A Pilot Study Investigating the Effect of Road Traffic and Wind Farm Noise on Electroencephalogram (EEG) Spectral Power During Stage 2 Sleep

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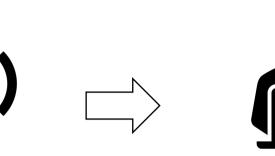
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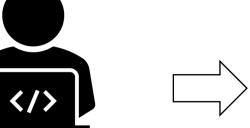


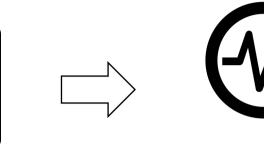
The Theory: The effect of wind farm relative to road traffic noise on human sleep microstructure remains unclear (1,2,3,4). To help guide further research in this area, this pilot study sought to

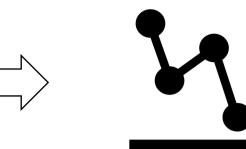
examine the impact of wind farm and short-range traffic noise (WFN and TN respectively), at varying sound pressure levels (SPLs), on sleep EEG using quantitative electroencephalogram (qEEG) analysis.













Overnight laboratory Brain signals sleep study recorded

Road traffic and wind farm noise played only whilst asleep

nd Manual scoring

Spectral power analysis

Artifact removal, normality assumptions (LOG10) transform

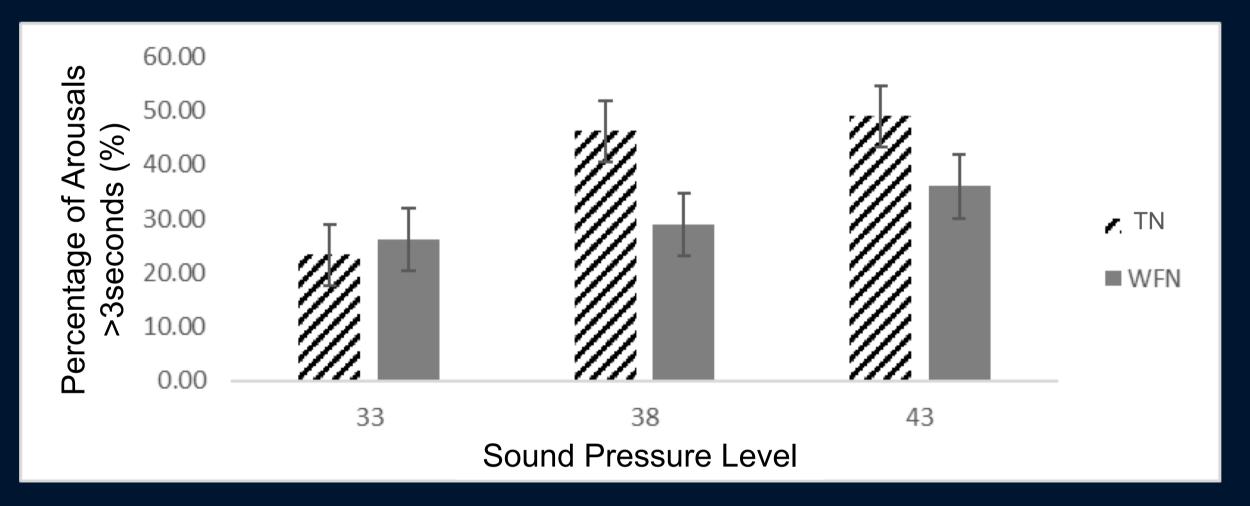
Data analysed using linear mixed model

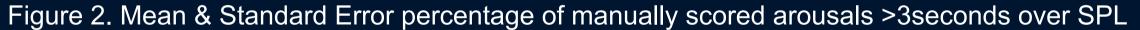
Results examined

Figure 1. Simple methodological breakdown of participation through to analyses.

Method: Twenty-three predominantly young healthy good sleepers (10 (41.7%) males, mean ± standard deviation; age 21.6±2.1 years, range 18-29) attended the Flinders University Sleep Laboratory. Participants were exposed to 3-minute WFN and TN stimuli played at sound pressure levels of 33, 38 and 43 dB(A) in randomised order throughout the night only during established sleep (>1 min stage 2 sleep or deeper). Power spectral analysis using a Fast Fourier transform method was conducted to noise events commencing in N2 sleep in 5-second epochs relative to stimulus onset before and throughout each 3-minute noise stimulus. The resulting EEG spectral power from delta to beta (0.5-4, 4-8, 8-12, 12-15, 15-32Hz) frequency ranges were normalised to baseline spectral power during the 15 seconds prior to stimulus onset and log10 transformed for each five second epoch and compared between noise types and SPLs within-subjects using linear mixed model analysis (Figure 1).

Short range nocturnal traffic noise resulted in more coinciding awakenings and increased fast frequency brain activity than low frequency wind farm noise with amplitude modulation at 43 dB(A) during stage 2 sleep. This may cause consequences for the establishment and maintenance of deep sleep which is not detected by traditional sleep scoring.





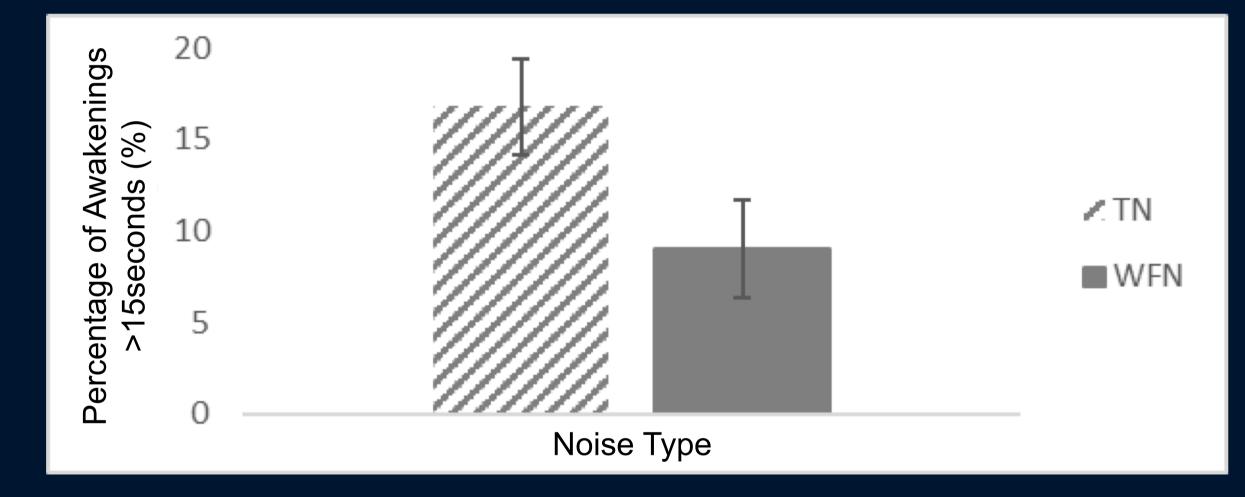


Figure 4. Mean & Standard Error percentage of manually scored awakenings >15seconds

and noise type.

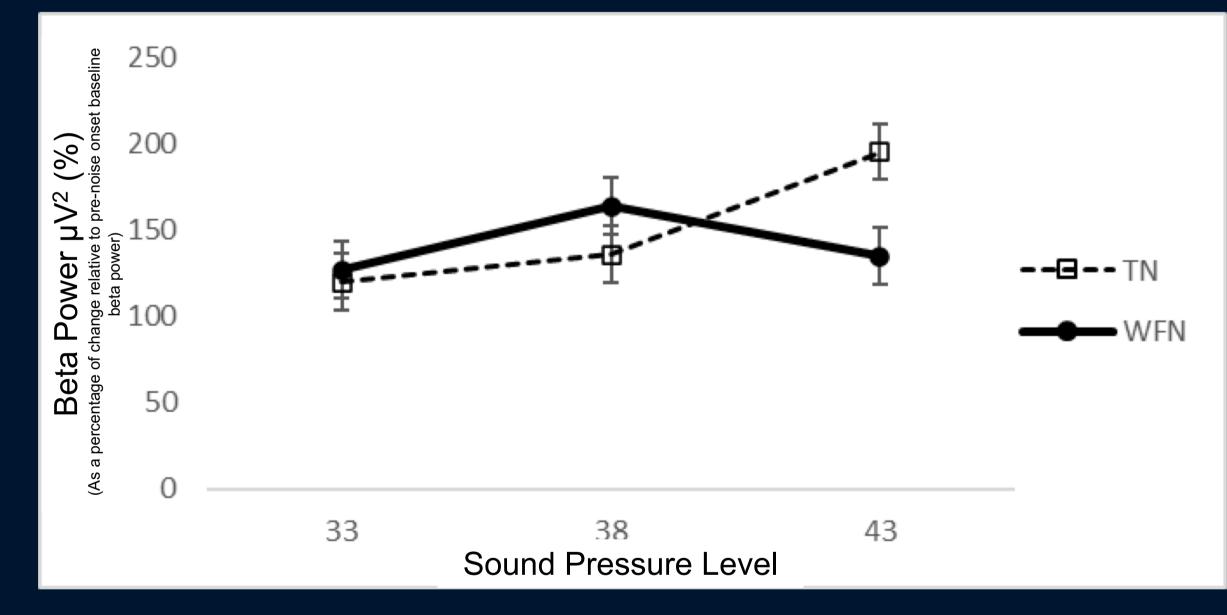


Figure 3. Mean & standard error of untransformed beta power as a percentage of baseline during traffic and wind farm noise with amplitude modulation at 33, 38 and 43 dB(A).

between noise type.

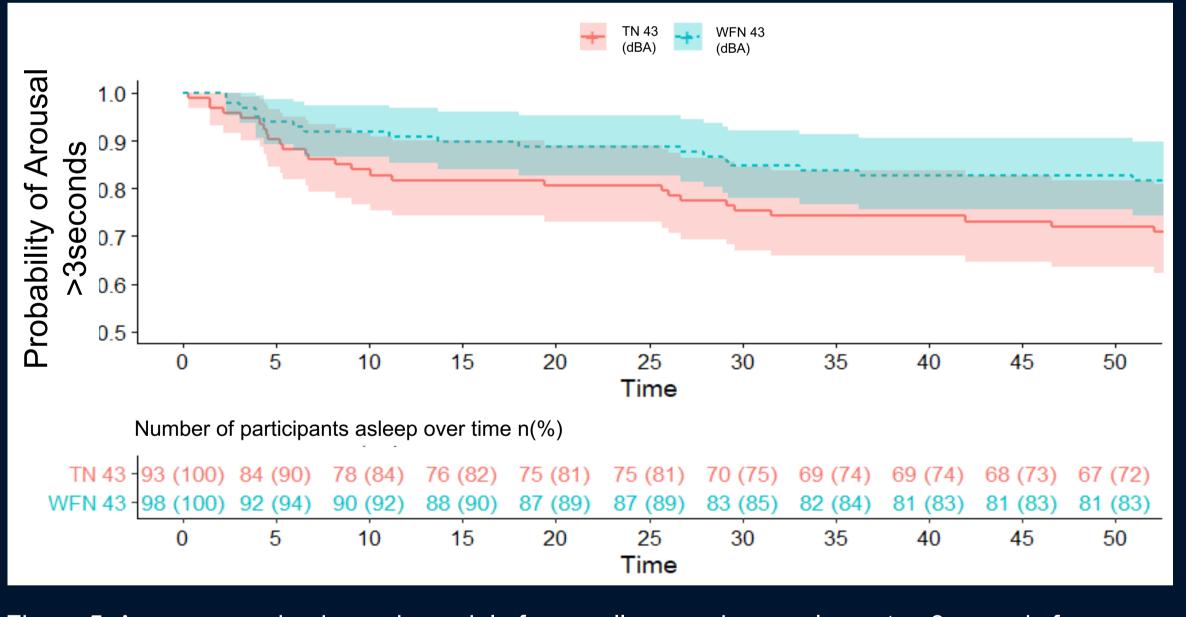


Figure 5. A cox regression hazards model of manually scored arousal events >3seconds for participants awake versus asleep between WFN and TN at 43dB(A) across the first 50 seconds of noise exposure depicting no significant probability to arouse between noise types.

Results: A significant noise type by sound pressure level interaction was found for beta power (15-32 Hz) during stage 2 sleep, where **traffic noise showed greater mean difference increase than** wind farm noise at high sound pressure level (Figure 2). At 43 dB(A) traffic noise was found to coincide with increased beta-power relative to wind farm noise. This is indicated by log10 transformed percentage of beta power relative to baseline power observed in the 15 seconds prior to noise onset at 43 dB(A). Log10 transformed percentage of beta power relative to baseline power relative to baseline power observed in the 15 seconds prior to noise onset at 43 dB(A). Log10 transformed percentage of beta power relative to baseline power relative to baseline power observed in the 15 seconds prior to noise onset increased significantly by 9.7% (0.097[0.048 to 0.146], *p*<0.001). This was a large effect, Cohen's *d*=1.01[0.40 to 1.62] (Figure 3).

A significant main effect of sound pressure level was found for percentage of arousals F(2,66.119)=6.522, p=.003. Percentage of arousals >3seconds was significantly higher during 38 dB(A) compared to 33 dB(A) (Mean difference ± Standard Error (12.82±5.22, p=.049)). And percentage of arousals were also greater during 43 dB(A) compared to 33 dB(A) (17.78±5.08, p=.003) (Figure 2).

Conclusion: These results suggest that TN leads to significantly greater increases in EEG beta

power during stage 2 sleep compared to WFN at 43 dB(A). However, further studies in larger samples of

A significant main effect of percentage of all awakenings was observed between noise types

(F(1,83.648)=4.521, p=.036), where significantly more awakenings occurred during exposure to

traffic noise compared to WFN (7.808±3.672, *p*=.036) (Figure 4).

WFN affected and unaffected individuals remain warranted. This study shows the value of assessing

microstructure events which are not observed in manual scoring but may significantly impact sleep.

The Future: Future studies should consider the importance of sleep microstructure in addition to

manual scoring and test for potential daytime impacts of the influence of disturbed sleep microstructure.

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PhD Scholarship funded by The Hospital Research Foundation



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