Long-term wind turbine noise exposure and incidence of myocardial infarction in the Danish nurse cohort

Elvira V. Bräunera,b, Jeanette T. Jørgensena, Anne Katrine Duun-Henriksena, Claus Backalarzc, Jens E. Laursen,c, Torben H. Pedersenc, Mette K. Simonsend,e, Zorana J. Andersen,f,⁎

a Section of Environmental Health, Department of Public Health, University of Copenhagen, Copenhagen, Denmark
b Juliane Marie Center, Department of Growth and Reproduction, Rigshospitalet, Copenhagen, Denmark
c DELTA Acoustics, Hørsholm, Denmark
d Diakonissestiftelsen, Frederiksberg, Denmark
e The Parker Institute, Copenhagen University Hospital, Bispebjerg, Frederiksberg, Denmark
f Centre for Epidemiological Research, Nykøbing F Hospital, Denmark

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ABSTRACT

Background: Growing evidence supports the concept that traffic noise exposure leads to long-term health complications other than annoyance, including cardiovascular disease. Similar effects may be expected from wind turbine noise exposure, but evidence is sparse. Here, we examined the association between long-term exposure to wind turbine noise and incidence of myocardial infarction (MI).

Methods: We used the Danish Nurse Cohort with 28,731 female nurses and obtained data on incidence of MI in the Danish National Patient and Causes of Death Registries until ultimo 2013. Wind turbine noise levels at residential addresses between 1982 and 2013 were estimated using the Nord2000 noise propagation model, as the annual means of a weighted 24-hour average (Lden) at the most exposed façade. Time-varying Cox proportional hazard regression was used to examine the association between the 11-, 5- and 1-year rolling means prior to MI diagnosis of wind turbine noise levels and MI incidence.

Results: Of 23,994 nurses free of MI at cohort baseline, 686 developed MI by end of follow-up in 2013. At the cohort baseline (1993 or 1999), 10.4% nurses were exposed to wind turbine noise (≥1 turbine within a 6000-m radius of the residence) and 13.3% in 2013. Mean baseline residential noise levels among exposed nurses were 26.3 dB, higher in those who developed MI (26.6 dB) than among those who didn’t develop MI (26.3 dB). We found no association between wind turbine noise and MI incidence: adjusted hazard ratios (HRs) and 95% confidence intervals (CIs) comparing nurses with 11-years mean residential noise levels of < 21.5 dB, 21.5–25.4 dB, 25.4–29.9 dB, and > 29.9 dB, to non-exposed nurses were 0.89 (0.64–1.25), 1.20 (0.82–1.77), 1.38 (0.95–2.01), and 0.88 (0.53–1.28), respectively. Corresponding HR (95% CI) for the linear association between 11-year mean levels of wind turbine noise (per 10 dB increase) with MI incidence was 0.99 (0.77–1.28).

Similar associations were observed when considering the 5- and 1-year running means, and with no evidence of dose-response.

Conclusions: The results of this comprehensive cohort study lend little support to a causal association between outdoor long-term wind-turbine noise exposure and MI. However, there were only few cases in the highest exposure groups and our findings need reproduction.

1. Introduction

Wind energy is presently the fastest-growing new source of electricity in the world, which is considered a “clean” alternative to fossil fuel generated electricity and often preferred by governments as their renewable energy option. Denmark is the world leader in total wind capacity and in 2016 wind power represented 37.6% of Denmark’s total electricity consumption (Global Wind Energy Council, 2017a). The Danish government has set a goal to generate 50% of the country’s electricity by wind energy by 2021, implying continuing increase in...
numbers and size of wind turbines, as well as proportion of the Danish population who live in close proximity to wind turbines (Global Wind Energy Council, 2017a).

The global benefits of wind energy in terms of reduced emissions of air pollutants are important, as wind power avoided over 637 million tons of CO2 emissions globally in 2016 (Global Wind Energy Council, 2017b). However, wind turbines are a source of environmental noise and the local-level potential risk to human health remains the subject of intense debate. There is comprehensive evidence that traffic noise (road, rail and air) leads to complications other than annoyance, including risk of cardiovascular disease, and hereby myocardial infarction (MI) (Münzel et al., 2017). However, these data cannot be extrapolated directly to the wind turbine noise, which is essentially different. Wind turbine noise is characterized by a more rhythmic modulation of sound than traffic sources, and seems to cause more annoyance and sleep disturbance than traffic noise (and other environmental noise sources) at similar noise levels (Pedersen and Waye, 2004; Pedersen et al., 2009). Wind turbines are typically located in rural areas in which background noise levels and sensitivity thresholds to noise may be lower. Additionally, road traffic noise is ubiquitous, affecting everyone, and a source of particulate or gaseous oxidative stressors (relevant for cardiovascular endpoints). Wind turbine noise is non-ubiquitous, predominantly a rural exposure, with around 800,000 homes (circa 12%) located within a 6000-m radius of at least one wind turbine in Denmark in 2016.

Epidemiological studies assessing the health impacts of wind turbines are few and have been inconsistent with a range of methodological weaknesses, including reliance on self-reported health effects and statistical power issues. Some of these studies report associations between wind turbine noise and idiopathic health symptoms, including sleep disturbance, fatigue, nausea, dizziness, headache, lack of concentration, as well as annoyance, (Hanning and Evans, 2012; Bakker et al., 2012; Kowano et al., 2013; Shepherd et al., 2011; McCallum et al., 2014) whilst others find no effects, (Pedersen et al., 2009; Knopper et al., 2014; Blanes-Vidal and Schwartz, 2016) arguing that annoyance due to visual rather than auditory aspects are the underlying cause leading to stress and sleep problems (Pedersen et al., 2009). In a previous study annoyance was reported to be strongly correlated with a negative attitude toward the visual impact of wind turbines on the landscape, and the authors further demonstrate that people who benefit economically from wind turbines have a significantly decreased risk of annoyance, despite exposure to similar sound levels, (Pedersen et al., 2009) as replicated in more recent studies (Jalali et al., 2016a; Jalali et al., 2016b; Janssen et al., 2011). A review of three cross-sectional studies, found that annoyance was consistently directly associated with wind turbine noise, but that no other measure of health or wellbeing (e.g. headache, tiredness, sleep disturbance) was consistently related to sound pressure levels (Pedersen, 2011). The effect of wind turbine noise on cardiovascular endpoints remains largely unexplored. Two Canadian cross-sectional studies, relying on self-reported data on health outcome found no association between long-term wind turbine noise exposure and high blood pressure or heart disease (Pedersen, 2011; Michaud et al., 2016a; Michaud et al., 2016b). The most recent large nationwide Danish study found no conclusive evidence of association between short-term exposure to wind turbine noise and MI (Poulsen et al., 2018). Rapidly increasing investments in wind-turbines worldwide, the intense debate regarding potential health effects and the lack of epidemiological studies, merit more research. Using a prospective design with long-term follow-up for MI in high quality and complete nationwide registries, we examine the association between long-term exposure to wind turbine noise and risk of MI in a large, nationwide cohort of Danish nurses.

2. Methods

2.1. Study population - the Danish nurse cohort

The Danish Nurse Cohort (Hundrup et al., 2012) was inspired by the American Nurses’ Health Study to investigate the health effects of hormone replacement therapy (HRT) in a European population. In 1993, the cohort was initiated by sending a questionnaire to 23,170 female members of the Danish Nurse Organization who were at least 44 years old at the time. The Danish Nurse Organization includes 95% of all nurses in Denmark. In total, 19,898 (86%) nurses replied, and the cohort was reinvestigated in 1999 when firstly 10,534 new nurses (who had reached the age of 44 years in the period 1993–99) were invited of which 8344 responded) and secondly 2231 non-responders from 1993 were re-invited of which 489 responded. The questionnaire included questions on socio-economic and working conditions, parents’ occupation, weight and height, lifestyle (diet, smoking, alcohol consumption and leisure time physical activity), self-reported health, family history of cardiovascular disease, use of oral contraceptives and hormone replacement therapy (HRT). In the present study we used the earliest baseline information from 1993 (19,898) or 1999 (8833) for 28,731 of nurses.

Since establishment of the Central Population Register in 1968, (Pedersen, 2011; Schmidt et al., 2014) all citizens of Denmark have been given a unique personal identification number, which allows accurate linkage between registers. The cohort members were linked to the Central Population Register (Pedersen, 2011; Schmidt et al., 2014) to obtain the nurses vital status information at 31st December 2013 (active, date of death/emigration). Using the unique personal identification number of the cohort members, all residential histories were traced in the Central Population Register between 1982 and 2013. Each residential address contained a unique identification code composed of a municipality-, road- and house number code. The dates the persons had moved to and from each address were noted. The addresses were then linked to a database of all official addresses and their geographical coordinates in Denmark.

2.2. Identification of outcome - National patient register and Danish registry of causes of death

The endpoint was incidence of MI (International Classification of Disease (ICD) 10: I21 and ICD 8:410), defined as first-ever hospital contact (emergency, in- or outpatient) for MI, identified in the Danish National Patient Registry, (Schmidt et al., 2015; Lyng et al., 2011) or out-of hospital death due to MI for those who did not have hospital contact for MI, identified in the Danish Registry of Causes of Death (Helweg-Larsen, 2011). The Danish National Patient Registry has collected nationwide data on all non-psychiatric hospital admissions since 1977, and since 1995, patients discharged from emergency departments and outpatient clinics have also been registered. Danish Registry of Causes of Death contains cause-specific mortality according to World Health Organizations guidelines since 1970 and ICD 10 codes since 1994. The Danish National Board of Health maintains the registers and assures the quality of the data. Participants with a discharge diagnosis or self-report of MI before enrolment into the Nurses Cohort were excluded.

2.3. Exposure assessment

2.3.1. Identification of Danish wind turbines

8768 on-shore wind turbines in operation at any time in Denmark from 1982 to 2013 (off-shore turbines were excluded, n = 510) were identified, using the administrative Master Data Register of Wind Turbines maintained by the Danish Energy Agency (Danish Energy Authority, 2017). It is mandatory for all wind-turbine owners to report to the register, which contains geographical coordinates, date of grid
connection, cancelation date for decommissioned turbines, and output for each Danish power producing wind turbine. Each of the turbines was classified into one of 99 noise spectra classes detailing the noise spectrum from 10 Hz to 10,000 Hz in thirds of octaves for wind speeds from 4 to 25 m/s, based on individual wind turbine data including height, model, type and operational settings (when relevant). These noise classes were formed from existing measurements of sound power for Danish wind turbines (Backalzr et al., 2016). At each wind-turbine location, the wind speed and direction at hub height was estimated, using mesoscale model simulations (Hahmann et al., 2015; Pena and Hahmann, 2017). Temperature and relative humidity at 2 m as well as the atmospheric stability were also estimated from these simulations.

2.3.2. Wind turbine noise exposure data

Each of the nurses’ homes was identified and geocoded. The noise assessment at each nurses’ homes from wind turbines was calculated according Nord2000 method (Backalzr et al., 2016). Each home address and each wind turbine were geocoded and the model takes into consideration, meteorological data for each wind turbine every hour throughout the years 1982–2013. The applied noise exposure modelling has been described in details elsewhere (Backalzr et al., 2016). In brief, wind turbine noise exposure was estimated for the all 1,278,630 different addresses the nurses had lived in using the Nord2000 noise propagation model which has been validated for wind turbines and previously detailed (Kragh et al., 2001; Sendergaard et al., 2009). For each home, the noise contribution from all wind turbines within a 6000 m radius was calculated hour by hour. Outdoor A-weighted sound pressure level (LAeq) at the most exposed façade of all buildings were calculated and exposure was aggregated as follows: day (Lday; 07:00–19:00 h), evening (Levening; 19:00–22:00 h), night (Lnight; 22:00–07:00 h), expressed as L10eq (the overall weighted 24-h noise level during the day, evening (+5 dB) and night (+10 dB)), and 24 h (un-weighted 24-h average), as yearly averages. Geographical coordinates were obtained for 99.9% of all the addresses.

2.3.3. Air pollution and noise from road traffic

As previously described in detail, (Jørgensen et al., 2016; Hansen et al., 2016) we used the newly updated, high-resolution Danish air pollution dispersion modelling system (AirGIS) to estimate exposure to outdoor air pollution at the residence (Jensen et al., 2001). Necessary input data for carrying out the exposure modelling has been established for the first time in Denmark (Kakosimos et al., 2010). Road traffic noise at residential addresses of the nurses was estimated using the Nord2000 model. The input variables for the traffic noise model include the geocodes of the location, the height of apartments above street level, road lines with information on yearly average daily traffic, traffic composition and speed, road type (motorway, rural highway, road wider than 6 m, and other road), building polygons for all surrounding buildings (height of buildings, etc.), and meteorology. Noise from road traffic was calculated at individual residential addresses for the period 1982–2013, as the equivalent continuous L10eq at the most exposed façade of the dwelling for the Lday L1evening, Lnight and L10eq as yearly averages.

2.4. Statistical analysis

We applied the Cox proportional hazards regression model to test the incidence of MI as a function of wind turbine noise exposure with age as the underlying time scale in all models, ensuring comparison of individuals of the same age. Start of follow-up was at the age on the date of recruitment (1st April 1993 or 1st April 1999), so nurses were considered at risk from recruitment, and end of follow-up was age at the date of first MI event, date of death, emigration or 31st December 2013, whichever came first. Nurses with an MI event before enrollment were excluded from the analyses. The effect of wind turbine noise was evaluated in several steps: Model 1) crude model, adjusted only for calendar year at recruitment into the cohort; Model 2) main, fully adjusted model, additionally adjusted for a-priori selected potential confounding variables that were both correlated to noise exposure and a risk factor for MI: smoking status (never, current, previous), smoking pack-years, alcohol consumption (g/week), physical activity (low, medium, high), the consumption of fruit (yes, no), avoidance of fatty meat consumption (yes, no), use of oral contraceptives, use of hormone therapy, employment status (employed, unemployed, retired, other) and marital status (married, separated, divorced, unmarried, widow). The main analysis was performed on the cohort with complete information on all the covariates included in Model 2.

We examined the following wind turbine noise exposures to assess chronic exposure using the 1-, 5- and 11-year rolling mean during follow-up prior to diagnosis/censoring. In each rolling mean window, we considered day-, evening-, night- and Ln10eq time exposure separately. To avoid enforcement of linearity between being exposed to wind turbine noise and not being exposed, two variables were used in these models; a binary variable distinguishing unexposed from exposed (0/1) and a continuous variable with the actual exposure for those exposed and the median exposure level for unexposed subjects. The continuous variable reflects the relative increase in hazard for ten units increase in exposure (10 dB) within the population of exposed nurses. A 10 dB increase in noise level is equivalent to a subjective doubling in loudness. (Stevens, 1955). Furthermore, we used categorical versions of wind turbine noise exposure based on type-specific baseline values. Wind turbine noise exposures were modelled as time-varying variables in all models.

We carried out sensitivity models to assess possible mediators of an association between wind turbine noise and MI in four additional separate models. Model 3) as for model 2, adjusted further for Body Mass Index (BMI); Model 4) as for model 2, adjusted further for self-reported hypertension at baseline; Model 5) as for model 2, adjusted further for self-reported diabetes at baseline and Model 6) as for model 2, adjusted further average gross income at the municipality at baseline, which we used as a proxy for socio-economic status. Continuous variables, year, smoking pack-years, alcohol consumption, BMI, and average gross income at the municipality were modelled with restricted cubic splines. Noise estimates and traffic air pollution were available for every calendar year at recruitment into the cohort; Model 2) main, fully adjusted model, additionally adjusted for a-priori selected potential confounding variables that were both correlated to noise exposure and a risk factor for MI: smoking status (never, current, previous), smoking pack-years, alcohol consumption (g/week), physical activity (low, medium, high), the consumption of fruit (yes, no), avoidance of fatty meat consumption (yes, no), use of oral contraceptives, use of hormone therapy, employment status (employed, unemployed, retired, other) and marital status (married, separated, divorced, unmarried, widow). The main analysis was performed on the cohort with complete information on all the covariates included in Model 2.

The potential effect modification of the association between wind turbine noise among exposed Nurses and MI incidence by age, night shift work, obesity, road traffic noise/N0x, traffic related air pollution and urbanicity index was examined by introducing interaction terms to the main linear model (model 2) and tested by the likelihood ratio test. Furthermore, the association between noise exposure and MI was also estimated as non-linear trends in wind-turbine noise concentrations using restricted cubic splines.

The cohort consists of elderly nurses (> 58 years old at the end of follow-up in 2013), thus the effect of non-MI death as a competing risk was also investigated as a function of wind turbine noise to assess whether time to MI in our main models was precluded by death.

All effects are reported as cause-specific hazard ratios (HRs) and 95% confidence intervals (CIs). All analysis and graphical presentations were performed using the R statistical software 3.2.0 (with packages: survival, rms, Epi., maptools, OpenStreetMaps).

Spearman correlation between metrics of wind-turbine noise and traffic noise and air pollution were estimated and these were not correlated (r = 0.14).

Research was conducted in accordance with principles of the Declaration of Helsinki and the Danish Nurses Cohort study was approved by the Scientific Ethics Committee for Copenhagen and Frederiksberg and written informed consent was obtained from all participants prior to enrollment. The present register based study was approved by the Danish Data Protection Agency (J.nr: 2016-41-4792).
3. Results

Of the total 28,731 recruited nurses in the Danish Nurses Cohort, we excluded 4 who died or emigrated before start of follow-up, 109 who had reported having MI via questionnaire at the cohort baseline, and additional 186 who didn’t report having MI, but were registered with a discharge diagnosis of MI event in the Danish National Patient Registry before baseline. We additionally excluded 4420 nurses with missing information on covariates and 18 due to missing address information or inability to geocode address, leaving 23,994 nurses for the final analyses.

Mean follow-up was 20.0 years giving a total of 412,386 person-years of observations, during which 686 nurses developed MI, with an incidence rate of 1.7 new cases per 1000 person-years, which is comparable to the standardized incidence rate for women in Denmark in a similar period (2.1 per 1000 person-years in 1984 and 1.3 per 1000 person-years in 2008) (Schmidt et al., 2012).

The nurses who were registered with MI were older, had higher BMI, smoked more, consumed less alcohol, were less physically active, ate more fatty meat, ate slightly less fruit, had higher rates of hypertension, diabetes and HRT usage, but lower rates of ever using oral contraceptives, tended to be retired, lived in areas with slightly lower income, were exposed to higher levels of NOx traffic related air pollution, but around the same levels of annual weighted road traffic noise at baseline than nurses who were not registered with an MI event within the follow-up period. At baseline, the nurses registered with MI were exposed to similar levels of wind turbine noise as those without MI (Table 1).

Nurses from Danish Nurse Cohort resided all around Denmark with wide geographical variation (Fig. 1), with 14.8% residing in urban areas (population density ≥ 5,220 persons/km²), 42.4% in provincial towns (180–5220 persons/km²) and 40.3% in rural areas (< 180 persons/km²) at the cohort baseline, which corresponds closely to the distribution of the Danish population. The estimated residential noise levels from wind turbines at baseline and distance to wind-turbine varied greatly, as did the proportion of population within the 600-m radius from wind turbine, throughout follow-up, with around 10% (n = 1748) exposed in 1993, almost 15% (n = 3971) in 2002 and 13% (n = 3058) in 2013 (Fig. 2). Mean (standard deviation) wind turbine noise levels among exposed nurses were 26.1 (6.5) dB in 1993, 26.3 (7.1) dB in 2002, and 26.4 (6.7) dB in 2013. At the cohort baseline in 1993 or 1999, mean baseline residential noise levels among exposed nurses were slightly higher in those who developed MI (26.6 dB) and among those who didn’t develop MI (26.2 dB).

Compared to 21,493 unexposed nurses at the cohort baseline, the 2501 exposed nurses were slightly younger, had higher BMI, smoked less, were less physically active, had slightly higher rates of diabetes and oral contraceptive use, but lower HT use, tended to still be working, lived in rural rather than urban areas, had slightly lower incomes, were exposed to higher levels of NOx traffic related air pollution, but around the same levels of annual weighted road traffic noise but were similar in regards to hypertension, avoiding consumption of fatty meats, fruit consumption, and diabetes rates. Among these baseline non-exposed nurses, 614 developed MI during a mean follow-up of 20 years and 370,226.5 person-years, and incidence rate of 1.7 per 1000 person-years whilst 72 of the baseline exposed nurses developed MI within 42,159.6 person-years, with an identical incidence rate of 1.7 per 1000 person-years (Supplementary Table S1).

Table 2 shows the associations between weighted wind-turbine noise L_{den} and MI (hospitalization or death due to MI) incidence (n = 686) among 23,994 Danish Nurse Cohort participants. We found no association between wind turbine noise and MI incidence: adjusted hazard ratios (HRs) and 95% confidence intervals (CI s) comparing nurses with 11-years running meanresidential noise levels of < 21.5 dB, 21.5–25.4 dB, 25.4–29.9 dB, and > 29.9 dB, to non-exposed nurses
were 0.89 (0.64–1.25), 1.20 (0.82–1.77), 1.38 (0.95–2.01), and 0.88 (0.53–1.28), respectively with no dose-response relationship (Table 2, Supplementary Fig. S1). HR estimation as non-linear trends in wind-turbine noise concentrations (L_{den} for 11-, 5- and 1-year rolling mean) using restricted cubic splines showed no linear effect with all P-values for non-linear effect above 0.05 (results not shown). The corresponding HR (95% CI) for the linear estimate of the association between 11-year mean levels of wind turbine noise (per 10 dB increase) with MI incidence was 0.99 (0.77–1.28). Similar associations were observed 5-, and 1-year running means, with almost all of the CI’s spanning one and with no evidence of dose-response.

There were no marked differences in HR of MI when comparing effects of outdoor A-weighted sound pressure wind turbine noise level (L_{Aeq}) at the most exposed façade of all buildings aggregated as day (L_d; 07:00–19:00 h), evening (L_e; 19:00–22:00 h), night (L_n; 22:00–07:00 h), and 24 h (unweighted 24-h average) (Supplementary Table, S2) when compared to these effects weighted as L_{den}. Alternative analyses with L_{den}, L_d, L_e, and L_{24hr unweighted} categorized with cut-off value at 20 dB (> and < 20 dB), with reference group including non-exposed nurses together with nurses who lived close to wind turbines and were exposed to noise levels below 20 dB, showed weak positive but statistically non-significant association with MI (Supplementary Table S3).

3.1. Identification of confounders, mediators and effect modifiers

The effect of the included a-priori selected confounders (smoking, alcohol, physical activity, diet, oral contraceptives, hormone therapy, employment status, marital status) in model 2 compared to the crude model was very minor (Table 2) and HRs were only slightly attenuated in the fully adjusted model.

There was no evidence of effect mediation by BMI, self-reported hypertension, diabetes or socio-economic status (municipal level gross average annual income as a proxy) in sensitivity analyses with no marked deviation from the main model 2 (Table 2, models 3–6). Nor was there evidence of effect modification by age, obesity, road traffic noise/pollution or urbanicity index (Table 3).

3.2. Competing risk by non-MI death

The number of competing events within the cohort during follow-up was high (non-MI death, n = 3745), compared to the outcome of interest (MI, n = 686), but when assessing competing risk in model 2 (the main model), there was no indication of association between wind turbine noise exposure and non-MI death in our data. Thus, providing no evidence supporting non-MI death as being a competing event that masks the association of interest in this study.

4. Discussion

In this nationwide, prospective cohort study of Danish female
nurses, we found little evidence to support a causal relationship between long-term exposure to wind turbine noise and MI incidence, within the exposure windows considered (11-, 5- and 1-year).

The relationship between wind turbine noise exposure and MI incidence is largely unexplored and the results of this comprehensive and large nationwide prospective cohort with a representative distribution of over 1,278,630 different present and historical addresses around entire Denmark sheds important new light to this relationship. We benefited from objective assessment of MI incidence based on high quality Danish registries with near 100% coverage, (Schmidt et al., 2015; Lyngø et al., 2011; Helweg-Larsen, 2011) as well as detailed information on MI risk factors. This assessment implies minimal possibility of recall and information bias and no selection bias, which has hampered previous studies on the topic relying on self-reports, as recruited persons in previous studies would be likely highly motivated to participate, if already annoyed or distressed by the wind turbine and their devalued homes. We furthermore benefited from the state-of-the-art high-resolution validated exposure models for wind turbine and road traffic noise (Backalarz et al., 2016; Kragh et al., 2001; Søndergaard et al., 2009) as well as air pollution, (Kakosimos et al., 2010; Ketzel et al., 2011) which were based on geocodes and also accounted for all address changes, meteorological conditions as well as the size and the type of wind turbines. Overall associations support no association with most CIs spanning one, and the few HRs above one are thought to be chance findings or due to residual confounding and not true effects. This is also supported by the lack of a dose-response association.

Two previous Canadian cross-sectional studies have investigated the association between long-term outdoor wind turbine noise exposure and cardiovascular disease (high blood pressure and heart disease) and reported no association in accordance with our study (Pedersen, 2011; Michaud et al., 2016a; Michaud et al., 2016b). However, those studies were small (ca. 1200 participants) and relied on self-reported outcome data. Most recently, a large nationwide Danish study used a case-crossover design, and MI incidence based on MI hospitalization and death registrations, (Poulsen et al., 2018) those authors only consider the short-term effects of low frequency nighttime indoor wind turbine noise exposure and report some suggestion of acute effects (Poulsen et al., 2018). Although that study is in line with ours, the results are not directly comparable, as we only study effects of long-term exposure to outdoor wind turbine noise with no inclusion of indoor noise exposure. The authors did however consider outdoor nighttime noise exposure, reporting a protective effect in relation to MI risk, which we could not replicate in our present study (Supplementary table S4), possibly due to the low numbers of cases in our cohort exposed to wind turbine levels > 42 dB(A).

In the present study the wind turbine noise levels were relatively low. Only 25% of nurses exposed nurses living within a 6000-m radius of one or more wind turbines (equivalent to around 3% of all included nurses) were exposed to levels over 29.9 dB(A) throughout follow-up, as a large proportion of the included nurses had never lived in proximity of a wind turbine. According to the World Health Organization it is not plausible that noise levels at and below 30 dB(A) would cause sleep disturbances, and that only modest health effects would be expected at and below 40 dB(A) (WHO, 2011). This may imply that the noise levels in our study may not have induced intermediates (high blood pressure, sleep disturbance, etc.) previously reported to be on the causal pathway from noise exposure to MI, (Munzel et al., 2017a; Munzel et al., 2017b; Munzel et al., 2017c; Babisch et al., 2006) and direct auditory effects leading to MI at these levels are not expected (Munzel et al., 2017c). These levels of wind turbine noise are also substantially lower than road traffic noise levels within the same cohort, which were over 50 dB(A) in average, noting that a 20 dB(A) difference between these two sources of noise levels is perceived as around four times the loudness, due to the logarithmic scale of sound. (Stevens, 1955)

We found no evidence of confounding or effect modification of the relationship between wind turbine noise and MI in the present study, implying a robust estimate. Although valid, the information on confounding and effect modifying variables were collected at cohort baseline, and we acknowledge that these may have changed throughout the 20-year average follow-up time.

A major limitation in our present study is the exposure

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**Fig. 2.** Average weighted $L_{den}$ for wind turbine noise exposure per year (right axis) and proportion of women living at wind turbine noise exposed addresses (left axis).
<table>
<thead>
<tr>
<th>Person-years (PY)</th>
<th>N cases</th>
<th>Incidence Rate per 1000 PY</th>
<th>Model 1 HR (95% CI)</th>
<th>Model 2 HR (95% CI)</th>
<th>Model 3 HR (95% CI)</th>
<th>Model 4 HR (95% CI)</th>
<th>Model 5 HR (95% CI)</th>
<th>Model 6 HR (95% CI)</th>
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<tr>
<td><strong>L_{den}, 11-year rolling mean</strong></td>
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<tr>
<td>Unexposed&lt; 21.5 dB(A)</td>
<td>344,749</td>
<td>577</td>
<td>1.7</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
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<tr>
<td>21.5–25.4 dB(A)</td>
<td>27,290</td>
<td>37</td>
<td>1.4</td>
<td>0.90 (0.65–1.26)</td>
<td>0.89 (0.64–1.25)</td>
<td>0.87 (0.62–1.22)</td>
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<tr>
<td>25.4–29.9 dB(A)</td>
<td>14,218</td>
<td>27</td>
<td>1.9</td>
<td>1.24 (0.84–1.82)</td>
<td>1.20 (0.82–1.77)</td>
<td>1.19 (0.81–1.75)</td>
<td>1.22 (0.83–1.80)</td>
<td>1.19 (0.81–1.76)</td>
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<tr>
<td>&gt; 29.9 dB(A)</td>
<td>13,190</td>
<td>29</td>
<td>2.2</td>
<td>1.39 (0.95–2.02)</td>
<td>1.38 (0.95–2.01)</td>
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<tr>
<td>Linear trend per 10 dB(A)</td>
<td>686</td>
<td>0.98 (0.77–1.25)</td>
<td>0.99 (0.77–1.28)</td>
<td>1.02 (0.79–1.31)</td>
<td>0.99 (0.77–1.24)</td>
<td>0.99 (0.77–1.27)</td>
<td>0.97 (0.75–1.25)</td>
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<tr>
<td><strong>L_{den}, 5-year rolling mean</strong></td>
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<tr>
<td>Unexposed&lt; 21.5 dB(A)</td>
<td>353,868</td>
<td>591</td>
<td>1.7</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
</tr>
<tr>
<td>21.5–25.4 dB(A)</td>
<td>17,722</td>
<td>25</td>
<td>1.4</td>
<td>0.95 (0.64–1.42)</td>
<td>0.93 (0.63–1.40)</td>
<td>0.93 (0.62–1.39)</td>
<td>0.93 (0.62–1.38)</td>
<td>0.93 (0.62–1.38)</td>
</tr>
<tr>
<td>25.4–29.9 dB(A)</td>
<td>13,235</td>
<td>26</td>
<td>1.9</td>
<td>1.31 (0.89–1.95)</td>
<td>1.28 (0.86–1.90)</td>
<td>1.27 (0.86–1.89)</td>
<td>1.31 (0.88–1.94)</td>
<td>1.28 (0.86–1.90)</td>
</tr>
<tr>
<td>&gt; 29.9 dB(A)</td>
<td>13,707</td>
<td>28</td>
<td>2.0</td>
<td>1.33 (0.91–1.94)</td>
<td>1.32 (0.90–1.93)</td>
<td>1.31 (0.89–1.92)</td>
<td>1.32 (0.90–1.94)</td>
<td>1.31 (0.89–1.92)</td>
</tr>
<tr>
<td>Linear trend per 10 dB(A)</td>
<td>686</td>
<td>1.02 (0.77–1.36)</td>
<td>1.04 (0.78–1.39)</td>
<td>1.04 (0.78–1.40)</td>
<td>1.05 (0.78–1.41)</td>
<td>1.04 (0.78–1.39)</td>
<td>1.02 (0.76–1.38)</td>
<td></td>
</tr>
<tr>
<td><strong>L_{den}, 1-year rolling mean</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Unexposed&lt; 21.5 dB(A)</td>
<td>361,902</td>
<td>604</td>
<td>1.7</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
<td>1 (ref)</td>
</tr>
<tr>
<td>21.5–25.4 dB(A)</td>
<td>10,566</td>
<td>13</td>
<td>1.2</td>
<td>0.83 (0.48–1.43)</td>
<td>0.83 (0.48–1.44)</td>
<td>0.83 (0.48–1.43)</td>
<td>0.82 (0.47–1.42)</td>
<td>0.81 (0.47–1.41)</td>
</tr>
<tr>
<td>25.4–29.9 dB(A)</td>
<td>12,154</td>
<td>30</td>
<td>2.5</td>
<td>1.66 (1.15–2.39)</td>
<td>1.60 (1.11–2.32)</td>
<td>1.59 (1.10–2.30)</td>
<td>1.63 (1.13–2.36)</td>
<td>1.60 (1.11–2.31)</td>
</tr>
<tr>
<td>&gt; 29.9 dB(A)</td>
<td>13,488</td>
<td>25</td>
<td>1.9</td>
<td>1.22 (0.82–1.82)</td>
<td>1.21 (0.81–1.82)</td>
<td>1.20 (0.80–1.79)</td>
<td>1.22 (0.81–1.82)</td>
<td>1.21 (0.81–1.80)</td>
</tr>
<tr>
<td>Linear trend per 10 dB(A)</td>
<td>686</td>
<td>0.82 (0.57–1.17)</td>
<td>0.83 (0.58–1.19)</td>
<td>0.83 (0.58–1.19)</td>
<td>0.83 (0.58–1.19)</td>
<td>0.83 (0.58–1.19)</td>
<td>0.77 (0.53–1.21)</td>
<td></td>
</tr>
</tbody>
</table>

HR: Hazard Ratio; CI: Confidence Intervals;
* Unexposed: these participants have not lived in proximity of a wind turbine within the rolling mean window. Cut-offs for wind-turbine noise exposures among exposed nurses are based on type-specific baseline quartiles.
  a Adjusted for age (underlying timeline) and calendar year at entrance into the cohort;
  b Main model, as for model 1 + smoking (status, pack-years), alcohol consumption, physical activity, avoid fatty meat consumption, fruit consumption, use of oral contraceptives, use of hormone therapy, marital status, employment status;
  c As for model 2 + body mass index; Note that the number of nurses in this analysis is different from model 2 due to missing information on body mass index;
  d As for model 2 + self-reported hypertension at baseline; Note that the number of nurses in this analysis is different from model 2 due to missing information on self-reported hypertension at baseline;
  e As for model 2 + self-reported diabetes at baseline; Note that the number of nurses in this analysis is different from model 2 due to missing information on self-reported diabetes at baseline;
  f As for model 2 + gross annual average income at the municipality level at baseline; Note that the number of nurses in this analysis is different from model 2 due to missing information on gross average income at the municipality level at baseline;
misclassification in modelled wind turbine noise concentrations since these are only proxies of personal exposure. Although our estimation of wind turbine noise exposure is based on complete residential histories, we cannot account for exposures via temporary migration to other destinations, at work in other regions in Denmark or whilst overseas in areas with either higher or lower noise exposures. Also, we had no access to individual information related to bedroom orientation to the closest wind turbine or noise exposure moderators such as façade insulation measures, window types. Additionally, the A-weighted nature of our estimates is not informative about any peaking characteristics of the wind turbine noise throughout follow up. So although, the average A-weighted wind turbine noise levels we report are in fact in accordance with the noise limits for wind turbines as specified by statutory order of the Danish Environmental Protection Agency; the limits of 44 dB(A) (wind speed of 8 m/s) and 42 dB(A) (wind speed of 6 m/s) and for dwellings in open country being for dwellings and summer cottages: 39 dB (wind speed of 8 m/s), (Statutory Order on Noise from Wind Turbines, Statutory Order no.1284 of 15. December 2011, 2011) there may have been peaks we didn’t address.

Another major weakness of our study is the small number of MI cases exposed to high levels of wind turbine noise, limiting the power to detect effects in this range of noise exposure. Furthermore, we had no available information on personal sensitivity to noise, levels of annoyance or sleep quality, which have all been reported to be on the casual pathway between noise exposure and health effects (Hanning and Evans, 2012; Bakker et al., 2012; Kuwano et al., 2013; Shepherd et al., 2011; McCallum et al., 2014). Albeit, these self-reports may have introduced bias as they include highly motivated persons with possible negative attitudes to wind turbines which have been repeatedly reported to play an important role as the underlying cause of reported health and sleep problems (Pedersen et al., 2009; Knopper et al., 2014; Blanes-Vidal and Schwartz, 2016; Pedersen, 2011). In our study it was not feasible to consider all noise sources including noise from neighbours, bedroom snoring, aircraft, railways and ventilation. Selander et al. found increased effects estimates of noise in relation to MI when excluding all those exposed to other noise sources as well as hearing impaired persons (Selander et al., 2009). Hence, we may have underestimated the effects of wind turbine noise in our present study. Another weakness is that we lacked data on personal and household income, important determinants of socio-economic status. Finally, we only consider women, and are thus unable to account for eventual differences in effect according to gender, albeit the effects of gender do remain unclear with some studies showing stronger effects among men (Babisch et al., 2005; Huss et al., 2016; Jarup et al., 2008) and others reporting no difference in gender (Selander et al., 2009; Sorensen et al., 2011; Beelen et al., 2009).

5. Conclusions

The results of this study infer no causal association between long-term exposure to wind turbine noise and MI in women above age 44. However, there were only few cases in the highest exposure groups and our findings need reproduction.

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Disclosures

None.

Author contribution statement

EVB drafted the manuscript. AKD performed statistical analyses and contributed to the manuscript preparation. ZJA contributed to the concept and design for the study, secured funding, prepared data for analyses, and supervised AKD in statistical analyses. JTJ contributed with data clean-up. MKS provided all nurse cohort data. CB, JEL and THP calculated all wind turbine noise estimations and provided expertise on noise and sound. All authors contributed to critical interpretation of data, and the final draft of the manuscript.

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Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2018.10.011.

References
