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Residential exposure to road and railway traffic noise and incidence of dementia: The UK Biobank cohort study

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ABSTRACT

Background: Evidence linking noise pollution and brain health, particularly at mid-to-late life, remains scarce. We investigated the associations between long-term exposure to road and railway traffic noise and incident dementia in the UK Biobank cohort.

Methods: Participants with available data for dementia incidence and linked traffic noise exposure during follow-up were included. Residential road traffic noise from both minor and major roads were calculated in accordance with CNOSSOS-EU framework; railway noise estimates were created by Extrium, with the raster datasets representing noise contributions from major railway corridors. Cox regression was used to quantify the associations between transport noise and incident dementia (incl. its subtypes), adjusting for potential confounders, air pollution and greenness.

Results: Of the full cohort (n = 502,416), 7668 participants had incident dementia during a median follow-up period of 9.67 years. No associations were found between all cause dementia incidence and road or railway noise. However, a 10-dB (dB) higher exposure in annual mean road traffic noise (L_{den}) was significantly associated with incident Alzheimer's disease (HR:1.150, 95 % CI: 1.022–1.294). The effect estimate was slightly higher when participants were exposed to night-time road noise above 45 dB (HR:1.188, 95 % CI:1.012–1.394) and this was mediated by the cardiovascular health profile. Railway noise (L_{den}) was significantly associated with incident Parkinson's disease related dementia (HR:1.042, 95 % CI:1.005–1.081), however, the effect estimate was slightly reduced after further adjustment of air pollution and residential greenness (HR:1.037, 95 % CI:0.998–1.077).

Conclusion: Distinct associations between different traffic noise exposures and incident dementia subtypes were found in this large UK prospective cohort study.

1. Introduction

There are currently over 50 million people worldwide living with dementia with this number being predicted to grow over the next decade (Nichols et al., 2022). In the United Kingdom (UK) alone, over 850,000 people currently live with dementia, with an estimated 20,000 new cases being reported each year (Wittenberg et al., 2019). The cause of this trend is in part attributed to a growing ageing population (Lewis et al., 2014), amongst many other risk factors. Prevention-oriented research remains crucial to enable and nourish the ageing brain and represents a formidable challenge to healthy ageing, an area of critical importance within society. A recent *Lancet*-commissioned review has identified 12 modifiable risk factors for dementia; air pollution was one of the three newly added risk factors based on the emerging evidence

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and was the only physical environmental exposure identified (Livingston et al., 2020).

A major gap in most previous air pollution-dementia studies is the lack of consideration of co-existing traffic noise pollution. In fact, both exposures share some overlapping biological pathways underlying their associations with brain health. For example, oxidative stress, systemic inflammation, and vascular dysfunction can be induced by both air pollution and traffic noise (Münzel et al., 2017, 2018). Animal studies have suggested that noise can accelerate the risk of cognitive impairment via promoting stress responses in various brain regions (Jafari et al., 2020). In humans, a novel study has found that chronic noise exposure can increase amygdalar activities in processing stress responses (Osborne et al., 2020).

Whilst a role of traffic noise pollution in cognitive development in children has been demonstrated (Clark and Paunovic, 2018), its role as a potentially modifiable risk factor in incident dementia at the other end of life has been far less explored. In older adults there is growing evidence showing an association between traffic noise pollution and cognitive impairment (Tzivian et al., 2016), which is generally considered a precursor to dementia. However, epidemiological evidence to date linking traffic noise and incident dementia, particularly at mid-to-late life, remains scarce and inconsistent (Andersson et al., 2018; Yu et al., 2023; Cantuaria et al., 2021; Carey et al., 2018; Lomme et al., 2023; Tuffier et al., 2024; Yuchi et al., 2020). This study aims to investigate the associations of both road and railway traffic noise exposures with incident dementia in the UK Biobank cohort, considering a range of individual covariates as well as environmental exposures such as air pollution and residential surrounding greenness.

2. Methods

2.1. Study cohort

UK Biobank enrolled over a half-million adults, aged 37–73 years, between 2006 and 2010 (Sudlow et al., 2015). Individuals who were registered with the National Health Service (NHS) and who resided within 25 miles of one of the 22 study assessment centres were invited to take part in the study. At baseline, participants provided detailed personal, demographic, and health-related information via a touchscreen questionnaire and nurse interview. Individuals' current and historic health records are retrieved from the NHS central repository as well as various national health registries. These data are linked centrally by UK Biobank to each participant following baseline assessment. Although there was a relatively low response rate (5.5 %), risk factor associations in UK Biobank are likely generalisable (Batty et al., 2020). All participants provided written consent and ethical approval was obtained from the Northwest Multi-Centre Research Ethical Committee and Patient Information Advisory Group.

2.2. Dementia outcomes

Since baseline recruitment, incident dementia and other clinical information pertaining to each study participant were ascertained via record linkages of hospital inpatient data, primary care data, mortality data, and baseline data on self-reported health conditions. Participants were followed until death or end of follow-up (December 31, 2021), whichever came first (event/censoring). Incident dementias were mapped by UK Biobank to the International Classification of Disease chapters (ICD10 and ICD9) (Supplementary Table 1a). For the present study, participants who were diagnosed with or who reported as having Alzheimer's disease, vascular dementia and Parkinson's disease related dementia during follow-up were included in the final analyses. Additionally, an all-cause dementia outcome category was generated using algorithmically defined outcome (ADO) fields conceived by UKB outcome adjudication group. This was done in conjunction with clinical experts, with positive predictive value (PPV, the proportion of cases identified that were true positives) being 82.5 % in validation studies (Wilkinson et al., 2019) (Supplementary table 1b).

2.3. Transport noise exposure

The annual mean road noise exposure estimates at address level were calculated in a simplified model of the 'Common Noise Assessment Methods' (CNOSSOS) (Kephalopoulos et al., 2012). The CNOSSOS were developed under the European Commission Noise Directive (2002/49/EC) and have been validated in epidemiological studies with relatively good performance on exposure ranking (Spearman ratio: 0.75) (Morley et al., 2015). For UK Biobank, these estimates were modelled using Annual Average Daily Traffic (AADT) counts and traffic speeds across the UK road-network, together with information relating to the surface roughness of land cover, building heights, wind profiles and average temperatures, as described in published protocols (Morley et al., 2015; Gulliver et al., 2015). Annual mean road traffic noise estimates for year 2013 were intersected with UKB participants baseline addresses to assign a noise exposure estimate from all major and minor road sources within 500 m. For railway noise, participant addresses were intersected with modelled railway noise estimates, created by Extrium on behalf of the UK Department for Environment, Food and Rural Affairs (DEFRA) (DEFRA, 2015). The underlying raster dataset represented the noise contributions from major rail corridors for the calendar year of September 2010 to September 2011, at a 10 m resolution (Fig. 1). For both transport noise sources, we used L_{den} and L_{night} noise metrics. L_{den} exposure represented the annual A-weighted equivalent noise level over 24 h, with a penalty of +10 dB(A) for night-time noise (22:00–07:00) and +5 dB(A) for evening noise (19:00–23:00). L_{night} represents an A-weighted equivalent noise level from 23:00 to 07:00 h.

2.4. Covariates

The covariates were selected *a priori* based on potential or established risk factors associated with dementia and other neurodegenerative diseases (Livingston et al., 2020). These factors included age, sex, education (with and without a university degree or equivalent), household income before tax (≤£31,000 vs >£31,000), current employment status at baseline (economically active (paid employment) or not), area-level Townsend deprivation index, cardiovascular risk score, air pollution (particulate matter with a diameter≤2.5 μm (PM2.5) and nitrogen dioxide (NO2)) and residential surrounding greenness. The cardiovascular risk score (CRS) was calculated as sum of seven health metrics (score range 0–14) and categorised into poor (score 0–6), intermediate (score 7–10), and optimal (score 11–14) cardiovascular health (Lloyd-Jones et al., 2010). These included four behavioural metrics (smoking, diet, physical activity, body mass index) and three biological metrics (fasting glucose, blood cholesterol, blood pressure), coded on a three-point scale (0, 1, 2) (Supplementary Table 2).

Annual average air pollution exposure ($PM_{2.5}$ and NO_2) for the year

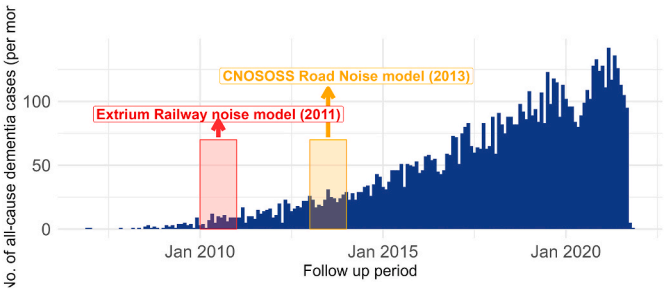

Fig. 1. Absolute incidence of all-cause dementia in UK Biobank. The figure show number of dementia cases per month across the follow-up study period (baseline to December 2021).

2010 was modelled for each address using the land use regression (LUR) model (Beelen et al., 2013; Eeftens et al., 2013). The percentage of home locations with proximity to greenspace was used for the present study. This was defined as the proportion of greenspace within 300 m of intermediate zone of UKB participants home location. We created a binary category with 20 % being used as a cut-off point for defining closeness to greenspace as informed by the previous literature (Cantuaria et al., 2021).

2.5. Statistical analysis

All participants enrolled in UK Biobank and with linked data on one or more source-specific transport noise exposure were included in the analyses. Baseline characteristics were summarised descriptively using the mean (sd) or median (interquartile range, IQR) for continuous variables or frequencies for categorical variables.

Cox proportional hazard regression model with follow-up time as the underlying timescale was used to quantify the associations between traffic noise exposure and incidence of dementia and its subtypes. Follow-up time was calculated using the date of baseline assessment attendance and the date of dementia first occurrence or death or December 31, 2021, whichever came first. Age was tested as underlying timescale and showed similar results as follow up time. Noise exposures from road and railway traffic (L_{den} and L_{night}) were analysed as continuous variables, assuming a linear effect and were each modelled separately. Hazard ratios (HR) calculated from the models were presented as per 10 dB higher of traffic noise exposure. In addition, to assess the effect different noise thresholds on the risk of dementia, road traffic noise measures were categorised as <50 dB, 50–55 dB, 55–60 dB, and ≥60 dB for L_{den} and <45, 45–50 dB, 50–55 dB, and ≥55 dB for L_{night}. For railway traffic noise, it was categorised as <50 and ≥ 50 dB for L_{den} and <45 and ≥ 45 dB for L_{night}.

For the unadjusted models, each noise metric and all-cause dementia were run first, then for each dementia subtype (Alzheimer's disease, vascular dementia, and Parkinson's disease related dementia). These models were then adjusted for age, sex, individual-level socioeconomic factors (education, household income, current employment status), Townsend deprivation index, and CRS (**Model 1**). To disentangle possible confounding from air pollution and greenness, model 1 was further adjusted for $PM_{2.5}$ and proximity to greenspace at buffer of 300 m (**Main model**). All covariables included in the modelling stage were within reasonable range of multicollinearity (variation inflation factor (VIF) = 1.0 to 1.9) and were selected to minimise confounding. The proportional hazard assumptions were assessed using the *stphplot* (ln (survival) vs ln (analysis time) and *estat phtest*. These tests assessed the proportional hazards assumption based on the Schoenfeld residuals. Furthermore, we assessed the dose-response relationships between traffic noise and dementia using a generalised additive cox regression model with two degrees of freedom. We assessed interactions between traffic noise and CRS, air pollution and proximity to greenspace on the risk of incident dementia. Significant interaction was set at two-tailed *p*-value of 0.05 or confidence interval that excluded the null. We also assessed mediating roles of CRS on the associations between road and railway noise and incident dementia and its subtypes using the main model. CRS was treated as a mediator in the modelling stage, not as a confounder. Additionally, the estimated mediated proportion was computed at the median of the CRS, and the exposure effect was compared across categorical levels of traffic noise exposures. Lastly, Cox regression was used to model the outcome, and linear regression to model the mediator.

The subgroup and sensitivity analyses based on the main model were conducted *a priori* by stratifying the effects of road and railway traffic noise on incident dementia based on age (<60 vs ≥ 60 years), sex (male and female), sleep duration (<6 vs ≥ 6 h of sleep) and presence of comorbidities (with either hypertension or diabetes or both). For the sensitivity analyses, we excluded participants who had a history of stroke at baseline, did further adjusting of sleep duration and hearing impairment, and restricted the analyses to non-movers only, using the main model. We also assessed the effects early road noise exposure estimates from 2009 on the risk of dementia with goal of seeing any changes of risk with time. All analyses were conducted in Stata18 and R Studio.

3. Results

3.1. Cohort characteristics

Of the full cohort (n = 502,416), a total of 7668 incident dementia cases were observed between baseline and end of follow up (median years of 9.67). This equated to an incidence rate (IR) of 11.86 cases per 10,000 person-years for all cause dementia. Alzheimer's disease (n = 3722) was the most common dementia with an incidence rate of 6.96 cases per 10,000 persons, followed by Parkinson's disease related dementia (n = 3466; IR: 5.69 cases per 10,000 person-years) and vascular dementia (n = 2,243, IR: 5.68 cases per 10,000 persons years). Incident dementia cases were more likely to be older, reside in deprived areas, and with a household income of less than £31,000 (Table 1). A higher proportion of incident cases of all-cause dementia had a history of smoking, alcohol intake, a higher systolic blood pressure, poorer cardiovascular health and more likely had multiple morbidities (diabetes, stroke, and hypertension).

Annual mean exposure to road L_{den} and L_{night} noise was similar between incident cases and non-cases (53 and 47 dB respectively). Additionally, road noise exposure measures (L_{den} and L_{night}) were moderately correlated with NO_2 (r = 0.22) and $PM_{2.5}$ (r = 0.28), while proximity to greenspace was negatively correlated with air pollution (NO_2 r = −0.64, and $PM_{2.5}$ r = −0.63) (Supplementary table 4).

3.2. Road traffic noise

The association between road noise and all cause dementia was positive in the adjusted models despite that statistical significance was not reached (HR per 10 dB higher: 1.041, 95 % CI: 0.952–1.137) (Table 2). In terms of the subtypes, a statistically significant association was found between road L_{den} and incident Alzheimer's disease (HR per 10 dB higher: 1.150, 95 % CI: 1.022–1.294) in the main model. Similar association was observed for road L_{night}. Subgroup analyses showed that those aged<60 years, male and those without a comorbidity presented a higher risk (Fig. 2). When noise metrics were modelled as a categorical exposure, it was found that those exposed to road L_{den} noise of 55 dB–60 dB, as compared to <50 dB, had a significantly higher risk of incident Alzheimer's disease (HR: 1.309, 95 % CI: 1.025–1.671) (Table 3). Similar associations were seen for L_{night} noise at 50–55 dB, when compared to <45 dB (HR per 10 dB: 1.365, 95 % CI: 1.07–1.741). L_{den} and L_{night} noise levels exceeding 60 dB and 55 dB showed moderate non-significant positive associations with all-cause dementia and Alzheimer's disease, respectively. There were no associations found with either vascular dementia or Parkinson's disease related dementia.

3.3. Railway traffic noise

The associations between railway traffic noise exposure and dementia outcomes were in general lacking except Parkinson's disease related dementia and vascular dementia. In model 1, railway L_{den} was significantly associated with Parkinson's disease related dementia (HR per 10 dB higher: 1.042, 95 % CI: 1.005–1.081); however, when further adjusted for air pollution and greenness, the association was attenuated (HR per 10 dB higher: 1.037, 95 % CI: 0.998–1.077) (Table 2). Further subgroup analyses generated using the main model revealed that this association was much more evident among those aged ≥60 years (HR per 10 dB:1.074, 95 % CI: 1.023–1.127) or male (HR per 10 dB: 1.067, 95 % CI: 1.021–1.115) (Fig. 3). Results of other subgroup analyses are

Table 1
Baseline characteristics of incident dementia in UK Biobank.

	All cause dementia	Alzheimer's disease	Vascular dementia	Parkinson's disease	Non cases
n	7668	3722	2243	3466	492,985
Age, mean (SD)	64.75 (4.81)	65.09 (4.30)	65.32 (4.15)	63.24 (5.40)	56.90 (8.09)
Sex (%), male	4034 (52.6)	1668 (44.8)	1298 (57.9)	2145 (61.9)	223,975 (45.4)
Follow up, years (median, IQR)	9.67 (7.45, 11.39)	9.62 (7.39,11.41)	9.47 (7.19,11.1)	9.85 (7.82, 11.41)	9.41 (6.71, 11.35)
Townsend deprivation index, n (%)					
1 (least deprived)	1393 (18.2)	896 (20.04)	398 (17.76)	773 (21.29)	99,205 (20.1)
5 (Most deprived)	1902 (24.8)	1017 (22.75)	582 (25.97)	703 (19.37)	98,386 (19.9)
Smoking status, n (%)					
Never, n (%)	3517 (46.4)	1813 (49.3)	948 (42.8)	1813 (52.7)	26,8901 (54.9)
Previous	3249 (42.9)	1547 (42.0)	989 (44.7)	1398 (40.7)	16,9092 (34.5)
Current	816 (10.8)	321 (8.7)	278 (12.6)	227 (6.6)	52,136 (10.6)
Alcohol consumption, n (%)					
Never, n (%)	535 (7.0)	256 (6.9)	150 (6.7)	186 (5.4)	21,789 (4.4)
Previous	539 (7.1)	216 (5.8)	167 (7.5)	189 (5.5)	17,522 (3.6)
Current	6545 (85.9)	3228 (87.2)	1914 (85.8)	3075 (89.1)	45,2067 (92.0)
Unemployment status, n (%)	111 (6.90)	49 (5.40)	30 (3.94)	47 (6.85)	9392 (3.19)
Average total household income before tax, n (%)					
Less than 31 K	4362 (76,45)	2535 (76.63)	1335 (79,37)	1850 (62.88)	200,848 (47.89)
More than 31 K	1344 (23,55)	773 (23.37)	347 (20.63)	1092 (37.12)	218,539 (52.11)
Degree, n (%)					
Yes	614 (8.35)	303 (8.5)	141 (6.6)	373 (11.0)	56,060 (11.6)
Sleep duration, n (%)					
<6 h	2005 (26.6)	916 (25.0)	604 (27.5)	810 (23.6)	120,899 (24.7)
6–8 h	4556 (60.5)	2323 (63.3)	1293 (58.9)	2222 (64.8)	330,789 (67.7)
>8 h	964 (12.8)	432 (11.8)	299 (13.6)	397 (11.6)	37,213 (7.6)
Road Lden, dB, n (%)					
Mean (SD)	53.35 (6.33)	53.27 (6.38)	53.52 (6.68)	53.21 (6.11)	53.35 (6.33)
Noise category					
<50	2113 (27.63)	1046 (28.19)	620 (27.74)	987 (28.52)	135,974 (27.68)
50–55 dB	3554 (46.49)	1720 (46.36)	1035 (46.31)	1581 (45.68)	227,357 (46.28)
55–60 dB	1094 (14.31)	522 (14.07)	298 (13.33)	498 (14.39)	70,949 (14.44)
≥60 dB	883 (11.55)	422 (11.37)	282 (12.62)	395 (11.41)	56,977 (11.60)
Road Lnight, dB (%)					
Mean, SD	46.72 (6.18)	46.65 (6.25)	46.89 (6.55)	46.58 (5.98)	46.73 (6.20)
Noise category					
<45 dB	3531 (46.19)	1753 (47.25)	1005 (44.97)	1619 (46.78)	225,807 (45.97)
45–50	2610 (34.14)	1236 (33.32)	779 (34.85)	1178 (34.04)	168,891 (34.38)
50–55	832 (10.88)	406 (10.94)	238 (10.65)	372 (10.75)	52,855 (10.76)
≥55	671 (8.78)	315 (8.49)	213 (9.53)	292 (8.44)	43,704 (8.90)
Rail Lden, dB (%)					
Mean, SD	15.86 (17.25)	15.30 (17.11)	16.01 (17.20)	15.66 (17.34)	16.24 (17.52)
Noise category					
<50 dB	6730 (97.6)	3183 (97.73)	1893 (97.68)	3054 (97.42)	424,094 (97.38)
≥50	165 (2.4)	74 (2.27)	45 (2.32)	81 (2.58)	11,401 (2.62)
Rail Lnight, dB (%)					
Mean (SD)	11.80 (13.98)	11.36 (13.81)	11.91 (13.96)	11.66 (14.07)	12.14 (14.24)
Noise category					
<45 dB	6781 (98.4)	3208 (98,5)	1904 (98.3)	3076 (98.1)	427,536 (98.2)
≥45	114 (1.7)	49 (1.5)	34 (1.8)	59 (1.9)	7959 (1.8)
Proximity to greenness, n (%)					
<20 %	2100 (31.5)	977 (30.1)	597 (30.9)	918 (29.5)	134,722 (31.2)
≥20 %	4777 (69.5)	2270 (69.9)	1337 (69.1)	2198 (70.5)	297,768 (68.8)
Nitrogen dioxide 2010 (median IQR)	26.63 (22.1,31.4)	26.29 (21.91,31.3)	26.86 (22.4,31.40)	26.00 (21.3,30.9)	26.25 (21.5,31.3)
Particulate matter ($PM_{2.5}$)2010 (median IQR)	10.00 (9.4, 10.6)	10.00 (9.4,10.6)	10.00 (9.4, 10.6)	9.90 (9.3, 10.5)	9.93 (9.3,10.6)
Systolic blood pressure (mean (SD)	144.16 (19.5)	144.36 (18.8)	145.21 (20.4)	142.00 (18.6)	137.71 (18.7)
Diastolic blood pressure (mean (SD)	81.78 (10.3)	81.67 (10.2)	81.98 (10.7)	82.22 (9.9)	82.22 (10.16)
Hypertension, n (%)	5113 (66.7)	2238 (60.1)	1772 (79.0)	2061 (59.5)	195,704 (39.7)
Diabetes, n (%)	1072 (14.1)	392 (10.6)	475 (21.3)	338 (9.8)	25,190 (5.1)
Stroke, n (%)	509 (6.6)	95 (2.6)	301 (13.4)	90 (2.6)	4884 (1.0)
Hearing difficulties, n (%)					
No	4627 (64.5)	2333 (66.5)	1287 (61.5)	2142 (65.7)	349,536 (74.6)
Yes	2541 (35.4)	1172 (33.4)	806 (38.5)	1117 (34.3)	118,924 (25.4)
Completely deaf	3 (0.01)	1 (0.0)	1 (0.0)	2 (0.1)	126 (0.0)

summarised in Supplementary 5. Lastly, exposure to railway L_{night} of ≥45 dB, as compared to <45 dB, was significantly associated with vascular dementia (HR: 2.151, 95 % CI: 1.206–3.835) (Table 3).

3.4. Exposure-response relationships

In general, exposure-response relationships between road L_{den} and all cause dementia, and each of the subtypes followed an upward trend at least until 70–80 dB; after which, the HRs started to decline with wide confidence intervals (Fig. 4). For railway noise, a levelling off of linear effects was observed, with peaks in the risk coefficient being seen at noise levels between 20 dB and 40 dB for all dementia outcomes.

3.5. Sensitivity analyses

The association between road L_{den} and Alzheimer's disease and between railways L_{den} and Parkinson's disease were robust to sensitivity analyses, particularly with further adjustment for sleep duration,

Table 2
Association between traffic noise (road and railway noise) and all-cause dementia and its subtype in UK Biobank.

Outcome	Exposure	Δ unit	Unadjusted		Model 1 [a]	Main model [b]
			Cases (n)	HR 95 % CI	HR 95 % CI	HR 95 % CI
All cause dementia	**Road noise**					
	Lden,24hrs	per 10 dB	7644	1.009 (0.974–1.046)	1.026 (0.944–1.115)	1.041 (0.952–1.137)
	Lnight	per 10 dB	7644	1.009 (0.973–1.047)	1.026 (0.942–1.117)	1.040 (0.95–1.139)
	Railway noise					
	Lden,24hrs	per 10 dB	6895	0 .996 (0 .982–1.01)	1.029 (0.996–1.063)	1.025 (0.991–1.059)
	Lnight	per 10 dB	6895	0.993 (0 .977–1.01)	1.036 (0.996–1.077)	1.030 (0.989–1.072)
Alzheimer's disease	**Road noise**					
	Lden,24hrs	per 10 dB	3710	0.997 (0.95–1.047)	1.101 (0.985–1.228)	**1.150 (1.022–1.294)**
	Lnight	per 10 dB	3710	0.998 (0.95–1.049)	1.100 (0.983–1.231)	**1.151 (1.021–1.298)**
	Railway noise					
	Lden,24hrs	per 10 dB	3257	0.986 (0.968–1.004)	1.001 (0.957–1.047)	0.993 (0.948–1.040)
	Lnight	per 10 dB	3257	0.983 (0.961–1.005)	0.998 (0.944–1.055)	0.989 (0.935–1.047)
Vascular dementia	**Road noise**					
	Lden,24hrs	per 10 dB	2235	1.063 (0.996–1.134)	0.987 (0.834–1.168)	1.030 (0.86–1.235)
	Lnight	per 10 dB	2235	1.065 (0.997–1.138)	0.985 (0.829–1.17)	1.030 (0.856–1.239)
	Railway noise					
	Lden,24hrs	per 10 dB	1938	1.002 (0.977–1.028)	1.052 (0.987–1.120)	1.041 (0.976–1.111)
	Lnight	per 10 dB	1938	0.999 (0.969–1.032)	1.066 (0.988–1.150)	1.054 (0.975–1.140)
Parkinson's disease	**Road noise**					
	Lden,24hrs	per 10 dB	3461	0.986 (0.935–1.04)	0.979 (0.887–1.082)	0.996 (0.895–1.107)
	Lnight	per 10 dB	3461	0.986 (0.934–1.041)	0.979 (0.885–1.084)	0.996 (0.894–1.11)
	Railway noise					
	Lden,24hrs	per 10 dB	3135	0.988 (0.970–1.009)	**1.042 (1.005–1.081)**	1.037 (0.998–1.077)
	Lnight	per 10 dB	3135	0.986 (0.962–1.010)	1.045 (0.999–1.093)	1.038 (0.991–1.087)

[a] Age, sex, individual-level SES (education, household income, current employment status) and area-level SES (Townsend), cardiovascular risk score (CRS).
[b] Fully adjusted model + PM2.5 and proximity to greenness.

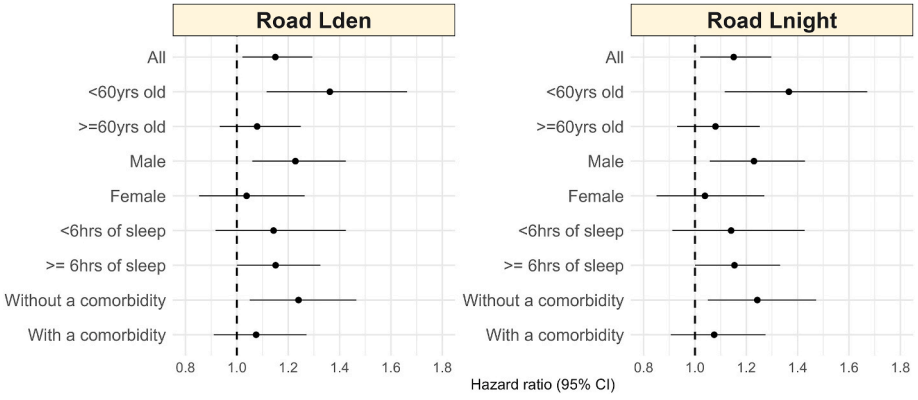

Fig. 2. Subgroup analyses showing the effects of road traffic noise (Lden and Lnight) per 10 DB on incident Alzheimer's disease based on the full cohort first then by age, sex, sleep duration and presence of comorbidities in UK Biobank.

hearing impairment, and after excluding participants with a history of stroke at baseline. However, when analyses were restricted to participants who did not move address during the study period (non-movers only), the association between road L_{den} and Alzheimer's disease and railway L_{den} and Parkinson's disease related dementia showed an increase, indicating the possible impact of cumulative noise exposure on these dementias (Supplementary table 6). There was no evidence of significant interactions between transport noise, air pollution, greenspace, on the risk of incident dementia (Supplementary Table 7). Lastly, the associations between road L_{den} noise and Alzheimer's disease were not found when using the baseline road noise data at 2009 (Supplementary Table 8).

3.6. Mediation analyses

Findings from the mediation analyses indicate that the association between L_{night} road traffic noise exposure above 45 dB and incident Alzheimer's disease was indeed mediated by CRS (Table 4). The total excess risk coefficient of 0.208 (95 % CI: 0.012–0.403) indicates that the excess risk due to CRS for Alzheimer's disease was higher for participants exposed to L_{night} road noise 45 dB and above compared with individuals exposed to lower than 45 dB. The control direct effect also indicated if all those with Alzheimer's disease had a poor cardiovascular health (CRS score of 6 or less), the excess risk associated with L_{night} road noise for those with exposure 45 dB and above would be higher (Coef:0.238, 95 % CI: 0.029–0.447).

4. Discussion

In this large prospective cohort study, while no statistically significant associations were observed of all cause dementia with either road or railway noise, we found distinct associations between road and railway traffic noise and subtypes of incident dementia. Specifically, we observe associations between road traffic noise (L_{den} and L_{night}) and incident Alzheimer's disease, and a possible association between railway noise and incident Parkinson's disease related dementia. The association between night-time road noise and Alzheimer's disease was mediated by cardiovascular risk profiles, with controlled direct effects

Table 3
Association between traffic noise (road and railway noise) and incident dementia based on different noise thresholds.

Outcome	Exposure	Noise categ.	Unadjusted		Model 1[a]	Main model [b]
			Cases (n)	HR 95 % CI	HR 95 % CI	HR 95 % CI
All cause dementia	**Road noise**					
	Lden,24hrs	<50 dB	2113	Ref	Ref	Ref
		50–55 dB	3554	1.002 (0.95–1.058)	0.976 (0.856–1.112)	0.982 (0.854–1.129)
		55–60 dB	1094	0.994 (0.924–1.070)	1.074 (0.907–1.271)	1.116 (0.934–1.333)
		≥60 dB	883	1.012 (0.935–1.094)	1.002 (0.832–1.206)	1.028 (0.841–1.256)
	Lnight	<45 dB	3531	Ref	Ref	Ref
		45–50 dB	2610	0.984 (0.936–1.036)	1.018 (0.901–1.149)	1.038 (0.913–1.181)
		50–55 dB	832	1.011 (0.938–1.091)	1.096 (0.921–1.303)	1.094 (0.908–1.317)
		≥55 dB	671	1.001 (0.921–1.087)	1.01 (0.832–1.226)	1.057 (0.858–1.301)
	Railway noise					
	Lden,24hrs	<50 dB	6730	Ref	Ref	Ref
		≥50 dB	165	0.933 (0.800–1.089)	1.106 (0.794–1.542)	1.096 (0.783–1.535)
	Lnight	<45 dB	6781	Ref	Ref	Ref
		≥45 dB	114	0.922 (0.766–1.11)	1.266 (0.875–1.83)	1.249 (0.858–1.817)
Alzheimer's disease	**Road noise**					
	Lden,24hrs	<50 dB	1265	Ref	Ref	Ref
		50–55 dB	2061	0.958 (0.893–1.028)	1.075 (0.898–1.286)	1.082 (0.888–1.317)
		55–60 dB	633	0.959 (0.871–1.055)	**1.259 (1.004–1.578)**	**1.309 (1.025–1.671)**
		≥60 dB	504	0.987 (0.89–1.094)	1.159 (0.90–1.488)	1.29 (0.985–1.691)
	Lnight	<45 dB	2118	Ref	Ref	Ref
		45–50 dB	1474	0.921 (0.862–0.985)	1.113 (0.944–1.312)	1.124 (0.94–1.344)
		50–55 dB	495	1.012 (0.917–1.116)	**1.332 (1.065–1.667)**	**1.365 (1.07–1.741)**
		≥55 dB	376	0.969 (0.868–1.081)	1.115 (0.858–1.449)	1.225 (0.924–1.625)
	Railway noise					
	Lden,24hrs	<50 dB	3812	Ref	Ref	Ref
		≥50 dB	98	0.993 (0.812–1.213)	1.325 (0.866–2.028)	1.294 (0.837–2.000)
	Lnight	<45 dB	3846	Ref	Ref	Ref
		≥45 dB	64	0.919 (0.718–1.177)	1.418 (0.875–2.296)	1.457 (0.899–2.36)
Vascular dementia	**Road noise**					
	Lden,24hrs	<50 dB	620	Ref	Ref	Ref
		50–55 dB	1035	0.99 (0.896–1.093)	0.941 (0.736–1.203)	0.956 (0.732–1.248)
		55–60 dB	298	0.925 (0.806–1.063)	0.927 (0.666–1.29)	0.905 (0.63–1.3)
		≥60 dB	282	1.114 (0.967–1.283)	0.843 (0.582–1.221)	0.932 (0.625–1.389)
	Lnight	<45 dB	1005	Ref	Ref	Ref
		45–50 dB	779	1.028 (0.936–1.129)	1.076 (0.856–1.353)	1.041 (0.813–1.332)
		50–55 dB	238	1.025 (0.89–1.18)	0.807 (0.555–1.175)	0.709 (0.463–1.087)
		≥55 dB	213	1.133 (0.977–1.314)	0.968 (0.661–1.416)	1.053 (0.701–1.582)
	Railway noise					
	Lden,24hrs	<50 dB	1893	Ref	Ref	Ref
		≥50 dB	45	0.915 (0.68–1.229)	1.633 (0.936–2.847)	1.676 (0.961–2.924)
	Lnight	<45 dB	1904	Ref	Ref	Ref
		≥45 dB	34	0.985 (0.702–1.383)	**2.088 (1.172–3.721)**	**2.151 (1.206–3.835)**
Parkinson's disease	**Road noise**					
	Lden,24hrs	<50 dB	1039	Ref	Ref	Ref
		50–55 dB	1646	0.939 (0.869–1.015)	0.926 (0.8–1.072)	0.887 (0.758–1.038)
		55–60 dB	530	0.979 (0.881–1.086)	0.934 (0.767–1.137)	0.925 (0.75–1.14)
		≥60 dB	415	0.975 (0.87–1.093)	0.988 (0.801–1.218)	1.027 (0.82–1.284)
	Lnight	<45 dB	1694	Ref	Ref	Ref
		45–50 dB	1233	0.968 (0.899–1.042)	1.014 (0.884–1.163)	1.016 (0.877–1.177)
		50–55 dB	393	0.999 (0.895–1.115)	0.971 (0.79–1.194)	0.982 (0.787–1.226)
		≥55 dB	310	0.977 (0.865–1.103)	0.971 (0.774–1.217)	1.015 (0.796–1.294)
	Railway noise					
	Lden, 4hrs	<50 dB	3204	Ref	Ref	Ref
		≥50 dB	84	1.002 (0.807–1.244)	0.91 (0.596–1.39)	0.894 (0.58–1.378)
	Lnight	<45 dB	3227	Ref	Ref	Ref
		≥45 dB	61	1.038 (0.805–1.337)	1.053 (0.66–1.679)	1.026 (0.634–1.658)

[a] Age, sex, individual-level SES (education, household income, current employment status) and area-level SES (Townsend), cardiovascular risk score (CRS).
[b] Main model (age, sex, individual-level SES (education, household income, current employment status) and area-level SES (Townsend), CRS, PM2.5, greenspace.

indicating that moving from optimal to poor cardiovascular health, was linked with excess risk that can be attributed to higher night-time road noise (45 dB or above).

Our finding showing the association between road and railway traffic noise with incident dementia subtype is consistent with the largest European study on transport noise and dementia. The nationwide study of nearly two million residents from Denmark aged 60 years old and older, included 103,500 incident cases of dementia during a 14 year follow up and with complete residential history of each participant and noise exposure estimate at the most and least exposed façade of the building, found that road and railway noise were significantly associated with the incidence of dementia and its subtype (Cantuaria et al., 2021). The study reported that exposure to road L_{den} noise of 50–55 dB compared to <45 dB was associated with increased risk of Alzheimer's disease (HR: 1.30, 95 % CI: 1.25–1.36). This association was independent of air pollution adjustment. While this study was powered by a very large sample size and high-quality noise exposure estimates, it did not consider many known risk factors for dementia at the individual level, particularly those related to cardiovascular health.

Furthermore, results from other studies and population of much lower sample size are less conclusive and inconsistent with our findings. This heterogeneity between studies may be explained by differences in

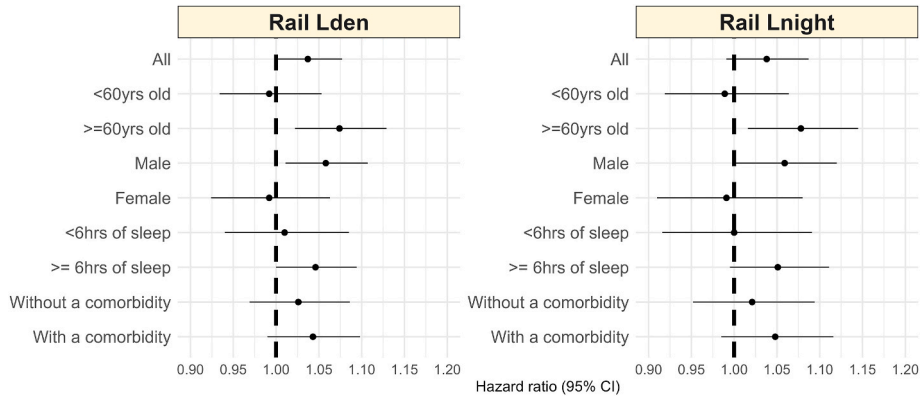

Fig. 3. Subgroup analyses showing the effects of railway noise (Lden and Lnight) per 10 DB on incident Parkinson's disease related dementia based on the full cohort first then by age, sex, sleep duration and presence of comorbidities in UK Biobank.

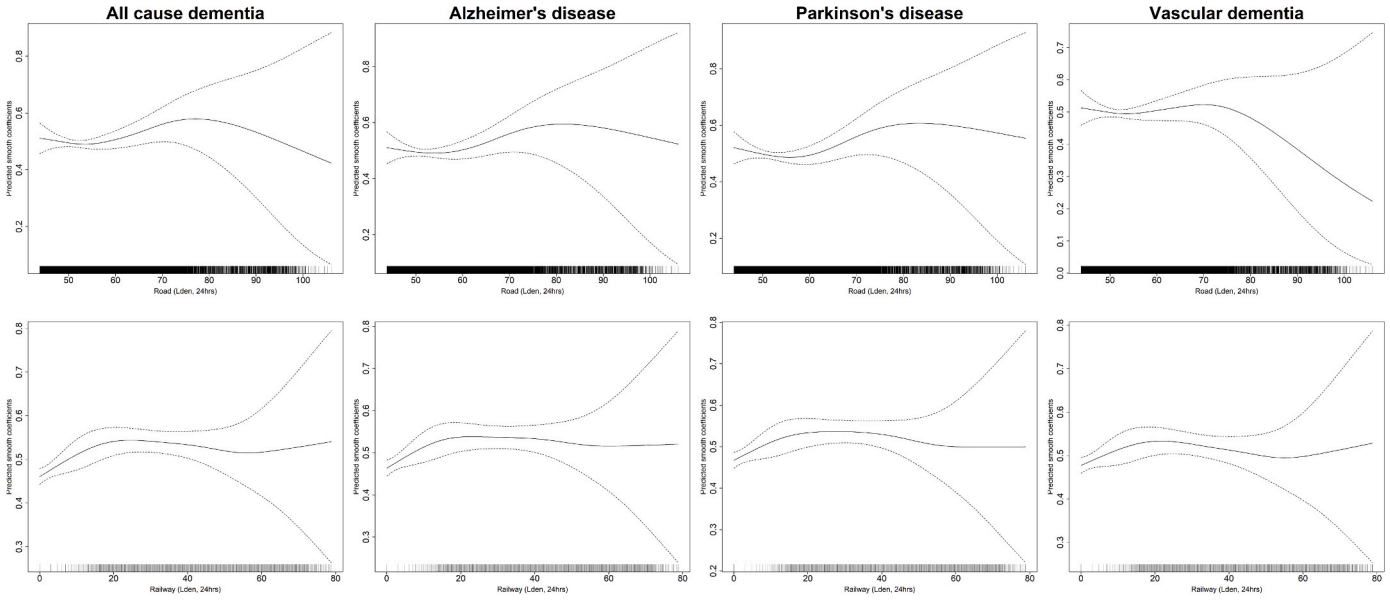

Fig. 4. Association between road and railway traffic noise and incidence of all-cause dementia and its subtypes in UK Biobank. The figure shows log-linear smooth function coefficients from a generalised additive cox model adjusted for age, sex, individual-level SES (education, household income, current employment status) and area-level SES (Townsend), cardiovascular risk score, air pollution ($PM_{2.5}$) and proximity to greenspace. In line with results from Table 2 and based on the smooth function parameters, Road noise and Railway noise (Rail $_{LDEN,\ 24hrs}$) showed significant association with incident Alzheimer's disease and Parkinson's disease related dementia, respectively.

noise modelling approach, window of noise exposure, levels of confounder adjustment and approach to case definition. For instance, a study of 1721 participants (mean age: 68.5 years, incident cases = 302 during a 15-year follow-up) in Umeå, Sweden did not find an association between road traffic noise and incident dementia (Andersson et al., 2018). Another study of 1612 Mexican American participants (mean age: 70 years, incident cases = 104 during a 10-year of follow-up) also reported no association with dementia incidence (Yu et al., 2023). However, the study found a synergistic effect with air pollution on dementia incidence, in particular when exposure to road noise level greater than 65 dB. Both of these two studies were limited by their small sample sizes. A large record-linkage study in Greater London of 130,978 participants aged 50–79 years (incident cases = 2181 during a 7-year of follow-up) found that night-time road traffic noise exposure was moderately associated with incident dementia (Carey et al., 2018). However, this association was lost after further adjustment for air pollution. More recently, the Danish Nurse Cohort (DNC) study of 25, 233 nurses (mean age: 53 years, incident cases = 1409 during a 23-year of follow-up) reported that the association between road traffic noise and incident dementia appeared to be attenuated by further adjustment of air pollution (Tuffier et al., 2024). Compared to the DNC study, our study is of a much larger sample size, but we reported very similar associations, with a HR per 10 dB higher of 1.04(95 % CI:0.95–1.14) compare with HR of 1.02(95 % CI:0.93–1.11) in the DNC study.

Given the larger sample size of our study, we were among the very few studies that had investigated the associations of traffic noise exposures with dementia subtypes. We herein reported a significant association of road L_{den} and incident Alzheimer's disease, with the effect size being very similar as that reported from the Danish national cohort study (Cantuaria et al., 2021). We also found that the association was slightly stronger for night-time noise, which was also observed in the Danish study when L_{den} at the least exposed façade (e.g., assuming where bedroom is located) was used in the analysis. These results may suggest that poor sleep quality due to higher road noise may at play for a higher risk of Alzheimer's disease (Livingston et al., 2020). We used data on sleep duration for our analysis, which may not necessarily capture sleep disturbance. Future studies are recommended to use high quality sleep data to explore this potential pathway. Another two large

Table 4
Estimated excess risk of association between transport noise and incident dementia, mediated by cardiovascular risk score.

Mediation analysis					
Mediator	**Outcome**	Road noise[a]		Railway noise[a]	
		Lden	**Lnight**	**Lden**	**Lnight**
		Coef (95 % CI)	**Coef (95 % CI)**	**Coef (95 % CI)**	**Coef (95 % CI)**
CRS	**All-cause dementia**				
	Total effect (TE)	0.1115 (-00191–0.2421)	0.0713 (−0.0541–0.1968)	0.1491 (−0.3445–0.6427)	0.1491 (−0.3445–0.6427)
	Controlled direct effect (CDE)	0.1404 (0.0007–0.2801)	0.1079 (−0.0275–0.2434)	0.1254 (−0.3973–0.648)	0.1254 (−0.3973–0.648)
	Reference interaction (INTref)	.0.029 (-0062 - 0.004)	**−0.0367 (-0.0727–0.0008)**	0.0241 (−0.1353–0.1834)	0.0241 (−0.1353–0.1834)
	Mediated interaction (INTmed)	0.0005 (-00006–0.0016)	0.0012 (−0.0004–0.0028)	−0.0002 (−0.0033–0.0028)	−0.0002 (−0.0033–0.0028)
	Pure indirect effect (PIE)	−0.0004 (-00012–0.0004)	−0.0011 (−0.0023–0.0002)	−0.0001 (−0.0016–0.0013)	−0.0001 (−0.0016–0.0013)
	Alzheimer's disease				
	Total effect (TE)	0.1842 (−0.0061–0.3744)	**0.2075 (0.0117–0.4033)**	0.3611 (−0.404–1.1262)	0.3611 (−0.404–1.1262)
	Controlled direct effect (CDE)	0.2054 (0.0032–0.4075)	**0.2382 (0.029–0.4474)**	0.3626 (−0.4434–1.1686)	0.3626 (−0.4434–1.1686)
	Reference interaction (INTref)	−0.0214 (−0.0627–0.0198)	−0.0313 (−0.0777–0.0152)	−0.0015 (−0.1747–0.1716)	−0.0015 (−0.1747–0.1716)
	Mediated interaction (INTmed)	0.0004 (−0.0007–0.0016)	0.0012 (−0.0008–0.0032)	0.0001 (−0.0023–0.0023)	0.0001 (−0.0023–0.0023)
	Pure indirect effect (PIE)	−0.0002 (−0.0008–0.0004)	−0.0007 (−0.002–0.0007)	0.0001 (−0.0003–0.0003)	0.0001 (−0.0003–0.0003)
	Vascular dementia				
	Total effect (TE)	0.0321 (−0.2085–0.2727)	0.0128 (−0.2191–0.2448)	0.6925 (−0.5262–1.9113)	0.6925 (−0.5262–1.9113)
	Controlled direct effect (CDE)	0.0967 (−0.1661–0.3594)	0.085 (−0.1724–0.3424)	0.6448 (−0.649–1.9386)	0.6448 (−0.649–1.9386)
	Reference interaction (INTref)	−0.0648 (−0.1324–0.0027)	−0.0724 (−0.1506–0.0058)	0.0485 (−0.3895–0.4865)	0.0485 (−0.3895–0.4865)
	Mediated interaction (INTmed)	0.001 (−0.0012–0.0033)	0.0022 (−0.0008–0.0051)	−0.0005 (−0.0067–0.0058)	−0.0005 (−0.0067–0.0058)
	Pure indirect effect (PIE)	−0.0008 (−0.0023–0.0008)	−0.0019 (−0.0042–0.0004)	−0.0003 (−0.0042–0.0035)	−0.0003 (−0.0042–0.0035)
	Parkinson's disease				
	Total effect (TE)	0.0447 (−0.0975–0.1869)	0.0159 (−0.1202–0.1519)	−0.2272 (−0.6749–0.2206)	−0.2272 (−0.6749–0.2206)
	Controlled direct effect (CDE)	0.077 (−0.0739–0.228)	0.0253 (−0.1191–0.1697)	−0.2879 (−0.7634–0.1876)	−0.2879 (−0.7634–0.1876)
	Reference interaction (INTref)	−0.0331 (−0.0594–0.0067)	−0.0101 (−0.0387–0.0186)	0.0611 (−0.1093–0.2315)	0.0611 (−0.1093–0.2315)
	Mediated interaction (INTmed)	0.0008 (−0.0008–0.0025)	0.0005 (−0.0009–0.0018)	−0.0007 (−0.0087–0.0073)	−0.0007 (−0.0087–0.0073)
	Pure indirect effect (PIE)	−0.0001 (−0.0006–0.0003)	0.0002 (−0.0008–0.0011)	0.0003 (−0.0029–0.0035)	0.0003 (−0.0029–0.0035)

[a] Adjusted for age, sex, individual-level SES (education, household income, current employment status) and area-level SES (Townsend), cardiovascular risk score, air pollution ($PM_{2.5}$) and proximity to greenspace.

record-linkage studies, in which noise exposures were assigned at post-code level rather than individual-level, observed non-significant or null associations (Yuchi et al., 2020). In our study, we did not find any associations between road L_{den} and vascular dementia or Parkinson's disease related dementia. The Danish study has reported associations with vascular dementia at magnitudes similar to those of Alzheimer's disease (Cantuaria et al., 2021). A recent Dutch study reported a much stronger association between road L_{den} and Parkinson's disease (OR: 1.64, 95 %CI: 1.13–2.43), particularly at higher noise levels (75th percentile: 58.07–78.3 dB vs 25th percentile:22.3 dB–51.8 dB) (Lomme et al., 2023). Findings from the Dutch study was particularly subject to exposure misclassification mainly due to the challenge of assigning individual level noise exposure for densely populated cities and countries like the Netherlands (Chen et al., 2024).

Previously only the Danish study reported the associations between railway noise and dementia outcomes (Cantuaria et al., 2021). In contrast to our findings, they found overall consistent associations between railway noise and incident all cause dementia, or Alzheimer's disease. The association with Parkinson's disease related dementia was only evident when comparing noise level of 55–60 dB to less than 40 dB of exposure (HR: 1.49, 95 % CI: 1.25–1.78). Our findings suggested a possible association between rail L_{den} and Parkinson's disease related dementia, and between rail L_{night} over 45 dB and vascular dementia. Given the scarcity of studies into railway noise and dementia, more studies are warranted, especially also considering the effects of railway-related vibration (Seidler et al., 2023).

Possible mechanisms to explain the effects of noise and dementia are hypothesised to be through an altered regulation of neuroendocrine system. The current noise-annoyance model shows that long-term exposure to noise is liked with an activated hypothalamic-pituitary-adrenal axis (HPA) that secretes stress hormones (adrenocorticotropic, corticosterone and catecholamine hormones) which affects memory and learning (Jafari et al., 2020). Our findings suggests that road and railway noise may have unique effects on the brain health. The sound pressure intensity and frequency of these noise sources may induce annoyance at the different subcortical regions of the brain, potentially leading to the development of dementia sub-types. The exact mechanism explaining the different effects of noise on dementia subtypes are not yet clear. Emerging evidence from population studies suggests that traffic noise, mainly road noise, is linked with a lower global cognitive performance, principally processing speed and executive function, important markers of Alzheimer's disease and cognitive decline (Weuve et al., 2021; Mac et al., 2021; Haran et al., 2024). Evidence from animal studies have also shown that chronic exposure to noise can induce oxidative stress that drive the deposition of hyperphosphorylated Aβ and tau proteins involved in neuronal loss and cognitive decline in Alzheimer's disease (Meng et al., 2022). Less known is the role of railway noise in the pathophysiology of Parkinson's disease, highlighting a need for future studies to investigate the impact of different traffic noise sources on brain structures and function.

Cardiovascular diseases are well-established risk factors for dementia outcomes and has been linked to traffic noise exposures. This is the first study to report the mediating effects of cardiovascular health on the relationship between night-time road traffic noise and incident Alzheimer's disease. The presence of stress hormones at night has the potential to drive long-term changes in sleep pattern and vascular function, thereby leading to an increased risk of poor cognitive health and dementia over time (Meng et al., 2022; Kuntić et al., 2024). It is important to note our mediation analysis results will need to be compared with future studies so to better understand the role of cardiovascular health on traffic noise and dementia.

Strengths of our study include a large sample size with data on many individual-level covariates covering a series of biological and behavioural risk factors that may mask the effect of noise exposure on

dementia. In addition, we used updated, high-quality modelling of road traffic noise from both minor and major roads. This study has limitations. The outcome data used in this study were derived from multiple sources to enable greater detection of dementia cases; however regardless of this effort, outcome misclassification is still possible. Most dementia cases were identified from the hospital admission records which mean these cases may be on severe spectrum of dementia. Cases with dementia subtype were broadly defined using relevant ICD-10 codes (Supplementary Table 1a), the distribution of these dementia subtypes may not be comparable to that of the general population. For instance, we used a definition for Parkinson's disease that included ICD-10 codes that covered Parkinson's disease classified elsewhere (ICD-22). This meant that cases of Parkinson's disease were over-represented in this study and may not be representative of the general population (Alzheimer's Research UK Dementia Statistic, 2025). Moreover, some of the subtype cases were derived from the primary care record and this data only covered 45 % of UK Biobank population. Our study also did not consider the mixed forms of several dementia subtypes given the relatively small sample size of this particular group (n = 866). Furthermore, the date of diagnoses or date of dementia first occurrences may not be representative of disease onset and this could affect our risk estimates.

In terms of covariate adjustment, we used a universal cardiovascular risk score in all the analyses relating to different subtypes of dementia. It should be noted that adjustment of this cardiovascular risk score may not necessarily account for the risk for development of some dementia subtypes. For example, for Parkinson's related dementia, coffee consumption could be a strong protective factor whilst occupational and residential pesticide exposure could be risk factors, all of these covariates were not considered in this study (Ren and Chen, 2020; Ascherio et al., 2006). In addition, there appeared to be a strong, negative association between cigarette smoking and Parkinson's disease (Hernán et al., 2002), which might further restrict the suitability of this cardiovascular risk score for the specific investigation of Parkinson's dementia. Exposure misclassification is inevitable for this type of study as we lacked information on personal behaviours or other noise sources that may modify participants' noise exposures. Road traffic noise was assigned to baseline address for a single year (2013) during the follow-up, assuming that noise levels remained temporally stable over the follow-up period. Associations were not found when using the baseline road noise data at 2009. Unlike the road traffic noise data for year 2013 which were modelled for both main and minor roads at residential address, the 2009 baseline road noise data only considered noise from major roads which in part may explain the null results. Railway noise estimates were derived from major railway networks only and also assigned to a single year. Finally, we have many associations in this study, some of these may be chance findings. We also did not consider genetic factors of dementia in this study.

5. Conclusion

We found an association between road traffic noise exposure and incident Alzheimer's disease in this large cohort study. Our findings would need further validations so to as allow for an improved knowledge about the long-term impact of road and railway traffic noise on dementia.

CRediT authorship contribution statement

Enock Havyarimana: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Formal analysis, Data curation. **Xiangpu Gong:** Writing – review & editing, Methodology, Data curation. **Calvin Jephcote:** Writing – review & editing, Methodology, Data curation. **Sarah Johnson:** Writing – review & editing, Methodology, Data curation. **Sana Suri:** Writing – review & editing, Investigation, Funding acquisition. **Wuxiang Xie:** Writing – review & editing, Methodology. **Charlotte Clark:** Writing – review & editing, Investigation, Funding acquisition. **Anna L. Hansell:** Writing – review & editing, Resources, Project administration, Investigation, Funding acquisition. **Yutong Samuel Cai:** Writing – review & editing, Writing – original draft, Supervision, Resources, Project administration, Methodology, Investigation, Funding acquisition, Data curation, Conceptualization.

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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.envres.2025.121787.

Data availability

UK Biobank data is publicly available and can be accessed by approved researchers. The road and railway traffic noise data will be made available upon request.

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