3N cohort. Estimates are presented for models 1 and 2. Model 1: Adjustment for age (as the time-scale) and region of residence. Model 2: Model 1 + family history of diabetes, education level smoking, alcohol consumption, total physical activity, BMI, postmenopausal hormone therapy, and western dietary pattern score.

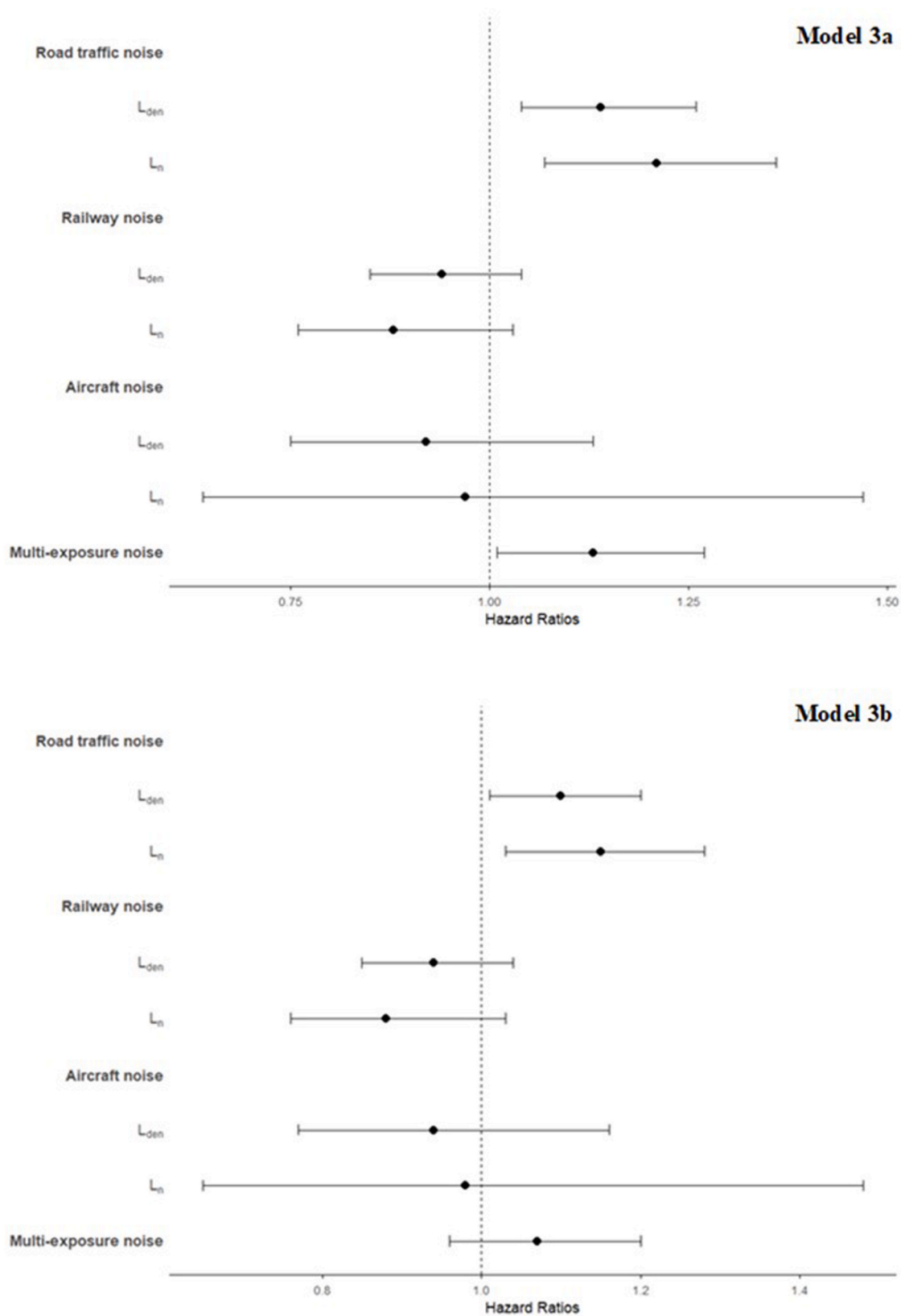


Fig. 4. Hazards Ratios (95 % confidence intervals) for the association between transportation noise exposure (Lden and Ln) and the risk of T2D in the E3N cohort. Estimates are presented for models 3a and 3b. Model 3a: Model 2 + NO₂ pollution. Model 3b: Model 2 + PM_{2.5} pollution.

Table 3Modification of the association between road traffic noise (per 10 dB(A)) in terms of L_{den} and the risk of T2D by NO_2 and $PM_{2.5}$ respectively.

	HR (95 % CI)	Cases (n)	p-Interaction
Exposure to NO_2 ($\mu g/m^3$) ^a			0.007
Q1: <27.4	1.10 (0.96, 1.27)	225	
Q2: 27.4–33.9	0.89 (0.74, 1.07)	198	
Q3: 33.9–43.7	1.21 (0.94, 1.58)	205	
Q4: >43.7	1.62 (1.12, 2.19)	197	
Exposure to $PM_{2.5}$ ($\mu g/m^3$) ^b			0.02
Q1: <12.2	1.05 (0.92, 1.19)	212	
Q2: 12.2–15.6	1.06 (0.87, 1.07)	207	
Q3: 15.6–20.3	0.92 (0.75, 1.14)	213	
Q4: >20.3	1.60 (1.20, 2.14)	193	

Abbreviations: NO_2 , nitrogen dioxide; $PM_{2.5}$, particulate matter with aerodynamic diameter less than or equal to 2.5 μm ; Q1, quartile 1; HR, hazard ratio.^a Adjustment for age (as the time-scale), region of residence, family history of diabetes, education level smoking status, alcohol consumption, total physical activity, BMI, postmenopausal hormone therapy, western dietary pattern score and NO_2 pollution.^b Adjustment for age (as the time-scale), region of residence, family history of diabetes, education level smoking status, alcohol consumption, total physical activity, BMI, postmenopausal hormone therapy, western dietary pattern score and $PM_{2.5}$ pollution.

incident cases among them, likely contributed to the variability observed in hazard ratio estimates across model specifications, especially for road traffic noise. This limitation underscores the need for further studies involving larger populations exposed to transportation noise in order to produce more stable effect estimates and enhance the robustness of the findings.

Our study has several strengths. It is the first epidemiological study in France to investigate the association between transportation noise exposure and the risk of T2D. It relies on a relatively large sample (18,926 participants, including 825 T2D cases) drawn from a well-established prospective cohort. The level of evidence provided by a prospective design is higher than that of cross-sectional or case-control studies (Mann, 2003). Attrition bias, often a major issue in longitudinal research, was minimal in the E3N cohort, where the average response rate to follow-up questionnaires remained around 83 %, and total loss to follow-up since 1990 is below 3 %.

The E3N data were regularly updated, and the availability of a comprehensive dataset enabled adjustment for numerous individual variables throughout follow-up, including air pollution (NO_2 and $PM_{2.5}$) which has been associated with T2D risk (Kutlar Joss et al., 2023; Liu et al., 2019), thereby limiting potential confounding. Incident T2D cases were validated using a well-defined validation algorithm, allowing differentiation from type 1 diabetes and minimizing information bias.

Most previous studies have assessed exposure to a single source of transportation noise, whereas few have considered the three main sources (road traffic, railway, and aircraft noise) simultaneously. Only three cohort studies (Dimakopoulou et al., 2017; Ohlwein et al., 2019; Shin et al., 2020) examined the night-time noise indicator (L_n). This distinction between total-day and night-time exposure is particularly relevant, as experimental studies have shown that night-time noise more strongly disrupts sleep, which may in turn increase T2D risk (Basner et al., 2011; Brink et al., 2008).

Our findings suggest that long-term exposure to road traffic noise may contribute to the development of T2D, particularly among populations simultaneously exposed to high levels of air pollution. This highlights the need for public health policies aimed at reducing urban environmental exposures through noise and air quality management strategies. Beyond regulatory thresholds, urban planning interventions, such as improving building insulation, and designing quieter transport infrastructures, could help mitigate the combined burden of these environmental risk factors. Further research should aim to clarify the underlying biological mechanisms linking noise exposure to metabolic outcomes, particularly the roles of sleep disturbance and chronic stress pathways. Studies integrating individual-level behavioral and physiological data (e.g., sleep duration, stress biomarkers) would be especially valuable. Additionally, future research should consider the impact of

multi-exposure scenarios combining different noise sources and pollutants, as well as potential sex-specific susceptibilities.

5. Conclusion

This study is the first conducted in France to investigate the association between transportation noise exposure and the incidence of T2D. Our findings are consistent with most previous cohort studies carried out in other countries, showing an increased risk of T2D with higher exposure to road traffic noise, but no association with railway or aircraft noise. These results were observed among middle-aged women living in Île-de-France and Auvergne-Rhône-Alpes, the two most densely populated regions of France. Moreover, the association between road traffic noise (L_{den}) and T2D incidence may be restricted to populations with high exposure to air pollution (NO_2 and $PM_{2.5}$). Further studies are needed to better understand the combined effects of transportation noise and air pollution, particularly for railway and aircraft noise exposures.

6. Finding sources

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Credit authorship contribution statement

Anita Houeto: Writing – review & editing, Writing – original draft, Visualization, Validation, Supervision, Methodology, Investigation. **Anne-Sophie Evrard:** Writing – review & editing, Validation, Methodology, Investigation, Conceptualization. **Claire Perrin:** Writing – review & editing, Validation, Software, Methodology, Investigation, Formal analysis, Data curation. **Fanny Artaud:** Writing – review & editing, Validation, Methodology, Conceptualization. **Adélie Boileau:** Writing – review & editing, Validation, Methodology. **Mathieu Hellot:** Writing – review & editing, Validation, Methodology. **Pierre Jamard:** Writing – review & editing, Validation, Methodology. **Fanny Mietlicki:** Writing – review & editing, Validation, Methodology, Conceptualization. **Céline Domergue:** Writing – review & editing, Validation, Methodology.

Valérie Janillon: Writing – review & editing, Validation, Methodology, Conceptualization. **Bruno Vincent:** Writing – review & editing, Validation, Methodology. **Thomas Coudon:** Writing – review & editing, Validation, Methodology. **Lény Grassot:** Writing – review & editing, Validation, Methodology. **Delphine Praud:** Writing – review & editing, Validation, Methodology. **Guy Fagherazzi:** Writing – review & editing, Validation, Methodology, Conceptualization. **Gianluca Severi:** Writing – review & editing, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization. **Élodie Faure:** Writing – review & editing, Visualization, Validation, Supervision, Project administration, Methodology, Investigation, Funding acquisition, Conceptualization.

Ethics approval and consent to participate

The study was approved by the French National Commission for Data Protection and Privacy ([ClinicalTrials.gov](https://clinicaltrials.gov) identifier: NCT03285230). All participants provided written informed consent.

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.2025.109972>.

Data availability

Data will be made available on request.

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