

Long-term exposure to road traffic noise and risk of heart failure: a cohort study

Mette Sørensen¹, Olav Wendelboe Nielsen², Ahmad Sajadieh², Matthias Ketzel³, Anne Tjønneland¹, Kim Overvad⁴, Ole Raaschou-Nielsen^{1,3}

¹ Diet, Genes and Environment, Danish Cancer Society Research Center, Copenhagen, Denmark (corresponding author)

² Department of Cardiology, Copenhagen University Hospital of Bispebjerg, Bispebjerg, Denmark

³ Department of Environmental Science, Aarhus University, Roskilde, Denmark

⁴ Section for Epidemiology, Department of Public Health, Aarhus University, Denmark, and Department of Cardiology, Aalborg University Hospital, Denmark

Corresponding author's e-mail address: mettes@cancer.dk

ABSTRACT

We aimed to investigate associations between road traffic noise and incident heart failure, which is an area receiving only little attention.

In a cohort of 57,053 people aged 50-64 years at enrolment in 1993-1997, we identified 2,550 incident heart failure cases during a mean follow-up of 13.4 years. Residential address history from 1987-2011 was found in national registers, and road traffic noise (L_{den}) and air pollution (NO_2) were modeled for all addresses. Analyses were done using Cox proportional hazard model.

An interquartile range increase in 10-years mean road traffic noise exposure (9.9 dB) was associated with an incidence rate ratio (IRR) of 1.14 (1.08-1.21) after adjustment for gender, lifestyle and socioeconomic status. After further adjustment for air pollution the IRR was 1.08 (1.00-1.16). In these mutually adjusted models air pollution was also associated with heart failure (IRR: 1.07 (1.01-1.14)). The association between noise and heart failure seemed strongest among men (1.19), hypertensive (1.17) and diabetics (1.20).

In conclusion, road traffic noise increased risk of heart failure, both before and after air pollution adjustment.

INTRODUCTION

Heart failure is a common disease, associated with morbidity and mortality. It ranks as the most frequent reason for hospitalization among older people [1]. Heart failure is a complex syndrome representing the end-stage of various cardiovascular diseases, including coronary diseases and hypertension.

Road traffic noise and ambient air pollution are environmental exposures that co-exist. They have both been associated with higher risk of cardiovascular diseases [2-4]. A recent meta-analysis found a positive association between short-term increases in air pollutants and risk of heart failure hospitalization or death [5]. With regard to long-term exposure, an expert position paper recently concluded that studies on long-term exposure to air pollution and incidence of chronic heart failure were still missing [6]. Also, few studies have found traffic noise to be associated with heart failure [7, 8].

Our aim was to study the association between long-term residential exposure to road traffic noise, before and after adjustment to air pollution, and risk of incident heart failure in a large Danish cohort.

METHODS

The study was based on the Danish Diet, Cancer and Health cohort, into which 57,053 residents of Copenhagen or Aarhus, between 50 and 64 years of age and without a history of cancer were enrolled in the period between 1993 and 1997 [9]. All participants completed self-administered, interviewer-checked, questionnaires at enrolment covering education, hypertension and lifestyle habits. Trained staff members measured height, weight, waist circumference and blood pressure according to standardized protocols. The study was conducted in accordance with the Helsinki Declaration, approved by local ethical committees, and all participants provided written informed consent

Heart failure cases diagnosed before death, emigration, or the end of follow-up (31st December 2011) were identified by linking the unique personal identification number of each cohort member to the nationwide Danish National Patient Register. Cases were identified using discharge diagnosis according to the International Classification of Diseases (ICD) 8 codes 4270 and 4271 or ICD-10 codes I50, I11.0, I42.0, I42.9 and I42.9. We excluded all participants with a diagnosis of heart failure before 1st July 1997.

Residential address history for all participants between 1st of July 1987 and 31st December 2011 were collected from the Danish Civil Registration System. All addresses were geocoded. Annual levels of road traffic noise were estimated using SoundPLAN, which is a calculation program that implements the joint Nordic prediction method for road traffic noise [10]. The noise model requires information on a number of variables: geocode and height for each address (at most exposed façade); information on annual average daily traffic for all Danish road links, distribution of light and heavy vehicles for all road links, travel speed and road type for all road links (motorway, express road, road wider than 6 m, road less than 6 m and more than 3 m, and other road); and polygons (3D) for all buildings in Denmark, obtained from the Danish Geodata Agency. We did not have information on noise barriers and road surface. We calculated road traffic noise as the equivalent continuous A-weighted sound pressure levels at the most exposed facade of each residence during day, evening and night, and expressed noise exposure as L_{den} .

We used an dispersion modelling system (AirGIS) to calculate exposure to annual levels of ambient NO_2 for all addresses [11]. This modelling system calculates air pollution at each address as the sum of: a) air pollution from the nearest street, calculated using information

on traffic, car fleet emission factors, geometry of streets and buildings as well as daily information on meteorology; b) urban background, which is calculated using information on emission density, city dimensions and heights of buildings; and c) regional background, estimated from rural monitoring data and national vehicle emissions [11]. For traffic input data, the information used were the same as described above for noise. NO₂ is a surrogate for a mix of traffic-related air pollutants.

Analyses were based on Cox proportional hazards model with age as underlying time. We used left truncation at age of 1st July 1997 (delayed entry), and right censoring at the age of heart failure, death, emigration or end of follow-up (31st December 2011). Exposure to road traffic noise (L_{den}) and air pollution (NO₂) were modelled as time-weighted means 1-, 5- and 10-years' preceding diagnosis, taking all present and historical addresses in these periods into account. The exposure windows were entered as time-dependent variables into the statistical risk model.

Incidence rate ratios (IRR) for heart failure in association with noise and air pollution were analysed in three models: Model 1 with adjustment for age and sex; Model 2 further adjusted for baseline information on smoking status, smoking intensity, smoking duration, intake of vegetables and red meat, years of school attendance, alcohol intake, sport during leisure time, calendar year and area level socioeconomic status (at baseline). In Model 3 we adjusted road traffic noise for air pollution and *vice versa*, (for same exposure time-window, 1-, 5- and 10 years). We estimated the association between noise and heart failure in predefined sub-groups by sex, baseline hypertension, baseline obesity, and a diagnose of myocardial infarction, or diabetes (before censoring). Potential modification were evaluated by introducing an interaction term into the model and tested by Wald test.

RESULTS

Of the study cohort of 57,053, we excluded 574 with cancer before baseline, 329 with heart failure before 1st July 1997, 316 who died or emigrated before start of follow up (1st July 1997), 3,119 without information on exposure, and 1,780 without information on one or more potential confounders, leaving a study population of 50,935 participants of whom 2,550 developed heart failure within a mean follow-up of 13.4 years.

When comparing with the total cohort we found that cases with heart failure more often men; they had fewer years of school attendance; they smoked more, they eat less vegetables and more red meat, they drank more alcohol, they were less physically active and more obese, and they were more likely to have hypertension as compared with the whole cohort. The correlation (R_{Spearman}) between 10-years exposure of road traffic noise and air pollution (NO₂) preceding start of follow-up was 0.65.

In crude analyses and in analyses with further adjustment for lifestyle, SES and calendar-year we found both exposures to be associated with a statistically significant higher risk of heart failure for all three exposure time-windows (Table 1). In two-pollutant models with mutual noise/air pollution adjustment the IRRs were lower, but there was still a statistically significant association with heart failure for both exposures for the 10-years exposure time-window: IRR of 1.08 (1.00-1.16) for road traffic noise and of 1.07 (1.01-1.14) for air pollution.

Our results indicated effect modification by gender (only association among men), baseline hypertension (only association among hypertensive) and diabetes (strongest association among patients with existing diabetes), although all interactions were insignificant (Table 2).

Table 1 Associations between 1-, 5- and 10-years exposure to road traffic noise and air pollution and risk for heart failure.

	Model 1 IRR (95% CI)	Model 2 IRR (95% CI)	Model 3 IRR (95% CI)
1-year exposure			
L _{den} , per IQR (10.0 dB)	1.22 (1.15-1.29)	1.13 (1.07-1.19)	1.06 (0.98-1.14)
NO ₂ , per IQR (6.6 µg/m ³)	1.17 (1.13-1.21)	1.10 (1.06-1.14)	1.07 (1.02-1.12)
5-year exposure			
L _{den} , per IQR (9.8 dB)	1.23 (1.17-1.30)	1.14 (1.08-1.21)	1.07 (0.99-1.15)
NO ₂ , per IQR (7.1 µg/m ³)	1.19 (1.14-1.23)	1.11 (1.07-1.16)	1.08 (1.02-1.14)
10-year exposure			
L _{den} , per IQR (9.9 dB)	1.24 (1.18-1.31)	1.14 (1.08-1.21)	1.08 (1.00-1.16)
NO ₂ , per IQR (7.5 µg/m ³)	1.20 (1.15-1.25)	1.11 (1.07-1.16)	1.07 (1.01-1.14)

Model 1: Adjusted for age and sex; Model 2: further adjusted for lifestyle, SES and calendar-year; Model 3: Further mutual exposure adjustment.

Table 2 Effect modification of associations between 10-years exposure (per IQR) to road traffic noise and risk for incident heart failure

	N cases	Road traffic noise (L _{den})	
		IRR (95% CI)	P-value
Gender			0.053
Men	1,622	1.19 (1.11-1.28)	
Women	928	1.06 (0.96-1.17)	
BMI			0.88
Obese (≥ 30)	696	1.12 (1.01-1.25)	
Non-obese (< 30)	1,854	1.13 (1.06-1.21)	
Hypertension			0.21
Yes	1,793	1.17 (1.09-1.25)	
No	757	1.08 (0.97-1.20)	
Myocardial infarction			0.81
Yes	787	1.12 (1.01-1.24)	
No	1,763	1.13 (1.06-1.21)	
Diabetes			0.30
Yes	573	1.20 (1.06-1.35)	
No	1,977	1.12 (1.05-1.19)	

Adjusted for age, sex, length of school attendance, municipality SES, smoking status, duration and intensity, intake of vegetables, red meat and alcohol, sport during leisure time and calendar-year.

DISCUSSION

We found that road traffic noise and air pollution was associated with higher risk of heart failure, both before and after mutually adjustment. The association between noise and heart failure seemed strongest among men, people with baseline hypertension and diabetics.

Heart failure is a condition that can develop slowly over time, and it is, therefore, relevant to investigate whether long-term exposure increases the risk of incident heart failure. We found long-term exposure to road traffic noise and air pollution to be associated with a higher risk of heart failure both before and after mutual adjustment. This indicates that both of these co-existing traffic pollutants affect the risk of developing heart failure. For air pollution, our results are supported by one other study, which found long-term exposure to different air pollutants, PM₁₀, NO₂ and SO₂, to be associated with a statistically significant increase in risk of heart failure [12].

Heart failure is a condition that represents the end stage of other cardiovascular diseases. We found that people who were hypertensive at baseline seemed more susceptible to the hazardous effects of noise on risk of heart failure. This indicates that noise may increase risk of heart failure among persons whose vascular systems are already known to be challenged by other factors. In support of this, the results also indicated a stronger relationship among people with pre-existing diabetes. Furthermore, the association between noise and heart failure was strongest among men. Again, this fits with the concept of an association among persons with a challenged vascular system, as men are known to have more advanced atherosclerosis than women [13].

The strengths of the present study include the prospective design with extensive confounder information, the large number of cases, inclusion of only the first hospitalization of heart failure and access to residential address history. Both exposures were estimated using high quality input data and state-of-the-art exposure models. Furthermore, cases with heart failure were identified using a high quality nationwide hospital register [14]. The present study also has some limitations. Estimation of air pollution and road traffic noise were based on exposure models, and although both models are state-of-the-art models, estimation of exposure is inevitably associated with some degree of uncertainty. As the exposure model does not distinguish between cases and non-cases, such misclassification is likely to be non-differential. We also lacked information on factors that influence the personal exposure to air pollution and road traffic noise, including time spent at home, information on commuting and occupational exposure, and direction of the bedroom, which may result in exposure misclassification. We would expect such misclassification to be unrelated to our outcome of interest and, therefore, draw the estimates towards the null. Residual confounding by unmeasured characteristics is always a risk, although we adjusted for many risk factors for heart failure.

In conclusion, we found that air pollution and road traffic noise was associated with higher risk of incident heart failure. The association with noise seemed strongest among persons with an impaired vascular system.

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