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Diurnal variability of transportation noise exposure and cardiovascular mortality: A nationwide cohort study from Switzerland

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ABSTRACT

Background: Most epidemiological noise studies consider 24 h average noise exposure levels. Our aim was to exploratively analyze the impact of noise exposure at different time windows during day and night on cardiovascular mortality.

Methods: We generated Switzerland-wide exposure models for road traffic, railway and aircraft noise for different time windows for the year 2001. Combined noise source equivalent continuous sound levels (L_{eq}) for different time windows at the most exposed façade were assigned to each of the 4.41 million Swiss National Cohort adult participants. Follow-up period was from 2000 to 2008. Hazard ratios (HR) of noise effects on various cardiovascular primary causes of death were computed by Cox regression models adjusted for potential confounders and NO_2 levels.

Results: For most cardiovascular causes of death we obtained indications for a diurnal pattern. For ischemic heart disease the highest HR was observed for the core night hours from 01 h to 05 h (HR per standard deviation of L_{eq}: 1.025, 95% CI: 1.016–1.034) and lower HR for the daytime 07 h to 19 h (1.018 [1.009–1.028]). Heart failure and daytime L_{eq} yielded the highest HR (1.047 [1.027–1.068]).

Conclusion: For acute cardiovascular diseases, nocturnal intermittent noise exposure tended to be more relevant than daytime exposure, whereas it was the opposite for chronic conditions such as heart failure most strongly associated with continuous daytime noise. This suggests that for acute diseases sleep is an important mediator for health consequences of transportation noise.

1. Introduction

Transportation noise has been shown to affect cardiovascular health (Münzel et al., 2014). With regard to road traffic noise, a meta-analysis reported a risk increase of 8% (95% CI: 4–13%) for coronary heart diseases per 10 dB increase in noise levels (Babisch 2014). Also with regard to myocardial infarction the majority of studies published to date have reported significant positive associations with road traffic noise (Babisch et al., 2005; Sørensen, 2012; Willich et al., 2006). Meta-analyses focusing on aircraft noise reported pooled relative risk increases of 1.63 (95% CI: 1.14, 2.33) and 1.06 (95% CI: 1.04, 1.08) per 10 dB increase in noise levels for hypertension (Huang et al., 2015) and ischemic heart disease (Vienneau et al., 2015), respectively. This evidence is based on studies that used 24 h average noise level metrics such as $L_{eq,24}$ (defined as the energetic average of continuous measurements over a 24 h period) or L_{den} (defined as the weighted energetic average of $L_{eq,day}$, $L_{eq,evening}$ and $L_{eq,night}$ with a penalty of 5 dB and 10 dB applied respectively to the latter).

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Using the exposure metric L_{den}, we found associations between different cardiovascular causes of death and noise exposure from road, railway and aircraft noise in a previous study of the Swiss National Cohort (SNC) using data from 2000 to 2008 (Heritier et al., 2017). For instance, increased risk of death from myocardial infarction (MI) per 10 dB increase in L_{den} of road, railway and aircraft noise exposure were 4.0% (95% CI: 2.1–5.9%), 2.0% (95% CI: 0.7–3.3%) and 2.7% (95% CI: 0.6–4.9%), respectively.

However, it has been hypothesized that the impact of exposure to transportation noise on cardiovascular health is at least in part mediated via sleep alterations (Münzel et al., 2014), and thus noise exposure during sleep may be more critical than during the day. In sleeping subjects, transportation noise can induce sleep disturbances (Frei et al., 2014), and short sleep duration is a predictor of coronary heart disease and stroke (Cappuccio et al., 2011). Further, noise during sleep has a direct impact on the cardiovascular system by an acute increase in heart rate and blood pressure (Jarup et al., 2007) as well as neurocortical arousals (Basner et al., 2011).

Despite this evidence, little is known from epidemiological research about cardiovascular effects of noise at different times of the day and night, particularly during the nighttime hours; likely due to the high correlations between the exposures. So far only a few studies report the difference between nighttime and daytime exposure (Dratva et al., 2012; Hansell et al., 2013; Jarup et al., 2007) and there is a lack of knowledge and considerable uncertainty about the most sensitive nighttime windows and their detrimental impact on cardiovascular health. Such knowledge, however, is needed for policy makers to create an effective regulatory framework for noise.

The aim of this paper was to use data from our previous study (Heritier et al., 2017) to explore the effects of transportation noise in different time windows during day and night on cardiovascular mortality. In particular, we aimed at providing exposure-response relationships between transportation noise and cardiovascular outcomes for specific time windows: 07–19 h, 19–23 h, 23–01 h, 01–05 h, 05–06 h, and 06–07 h.

2. Material and methods

2.1. Study population

The SNC links national census data with mortality and emigration records (Spoerri et al., 2010). The census data contains personal, household and building information. Date of death, is included in the mortality records. The data used in our study is based on the 4 December 2000 census and on mortality and emigration data for the period 5 December 2000–31 December 2008 and contains 7.28 million people. We excluded subjects below 30 years of age (n = 2.59 million), observations for which residential coordinates were missing (n = 0.19 million), subjects living in an institution (n = 0.25 million), and observations for which the cause of death was imputed (0.03 million) leaving 4.41 million observations for the analyzes.

The outcomes under investigation were primary causes of death from all cardiovascular diseases (CVD) (ICD-10: I00-I99), ischemic heart diseases (IHD) (ICD-10: I20-I25), stroke (ICD-10: I60-I64), myocardial infarction (MI) (ICD-10: I21-I22), heart failure (ICD-10: I50) and hypertensive disease related death (ICD-10: I10-I15, comprising primary hypertension, hypertensive heart diseases, hypertensive renal disease, and hypertensive heart and renal disease). For 37% of all stroke deaths we had information about type of stroke (ICD-10: I60-I64), hemorrhagic (ICD 10: I60-I62) or ischemic (ICD 10: I63).

The SNC was approved by the cantonal ethics boards of Bern and Zurich.

2.2. Noise exposure data

Within the framework of the SiRENE project (Short and Long Term Effects of Transportation Noise Exposure), we built a Swiss-wide noise exposure database for the reference year 2001 which includes the three major transportation noise sources in Switzerland (road traffic, railway and aircraft noise) in different time windows (for details see (Karipidis et al., 2014)). For these analyzes we calculated combined noise exposure ($L_{eq,Comb}$) from all three sources (i.e. the energetic sum of road, railway, and aircraft noise) for the following time windows: 07–19 h ($L_{eq,Comb}$(07-19)), 19–23 h ($L_{eq,Comb}$(19–23)), 23–01 h ($L_{eq,Comb}$(23-01)), 01–05 h ($L_{eq,Comb}$(01-05)), 05–06 h ($L_{eq,Comb}$(05-06)), and 06–07 h ($L_{eq,Comb}$(06-07)). The time windows in the night were selected to roughly represent the different aspects of sleep at the population level, which is falling asleep (23:00–01:00), persistent sleep (01:00–05:00) and wake up phase (05:00–06:00 and 06:00–07:00) (Tinguely et al., 2014).

To consider the different characteristics of the three transportation noise sources, we additionally calculated the intermittency ratio (IR) quantifying the contribution of individual noise events above the background level to the combined noise exposure (Wunderli et al., 2016). IR values range from 0% to 100%, where 0% IR means that no single event occurs 3 dB above background (i.e. continuous noise) and 100% IR means that all noise energy is produced by “individual” noise events. Background corresponds to hourly L_{eq}. We estimated IR from all transportation sources combined in the same time periods as the L_{eq}.

For each building and each floor in Switzerland, noise exposure was estimated at pre-defined façade points. For each façade point, we calculated the $L_{den,Comb}$ from all transportation sources combined. $L_{eq,Comb}$ and IR_{Comb} for the specific time ranges were assigned on the basis of the façade point with the highest $L_{den,Comb}$ value. Using the available geocodes for each SNC participant and the information about floor of residence, we linked participants to their respective façade points. If information on floor of residence was not available (n = 1,290,042), we assigned the noise estimates of the middle floor of the building.

2.3. Statistical analysis

We analyzed the data using the Cox proportional hazards model with age as the underlying time variable. Participants were followed until emigration, death or end of follow-up in 2008, whatever occurred first. For all time windows $L_{eq,Comb}$ values below 35 dB were set to 35 dB for the analyzes. In order to calculate comparable hazard ratios (HR) for different times of the day and night with different noise exposure distributions, the L_{eqs} for each time window of interest were standardized and HRs per one standard deviation (SD) of exposure reported. To adjust for the noise peaking characteristics, we included a categorical (quintile) term for IR_{Comb}(Night) (19:00–7:00) in all models based on previous findings, where nighttime IR was significantly associated with CVD in a bell shaped relationship with highest HR for the 3rd and 4th quintile (Heritier et al., 2017). Additional to age (timescale), the following confounders were included in the model: sex (female/male), neighborhood index of socio-economic position(Panczak et al., 2012) (tertiles low, medium, high), civil status (single, married, widowed, divorced), education level (compulsory education or less, upper secondary level education, tertiary level education, not known), average NO_2 concentration (estimated outdoor residential levels using PolluMap, a 100 × 100 m dispersion model for Switzerland for the year 2010 (FOEN, 2013)), mother tongue (German and Rhaeto-Romansch, French, Italian, other language) and nationality (Swiss, rest of Europe, rest of the world/unknown).

In addition to exploring linear relationships with standardized $L_{eq,Comb}$ at during different time windows, we also conducted categorical noise analyzes using absolute values of $L_{eq,Comb}$ in 5 dB categories to explore the form of the exposure response at different times of the day and night.

In a second analysis we evaluated the impact of IR in different time windows during day and night adjusted for $L_{den,Comb}$ and the other adjustment variables mentioned above. IR_{Comb} at different time

Table 1
Study population characteristics.

Characteristics at baseline	Study population (n = 4,415,206)
Males (%)	47.9
Age: mean (SD)	52.4 (15.1)
Education level (%)	
Compulsory education or less	23.8
Upper secondary level education	51.6
Tertiary level education	22.2
Not known	2.2
Civil status (%)	
Single	13.9
Married	69.6
Widowed	7.9
Divorced	8.4
Socio-economic position (%) (Panczak et al., 2012)	
low	33.3
medium	33.3
high	33.3
Mother tongue (%)	
German and Rhaeto-Romansch	64.7
French	19.4
Italian	7.3
Other	8.4
Nationality	
Swiss	82.2
Rest of Europe (inclusive ex-USSR)	15.9
Other/unknown	1.9
NO_2 concentration in µg/m^3: mean (SD)	20.4 (6.2)
Outcomes	
All cardiovascular diseases (CVD) (ICD-10: I00-99)	142,955 (37.4%[a])
Hypertensive diseases (ICD-10: I10-I15)	13,549 (9.4%[b])
Ischemic heart diseases (IHD) (ICD-10: I20-I25)	60,261 (42.2%[b])
Myocardial infarction (MI) (ICD-10: I21-I22)	19,313 (13.5%[b])
Heart failure (ICD-10: I50)	12,345 (8.6%[b])
Stroke (ICD-10: I60-I64)	22,377 (15.7%[b])
Hemorrhagic stroke (ICD-10: I60-I62)	5354 (3.7%[b])
Ischemic stroke (ICD-10: I63)	2991 (2.1%[b])

[a] % of total causes of death.
[b] % of all CVD causes of death.

windows was split into quintiles to visualize non-linear relationships.

All analyzes were conducted with the statistical software R and the package "survival" (R Development Core Team 2008; Therneau, 2000).

3. Results

3.1. Study population

The study sample amounted to 4.41 million observations, with 33.85 million person-years for the period 5th December 2000–31 st December 2008. Median (mean) time of follow-up was 8.07 (7.64) years. Characteristics of the study sample are displayed in Table 1.

The cohort contained 142,955 deaths from CVD, of which 42.2%, 15.7%, 9.4%, 8.6% were deaths from IHD, stroke, hypertensive disease and heart failure. Increase in crude incidence rates by increasing daytime and nighttime noise were observed for all outcomes except for stroke (eTable 1*)*.

3.2. Noise exposure

The percentage of the study population exposed to levels > 45 dB and > 55 dB was higher during daytime than nightime and consequently the percentage of observations below 35 dB was highest during the core night from 01 h to 05 h (Table 2).

The distribution of transportation noise exposure $L_{eq,Comb}$ in the different time windows was slightly skewed as displayed in Fig. 1 and summarized in eTable 2.

Table 2
Percentage of the study population exposed to noise < 35 dB, > 45 dB and > 55dB.

Exposure variable	% of population < 35 dB	% of population with > 45 dB	% of population with > 55 dB
$L_{eq,Comb}$(07–19 h)	1.2	89.6	47.7
$L_{eq,Comb}$(19–23 h)	2.7	77.9	32.4
$L_{eq,Comb}$(23–01 h)	7.8	54.2	15.9
$L_{eq,Comb}$(01–05 h)	18.2	37.7	7.3
$L_{eq,Comb}$(05–06 h)	4.9	64.9	22.8
$L_{eq,Comb}$(06–07 h)	1.8	84.1	38.9

Spearman's rank correlations between the $L_{eq,Comb}$ for different time windows were high (≥0.94, eTable 3 *Supplementary material*), whereas Spearman's rank correlations between the IR_{Comb} for different time windows were somewhat lower (eTable 4 *Supplementary material*). Road traffic is the most prevalent transportation noise source and thus correlation between $L_{eq,Road}$ with $L_{eq,Comb}$ at different time windows was higher than corresponding correlations of $L_{eq,Rail}$ and $L_{eq,Air}$ with $L_{eq,Comb}$ (eTable 5). Correlation between $L_{eq,Comb}$ and IR_{Comb} at different time periods was generally low and did not exceed 0.4.

3.3. Diurnal effect pattern for $L_{eq,Comb}$

For IHD, we observed the highest HR for standardized noise exposure during the core night ($L_{eq,Comb}$(01–05 h)) and the lowest HR for the time window 06–07 h and during the day, although differences were not statistical significant (Fig. 2). The HR per standard deviation of $L_{eq,Comb}$ was 1.025 (95% CI: 1.016–1.034) for the core night, 1.017 (95% CI: 1.008–1.026) for the time window 06–07 h and 1.018 (95% CI: 1.009–1.028) for daytime exposure (eTable 6). A similar pattern was seen for all CVD deaths combined (eFigure 1). The diurnal pattern was not pronounced for MI (eFigure 1*)*. For heart failure, an inverse pattern was observed, the risk being lowest for $L_{eq,Comb}$(01–05 h) and highest during the day (07–19 h, HR = 1.047 [1.027–1.068]) (Fig. 2). For hypertensive disease highest HR was observed in the early morning (05–06 h) and lowest HR in the early night (23–01 h) (Fig. 2). For all stroke deaths, no overall association and no overall diurnal pattern was seen (Fig. 2). However, for hemorrhagic stroke, HRs tended to be slightly increased during night but not during the day (eFigure 1*)*. For ischemic stroke, HRs were highest during daytime and only slightly above unity in the core night (*eFigure 1*).

Non-standardized HR for each time window in absolute 5 dB $L_{eq,Comb}$ categories demonstrated a fairly linear exposure-response pattern for hypertensive disease, IHD and heart failure (Fig. 3), CVD and MI (eFigure 2). During the core night (01–05 h), HRs tended to start increasing from a lower level than during daytime. This pattern also held for heart failure (Fig. 3) where a different diurnal pattern was seen for the standardized exposure analysis.

3.4. Day-night pattern for the intermittency ratio (IR)

For IR_{Comb} during night (23:00–6:00) the categorical HRs followed a bell shaped curve with highest risk in the 3rd and 4th quintiles for cardiovascular disease (eFigure 3). For IR_{comb} during daytime windows the HR curves were somewhat flatter and skewed toward the 2nd quintile (eTable 7), which corresponds to a more continuous noise level. A similar pattern was observed for ischemic heart disease but an opposite pattern for hypertensive diseases (Fig. 4): noise events were relevant during daytime as whereas during core night (01:00–05:00), continuous noise (1st quintile) tended to be more detrimental. For heart failure highly intermittent noise (5th quintile) produced somewhat reduced HR except during the core night. Highly intermittent noise implies relatively few noise events with longer quiet phases between the

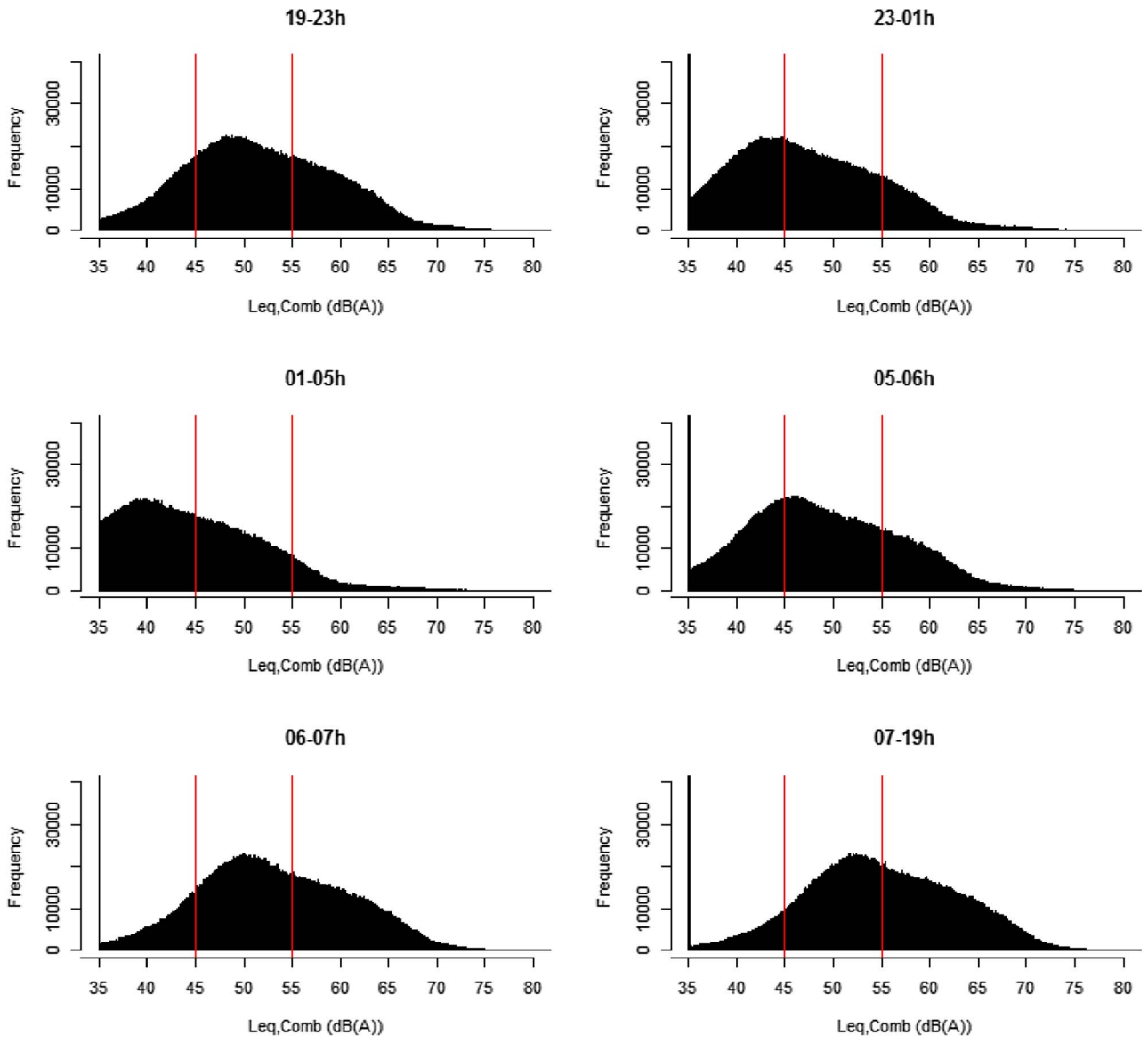

Fig. 1. Distribution of Leq,Comb(19–23 h), Leq,Comb(23–01 h), Leq,Comb(01–05 h), Leq,Comb(05–06 h), Leq,Comb(06–07 h), and Leq,Comb(07–19 h) in the study population. The vertical red lines show the 45 dB (left) and 55 dB (right) threshold. (For interpretation of the references to colour in this figure legend, the reader is referred to the web version of this article.)

events.

4. Discussion

Similar to other studies, we previously observed in this SNC dataset associations between various cardiovascular causes and deaths and average noise exposure expressed as L_{den} (Heritier et al., 2017). In this follow-up paper we could investigate exposure effects from transportation noise in different time windows during day and night, which is useful information for an effective regulatory framework of noise. Our results point toward a stronger association between transportation noise exposure during the nighttime hours 23–06 h and acute heart diseases, (e.g. IHD) than for daytime. Thereby intermittent noise seems to be more critical than continuous noise exposure. This pattern tended to be reversed when considering chronic heart disease such as heart failure. For hypertensive disease, the diurnal pattern was less distinct with highest HRs in the early morning and during daytime. No association at all was found with stroke, although noise tended to be associated with ischemic stroke, mainly during daytime. During night, cardiovascular disease risk started to increase as low as 35 dB.

Different outcome related impacts of the timing of noise exposure may indicate that different mechanisms are at play in the aetiology of cardiovascular outcomes. With regard to IHD, our finding supports the hypothesis that noise associated sleep disturbances impair cardiovascular health in the long run. The core night contains more deep sleep than the early morning hours and thus disturbances from noise events during this time interval seem to be considerably critical (Webb and Agnew 1971). Sleep disturbances have been linked with higher risk for obesity and diabetes (Eze et al., 2017; Knutson and Van Cauter, 2008) which are known risk factor for IHD (Lavie et al., 2009; Wilson et al., 1998) and also recently found to be related to transportation noise exposure (Eze et al., 2017; Sörensen et al., 2013); thus further supporting the link between nighttime noise and this particular outcome. The onset of acute coronary events shows a strong morning peak during the time of sympathetic nervous system activation (Muller 1999) going along with endothelial dysfunction, arterial stiffening, platelet aggregation and thrombus formation (Chen and Yang 2015). Thus, noise exposure in the previous night may act as an additional stressor (Münzel et al., 2014) which triggers the onset of fatal events.

Interestingly, for heart failure an opposite diurnal pattern was seen

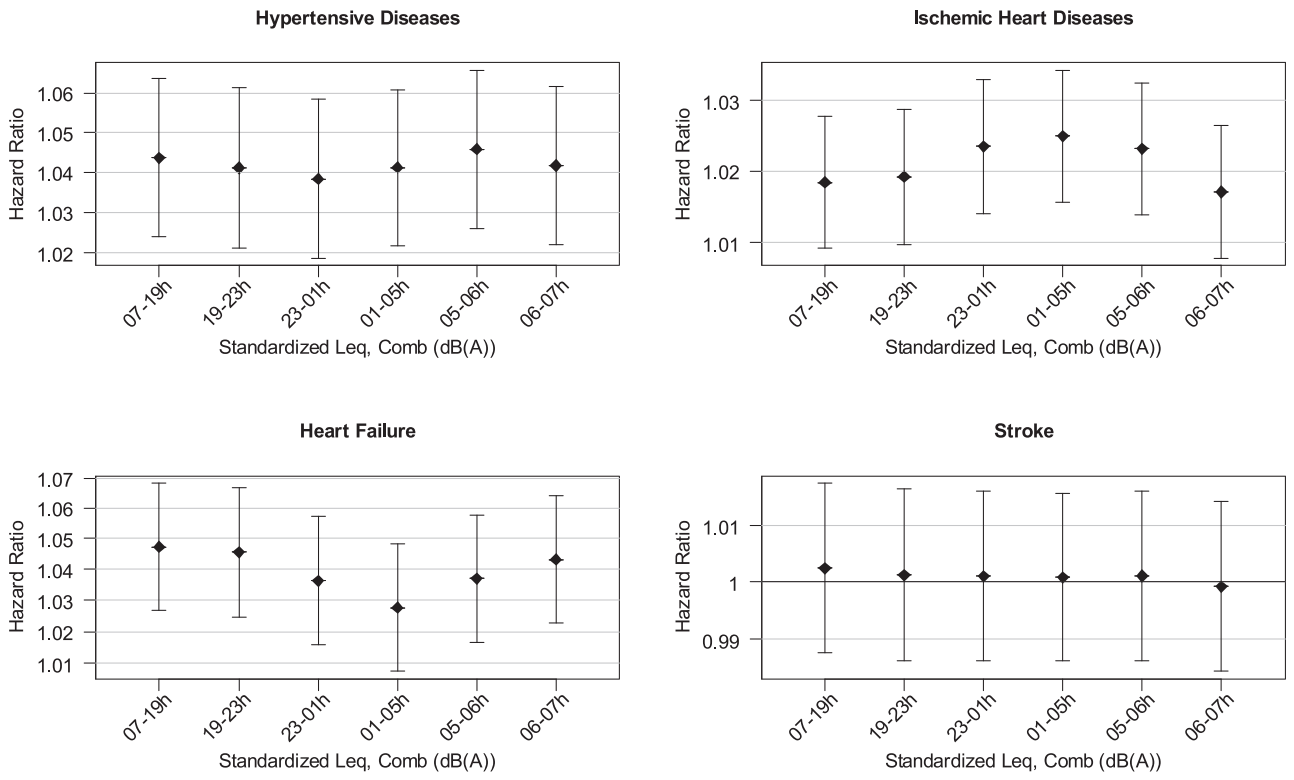

Fig. 2. Adjusted[a)] hazard ratio and 95% confidence intervals per increase of 1 standard deviation[b)] of standardized $L_{eq,Comb}$ in the different time windows for the following causes of death: Hypertensive diseases, IHD, heart failure and stroke. Note different y-axis for all the figures.
[a)] adjusted for age (timescale), sex, neighborhood index of socio-economic position, civil status, education level, mother tongue, nationality, NO_2 exposure and IR_{comb}(Night), [b)] SD is between 7.35 and 8.01 dB (see eTable 6)

compared to death due to IHD. Daytime noise and continuous noise exposure was most critical. Heart failure has only recently been linked to noise exposure (Correia et al., 2013; Seidler et al., 2016). Death by heart failure is caused in approximately 50% of cases by sudden cardiac death and in 35% by acute worsening of ischemia (Ørn and Dickstein, 2002). Deaths from sudden cardiac death occur most often during 6 am and 12 noon (Moser et al., 1994) and thus it is plausible that noise associated stress during daytime is a relevant trigger.

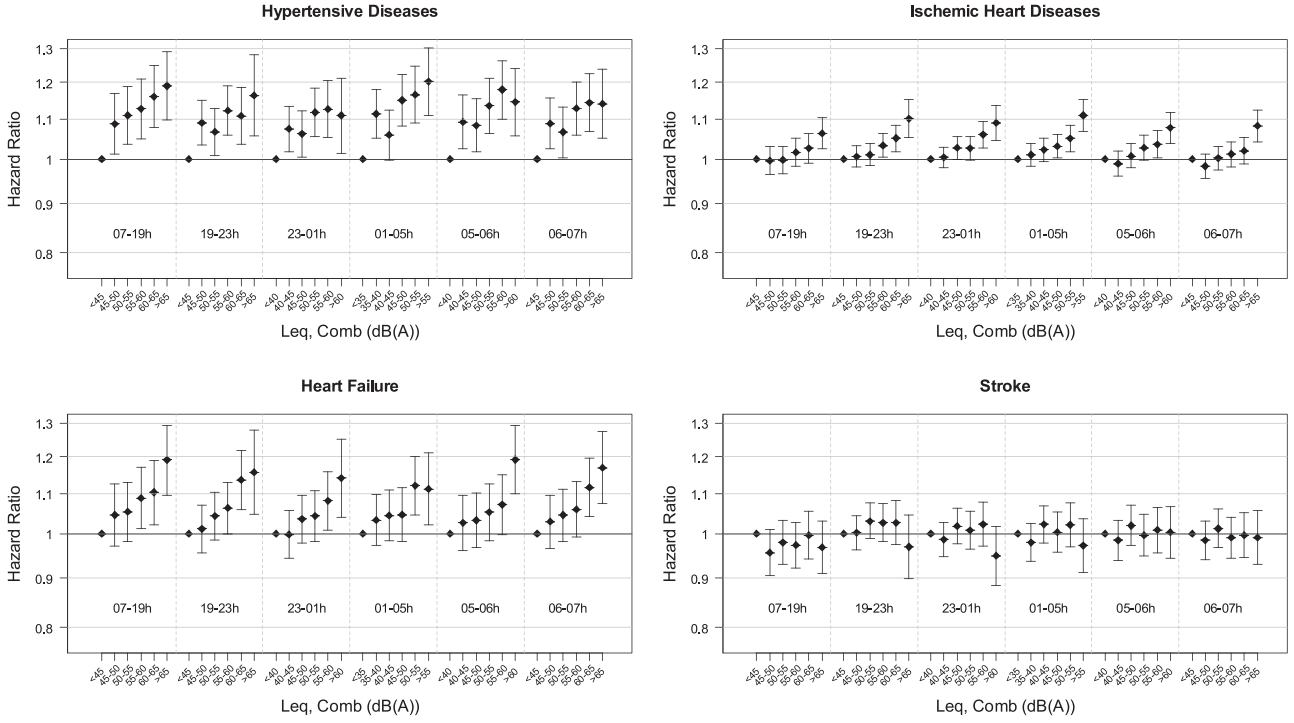

Fig. 3. Adjusted[a)] categorical HRs for specific time windows. Models for the following causes of death: Hypertensive diseases, IHD, heart failure and stroke.
[a)] adjusted for age (timescale), sex, neighborhood index of socio-economic position, civil status, education level, mother tongue, nationality, NO_2 exposure and IR_{comb}(Night)

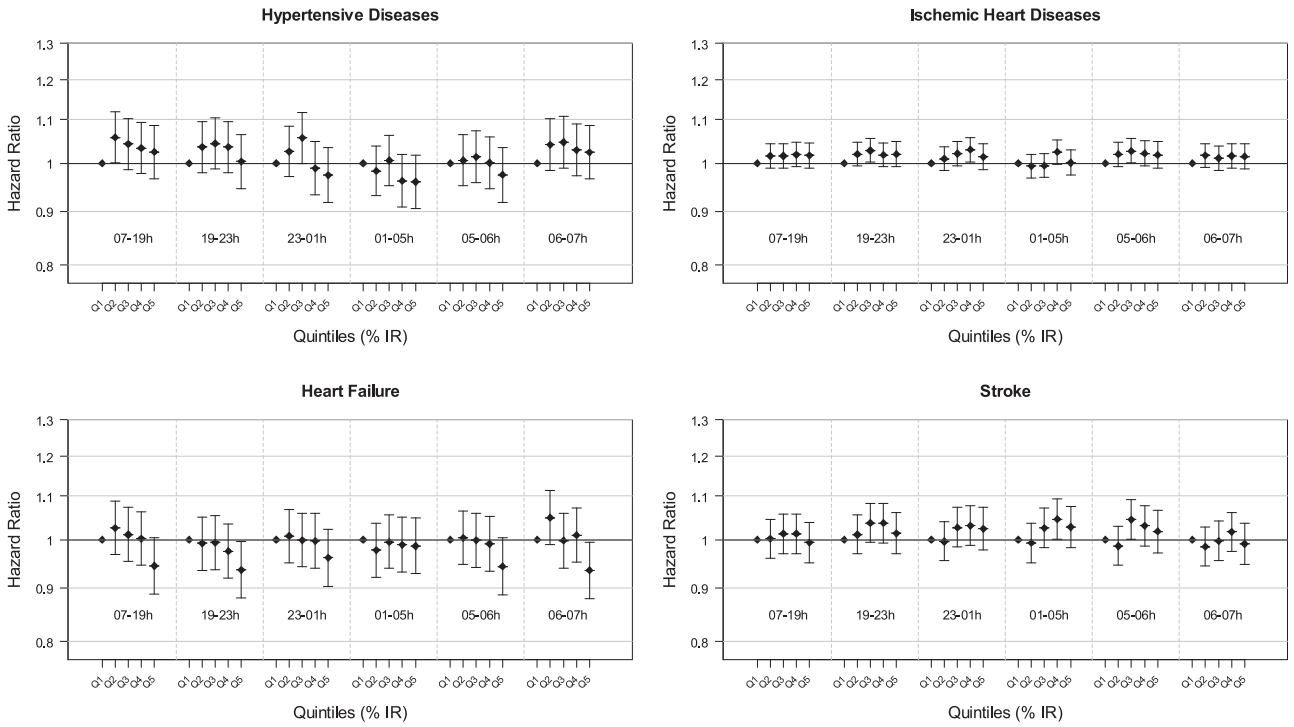

Fig. 4. Adjusted[a)] categorical Hazard ratio and 95% confidence intervals per quintiles of IR at different time windows for the following causes of death: Hypertensive diseases, IHD, heart failure and stroke.

[a)] adjusted for age (timescale), sex, neighborhood index of socio-economic position, civil status, education level, mother tongue, nationality, NO_2 exposure and L_{den}.

For hypertensive disease related mortality the diurnal pattern was most similar to heart failure but less distinct. Early morning hours and daytime exposure may be most critical. The start of activity phase in the early morning hours coincides with the diurnal peak levels of glucocorticoids (Dickmeis, 2009). Additional external noise stressors may thus affect body homeostasis with higher risk for a fatal event. Daytime noise exposure is expected to lead to vegetative arousals with increases in blood pressure and stress hormone levels, which may result arterial hypertension (Münzel et al., 2014). Our results suggest that for such arousals during the day noise events are more critical than continuous noise exposure levels. Conversely, during night continuous noise exposure might be most critical for hypertensive diseases.

Only a few epidemiological studies systematically addressed the different impacts of daytime and nighttime noise on cardiovascular outcomes. Halonen et al. (2015) found higher risk of cardiovascular hospital admissions in the elderly for road traffic noise during daytime. Since cardiovascular admissions comprised heart failure, this observation may be in line with our findings for heart failure related deaths during the day. Others observed stronger effects at night for hypertension and aircraft noise (Jarup et al., 2007) as well as for systolic blood pressure and railway noise (Dratva et al., 2012). However, the difference in exposure assessment of those studies limits comparison with ours. It is well possible that the exact timing of a fatal event also depends on the typical sleeping attitudes in a population. In Switzerland people wake up on average at 06:30 (IQR:06:00–07:00) (Tinguely et al., 2014). Critical time windows may be different for other populations with a different sleeping pattern.

A particular challenge for such an evaluation in observational research is the inherent high correlation between exposure levels at different time windows which limits causal inference and produces largely overlapping confidence intervals as shown in Fig. 2. Thus, chance cannot be completely ruled out as a potential explanation for the observed findings. However, HR of L_{eq} for IHD, hypertensive diseases and heart failure showed relative smooth cosine-shaped diurnal patterns, which speak against random fluctuations of the risk estimates.

By standardizing the noise exposure during different time windows we were able to directly compare the HRs. We also combined exposure from the three traffic sources road, railway and aircraft. This is expected to reduce within day correlation since the diurnal pattern is different for the three traffic sources (e.g. night curfews for aircraft, heavy nighttime railway traffic from freight trains). On the other hand, this may create confounding if one assumes different health effects for different noise characteristics emitted by different sources. To prevent, or at least minimize, such confounding we have additionally included in all our models the IR and could indeed demonstrate that – independent on the noise levels – IR is related to cardiovascular mortality. For IHD we found in tendency a pattern of larger effects for nighttime IR compared to daytime IR and a shift of the risk toward higher IR quintiles. This indicates that noise events may be more problematic for nighttime; whereas during daytime continuous noise with absence of quiet phases may be most detrimental.

We did not find an association between stroke and noise exposure. In line with a Danish cohort study (Sorensen et al., 2014) noise was not associated with hemorrhagic stroke, but some indication for an association with ischemic stroke was observed, in particular for daytime noise (HR:1.029, 95% CI: 0.988–1.072, eTable 6). However, information about type of stroke was only available for 37% of the 22,377 stroke deaths, which explains why the subgroup results for hemorrhagic and ischemic stroke do not add up to the results for all types of stroke, which also includes non-specified strokes (ICD-10: I64). In addition, stroke does not share a common etiology with the other CVD if no information about cardiac embolism is available (Arboix and Alió, 2012; Ferro, 2003). Therefore including stroke in the broader CVD category, as most often done in cardiovascular noise research, will have an impact on the strength of association for CVD in general. Indeed, in a sensitivity analysis excluding stroke from all CVD yielded a 0.2% increase of the HR for CVD per 10dB of $L_{eq,Comb}$(01–05 h).

4.1. Exposure misclassification

One might argue that the diurnal pattern of HRs throughout the different time windows may reflect exposure misclassification. During

the core night, the exposure assigned to the study population may be closer to the true exposure since people are at home, while during the day noise exposure is not relevant for the true exposure since people may be elsewhere for example at work. However, if there was only exposure misclassification at play, this would affect all estimates for all outcomes. This is not the case for hypertensive disease, ischemic heart disease and heart failure for which the estimates of the core night (01–05 h) and the day (07–19 h) have either the same or even a lower magnitude. This suggests that the observed pattern reflects a true underlying risk pattern and is not just caused by a common underlying bias.

4.2. Strength and limitations

The strengths of this study are the large study population and the long follow-up time providing a high number of cases. We developed an extensive and detailed noise exposure model, which allowed an individual exposure assessment at the address and floor level, which has rarely been done in previous large population studies. Detailed input data of the noise models such as traffic flow statistics could be used to calculate L_{eq} and IR with high temporal resolution. Potential selection bias is minimal in this nationwide study based on census data.

Though our models are adjusted for socioeconomic status and other demographic variables, we could not adjust for lifestyle and smoking as this information is not available in the SNC. We therefore cannot rule out that residual confounding of lifestyle may play a role for our analyses, although no indications for this were seen in a previous SNC noise study (Huss et al., 2010). Further, as in every study on long term noise exposure, exposure misclassification is unavoidable due to uncertainty in the input data (e.g., traffic information). A validation of the road traffic model using 123 weekly noise measurements demonstrated a good agreement with an average difference between modeling and measurements of +0.4 dB with an interquartile range (IQR) of −1.7 to 3.1 dB (Schlatter, 2017). Most relevant, even during night (23:00–7:00) when other sources of noise may have a larger impact on the measurements due to low traffic density, a good model performance was observed in the exposure range between 30 and 70 dB (mean deviance: 0.2 dB, IQR: −1.8 to 2.7 dB. Note that noise exposure was assigned based on estimates for the loudest outdoor façade point while no information was available regarding indoor noise levels. We did not have information on sleeping room orientation nor on factors relevant for the noise propagation into the residency such as sound proof window installation and window closing behavior. However, such misclassification is more likely to dilute the association than introduce a bias.

5. Conclusion

This study indicates diurnal patterns for the association between transportation noise and various cardiovascular causes of mortality. This suggests that different mechanisms are at play in the aetiology of various noise associated cardiovascular causes of deaths. Our results provide more support for protecting the population against nighttime noise as risk factor for CVD.

Conflict of interests

None to declare

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Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.ijheh.2018.02.005.

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