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Short-term nighttime wind turbine noise and cardiovascular events: A nationwide case-crossover study from Denmark

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ABSTRACT

Aims: The number of people exposed to wind turbine noise (WTN) is increasing. WTN is reported as more annoying than traffic noise at similar levels. Long-term exposure to traffic noise has consistently been associated with cardiovascular disease, whereas effects of short-term exposure are much less investigated due to little day-to-day variation of e.g. road traffic noise. WTN varies considerably due to changing weather conditions allowing investigation of short-term effects of WTN on cardiovascular events.

Methods and results: We identified all hospitalisations and deaths from stroke (16,913 cases) and myocardial infarction (MI) (17,559 cases) among Danes exposed to WTN between 1982 and 2013. We applied a time-stratified, case-crossover design. Using detailed data on wind turbine type and hourly wind data at each wind turbine, we simulated mean nighttime outdoor (10–10,000 Hz) and nighttime low frequency (LF) indoor WTN (10–160 Hz) over the 4 days preceding diagnosis and reference days. For indoor LF WTN between 10 and 15 dB (A) and above 15 dB(A), odds ratios (ORs) for MI were 1.27 (95% confidence interval (CI): 0.97–1.67; cases = 198) and 1.62 (95% CI: 0.76–3.45; cases = 21), respectively, when compared to indoor LF WTN below 5 dB(A). For stroke, corresponding ORs were 1.17 (95% CI: 0.95–1.69; cases = 166) and 2.30 (95% CI: 0.96–5.50; cases = 15). The elevated ORs above 15 dB(A) persisted across sensitivity analyses. When looking at specific lag times, noise exposure one day before MI events and three days before stroke events were associated with the highest ORs. For outdoor WTN at night, we observed both increased and decreased risk estimates.

Conclusion: This study did not provide conclusive evidence of an association between WTN and MI or stroke. It does however suggest that indoor LF WTN at night may trigger cardiovascular events, whereas these events seemed largely unaffected by nighttime outdoor WTN. These findings need reproduction, as they were based on few cases and may be due to chance.

1. Introduction

As the number of wind turbines (WT) has increased so has concern about potential health effects, particularly since WT noise (WTN) has been reported to be more annoying than noise from other sources at similar levels (Janssen et al., 2011). Also, some (Schmidt and Klokker, 2014) but not all (Jalali et al., 2016; Michaud et al., 2016c) studies have found an association with sleep disturbances.

Noise can act as a stressor and provoke a typical stress response, including hyperactivity of the sympathetic autonomic nervous system and activation of the hypothalamus-pituitary-adrenal axis. Nighttime noise exposure is considered particularly hazardous (Babisch et al., 2005; WHO, 2009) and has been associated with disturbance of sleep, from full awakenings to unconscious autonomic perturbations, such as sleep stage changes and body movements (Griefahn et al., 2008; Miedema and Vos, 2007); the latter from outdoor noise levels of down to 30 dB (WHO, 2009). Nighttime noise exposure has been associated with reduced cardiac parasympathetic tone, high blood pressure, endothelial dysfunction, oxidative stress and increased levels of stress hormones shortly after noise exposure or on the morning after (Graham et al., 2009; Schmidt et al., 2013). Evidence from cardiac arousals does not suggest pronounced habituation to nighttime noise (Basner et al., 2011; Muzet, 2007). Long-term residential exposure to transportation noise has consistently been associated with increased risk of cardiovascular diseases (Halonen et al., 2015; Sorensen et al., 2011; Vienneau et al., 2015), whereas it is unknown whether short-term exposure to

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noise can trigger a cardiovascular event due to lack of studies (Recio et al., 2016). These results are, however, not readily applicable to WTN: WTN levels are typically lower than those reported in relation to health effects of traffic noise. and WTN is reported as more annoying than traffic noise at similar sound levels (Janssen et al., 2011). Also, WTs are typically erected in rural areas and amplitude modulation gives WTN a rhythmic quality different from that generated by car tires. Furthermore, levels of WTN depend on wind speed and direction and hence vary more unpredictably than road traffic noise, permitting investigation of acute effects of noise exposure. Such effects are virtually unexplored, even though factors affected by noise exposure, including increased blood pressure and oxidative stress, are believed to be important triggers of stroke and myocardial infarction (MI) (Biasucci et al., 2008; McColl et al., 2009).

Studies from Canada (1238 participants) and Sweden and the Netherlands (1755 participants) investigated associations between long-term outdoor WTN and self-reported cardiovascular diseases (high blood pressure and heart disease) (Michaud et al., 2016b; E Pedersen 2011). The study from Canada additionally investigated hair cortisol levels, resting heart rate and blood pressure collected at the time of interview (Michaud et al., 2016a). Neither study found any association. However, as most scientific literature on WTN (Schmidt and Klokker, 2014), the studies were cross-sectional, relied on self-reported data and had few participants potentially exposed to WTN levels above 40–45 dB. Also, their exposure metrics did not reflect day-to-day variations in WTN, making the results relevant mainly for long-term health effects.

Denmark is a densely-populated country with a high number of residents living close to WTs. This provides a unique opportunity to investigate acute effects of WTN on stroke and MI.

2. Methods

2.1. Study base and noise exposure assessment

The study was based on the Danish population, where all citizens since 1968 have been assigned a personal identification number by the Central Population Register, allowing residents to be tracked in and across all Danish health and administrative registers (CB Pedersen 2011).

We identified all WTs (7860) in operation in Denmark any time between 1980 and 2013, from the administrative Master Data Register of Wind Turbines maintained by the Danish Energy Agency. The register, to which reporting is mandatory for all WT owners, contained cadastral codes and geographical coordinates for each WT from the WT owner. For WTs in operation at the time of data extraction, the register also contained coordinates from the Danish Geodata Agency. In case of disagreement between the recorded geographical locations, the WT location was validated against aerial photographs and historical topographic maps of Denmark. We excluded 517 offshore WTs and 87 WTs for which a credible location could not be established. Moreover, 314 WTs wrongly recorded in the Master Data Register were assigned coordinates based on maps and aerial photographs. Information on height, model, type and operational settings (when relevant) was gathered for all WTs, based on which each WT 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. These noise classes were formed from existing measurements of sound power for Danish WTs (details in (Backalarz et al., 2016; Søndergaard and Backalarz, 2015)).

For each WT location, we estimated the hourly wind speed and direction at hub height for the period 1982–2013, using mesoscale model simulations performed with the Weather Research and Forecasting model (Hahmann et al., 2015; Peña and Hahmann, 2017). From these simulations, we also extracted the temperature and the relative humidity at 2 m height as well as the atmospheric stability at each WT location.

The applied noise exposure modelling has been described in details elsewhere (Backalarz et al., 2016). In summary, we used a two-step approach. First, we identified buildings eligible for detailed noise modelling, corresponding to all dwellings in Denmark that could experience at least 24 dB(A) outdoor noise or 5 dB(A) indoor low frequency (LF, 10–160 Hz) noise under the unrealistically extreme scenario that all WTs ever standing in Denmark were simultaneously operating at a wind speed of 8 m/s with downwind sound propagation in all directions. In the second step, we performed a detailed modelling of noise exposure for the 553,066 buildings identified in step one: We calculated noise levels in 1/3 octave bands from 10 to 10,000 Hz using the Nord2000 noise propagation model (Kragh et al., 2001), taking into account time varying wind speed and direction, temperature, relative humidity and atmospheric stability. The model has been successfully validated for WTs (Søndergaard et al., 2009). For each dwelling, the noise contribution from all WTs within a 6000 meters radius was calculated hour by hour. For each night these modelled values were then aggregated over the period 10 pm to 7 am (nighttime), which is considered the most relevant time-window, because people are most likely to be at home as well as sleep during these hours. We calculated outdoor A-weighted sound pressure level at the front door of all buildings. We also calculated A-weighted indoor LF (10–160 Hz) sound pressure level for each dwelling using existing data on sound attenuation in this frequency range. All dwellings were classified into one of six sound insulation classes based on building attributes in the Building and Housing register (Christensen, 2011): “1½-story houses” (residents assumed to sleep on the second floor), “light façade” (e.g. wood), “aerated concrete” (and similar materials including timber framing), “farm houses” (remaining buildings in the registry classified as farms), “brick buildings” and “unknown” (assigned the mean attenuation value of the five previous classes). For each of the six classes, the frequency-specific attenuation values subtracted from the outdoor noise can be found elsewhere (Backalarz et al., 2016).

For each dwelling, we determined a validity score for the noise estimate for each night. This score summed up information for all contributing WTs on the number of measurements used to determine the WTN spectra class, and how closely the simulated meteorological conditions of each night resembled the conditions under which the relevant WTN spectra were measured.

2.2. Study population and identification of outcomes

From the Danish Civil Registration System (CB Pedersen 2011), we identified our study population defined as all adults (≥18 years) living in a dwelling that had on two separate days over the period 1982–2013 experienced at least 1 h with outdoor WTN above 30 dB(A). The last criteria reduced the population while retaining all potentially high exposed. In this study population, we identified all diagnoses of stroke (International classification of disease (ICD) 10: I61, I63, I64 and ICD 8: 431–434 and 436) or MI (ICD 10: I21 and ICD 8: 410) from the Danish National Patient Register (Lynge et al., 2011) and the Danish Register of Cause of Death (Helweg-Larsen, 2011). We excluded outpatients and patients found dead, because an exact date of event could not be reliably established. Admissions separated by at least 28 days were counted as separate events. We additionally required no hospitalisation for any reason in the 28 days preceding diagnosis. Also, we excluded cases who, at the time of diagnosis, had lived < 18 months at their present address or if the closest WT had not been the same for the past 18 months (to ensure that any observed effect was unrelated to environmental changes from moving address or changes in nearby WTs).

The study was approved by the Danish Data Protection Agency (J.nr: 2014-41-2671). By Danish Law, ethical approval and informed consent are not required for entirely register-based studies not involving contact with study participants.

Table 1
Characteristics of each of the study populations for all case events and for those associated with high levels of nighttime wind turbine noise exposure.

	Myocardial infarction			Stroke		
	All case events (N = 15,092)	> 36 dB(A) outdoor noise (N = 374)	> 10 dB(A) LF indoor noise (N = 219)	All case events (N = 14,623)	> 36 dB(A) outdoor noise (N = 302)	> 10 dB(A) LF indoor noise (N = 181)
Women	32%	28%	30%	45%	35%	40%
First hospital admission[a]	81%	83%	80%	83%	81%	89%
Age at diagnosis						
< 65 years	36%	41%	38%	27%	33%	33%
65–80 years	41%	42%	41%	42%	46%	44%
≥80 years	22%	17%	21%	31%	21%	23%
Year of diagnosis						
1982–1992	22%	32%	9%	13%	18%	7%
1993–2003	38%	39%	27%	41%	46%	31%
2004–2013	40%	30%	64%	46%	36%	62%
Living duration at same residence						
1.5–5 years	15%	11%	10%	18%	11%	7%
5–10 years	15%	12%	11%	15%	15%	10%
≥10 years	70%	77%	79%	67%	74%	82%
Type of dwelling						
Farm	14%	34%	38%	13%	34%	40%
Single–family detached homes	64%	55%	54%	58%	56%	57%
Others	22%	11%	7%	28%	11%	2%
Sound insulation class[b]						
1 1/2 story building	32%	48%	65%	28%	41%	66%
Light façade	1%	1%	3%	2%	1%	1%
Aerated concrete	4%	3%	14%	4%	4%	18%
Farmhouse	6%	13%	10%	6%	16%	10%
Brick façade	47%	29%	4%	19%	33%	4%
Unknown	10%	6%	5%	12%	5%	1%
Distance to closest wind turbine						
< 500 m	17%	95%	67%	16%	93%	55%
500–1000 m	47%	5%	29%	46%	7%	43%
1000–2000 m	35%	–	4%	37%	0%	3%
≥2000 m	1%	–	0%	1%	–	–
Total height, closest wind turbine						
< 25 m	16%	14%	5%	14%	18%	4%
25–50 m	53%	64%	21%	52%	54%	19%
50–75 m	25%	21%	59%	26%	25%	65%
75–100 m	5%	2%	12%	7%	3%	12%
≥100 m	1%	0%	2%	1%	0%	1%
Tree coverage, 500 meters radius from dwelling						
< 1%	13%	13%	14%	14%	16%	15%
1–10%	40%	56%	57%	39%	55%	52%
10–25%	39%	29%	24%	40%	27%	29%
≥25%	8%	2%	5%	8%	2%	3%
Distance to major road[c]						
< 2000 m	59%	52%	53%	60%	55%	49%
≥2000 m	41%	48%	47%	40%	45%	51%

[a] First time diagnosed with myocardial infarction or stroke respectively.
[b] All dwellings where classified into one of six sound insulation classes based on building attributes.
[c] Major road defined as ≥5000 vehicles per day.

2.3. Potential confounders and effect modifiers

Based on review of existing literature and biological plausibility we a priori selected the following potential confounders: temperature, humidity and air pollution, which have been found or suggested to be associated with cardiovascular events (Claeys et al., 2017; Zeng et al., 2017), as well as associated with WTN through associations with sound propagation or wind speed. As potential effect modifiers, we investigated tree coverage near each dwelling and road traffic. Additionally, we included wind speed that could conceivable mask WTN and affect cardiovascular risk.

We divided Denmark into 25 km * 25 km cells, providing cells that contained at least one WT. From the simulated data for all WT locations within each cell (Hahmann et al., 2015), the daily mean temperature and relative humidity at 2 m height as well as wind speed at 10 m height were calculated. Furthermore, for each dwelling we calculated the percentage of land covered by forest, thicket, groves, single trees and hedgerows within a 500 m radius using GIS data from the Danish Geodata Agency. For each dwelling, we assigned daily, regional background concentration of air pollution (NO_x) from the nearest of seven Danish locations for which background air pollution was modelled as hourly time series for the complete investigation period, using the validated Danish THOR air pollution modelling system. Lastly, as a proxy for local air pollution and road traffic noise exposure, we identified the distance from each dwelling to the nearest road with an average daily traffic count of ≥5000 vehicles in 2005.

2.4. Statistical analysis

We calculated odds ratios (ORs) for stroke and MI in a time-stratified case-crossover design using conditional logistic regression. The case-crossover study is well-suited to investigate effects of an intermittent exposure on the onset of acute disease outcomes, and is unique in that the case serves as his/her own control (Janes et al., 2005;

Table 2
Associations between short-term residential exposure to outdoor and indoor wind turbine noise during nighttime and myocardial infarction (MI) and stroke.

Exposure	Outcome	Mean lag 1–4 days			Lag 1		Lag 2		Lag 3		Lag 4	
		Case events	OR[a]	95% CI[b]	OR[a]	95% CI[b]	OR[a]	95% CI[b]	OR[a]	95% CI[b]	OR[a]	95% CI[b]
Outdoor wind turbine noise	MI											
< 24 dB(A)		8862	1		1		1		1		1	
24–30 dB(A)		4418	0.96	(0.90–1.02)	1.00	(0.95–1.05)	0.93	(0.88–0.98)	1.01	(0.96–1.06)	0.97	(0.92–1.03)
30–36 dB(A)		1438	0.94	(0.84–1.06)	1.02	(0.93–1.11)	0.95	(0.87–1.04)	0.96	(0.88–1.06)	1.03	(0.94–1.13)
36–42 dB(A)		310	0.95	(0.74–1.20)	0.98	(0.81–1.19)	0.95	(0.79–1.15)	0.96	(0.79–1.16)	1.07	(0.89–1.30)
≥ 42 dB(A)		64	0.54	(0.30–0.95)	1.13	(0.75–1.72)	0.94	(0.61–1.42)	0.87	(0.57–1.33)	0.79	(0.52–1.18)
Outdoor wind turbine noise	Stroke											
< 24 dB(A)		8525	1		1		1		1		1	
24–30 dB(A)		4431	1.01	(0.95–1.07)	0.97	(0.92–1.03)	0.99	(0.94–1.05)	1.03	(0.98–1.08)	1.01	(0.95–1.06)
30–36 dB(A)		1364	0.95	(0.84–1.08)	1.08	(0.98–1.18)	0.96	(0.88–1.06)	0.97	(0.88–1.07)	0.97	(0.88–1.07)
36–42 dB(A)		267	1.19	(0.92–1.54)	1.08	(0.88–1.32)	1.03	(0.84–1.25)	1.09	(0.90–1.33)	0.97	(0.79–1.18)
≥ 42 dB(A)		35	1.32	(0.65–2.67)	1.17	(0.66–2.08)	1.32	(0.77–2.27)	1.71	(0.97–3.01)	1.25	(0.70–2.26)
Indoor LF[c] wind turbine noise	MI											
< 5 dB(A)		14,042	1		1		1		1		1	
5–10 dB(A)		831	1.04	(0.91–1.18)	1.04	(0.94–1.17)	0.96	(0.86–1.08)	0.93	(0.83–1.04)	1.05	(0.94–1.18)
10–15 dB(A)		198	1.27	(0.97–1.67)	1.20	(0.96–1.51)	1.17	(0.93–1.47)	0.90	(0.72–1.14)	1.23	(0.98–1.53)
≥ 15 dB(A)		21	1.62	(0.76–3.45)	1.54	(0.87–2.72)	1.28	(0.71–2.34)	0.95	(0.54–1.67)	1.03	(0.58–1.82)
Indoor LF[c] wind turbine noise	Stroke											
< 5 dB(A)		13,682	1		1		1		1		1	
5–10 dB(A)		759	1.02	(0.89–1.17)	0.95	(0.85–1.07)	0.99	(0.88–1.11)	0.95	(0.85–1.07)	1.07	(0.95–1.20)
10–15 dB(A)		166	1.27	(0.95–1.69)	1.03	(0.81–1.31)	1.39	(1.10–1.75)	1.24	(0.98–1.56)	1.09	(0.86–1.38)
≥ 15 dB(A)		15	2.30	(0.96–5.50)	0.96	(0.45–2.04)	1.40	(0.72–2.73)	1.85	(0.97–3.54)	1.35	(0.69–2.66)

[a] OR: odds ratio; adjusted for ambient temperature (°C), relative humidity (%) and air pollution (NO_X); all included linearly.
[b] CI: confidence interval.
[c] Low frequency: 10–160 Hz.

Maclure, 1991). For each case event day, reference days were defined as all days within the month of diagnosis that were the same weekday. Our a priori defined main exposure metric was mean nighttime WTN (L_{pA} and L_{pALF}) over the period 1–4 days before the event; a time-span previously investigated for noise exposure (Recio et al., 2016) and air pollution (Andersen et al., 2010). We also investigated exposure on each of the four preceding days separately. Noise exposure was included categorically in the models: outdoor (< 24, 24– < 30, 30– < 36, 36– < 42 and ≥ 42 dB(A)) and indoor LF (< 5, 5– < 10, 10– < 15, and ≥ 15 dB(A)). We calculated ORs crude and adjusted for NO_x, temperature and relative humidity (averaged over same time-period as WTN in each model). In two additional analyses, we included wind speed and the second-degree polynomial of covariates.

We performed sensitivity analyses restricting the analyses to: 1) cases with no previous records of stroke or MI, 2) cases diagnosed in year 2000 or later, 3) cases living with < 1% tree coverage within 500 m of the dwelling (to avoid masking of the WTN noise from nearby vegetation; we applied a 500 m buffer as we assumed that vegetation further apart would be near indiscernible from background noise) and 4) cases living > 2000 m from a road with ≥ 5000 vehicles per day. Finally, we conducted analyses restricted to WTN estimates with high validity scores. Data were analysed using SAS 9.3 (SAS Institute Inc., Cary, NC, USA).

3. Results

We identified 17,559 events of MI and 16,913 events of stroke in the study population and excluded case events where the address (816 MI and 857 strokes) or nearest WT (1651 MI and 1433 strokes) had changed in the 18 months preceding diagnosis, yielding for analysis 15,092 MI events and 14,623 stroke events, corresponding to 13,343 and 13,026 persons, respectively.

Compared to all events, persons with high levels of nighttime WTN prior to their event were more likely to be male, younger, live in a building classified as a farm, have lived at the same address for > 10 years and have lived further from roads with dense traffic (Table 1). In addition, those with high indoor LF WTN levels had taller WTs near their home and were more likely to live in 1½-story houses. We found high correlations between all investigated WTN exposures (Supplement Table 1).

Table 2 shows associations between short-term exposure to nighttime outdoor WTN and hospitalisation or death from MI and stroke. The highest level (≥ 42 dB(A)) over the past 4 days was negatively associated with risk for MI (OR: 0.54, 95% confidence interval (CI): 0.30–0.95) and positively with risk for stroke (1.32, 95% CI: 0.65–2.67). Most of the study population was exposed to outdoor nighttime WTN below 24 dB(A), and only 64 MI events and 35 stroke events were associated with exposures exceeding 42 dB(A).

For nighttime indoor LF WTN above 10 dB(A), the risk tended to increase with increasing noise exposure over the past four days, with ORs for MI of 1.27 (95% CI: 0.97–1.67) for 10–15 dB(A) and 1.62 (95% CI: 0.76–3.45) for > 15 dB(A), and similarly for stroke the corresponding ORs were 1.27 (95% CI: 0.95–1.69) and 2.30 (95% CI: 0.96–5.50). In trend tests where we included mean exposure over past 4 days as a linear variable, we found no statistically significant trends (results not shown). Most of the study population was exposed to indoor LF WTN below 5 dB(A), and only few cases where exposed to > 15 dB (A). Analyses without adjustment for covariates (Supplementary Table 2), or further adjusted for their second-degree polynomial or for wind speed, did not substantially change risk estimates (results not shown).

When looking at the lag times for both outdoor and indoor nighttime WTN, the highest ORs were associated with noise exposure one day before MI events and three days before stroke events (Table 2).

Results of sensitivity analyses according to diagnosis after year 2000, first hospitalisation, tree coverage, and proximity to major roads did not deviate markedly from the main analysis results for indoor LF WTN (Table 3 and Supplement Table 3). Analyses of indoor LF WTN with high validity score reduced the ORs in the 10–15 dB(A) category but increased the ORs associated with exposures above 15 dB(A).

4. Discussion

This study found high levels of indoor nighttime LF WTN over the

Table 3
Sensitivity analyses of the associations between short-term residential exposure to indoor wind turbine noise exposure during nighttime (mean from day 1 to 4) and myocardial infarction (MI) and stroke.

Sensitivity analysis	Indoor LF[a] wind turbine noise (mean lag 1–4)	Myocardial infarction			Stroke		
		Case events	OR[b]	95% CI[c]	Case events	OR[b]	95% CI[c]
Main analysis (all)							
	< 5 dB(A)	14,042	1		13,682	1	
	5–10 dB(A)	831	1.04	(0.91–1.18)	759	1.02	(0.89–1.17)
	10–15 dB(A)	198	1.27	(0.97–1.67)	166	1.27	(0.95–1.69)
	≥ 15 dB(A)	21	1.62	(0.76–3.45)	15	2.30	(0.96–5.50)
Diagnosed after year 2000							
	< 5 dB(A)	7264	1		8286	1	
	5–10 dB(A)	638	1.13	(0.98–1.31)	605	0.99	(0.85–1.15)
	10–15 dB(A)	153	1.37	(1.01–1.87)	132	1.17	(0.86–1.59)
	≥ 15 dB(A)	16	1.62	(0.68–3.88)	12	2.48	(0.97–6.31)
First hospital admission for stroke/MI							
	< 5 dB(A)	11,379	1		11,395	1	
	5–10 dB(A)	694	1.10	(0.95–1.27)	646	1.02	(0.89–1.18)
	10–15 dB(A)	158	1.21	(0.89–1.65)	150	1.29	(0.95–1.74)
	≥ 15 dB(A)	18	1.59	(0.70–3.58)	11	2.01	(0.75–5.41)
< 1% tree coverage within 500 meters radius							
	< 5 dB(A)	1891	1		1886	1	
	5–10 dB(A)	104	0.80	(0.57–1.13)	93	1.07	(0.75–1.53)
	10–15 dB(A)	30	1.57	(0.74–3.35)	24	1.18	(0.55–2.54)
	≥ 15 dB(A)	–	–[d]		4	2.79	(0.47–16.61)
> 2000 m to major road[e]							
	< 5 dB(A)	6698	1		6360	1	
	5–10 dB(A)	492	1.11	(0.94–1.32)	431	0.98	(0.82–1.17)
	10–15 dB(A)	124	1.29	(0.91–1.82)	100	1.34	(0.94–1.93)
	≥ 15 dB(A)	7	1.11	(0.40–3.04)	12	2.68	(0.99–7.24)
Only exposure estimates with high validity score[f]							
	< 5 dB(A)	4398	1		4275	1	
	5–10 dB(A)	447	1.08	(0.91–1.28)	390	0.91	(0.76–1.09)
	10–15 dB(A)	92	0.92	(0.64–1.33)	88	1.10	(0.75–1.60)
	≥ 15 dB(A)	8	2.34	(0.67–8.24)	9	2.72	(0.62–11.89)

[a] Low frequency: 10–160 Hz.
[b] OR: odds ratio; adjusted for ambient temperature (°C), relative humidity (%) and air pollution (NOX); all included linearly.
[c] CI: confidence interval.
[d] This category contained less than three cases and was, therefore, pooled with the category above (10–15 dB(A)), because of Danish anonymity legislation.
[e] Major road: road with ≥ 5000 vehicles/day.
[f] Includes only case and reference periods with validity score better than the median among those with exposures above 10 dB(A). The validity score reflects the estimated uncertainty associated with all aspects of noise estimation at a specific address and day.

preceding days to be associated with increased risk estimates for both MI and stroke, whereas for outdoor nighttime WTN we observed higher risk estimates for stroke and lower risk estimates for MI. The number of cases exposed to > 15 dB(A) indoor LF WTN was, however, small, and the CIs generally spanned one.

While two studies have investigated long-term exposure to WTN and cardiovascular disease (Michaud et al., 2016b; E Pedersen 2011), no study has investigated short-term associations. For transportation noise, a recent case-crossover study from Madrid, using a citywide noise measure, found death from MI and cerebrovascular events (in people above 65 years of age) to be associated with noise exposure 0–1 days before an event (Recio et al., 2016). The study, however, investigated a range of exposure/endpoint combinations, and direct comparison to the present study is difficult, as they investigated only mortality and used ecological exposure data. Also, noise exceeded 55 dB on all nights, which is substantially higher than the noise levels in the present study.

We found no short-term associations between nighttime exposure to outdoor WTN below 36 dB(A) or indoor LF WTN below 10 dB(A) and risk of hospitalisation or death from MI or stroke. For outdoor WTN above 42 dB(A) the OR was decreased for MI but increased for stroke. We are not aware of plausible biological mechanisms to explain a protective effect of WTN; particularly one that would affect the risk of MI and stroke in opposite direction, and we consider that the association between MI and high levels of nighttime WTN is likely to be a chance finding. Among cases exposed to indoor nighttime WTN above 10 dB(A), we found all ORs to be above unity, with the highest estimates in the highest exposure categories. This tendency was seen for both MI and stroke, and while the elevation in 10–15 dB(A) category disappeared in a sensitivity analysis of only the most valid WTN data, the elevated risk associated with the highest exposure was consistent across a range of sensitivity analyses. However, we cannot rule out chance as an explanation for the observed results, as the number of case events preceded by the highest exposure levels was small and the wide CIs spanned one.

An important strength of our study is the nationwide design, including all cases of MI and stroke in Denmark with relevant WTN exposure since 1982. The identification of cases and addresses from high quality registers (Christensen, 2011; Lynge et al., 2011; CB Pedersen 2011) and modelled noise and weather parameters minimized the potential for participation or information biases. Additionally, we estimated noise levels specifically for nighttime, when people are most likely to be at home sleeping. A further strength is the detailed modelling of day-to-day residential nighttime WTN, based on hourly wind speed and direction at each WT position, combined with detailed WTN spectra. Furthermore, we estimated the potentially more biologically relevant indoor noise exposure, accounting for different housing sound insulation properties. We were, however, only able to differentiate into few insulation categories and had to classify each dwelling based on

relatively crude information. There is inevitably uncertainty in the modelled indoor and outdoor exposure metrics. As this is unrelated to case status, it is unlikely to bias the highest exposure category away from the null, while it may do so for intermediate categories. Accordingly, sensitivity analyses restricted to the most valid noise exposure measures did not decrease the ORs pertaining to LF WTN above 15 dB (A).

The applied case-crossover design, with reference days temporally close to the date of diagnosis, controls perfectly for constant and slow varying characteristics related to individuals (such as gender, education and medical conditions) or dwellings. We adjusted for potential environmental confounders, including wind speed, and although residual confounding due to the spatial resolution of these cannot be ruled out entirely, it is unlikely to have had a major effect on the risk estimates, as these were virtually unaffected by adjustment for our environmental confounders. Finally, the main limitation of the study is that despite including all relevant cases in Denmark, statistical power was impaired by having relatively few cases with high WT noise exposure. As the number of highly exposed cases is likely to be low in all populations, more studies should be conducted to facilitate meta-analysis. In addition, laboratory or field studies with direct monitoring of cardiovascular parameters and noise exposure might be informative.

4.1. Conclusions

The results did not show conclusive evidence of an association between nighttime WTN and MI or stroke. However, for the relatively few situations with high indoor LF WTN, higher risk estimates were consistently observed. A similar association was not consistently seen for outdoor WTN. The results indicate that WTN penetrating residences at night may act as a trigger of MI and stroke. The results may be due to chance and justify no firm conclusion before reproduced in other populations.

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Conflict of interests

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Authors' contributions

MS conceived the study. AHP and ORN contributed to study conception and design. AHP analysed the data and drafted the manuscript. AP and AH provided wind and climate data. MK and JB provided road traffic and air pollution data. RBN provided GIS data. All authors participated in interpreting results, revising the manuscript and approved the final submitted version of the paper.

Appendix A. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.envint.2018.02.030.

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