A comparison of cardiovascular activation responses to environmental noise during sleep

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ABSTRACT

Frequent cardiovascular activation responses associated with micro-arousals and sub-cortical sensory processes may negatively impact on cardiovascular health. Therefore, simple measures such as pulse wave amplitude (PWA) and heart rate (HR) may be sensitive cardiovascular markers of sleep disturbance to environmental noise. This study sought to compare the magnitude of automated measures of PWA and HR to noise presentations during sleep. Twenty-four participants attended a sleep laboratory for one overnight recording of electroencephalogram, electrocardiogram (ECG) and finger pulse oximetry.

Randomized 20-second noise batteries of multiple types of noise were played at different sound pressure levels (SPLs) (33-48 dB(A)) during established N2 and N3 sleep. Automated measures of HR and PWA were derived from ECG and oximetry signals and compared between noise levels and types using response-free survival analysis.

The principal findings support that noise stimuli induced a more prominent and reliable PWA response compared to HR responses. SPLs, noise type and sleep stage all influenced PWA responses due to noise. These findings support that relatively simple automated markers of cardiovascular activation responses, particularly PWA, provide useful markers of sleep disturbance.

INTRODUCTION

The World Health Organization [1] estimated that at least one million of healthy life years are lost every year due to environmental noise, with sleep disturbance as one of the main confounding factors. While several studies have shown an association between nocturnal noise exposure and cardiovascular disease [2,3,4], the underlying mechanism remains unclear.

Road traffic noise (RTN) [5] and wind farm noise (WFN) [6,7] studies aiming to understand the potential relationship between sleep fragmentation and noise have mainly focused on the macro-structure of sleep and arousal. However, it is possible that the micro-structure of sleep might play a more important role in potential sleep impairment. The electroencephalogram (EEG), electrocardiogram (ECG) and finger pulse oximetry measurements likely contain more information on parameters related to sleep disturbance effects [8,9]. Heart rate (HR) is calculated from the R-peak interval in the ECG signal. Finger vasoconstriction is determined by a decrease of pulse wave amplitude (PWA) signal recorded with finger pulse oximetry which provides an index of pulsatile light transmission and thus blood volume changes in a skin vascular bed. The two cardiovascular markers seem to be related to one another during sleep [10].

Catcheside et al. [11] studied the ability of different cardiovascular markers to accurately detect acoustically induced arousals. During sleep, when the HR accelerates, the PWA seems to fall [10]. It has also been reported that HR responses could be an indicator of a stress response to noise, whereas PWA responses may be more closely related to both cardiovascular and auditory protective mechanisms such as the auditory reflex [12]. However, they are both potentially useful markers of sleep disturbance.

Autonomic or cortical responses to noise during sleep such as HR and finger vasoconstriction seem to depend on acoustical characteristics, however their relative prominence is unknown [12,13]. The sound pressure level (SPL) seems to be an important acoustical characteristic for sleep disturbance. Basner et al. [14] observed that transportation noise with L_{Amax} as low as 33 dBA could provoke arousals in the autonomous nervous system. Moreover, Di Nisi et al. [10] showed that HR and PWA responses were proportional to the noise intensity regardless of noise types. For instance, airplanes (67.7 dBA) and railways (68.2 dBA) had larger responses compared to trucks (61.9 dBA) and motorcycles (52.7 dBA). Noise seems to provoke autonomous nervous system activation, additionally the cortical nervous system also seems to be activated.

Therefore, the PWA and HR responses could be sensitive sleep disturbance markers to environmental noises. Despite complaints regarding WFN, researchers have not studied the objective responses to this noise.

METHODOLOGY

Data collection

Participants and experimental conditions

Twenty-five individuals, including 11 males (26.4 ± 16.3 years, age range: 18 - 75 years) and 14 females (24.1 ± 9 years, age range: 19 - 55 years) were recruited for a one-night sleep study. Participants were screened to select good sleepers with normal hearing and without significant medical and/or psychiatric conditions. Basic auditory assessments were conducted via an audiometer for assessing hearing acuity. Participants' lights out time was determined by averaging habitual bedtime from a one week sleep diary kept at home prior to the laboratory study. Wake-up time was not controlled.

Physiological recordings

For the sleep study, participants were instrumented with polysomnography (PSG) equipment including electroencephalogram (EEG; F3, F4, C3, C4, Cz, O1 and O2), electro-oculogram (EOG; E1 and E2), chin electromyogram (EMG), leg movements (leg EMG),

electrocardiogram (ECG) and finger pulse oximetry. Simultaneous acoustic and sleep study recordings were time-locked via timing marks recorded simultaneously on both devices.

Auditory tones and controls

For the sleep study, a battery of block-randomised noise stimuli of 20 second duration, interspersed with 20-second silent periods were presented only when participants were asleep (at least 5 minutes into sleep at the start of the protocol and after at least 1 minute of stage 2 or deeper sleep on any subsequent return to sleep after an awakening). The 20 s noise battery during sleep included noise levels ranging from 33 to 48 dB(A), in 3 dB(A) increments, of the following noise types:

- traffic noise short range (TFN short-range) recorded 20 m from a main road,
- traffic noise long range (TFN long-range) recorded 700 m from a main road,
- WFN with amplitude modulation (AM) (WFN AM) recorded 3.3 km from a South Australian wind farm,
- WFN without AM (WFN NOAM) recorded 3.3 km from the same wind farm,
- “Swish” – WFN AM recorded 500 m from a wind farm,
- silence (background noise control).

During wakefulness, participants were exposed to either silence or WFN AM at 33 dB(A) in random order. The lowest sound pressure level was set at 33 dB(A) to ensure a minimal 10 dB(A) difference between the background noise (23 dB(A)) and noise stimuli. Each noise stimulus was ceased if the participant woke during the night (any EEG return to wake lasting 15 seconds or more) until the participant fell back to sleep, at which point the noise battery was re-started. An independent qualified sleep technician, blinded to the study aims and conditions, scored the sleep data according to American Academy of Sleep Medicine (AASM) criteria.

Cardiovascular markers

The R-wave peaks in the ECG signal were detected with the Hamilton-Tompkins algorithm [15] from where the instantaneous beats-per-minute (BPM) were evaluated. Given that the instantaneous beat-to-beat measures of HR and PWA occur at unevenly spaced R-R intervals, a cubic spline interpolation was used to align the responses relative to the noise onset to allow for ensemble averaging.

To help account for substantial variability in signals from heart beat-to-beat and over time, HR and PWA signals were normalised by expressing values as a percentage of the preceding 5 or 10 seconds prior to stimuli onset baseline for HR and PWA, respectively. Note that 20 seconds out of 30 seconds represents the stimuli length.

Statistical analysis

The hazard ratios for sound pressure level and noise type groups, as compared with silent controls, and the corresponding confidence intervals were estimated with the use of a stratified Cox proportional-hazard model. Survival curves for each group were estimated with the use of the Kaplan–Meier method and pairwise comparison was performed using the log-rank test. Rates at fixed time points were derived from the Kaplan–Meier estimate, along with their corresponding 95% confidence interval. Null hypotheses were rejected when $p < 0.05$.

The survival curves using the Kaplan-Meier method were estimated for the following groups:

- 7 groups of sound pressure level: Silence/Control, 33 dB(A), 36 dB(A), 39 dB(A), 42 dB(A), 45 dB(A) and 48 dB(A),
- 6 groups of noise type: Silence/Control, TFN short-range, TFN long-range, WFN AM, WFN NOAM and “Swish”,

Survival curves show the probability of a response, PWA-drop or EEG arousal, occurring up until a specific point in time. When a survival curve decreases more abruptly than another, it means that participants experienced more PWA responses due to noise in this condition than the other conditions. Numbers at risk were also determined to show the absolute number of participants still event-free and still at risk each 5 seconds out of the total 40 second period of analysis. The Cox proportional hazards regression model allows testing for differences in survival times of multiple groupings of predictor variables. Cox regression models were performed at 5 second cut-off time values. The sound pressure levels (SPLs) of the noise and the noise types in which the noise occurred used in the Kaplan-Meier method were studied as potential predictors of evoked PWA responses. The hazard ratio of each variable relative to the relevant reference category is an indicator of predictive utility, where a hazard ratio significantly below 1 or above 1 indicates a predictor of reduced or increased incidence of the selected outcome event, respectively. Statistical analysis was performed using packages Survival version 2.44-1.1 and Survminer version 0.4.6 in R.

RESULTS

Characteristic PWA and HR responses

This section presents results of the two cardiovascular marker responses occurring during Stage 2, Stage 3 and REM sleep grouped into “quiet” and “loud” conditions for one participant as an example. Table 1 shows the number of delivered noise samples which triggered a PWA-drop within the first 5 seconds after noise onset. The 5 second cut-off value was chosen because it was previously shown that PWA-drops occur within that time frame [11][16]. PWA-drops occurred in response to 17% of the noise stimuli with no statistically significant difference in propensity between “quiet” and “loud” groups (Fisher’s test, $p = 0.404$).

Table 1: Summary of the PWA-drops in “quiet” and “loud” groups occurring in Stage 2, Stage 3 and REM sleep for one participant.

	Total N	Present	Absent
“Quiet” (33-39 dB(A))	246	38 (15.4%)	208 (84.6%)
“Loud” (42-48 dB(A))	250	46 (18.4%)	204 (81.6%)
Total	496	84 (16.9%)	412 (83.1%)

In the “quiet” and “loud” conditions, the average PWA responses, when present 5 seconds after noise onset, were similar with a decrease of approximately 50% amplitude compared to the baseline, as shown in Figure 1. All PWA-drops in this figure were centred with their beginning at time 0 (see Figure 1A and Figure 1C). They were then assemble averaged, and the resulting signals are shown in Figure 1B and Figure 1D. The HR response showed a brief

transient acceleration of around 6% up to approximately 5 seconds after noise onset, then returned to baseline around 10 seconds after noise onset.

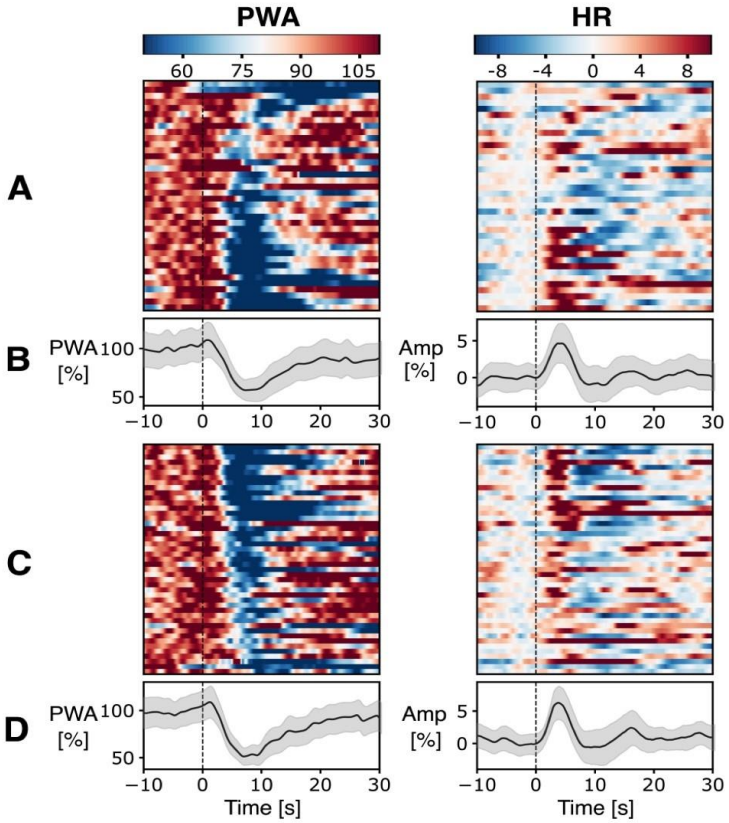

Figure 1: Typical PWA and HR in response to “quiet” (A and B) and “loud” (C and D) noise stimuli together with a spectrogram showing all responses during Stage 2, Stage 3 and REM sleep when a PWA-drop was present 5 seconds after noise onset. Time 0 indicates onset of a 20-second long noise stimuli.

In the case where a PWA response was not present 5 seconds after noise onset, the two cardiovascular markers both fluctuated around the baseline with minimal indication of systematic changes temporally related to the noise onset.

Survival probability of PWA-drops for SPLs

Table 2 summarises the results from a pairwise comparison of PWA-drop occurrence for the SPL factor at 5 seconds after noise onset. The SPL factor seems to impact PWA responses during the 5 first seconds after noise onset, however only SPLs equal or higher than 39dBA were significantly different from the control.

Table 2: P-values of a pairwise comparison between noise levels at 5 seconds after noise onset.

	Control	33dBA	36dBA	39dBA	42dBA	45dBA
33dBA	0.4015	-	-	-	-	-
36dBA	0.1812	0.5590	-	-	-	-
39dBA	0.0050	0.0665	0.1924	-	-	-
42dBA	3.9e-05	0.0024	0.0126	0.2289	-	-
45dBA	5.2e-13	2.1e-09	5.0e-08	3.8e-05	0.0039	-
48dBA	< 2e-16	4.4e-13	1.6e-11	5.0e-08	2.5e-05	0.1924

A Cox regression model performed with SPL showed that SPLs above 39 dB(A) showed statistically significantly higher hazard ratios compared to silence ($p < 0.05$) as shown in Figure 2. The figure also shows an increasing hazard ratio with increasing SPL. At 33 dB(A), the hazard ratio was not statistically significantly different compared to control ($p = 0.39$), but at 39 dB(A) and 48 dB(A), the hazard ratios were 50% and 190% compared to control, respectively ($p = 0.003$ and $p < 0.001$). This means that a PWA-drop had 50% and 190% more chance of occurring during the first 5 seconds after a noise onset at 39 dB(A) and 48 dB(A), respectively, compared to silence. In comparison, a Cox regression model was also performed with EEG arousals (Figure 3), where only SPLs at 42 dB(A) and 48 dB(A) SPLs were associated with a significantly higher probability of provoking an EEG arousal within the first 5 seconds of noise onset ($p < 0.05$).

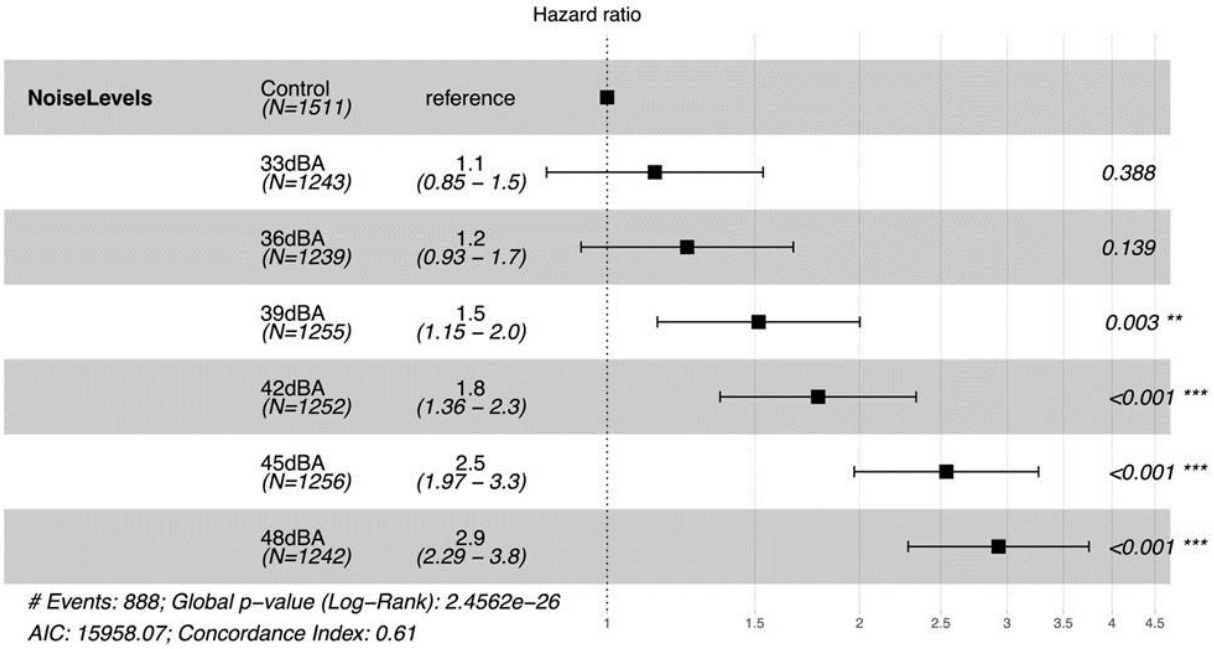

Figure 2: Comparison between PWA-drop hazard ratios for environmental noise at several sound pressure levels. Squares represent point estimates; bars represent 95% confidence limits. Ratios more than 1 indicate that PWA-drops occur more often with noise than when no noise is played during sleep.

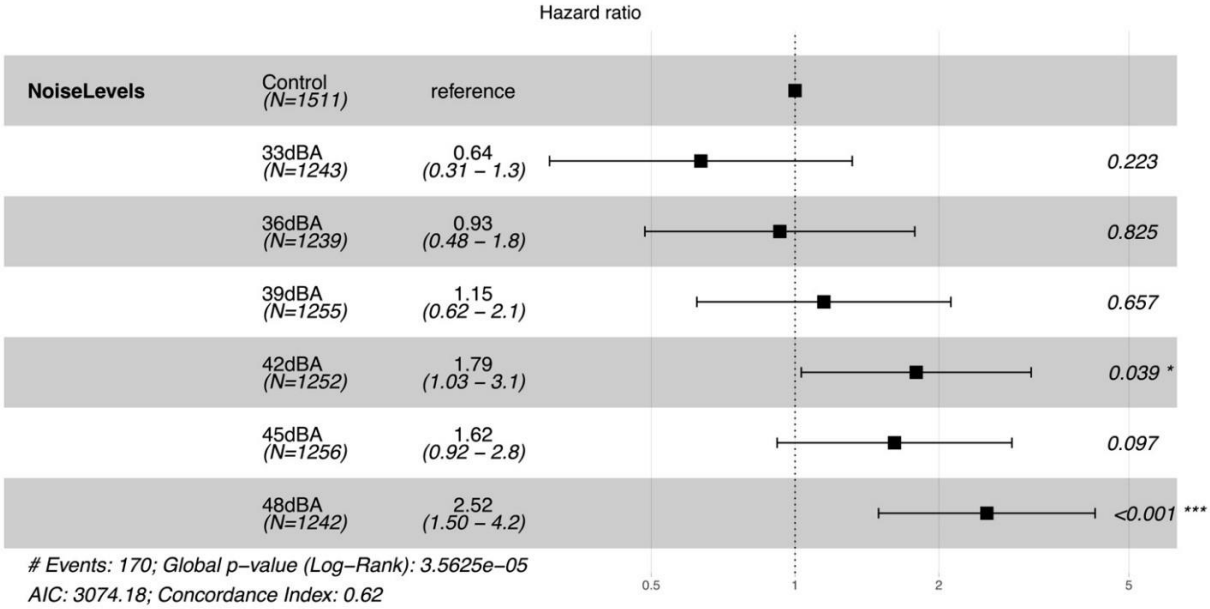

Figure 3: Comparison between EEG arousal hazard ratios for environmental noise at several sound pressure levels during sleep. Squares represent point estimates; bars represent 95% confidence limits. Ratios more than 1 indicate that EEG arousals occur more often with noise than when no noise is played.

Another Cox regression model was performed adjusted for grouped noise SPLs, also with a cut-off value of 5 seconds. The SPL groups were as follows:

- control with “silence”,
- “quiet” with SPLs between 33 dB(A) and 39 dB(A),
- “loud” with SPLs between 42 dB(A) and 48 dB(A).

In both groups the PWA-drop occurrence was significantly higher compared to the control (hazard ratio = 1.3, $p = 0.03$ for “quiet” and hazard ratio = 2.4, $p < 0.001$ for “loud”). The corresponding Kaplan-Meier curves are shown in Figure 4, demonstrating that PWA-drop responses occurred 14%, 8% and 6% of the time for “loud”, “quiet” and control conditions, respectively. After 5 seconds, the survival differences between groups clearly reduce and diminish by around 20 seconds, which is the time when the noise samples stopped playing. At that time, the PWA-drop responses occurred for 24%, 23% and 24% for “loud”, “quiet” and control groups.

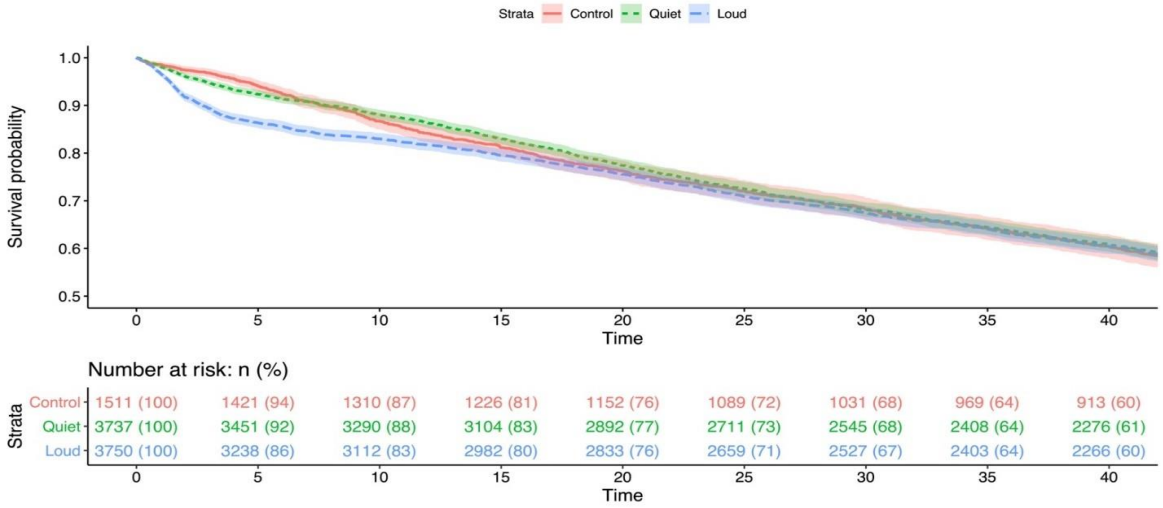

Figure 4: Kaplan-Meier survival curves for PWA-drop occurrence after noise onset adjusted for 3 sound pressure level groups; silence (control), “quiet” and “loud”. Each noise stimulus lasted 20 s after which there was 20 s of silence prior to the next noise stimulus.

The survival functions for noise levels appear to converge after 20 seconds suggesting that noise-evoked and spontaneous responses may not be additive. This could potentially indicate that noise-evoked responses reduce subsequent spontaneous response probability, or that noise-evoked response probability is relatively low and thus potentially difficult to distinguish from that of more common spontaneous responses over time.

Survival probability of PWA-drops for noise type

Another Cox regression model was performed on the noise types with their associated level groups (“quiet” or “loud”), as shown in Figure 5. These results suggest that wind farm noise with and without amplitude modulation only affected PWA responses in the “loud” condition. “Swish” noise, TFN short-range and TFN long-range affected the responses for all group level conditions.

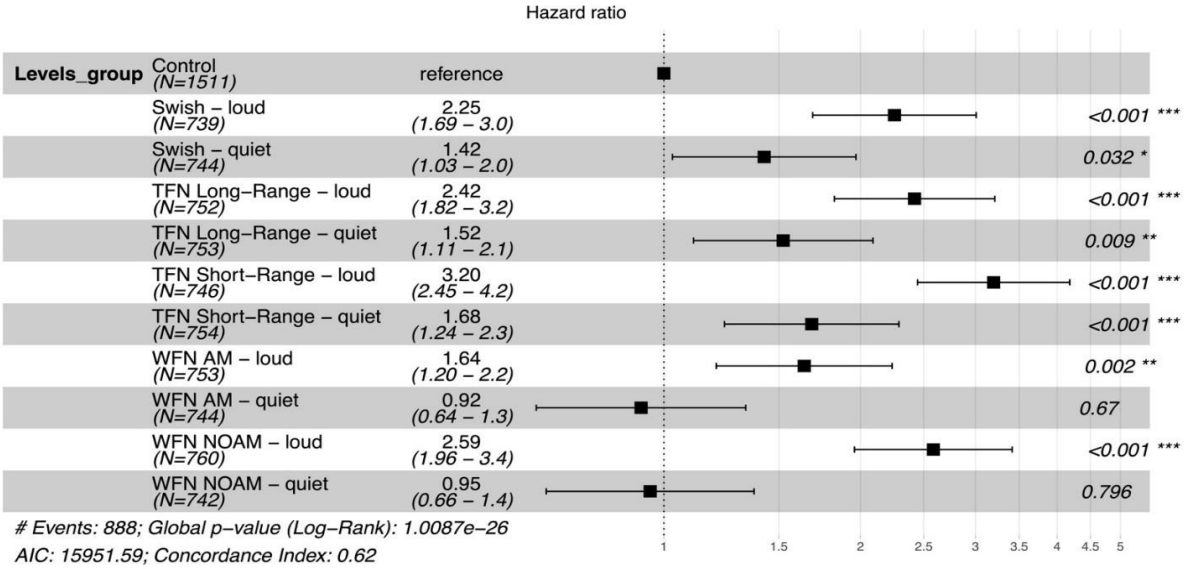

Figure 5: Comparison between PWA-drop hazard ratios for different noise types with their associated level group. Squares represent point estimates; bars represent 95% confidence limits. Ratios more than 1 indicate that PWA-drops occur more often with noise than when no noise is played during sleep.

Kaplan-Meier plots (Figure 6) revealed that all noise types are associated with an increased probability of PWA-drops after noise onset relative to control. PWA responses occurred 205 times (14%) out of all traffic noise short-range presentations at 5 seconds after noise onset. Traffic noise short-range was the noise type with the most PWA responses. On the other hand, wind farm noise with amplitude modulation was the type of noise with the least PWA responses at 5 seconds after noise onset; 113 times (8%). Moreover, as seen with the pairwise comparison between noise types (Table 3), all noise types showed statistically significant differences compared to the control except for wind farm noise with amplitude modulation.

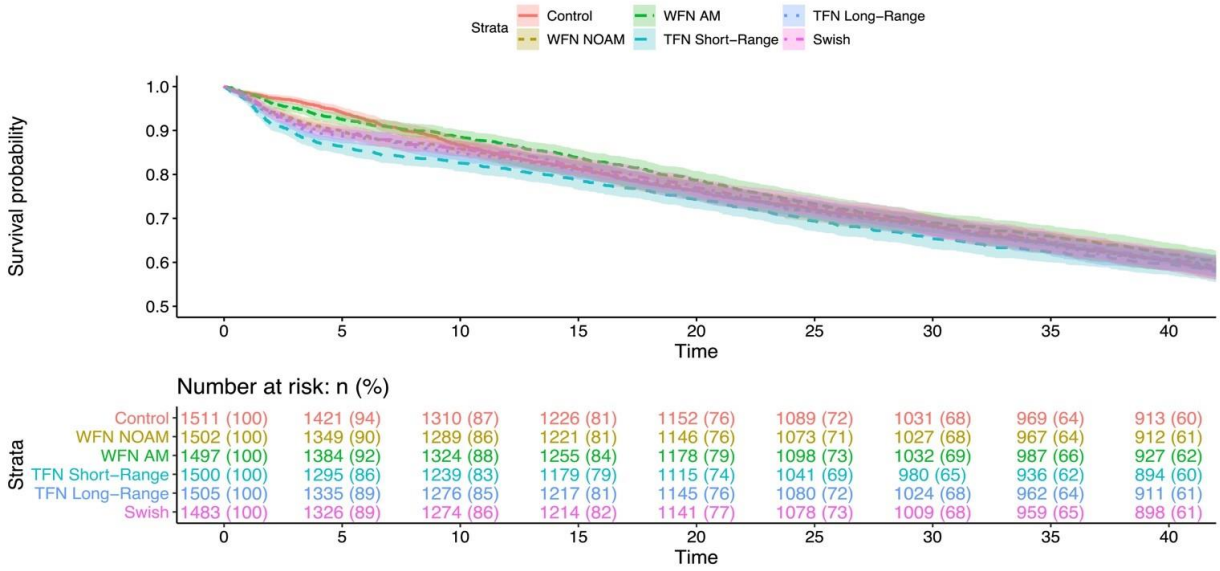

Figure 6: Kaplan-Meier survival curve showing PWA-drop occurrence after noise onset (20 s stimuli followed by 20 s of silence) adjusted for 7 noise types; silence, WFN NOAM, WFN AM, TFN short-range, TFN long-range and “Swish”.

Table 3: P-values of a pairwise comparison between noise types at 5 seconds after noise onset.

	Control	Swish	TFN Long-Range	TFN Short-Range	WFN AM
Swish	1.2e-05	-	-	-	-
TFN Long-Range	6.3e-07	0.5699	-	-	-
TFN Short-Range	9.3e-12	0.0149	0.0637	-	-
WFN AM	0.0990	0.0063	0.0009	2.7e-07	-
WFN NOAM	4.9e-05	0.7166	0.3710	0.0063	0.0149

DISCUSSION

The principal finding of this study was that environmental noise with an SPL of 39 dB(A) or higher evokes more PWA-drops than occur without noise presentations during sleep. The PWA-drops generally seem to occur in the first 5 seconds after noise onset. This is the first time that such a response has been measured to such low levels of environmental noise.

The amplitude of the average PWA response decreased for a period greater than 30 seconds, and the average HR response accelerated to its maximum around 5 seconds and then decelerated back to baseline within around 10 seconds, consistent with previous findings [10,15,16]. These results likely reflect sympathetic nervous system activation which largely controls PWA and HR responses [10]. Moreover, the magnitude of signal decrease (approximately 50%) for the PWA response was similar to previous findings [11] despite substantially lower SPLs and stimuli type used in this study 33 to 48 dB(A) versus 54 to 90 dB [11]. Furthermore, no strong differences between “quiet” and “loud” noise groups are also consistent with an “all or none” PWA response phenomenon.

The strong relationship between SPL and PWA response is perhaps not surprising given previous findings [16] and is consistent with an increased likelihood of response with increasing SPLs shown by Catcheside et al. [11] and Tassi et al. [17]. Moreover, the PWA response seems to be more sensitive at lower SPLs than evoked EEG arousals.

In this study, short-range traffic noise was found to have the highest impact on the PWA response. This noise type has a spectrum dominated by mid-frequency energy. On the other hand, wind farm noise with low-frequency amplitude modulation showed the least impact on the PWA response.

CONCLUSION

In summary, this study supports that PWA-drops are a more sensitive marker of noise disturbances during sleep compared to heart rate changes and EEG responses. SPL seems to be the most influential factor on PWA responses. Nonetheless, the noise type also seems to have an impact on the PWA response. Future work should examine a larger population exposed to more different noise types, which would be useful to help establish which component of the noise (amplitude modulation, low frequency, etc.) is the most influential on the cardiovascular system during sleep.

Acknowledgements

This research was supported by grants from the National Health and Medical Research Council (NHRMC; Project 1113571) and the Australian Research Council (ARC; Project DE120102185). The corresponding author would like to acknowledge the team that worked on this project as well as the whole team from the Adelaide Institute for Sleep Health.

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