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Long-term exposure to road traffic noise and all-cause and cause-specific mortality: a Danish Nurse Cohort study

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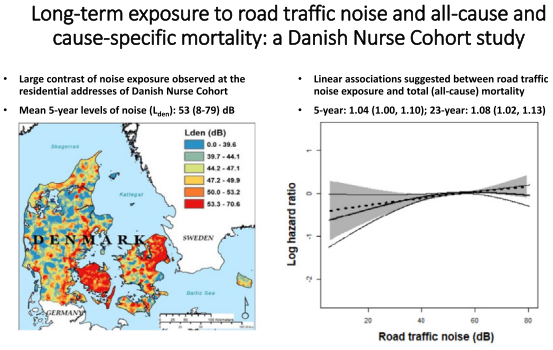
HIGHLIGHTS

- Road traffic noise (RTN) is linked to cardiometabolic disease morbidity.
- Evidence on the link between RTN and mortality remains limited.
- We examine how long-term RTN exposure links with total and cause-specific mortality.
- We include 24,994 nurses recruited in 1993 or 1999 from the Danish Nurse cohort.
- We find significant links between RTN and mortality in total and specific causes.

GRAPHICAL ABSTRACT



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ABSTRACT

Background: Long-term road traffic noise exposure is linked to cardio-metabolic disease morbidity, whereas evidence on mortality remains limited.
Objectives: We investigated association of long-term exposure to road traffic noise with all-cause and cause-specific mortality.
Methods: We linked 22,858 females from the Danish Nurse Cohort (DNC), recruited into the Danish Register of Causes of Death up to 2014. Road traffic noise levels since 1970 were modelled by Nord2000 as the annual mean of a weighted 24 h average (L_{den}). Cox regression models examined the associations between L_{den} (5-year and 23-year means) and all-cause and cause-specific mortalities, adjusting for lifestyle and exposure to $PM_{2.5}$ (particulate matter

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with diameter < 2.5 μm) and NO_2 (nitrogen dioxide).
Results: During follow-up (mean 17.4 years), 3902 nurses died: 1622 from cancer, 922 from CVDs (289 from stroke), 338 from respiratory diseases (186 from chronic obstructive pulmonary disease, 114 from lower respiratory tract infections [ALRIs]), 234 from dementia, 95 from psychiatric disorders, and 79 from diabetes. Hazard ratios (95% confidence intervals) for all-cause mortality from fully-adjusted models were 1.06 (1.01, 1.11) and 1.09 (1.03, 1.15) per 10 dB of 5-year and 23-year mean L_{den}, respectively, which attenuated slightly in our main model (fully-adjusted plus $PM_{2.5}$: 1.04 [1.00, 1.10]; 1.08 [1.02, 1.13]). Main model estimates suggested the strongest associations between 5-year mean L_{den} and diabetes (1.14: 0.81, 1.61), ALRIs (1.13: 0.84, 1.54), dementia (1.12: 0.90, 1.38), and stroke (1.10: 0.91, 1.31), whereas associations with 23-year mean L_{den} were suggested for respiratory diseases (1.15: 0.95, 1.39), psychiatric disorders (1.11: 0.78, 1.59), and all cancers (1.08: 0.99, 1.17).
Discussion: Among the female nurses from the DNC, we observed that long-term exposure to road traffic noise led to premature mortality, independently of air pollution, and its adverse effects may extend well beyond those on the cardio-metabolic system to include respiratory diseases, cancer, neurodegenerative and psychiatric disorders.

1. Introduction

Noise is one of the main environmental stressors on physical and mental health and wellbeing within urban environments (WHO Regional Office for Europe, 2018). Long-term exposure to environmental noise places a substantial burden on public health through physiological and psychological distress (WHO Regional Office for Europe, 2018). In Western Europe, at least one million healthy years of life are lost due to traffic-related noise, through morbidity related to ischemic heart disease (IHD), sleep disturbance and annoyance – the second highest burden following air pollution (WHO Regional Office for Europe, 2018). Traffic noise is projected to continue rising in Europe (EEA, 2020); and, it is being linked to a greater diversity of outcomes through sleep disturbance, inflammation, oxidative stress and allostatic load, including mental (Clark et al., 2020) and respiratory (Liu et al., 2020) health, independent of air pollution (So et al., 2020).

Road traffic noise is linked to cardiovascular disease (CVD) and ischemic heart disease (IHD) morbidity, with emerging yet inconsistent evidence on cerebrovascular, metabolic, and respiratory disease (Cai et al., 2020). As highlighted in a 2020 systematic review and meta-analysis, ischemic heart disease mortality is the most studied and most strongly associated outcome with long-term exposure to traffic noise (Cai et al., 2020). Evidence on the link between road traffic noise and mortality is still limited, with most studies finding positive associations between traffic-related noise and mortality (Gan et al., 2012; Halonen et al., 2015; Barceló et al., 2016; Héritier et al., 2017, 2018, 2019; Thacher et al., 2020) but not all (Beelen et al., 2009; Nieuwenhuijsen et al., 2018; Andersson et al., 2020; Klompmaker et al., 2020). The latter studies, finding inconsistent associations, consider comparatively short exposure periods of up to 5 years (see Supplemental Table S1). The causes of mortality for which less evidence exists, possibly due to a delayed mechanism from disease development to death, such as cancer, dementia, etc., may require longer-term exposures to see an effect.

The length of follow-up (exposure) period used to associate traffic noise with mortality outcomes has so-far been limited to less than 15 years (Cai et al., 2020), although in Denmark this period is extending to 23 years (Lim et al., 2021). Using a shorter exposure period may inadequately capture the development and ultimate outcome (mortality) of progressive diseases including cancer, which has earlier been associated with road traffic noise among Danish cohorts (Andersen et al., 2018; Thacher et al., 2020). As such, we aimed to augment the aforementioned emerging yet inconsistent evidence (on cerebrovascular, metabolic, and respiratory disease) by examining the association between longer-term (5-year and 23-year mean) exposure to road traffic noise, while adjusting for air pollution, and the risk of premature mortality by all-cause and specific causes: cardiovascular (CVD), respiratory, and psychiatric disorder, dementia, all (including breast) cancers, stroke, and diabetes. We hypothesised that longer road traffic noise exposure periods would be more strongly associated with mortality due to progressive diseases such as cancer, and potentially other less studied diseases such as psychiatric disorders, respiratory diseases and acute lower respiratory tract infections (ALRIs).

2. Methods

2.1. Cohort description

Established in 1993, and inspired by the American Nurses Health Study, the Danish Nurse Cohort (DNC) is a nationwide cohort which recruited 19,898 (86% of the invited 23,170) female nurses over 44 years of age responded with a completed questionnaire to begin participation (Hundrup et al., 2012). In 1999, the DNC recruited 8833 more nurses, after inviting both non-responding individuals from 1993 (489) and new individuals aged >45 years (8344) (Hundrup et al., 2012).

The questionnaire included a detailed inquiry on individual lifestyle factors (e.g., alcohol consumption, physical activity), body weight, hormone replacement therapy, reproductive history, health perception, and psycho-social work environment at baseline, which have been described in detail previously (Hundrup et al., 2012).

Using a unique personal identification number, participants were linked to the Civil Registration System to obtain a full residential address history and vital status from 1970 up to 31 December 2013 (Pedersen, 2011).

2.2. Outcome definition

An individual's cause of death was defined by linking the cohort participants to the Danish Register of Causes of Death (Helweg-Larsen, 2011), including all-cause (all deaths during the follow-up, not including death from 'accidents' and 'murder' [International Classification of diseases, 8th revision {ICD-8} prior to 1994: 8870; or ICD-10 after 1994: V01-X59, X85-Y84], including death from 'unknown' and 'suicide' [ICD-8: 9503, 9508, 9530 or ICD-10: X60–84]) and cause-specific mortality: CVD (ICD-8: 4100, 4129, 4279, 4339, 4369, 4412, 4500, ICD-10: I00–99), stroke (ICD-8: 4339, 4369, ICD-10: I60–69), ischemic heart disease (ICD- 8: 4100, 4129, ICD-10: I20–25), diabetes (ICD-10: E10-E14), respiratory disease (ICD-10: J00–99), COPD (ICD-10: J41–44), ALRIs (ICD-10: J09-J18, J20-J22), dementia (ICD10: F00–03, G30, G31.8, G31.9), psychiatric disorder (ICD-8: 2990, 3032, 3040, ICD-10: F04–99), all cancers (ICD-8: 1538, 1578–79, 1618, 1621, 1740, 1800,1820, 1830, 1991 2022, 2030, 2041, ICD-10: C00–97) and breast cancer (ICD-8:174, ICD-10: C50). Diabetes, dementia, and psychiatric disorder were based on primary and contributing cause of death.

2.3. Exposure assessment

We used air pollution and noise modelling systems with very fine spatial resolution that have estimated exposure to both pollutants at the individual participants' residential addresses (1 × 1 m, address point estimate).

2.3.1. Road traffic noise at residence

Residential road traffic noise levels from 1970 onwards were modelled using the Nord2000 model which estimated noise contribution from roads within a 3-km radius from the subjects' residential addresses, and expressed as the annual mean of a weighted 24 h average (L_{den}), calculated based on

noise levels of daytime (07:00–19:00 h), the evening (19:00–22:00 h), and the night-time (22:00–07:00 h) with a 5 dB and 10 dB penalty to the evening and night-time noise levels, respectively. The main input variables of this model are geocodes of location, street building geometry, road lines with information on yearly average daily traffic, traffic composition and speed, road type, and meteorology. Details of the Nord2000 model have been previously published (Kragh et al., 2001; Amini et al., 2020). In this study, we used exposure windows of 5-year (most commonly found across the literature) and 23-year (longest possible by our method) running means of L_{den} with 1-year lag as main exposure proxy of residential road traffic noise. We applied a 1-year lag to account for the fact that the cohort did not start at the beginning of the year, but on the 1st April for either 1993 or 1999.

2.3.2. Air pollution at residence

Residential $PM_{2.5}$ and NO_2 levels for the period 1970–2013 ($PM_{2.5}$ since 1990) were modelled using DEHM/UBM/AirGIS, a multi-scale and high-resolution (1 km × 1 km) Danish air pollution modelling system (Brandt et al., 2001a, 2001b). The system comprises of three air pollution models, which include the:

1) Danish Eulerian Hemispheric Model (DEHM), covering the entire Northern Hemisphere with a two-way nested grid (areas with higher resolution) going from 150 km × 150 km in an outer domain, to 50 km × 50 km over Europe and 5.6 km × 5.6 km over Denmark, to assess long-range transport components (Christensen, 1997; Brandt et al., 2012);
2) Urban Background Model (UBM), covering Denmark with a 1 km × 1 km grid resolution, to estimate the local background on a 1 km × 1 km resolution grid overlaying Denmark (Brandt et al., 2003); and,
3) Operational Street Pollution Model (OSPM), computing pollutant levels at the door step, which estimates the residential address' front door concentrations (Berkowicz, 2000; Ketzel et al., 2013).

All three models are linked meaning that UBM uses DEHM data as a boundary condition for UBM and UBM is used as boundary condition for OSPM. Further details and performance evaluation of the models have been previously published (Brandt, 2001; Khan et al., 2019). We calculated running means with 1-year lag for a period of three years for $PM_{2.5}$ (longest possible window since models for $PM_{2.5}$ started from 1990), and five or 23 years for NO_2, and used it as confounding variable for noise exposure with the corresponding window (3-year running with 1 year lag for $PM_{2.5}$ were used both for 5- and 23 year running mean with 1 year lag of noise exposure as it is the longest available window of $PM_{2.5}$ exposure).

2.4. Statistical analyses

We used time-varying Cox proportional hazards regression models with age as the underlying time scale to investigate mortality as a function of residential road traffic noise exposure while adjusting for air pollution. Exposure to road traffic noise and air pollution was included in the models as a time-varying exposure. Estimated effects of this association were expressed as hazard ratio (HR) with 95% confidence interval (95% CI) per 10 dB increase in L_{den}. Follow-up started at the age of the cohort entry (1 April 1993 or 1 April 1999), and ended at the age of the date of death (event), emigration, or 31 December 2013, whichever occurred first. For covariates in the main model, the Cox proportional hazard assumption was examined using a Kaplan-Meier curve for categorical variables, and a statistical test based on the scaled Schoenfeld residuals for numerical variables. Following this, we considered the assumption to be valid (data not shown).

We modelled the associations with increasing level of adjustment: (Model 1) Crude model, including age (time-scale) and baseline year (strata); (Model 2) Adjusted model, further adjusted for smoking (never, previous, or current), alcohol consumption in drinks per week (none: 0, moderate: 1–15, or heavy: >15), working status (no or yes), and marital status (married, separated/divorced, single, or widowed), as well as level of urbanization (rural: <180 persons/km^2, suburban: 180–5220 persons/km^2, urban: >5220 persons/km^2) and municipality-level average income (as the only socioeconomically-relevant variable available for this cohort); (Model 3) Adjusted model plus 3-year rolling mean of $PM_{2.5}$ (spline term with two df); (Model 4) Adjusted model plus 5-year or 23-year rolling mean of NO_2 (spline term with two df). We considered Model 3 to be our main model. We do not adjust for body mass index or physical activity as they may be on the causal pathway between road traffic noise and mortality (Pyko et al., 2015; Foraster et al., 2016; Roswall et al., 2017; Cramer et al., 2019).

We visualized the shape of an association between road traffic noise and mortality based on Model 3, with the spline term of road traffic noise with two degrees of freedom. A likelihood ratio test was applied to evaluate the deviations for linearity assumption.

Effect modification was explored for the association between L_{den} and mortality by including an interaction term between the modifier and the exposure in the model and using likelihood ratio test. These include age (< 65 year or ≥ 65 year), obesity (body mass index [BMI] < or ≥ 30 kg/m^2), history of self-reported diagnosis or medication for hypertension and diabetes (no or yes), history of self-reported diagnosis or hospitalization for myocardial infarction (no or yes), and urbanicity (rural: < 180 persons/km^2, suburban: 180–5220 persons/km^2, urban: >5220 persons/km^2).

All statistical analyses and graphical presentations were produced in R version 4.0.3 (R Core Team, 2016), using the following packages: dplyr, stats, survival, splines, mets, gplots, plotCI, and Hmisc. All statistical tests were two-sided, and p-value <0.05 was considered statistically significant. Exposure maps for each pollutant at cohort baseline were created using ArcGIS® software by ESRI.

3. Results

3.1. Study population

From a possible pool of 28,731 cohort participants, a total of 5873 individuals were excluded as: 4 individuals were unidentifiable; 907 individuals had missing information both on noise and air pollution data, and; 2826 individuals lacked covariate information. Following these exclusions, 24,994 individuals were included in the final analysis, of whom 3902 died by the end of follow-up. Those that died by the end of the study were more likely to be older, underweight or obese, current smokers, lower consumers of alcohol, and less likely married (single or widowed), compared to those that lived. Further, those that died were more likely living in areas that are urban yet of lower average income, and living in areas of higher noise and air pollution levels, compared to those that lived. Those that died by the end of follow-up were also more likely to have had hypertension, diabetes, or myocardial infarction at the study baseline (Table 1). Excluded individuals were more likely recruited earlier into the cohort (1993 versus 1999), older, have a higher BMI, current or previous smokers, consumed less alcohol, not working, widowed, living in rural areas and areas of higher air pollution, and had hypertension, diabetes or myocardial infarction (Supplemental Table S2).

3.2. Exposure characterisation

A large contrast of exposures was observed at the residential addresses of the cohort participants at baseline, for both noise and air pollution. Five-year mean level of noise (L_{den}) was 53 dB, ranging from 8 to 79 dB. Five-year mean level of NO_2 was 13 $\mu g/m^3$, ranging from 3 to 81 $\mu g/m^3$. Three-year mean level of $PM_{2.5}$ was 21 $\mu g/m^3$, ranging from 6 to 48 $\mu g/m^3$. See Table 1 for characterisation of exposure levels (Supplemental Table S3 according to vital status), and Fig. 1 for a spatial representation of the contrast in exposures. We observed limited temporal variation for L_{den} and NO_2, however a gradual reduction in $PM_{2.5}$, from 1991 to 2013 (see Supplemental Figs. S1, S2, and S3). L_{den} was moderately correlated with NO_2, however not with $PM_{2.5}$ (Table 2).

Table 1
Descriptive statistics for 24,994 female nurses from the Danish Nurse Cohort at the cohort baseline in 1993 or 1999: all and by vital status at end of follow-up.

Characteristics	All *N* = 24,994	Alive[f] *N* = 21,092	Dead[a] *N* = 3902	p-value[b]
Age at study start (years), mean ± SD	53.3 ± 8.2	51.6 ± 6.5	62.5 ± 10.0	<0.0001
Body mass index (kg/m^2), mean ± SD	23.7 ± 3.5	23.7 ± 3.5	23.6 ± 3.8	0.0061
Body mass index (kg/m^2), n (%)				
Underweight (<18.5)	627 (2.5)	409 (2.0)	218 (5.7)	<0.0001
Normal weight (18.5–24.9)	17,043 (69)	14,579 (69.7)	2464 (64.9)	
Overweight (25–29.9)	5631 (22.8)	4760 (22.8)	871 (22.9)	
Obese (≥30)	1400 (5.7)	1156 (5.5)	244 (6.4)	
Smoking status, n (%)				<0.0001
Never	8574 (34.3)	7707 (36.5)	867 (22.2)	
Previous	7694 (30.8)	6533 (31.0)	1161 (29.8)	
Current	8726 (34.9)	6852 (32.5)	1874 (48.0)	
Alcohol consumption, n (%)				<0.0001
None (0 drinks/week)	3974 (15.9)	2953 (14.0)	1021 (26.2)	
Moderate (1–14 drinks/week)	15,346 (61.4)	13,314 (63.1)	2032 (52.1)	
Heavy (≥ 15 drinks/week)	5674 (22.7)	4825 (22.9)	849 (21.8)	
Working status, n (%)	19,466 (77.9)	17,905 (84.9)	1561 (40.0)	<0.0001
Marital status, n (%)				<0.0001
Married	17,513 (70.1)	15,554 (73.7)	1959 (50.2)	
Separated/divorced	3244 (13)	2722 (12.9)	522 (13.4)	
Single	2528 (10.1)	1754 (8.3)	774 (19.8)	
Widow	1709 (6.8)	1062 (5.0)	647 (16.6)	
Degree of urbanicity, n (%)				<0.0001
Rural	10,210 (40.8)	8656 (41.0)	1554 (39.8)	
Suburban	10,961 (43.9)	9312 (44.1)	1649 (42.3)	
Urban	3823 (15.3)	3124 (14.8)	699 (17.9)	
Municipality- level average income[c], mean ± SD	158.8 ± 22.0	158.9 ± 22.2	158 ± 20.8	0.0120
Has hypertension, n (%)	3238 (13.0)	2344 (11.1)	894 (23.0)	<0.0001
Has diabetes, n (%)	307 (1.2)	201 (1.0)	106 (2.7)	<0.0001
Had MI, n (%)	195 (0.8)	80 (0.4)	115 (3.0)	<0.0001
L_{den}[d], mean ± SD (dB)	52.8 ± 7.9	52.7 ± 7.9	53.7 ± 7.8	<0.0001
$PM_{2.5}$[e], mean ± SD (μg/m^3)	20.6 ± 3.8	20.4 ± 3.8	21.5 ± 4.0	<0.0001
NO_2[d], mean ± SD (μg/m^3)	13.2 ± 7.7	13.1 ± 7.5	14.1 ± 8.7	<0.0001

Abbreviations: SD, Standard deviation; $PM_{2.5}$, Particulate matter aerodynamic diameter < 2.5 μm; NO_2, Nitrogen dioxide; L_{den}, The annual weighted noise levels of 24-h average; MI, Myocardial infarction.

[a] Total (all-cause) mortality (see Methods for details).

[b] Mean difference, from *t*-test for continuous variables and chi-square test for discrete variables.

[c] Danish Kroner multiplied by 1000.

[d] Five-year running mean at baseline with 1-year lag.

[e] Three-year running mean at baseline with 1-year lag.

[f] Alive, emigrated, or censored if died from 'accident' or 'murder' (see Methods for details).

3.3. Model outcomes

We observed that 5-year running means of linear road traffic noise exposure estimated strong (either statistically-significant or suggestive) associations with all-cause mortality, and mortality from stroke, diabetes, all respiratory diseases, and ALRIs. These associations were robust to full model adjustment by the air pollutants $PM_{2.5}$ and NO_2. Moreover, we observed that 23-year running means of linear exposure estimated stronger associations with all-cause mortality, and mortality from all respiratory diseases, breast cancer and all cancers; however, associations were weaker (compared to 5-year running mean) for mortality from stroke, diabetes, and ALRIs. The association with dementia was suggested to strengthen when adjusting for either air pollutant but especially so for NO_2. See Table 3.

Exposure-response curves suggest linear associations between road traffic noise exposure and total (all-cause) mortality, as well as the specific causes of CVD, ALRIs, and all cancers (Fig. 2). Such a linear exposure-response curve was not seen for ischemic heart disease, all respiratory diseases, chronic obstructive pulmonary disease, dementia, psychiatric disorders, and breast cancer (Supplemental Fig. S4).

Effect modification, of the association between exposure to road traffic noise (23-year running mean with 1 year lag, per 10 dB increase in L_{den}) and all-cause mortality, was not significant for age, obesity, urbanicity, or diagnosis/medication by hypertension or diabetes; however, was suggested to be significant for diagnosis/hospitalization by myocardial infarction (*p*-value = 0.054). See Fig. 3 and Supplemental Table S4.

4. Discussion

We found that long term exposure to road traffic noise was suggestively associated with premature mortality from all causes, and specifically with mortality from stroke and diabetes, as reported earlier. We also presented novel findings of a suggestive association of chronic exposure to road traffic noise with infectious respiratory diseases, psychiatric disorders, dementia, and cancer, suggesting that the adverse effects of noise extend beyond those on the cardiometabolic system. These findings go beyond the call for more research and, along with existing evidence, confirm that any policies aimed at reduction of road traffic noise as well as air pollution would prevent premature mortality and improve public health.

In our large cohort study, we considered the longest currently known (23-year mean) exposure period for road traffic noise and its association with a comprehensive selection of mortality outcomes. The observed positive association with all-cause mortality was stronger when using a 23-year compared to a 5-year period of noise exposure, which was robust for adjustment for air pollution (e.g., our main model including $PM_{2.5}$, but also NO_2). Specifically, a 23-year period of road traffic noise exposure was stronger (than a 5-year period) and significantly positively associated with mortality from psychiatric disorders, all respiratory diseases, ALRIs, and all cancers, and robust for adjustment by air pollution. More generally, positive associations of noise (using either exposure period) were suggested with mortality by other specific causes including CVD, stroke, ischemic heart disease, and diabetes. In addition, we report suggestive associations with 5-year mean noise exposure and stroke, diabetes, all respiratory diseases, ALRIs, and dementia.

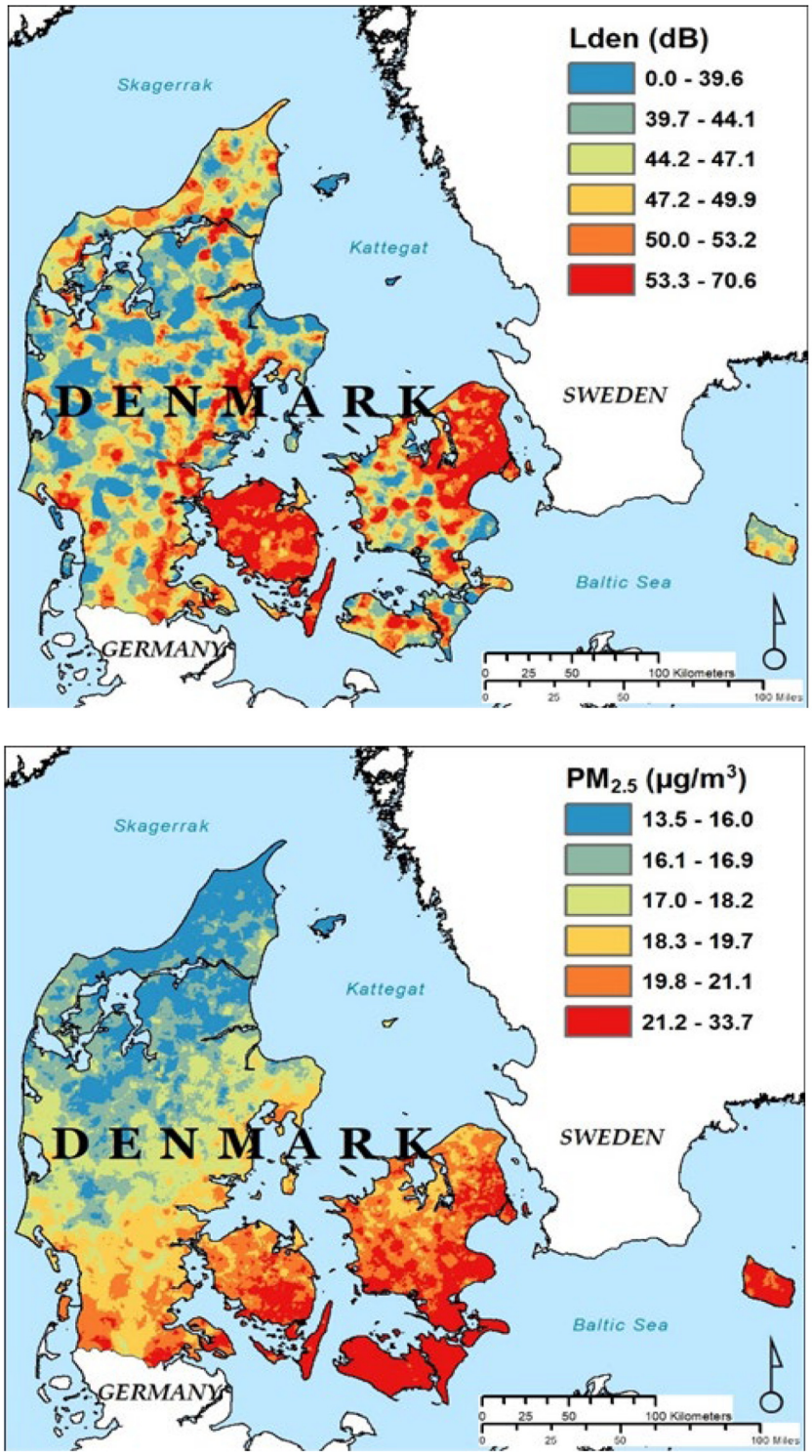

Fig. 1. Spatial variability of annual mean exposure levels at residences of Danish Nurse Cohort participants in the year before cohort enrolment (1993/1999) Caption: Visualization of cohort participant baseline residential annual (year preceding 1993/1999) exposure mean levels: interpolation on a 1 km × 1 km spatial resolution).

A recent systematic review and meta-analysis of the epidemiological evidence published in the last two decades found overall (pooled) positive suggestive associations between long-term road traffic noise (per 10 dB L_{den}) and mortality outcomes (Cai et al., 2020). Their evaluation of the published evidence was mostly as low to very low in quality, except for CVD and ischemic heart disease which was moderate in quality. While our study adds to the weight of evidence by using a large study cohort, a large exposure contrast and a long history of exposure to road traffic noise, we did not find suggestive (nor significant) associations between road traffic noise and mortality by ischemic heart disease. We did, however, find suggestive and some significant associations with mortality, as stated previously. The strength of these associations can depend on exposure period (duration) terms used.

While we found that associations were strengthened with a longer (23-year compared to 5-year mean) exposure window of L_{den} for all-cause mortality and mortality by all cancers, we found the opposite for stroke

Table 2
Pearson's correlation coefficient between road traffic noise and traffic-related air pollutants at the cohort baseline (1993 or 1999) for 24,994 female nurses from Danish Nurse Cohort.

	L_{den}[a]	$PM_{2.5}$[b]	NO_2[a]
L_{den}[a]	1		
$PM_{2.5}$[b]	0.34	1	
NO_2[a]	0.61	0.56	1

Abbreviations: $PM_{2.5}$, Particulate matter aerodynamic diameter < 2.5 μm; NO_2, Nitrogen dioxide; L_{den}, annual weighted noise levels of 24-h average.
[a] Five-year running mean at baseline with 1 year lag.
[b] Three-year running mean at baseline with 1 year lag.

mortality: associations were stronger with a shorter exposure window (5-year compared to 23-year mean) of L_{den}. Whether an association is strengthened by a shorter or longer exposure period could be partly explained by the duration of which certain diseases take to develop and become fatal. One previous study has investigated up to a maximum exposure window of 15 years (Sørensen et al., 2014a), with most other investigating shorter exposure windows between 5 and 10 years. See supplemental Table S1 for a full overview of previous, related literature. Thacher et al. (2020) performed one of the only analyses exploring the relationship with different exposure windows (1-, 5-, and 10-year exposure duration terms). In their slightly larger cohort study, they did not see marked differences in hazard ratio when expanding exposure windows from 1 to 5 to 10 year means, for all or any cause-specific mortality (Thacher et al., 2020). They found (based on data measured at the most exposed building façade, at an interquartile range close to 10 dB [equivalent to our continuous exposure level term]) a similar trend to ours (strengthened association when using a shorter exposure period) for stroke, and the same effect size for 5-year means of noise exposure.

Among this cohort, independent associations with both air pollution and road traffic noise were suggested. This independence of effect has been noted in previous syntheses of the evidence, that traffic-related air pollution is unlikely to confound the effect of traffic noise on CVD outcomes (Vienneau et al., 2015; Cai et al., 2020). While a high correlation between emissions or exposure concentrations of noise and air pollution from common sources such as road traffic (Davies et al., 2009) might be expected to confound associations of either to cardio- and cerebrovascular outcomes (Sørensen et al., 2014b; Héritier et al., 2019), we observed only moderate correlation between road traffic noise and NO_2, and weak correlation between road traffic noise and particulate matter ($PM_{2.5}$). When adjusting for NO_2, we observed enhanced effects on mortality from ALRIs when compared to adjustment for $PM_{2.5}$. Thacher and colleagues found little reduction in risk estimates of mortality from noise exposure with adjustment for air pollution, however called for further study of this association (Thacher et al., 2020).

We presented novel findings of suggestive association between road traffic noise and respiratory diseases including ALRIs, which persisted, although, attenuated when adjusting for air pollution. Biological plausibility of this association has been explained as due to an effect of noise, or the stress response it elicits, causing oxidative stress and pulmonary

Table 3
Associations between road traffic noise and all- and specific-cause mortality among 24,994 Danish Nurse Cohort participants: linear exposure.

Outcome (Cases)	Exposure window	Model 1: Crude	Model 2: Fully adjusted	Model 3 (Main): Model 2 + $PM_{2.5}$	Model4: Model2 + NO_2[b]
All-cause (N = 3902)					
	5-year	1.07 (1.02, 1.11)	1.06 (1.01, 1.11)	1.04 (1.00, 1.10)	1.05 (0.99, 1.11)
	23-year	1.10 (1.05, 1.15)	1.09 (1.03, 1.15)	1.08 (1.02, 1.13)	1.06 (0.99, 1.12)
All cardiovascular diseases (N = 922)					
	5-year	1.03 (0.94, 1.12)	1.05 (0.95, 1.16)	1.01 (0.91, 1.11)	0.99 (0.88, 1.11)
	23-year	1.04 (0.94, 1.14)	1.05 (0.94, 1.17)	1.01 (0.91, 1.13)	1.01 (0.89, 1.15)
Stroke (N = 289)					
	5-year	1.10 (0.94, 1.30)	1.13 (0.94, 1.35)	1.10 (0.91, 1.31)	1.07 (0.86, 1.33)
	23-year	1.04 (0.88, 1.24)	1.04 (0.86, 1.26)	1.01 (0.83, 1.23)	0.97 (0.77, 1.23)
Ischemic heart disease (N = 317)					
	5-year	0.95 (0.81, 1.10)	1.01 (0.85, 1.19)	0.94 (0.80, 1.11)	0.94 (0.77, 1.14)
	23-year	0.97 (0.83, 1.14)	1.05 (0.88, 1.26)	0.98 (0.82, 1.17)	1.02 (0.83, 1.27)
Diabetes (N = 79)					
	5-year	1.31 (0.96, 1.79)	1.30 (0.92, 1.84)	1.14 (0.81, 1.61)	1.05 (0.69, 1.58)
	23-year	1.25 (0.90, 1.74)	1.21 (0.83, 1.76)	1.07 (0.74, 1.54)	1.05 (0.68, 1.62)
All respiratory diseases (N = 338)					
	5-year	1.19 (1.03, 1.39)	1.15 (0.97, 1.37)	1.14 (0.96, 1.36)	1.18 (0.96, 1.45)
	23-year	1.22 (1.04, 1.43)	1.16 (0.97, 1.40)	1.15 (0.95, 1.39)	1.11 (0.89, 1.38)
COPD (N = 186)					
	5-year	1.12 (0.92, 1.37)	1.07 (0.86, 1.33)	1.06 (0.85, 1.33)	1.07 (0.82, 1.40)
	23-year	1.15 (0.93, 1.43)	1.08 (0.85, 1.38)	1.08 (0.84, 1.38)	1.00 (0.75, 1.34)
ALRIs (N = 114)					
	5-year	1.20 (0.92, 1.57)	1.16 (0.86, 1.57)	1.13 (0.84, 1.54)	1.22 (0.84, 1.77)
	23-year	1.20 (0.90, 1.59)	1.14 (0.83, 1.57)	1.11 (0.80, 1.53)	1.08 (0.74, 1.59)
Dementia (N = 234)					
	5-year	1.09 (0.91, 1.31)	1.09 (0.89, 1.34)	1.12 (0.90, 1.38)	1.23 (0.95, 1.59)
	23-year	1.07 (0.88, 1.30)	1.07 (0.86, 1.33)	1.09 (0.87, 1.36)	1.25 (0.96, 1.63)
Psychiatric disorders (N = 95)					
	5-year	1.17 (0.89, 1.54)	1.06 (0.79, 1.44)	1.04 (0.77, 1.41)	1.01 (0.71, 1.44)
	23-year	1.29 (0.95, 1.74)	1.14 (0.80, 1.62)	1.11 (0.78, 1.59)	1.02 (0.67, 1.53)
All cancers (N = 1622)					
	5-year	1.05 (0.98, 1.12)	1.05 (0.98, 1.13)	1.03 (0.96, 1.11)	1.04 (0.95, 1.13)
	23-year	1.09 (1.02, 1.17)	1.10 (1.01, 1.19)	1.08 (0.99, 1.17)	1.06 (0.96, 1.16)
Breast cancer (N = 330)					
	5-year	0.98 (0.86, 1.13)	1.01 (0.86, 1.18)	0.98 (0.84, 1.15)	0.97 (0.80, 1.16)
	23-year	1.03 (0.88, 1.21)	1.06 (0.89, 1.27)	1.03 (0.87, 1.23)	1.01 (0.82, 1.25)

Footnote: Road traffic noise is considered in the models as a 10 dB increase in 5- and 23- year running mean with a 1-year lag. $PM_{2.5}$ and NO_2 is considered in the models as running means from baseline (with 1-year lag for a period of three years for $PM_{2.5}$, and five or 23 years for NO_2), as a confounding variable for noise exposure with the corresponding window. A 3-year running mean with 1-year lag for $PM_{2.5}$ was used for both 5- and 23-year noise exposure windows as three years is the longest available window of $PM_{2.5}$ exposure.
Abbreviations: ALRIs, acute lower respiratory tract infections; COPD, chronic obstructive pulmonary disease.

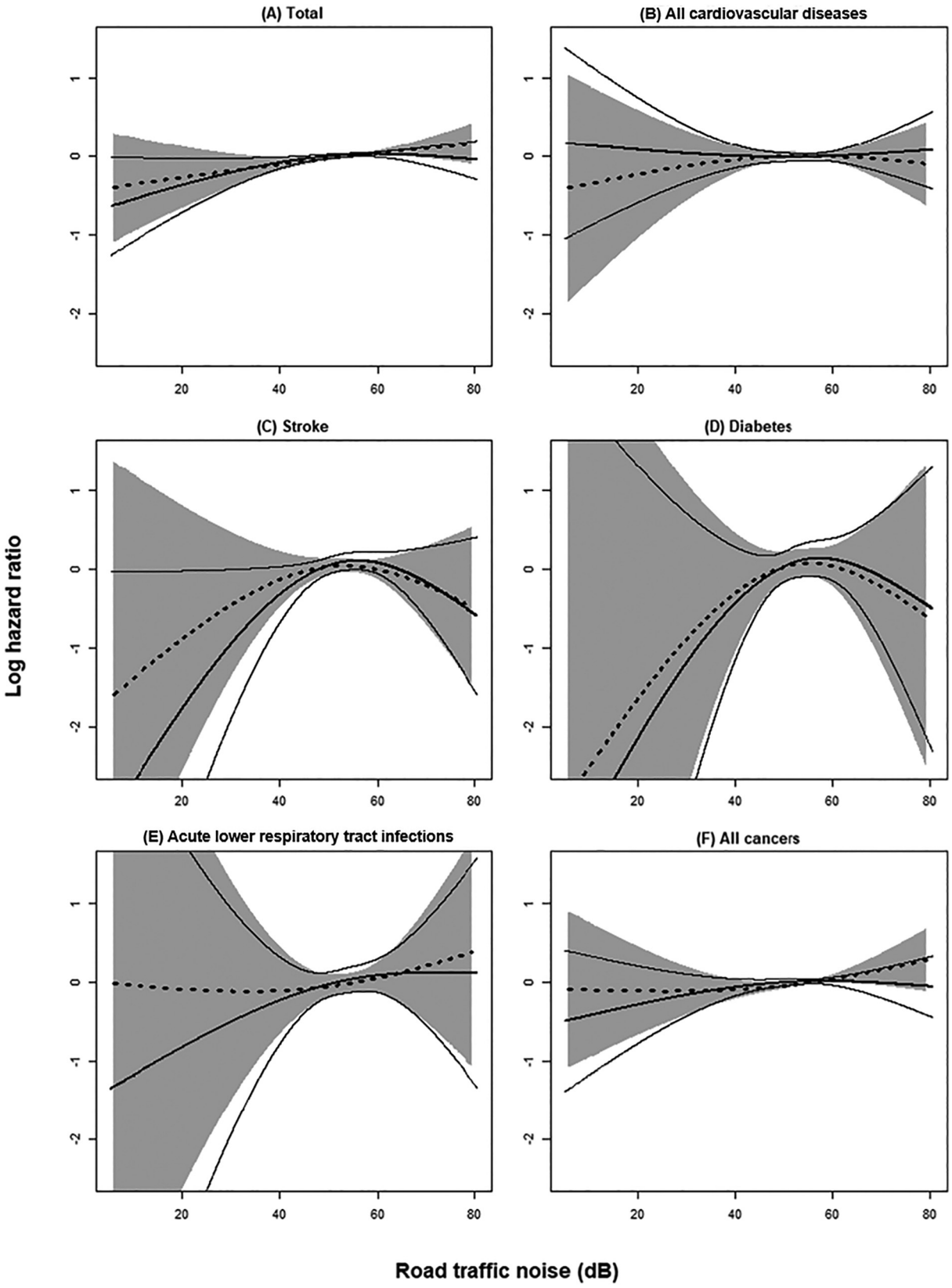

Fig. 2. Estimated exposure-response curves for the associations between road traffic noise (5- and 23-year running mean) and total (all-cause: A) and cause-specific mortality (BF) Caption: Natural spline with two degrees of freedom. Black bold solid line with upper and lower solid line indicates log hazard ratio and their 95% confidence interval for the association with 5-year running mean of road traffic noise. Black dotted line with the grey shade indicates log hazard ratio and their 95% confidence interval for the association with 23-year running mean of road traffic noise.

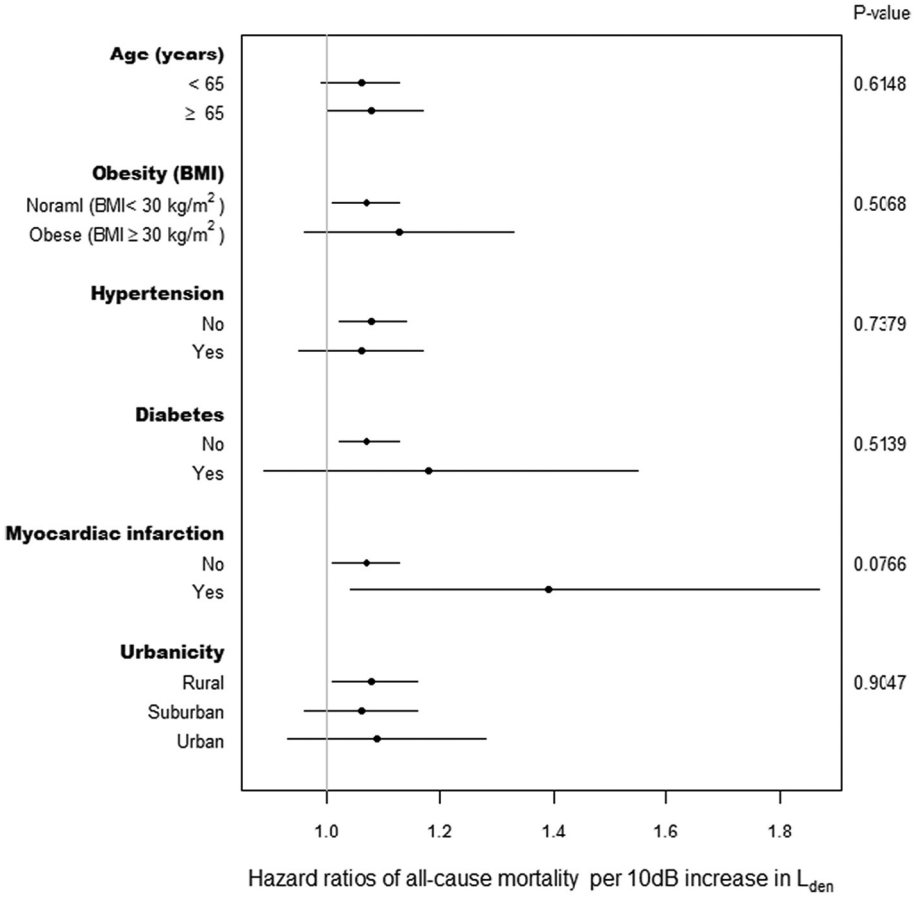

Fig. 3. Effect modification of associations between exposure to road traffic noise (23-year running mean with 1 year lag, per 10 dB increase in L_{den}) and all-cause mortality within the Danish Nurse Cohort study (N = 24,994, events = 3902) Caption: Effect modification was explored for the association between L_{den} and mortality by factors including age, obesity, self-reported hypertension, diabetes, myocardial infarction, and urbanicity. Numerical values are provided in Supplemental Table S4.

inflammation, as well as immune system dysregulation which increases the susceptibility to respiratory infection (Recio et al., 2016a). Other plausible pathways by which noise may increase risk of respiratory diseases is via sleep disturbance and stress related altering of smoking behavior (Roswall et al., 2018), reduction of physical activity (Roswall et al., 2017), increase in obesity (Foraster et al., 2018), and enriching DNA methylation related to C-reactive protein (Eze et al., 2020). In line with these proposed mechanisms, we have recently in this cohort detected associations between long term exposure to road traffic noise and COPD (Liu et al., 2021), in contrast to another study that found no such association in London (Carey et al., 2016). Furthermore, short-term exposure to road traffic noise was found to trigger COPD and respiratory mortality in two studies (Tobias et al., 2014; Recio et al., 2016b), and one study in children found that noise could enhance the association between traffic related air pollution and lung function (Franklin and Fruin, 2017) which corroborates other recently emerging idea of possible relevance of noise for respiratory diseases (Recio et al., 2016a; Linares and Díaz, 2019). Mechanisms related to effect of noise on CVD have been studied more than those on other outcomes. A recent review on the latest clinical and experimental studies shows how noise-induced stress increasing blood pressure, stress hormone levels, endothelial dysfunction, and oxidative stress may initiate the pathway from noise exposure to cardiovascular and metabolic outcomes including ischemic heart disease, stroke, and diabetes (Münzel et al., 2021).

Effects of noise on neurodegenerative and psychiatric disorders presented in this study are more novel, as evidence on this topic is just emerging. Our novel findings of association between road traffic noise and dementia mortality contrast findings of two studies which failed to detect associations with dementia incidence (Andersson et al., 2018; Carey et al., 2018). Similarly, evidence on effects of noise on psychiatric disorders is just emerging, although it is plausible that noise induced annoyance, stress response, as well as sleep disturbances can lead to increased risk of the development of neurocognitive, as well as emotional and behavioral disorders such as depression and anxiety (Lim et al., 2018; Schubert et al., 2019). One recent meta-analysis pointed at a likely association of road traffic noise with depression (Hegewald et al., 2020), while another pointed at a possible link with anxiety (Lan et al., 2020), however both concluded that higher quality and quantity of evidence was required to confirm such hypotheses.

Notwithstanding, road traffic noise is a risk factor for premature mortality, and a potentially relevant confounder for associations between air pollution and mortality that should be considered in future studies.

4.1. Strengths and limitations

A main strength and a novelty of our study is that we used the longest ever window of exposure to validated road traffic noise data (historical estimates up to 23 years) for association with all-cause and cause-specific mortality. This historical noise data has been earlier associated among the same cohort to morbidity/incidence of stroke (Cole-Hunter et al., 2021), myocardial infarction (Lim et al., 2021), diabetes (Jørgensen et al., 2019), atrial fibrillation (Andersen et al., 2021), breast cancer (Andersen et al., 2018), and chronic obstructive pulmonary disease (COPD) (Liu et al., 2021), as well as obesity (Cramer et al., 2019). Furthermore, the road traffic noise data have been used as a confounder in a number of studies on effects of air pollution on stroke (Amini et al., 2020) and mortality (So et al., 2020). We used a nationwide cohort, with residences across both urban and rural areas covering large contrasts in exposure levels of noise and air pollution. We

also were able to use detailed information on relevant confounders (individual covariates) plus specific air pollutants including NO_2 and $PM_{2.5}$. Since by cohort design we only included middle-age female nurses, we have reduced confounding by socioeconomic status such as the expected difference across age as a proxy for career stage.

A main limitation of our study is the lack of data on factors related to road traffic noise exposure, including annoyance by noise, bedroom placement, and levels of occupational noise and indoor noise. Additionally, we were limited to 3-year rolling means for exposure estimates of $PM_{2.5}$ (since models for $PM_{2.5}$ started from 1990), while this was not the case for L_{den} or NO_2. Our findings are limited in their generalizability to younger individuals (<44 years of age) and males, as well as populations in cities of higher traffic-related emission or exposure concentration levels, which are not represented in our study. Only municipality-level average income was available to be included as a SES measure. Some cause-specific outcomes (e.g., diabetes, stroke) were limited in the number of cases (and therefore statistical power), and therefore risk estimates should be interpreted with caution – this could be addressed in larger cohorts. Further, future studies should consider accounting for greenspace exposure if possible, to help understand any antagonistic or mitigating effects against traffic emissions expected from this potentially protective urban feature (Cai et al., 2020; Bauwelinck et al., 2021) – we did not have information on greenspace available for our cohort.

5. Conclusions

In conclusion, in this cohort of female nurses, we observed that long-term exposure to road traffic noise was associated with all-cause mortality, as well as suggestively so for mortality due to cardio- and cerebrovascular disease (stroke) and diabetes, independently from air pollution. Road traffic noise was more strongly associated with all-cause mortality, and mortality caused specifically by psychiatric disorders, respiratory diseases, ALRIs and all cancers when using a 23-year compared to a 5-year exposure mean.

Our findings suggest that the adverse effects of noise vastly extend beyond those of the cardiometabolic system, and that effects may be strengthened with prolonged exposure, which calls for further research and possible regulation.

CRediT authorship contribution statement

All authors made substantial contributions to methodology and validation of the manuscript.

ZJA further contributed with conceptualization, funding acquisition, and supervision. TCH further contributed with writing – original draft – of the manuscript. RS further contributed by performing the formal analysis and visualization.

All other authors further contributed to writing – review and editing – of the manuscript.

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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This study was conducted in accordance with The Code of Ethics of the World Medical Association (Declaration of Helsinki) for experiments involving humans. The locally appointed ethics committee has approved the research protocol and informed consent has been obtained from the subjects (or their legally authorized representative). We thank the Danish Nurse Cohort (DNC) participants and the DNC steering group for providing us with access to their data.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.scitotenv.2022.153057.

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