Establishing the Sleep Disruption Characteristics of Wind Turbine compared to Traffic Noise Using Quantitative Electroencephalography with Spectral Power Analysis

By

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Summary

Sleep is important for health and normal physiological and psychological wellbeing and daytime function. A well-known source of sleep disruption is nocturnal exposure to noise such as from air, road, and rail traffic. The consequences of consistently disrupted sleep can result in serious health deficits including hypertension, cardiovascular disease, impaired mental health, and daytime functioning. Therefore, all reports of significant sleep disruption warrant examination using appropriate sleep and noise assessment methods. Another source of nocturnal noise, increasing in its presence as the world attempts to reduce carbon emissions, is from wind farms. Noise from wind farms has more dominant low frequency components compared to other noise sources and its effects on sleep are currently unclear and need further investigation. Subjective reports of impaired sleep in some individuals living in the vicinity of wind farms have prompted the need for comprehensive investigation of the possible impact of wind farm noise (WFN) on sleep using objective measures of sleep in well controlled experimental studies. The gold-standard objective measure of sleep is polysomnography (PSG). However, the standard macrostructure sleep measures such as total sleep time and time spent in different sleep stages may not be sufficiently sensitive to capture more subtle changes within the EEG that could potentially differentially impact effective sleep quality and measures of daytime functioning.

This thesis used quantitative electroencephalography (qEEG) to objectively assess and compare the impact of traffic noise and wind farm noise on sleep. qEEG is likely to be more sensitive than traditional sleep assessment methods for evaluating noise effects on the sleep EEG. For example, traditional PSG analysis may find no effects of WFN on total sleep time, or the amount of time spent in individual sleep stages. However, it must be recognised that the definition of deeper sleep stages as distinguished from lighter stages of sleep is based on manual scoring of 30 second epochs and somewhat arbitrary and crude criteria dividing sleep stages. Potentially important differences within any given sleep stage in terms of amplitude, frequency, and power could easily be missed. These differences may importantly contribute to the functional effects of

deep sleep on the overall recuperative properties of the whole sleep period. If qEEG is sensitive to noise exposure but macrostructural analysis is not, qEEG analysis may be recommended for more comprehensive assessments of sleep beyond traditional macrostructure sleep analysis. Furthermore, such results would provide valuable feedback for informing noise guidelines and mitigation strategies which are currently based on more typically mid to high frequency dominated noise sources such as road traffic noise.

The first chapter of the current thesis introduces and examines the currently available evidence around nocturnal noise exposure and commonly used assessment methods with a focus on windfarm noise.

In the second chapter, 3-minute samples of road traffic noise (RTN) and wind farm noise with amplitude modulation were directly compared within 23 young healthy sleepers using quantitative EEG analysis across delta, theta, alpha, sigma and beta frequency bands (0.5-30Hz) during established N2 (non-REM stage 2) and N3 (non-REM stage 3) sleep. Three different sound pressure levels (33 dBA, 38 dBA and 43 dBA) were presented of both noise types (WFN and RTN). Despite minimal differences in traditional measurements of overall sleep macrostructure, there were significant noise sound pressure level (SPL) dependent increases in EEG alpha activity and delta activity. Responses were most evident in the first 5 seconds following noise sample onset with EEG predominantly rapidly returning to pre-noise onset states by 30 seconds post onset. The study also showed that at lower levels of noise exposure (33 dBA) in N2 sleep wind farm noise increased alpha EEG activity relative to road traffic noise. This study was among the first to directly compare WFN and RTN at variable sound pressure levels and showed the value of using qEEG to look beyond traditional measures of sleep macrostructure.

In the third chapter similar methodology was used to test qEEG responses during sleep to 3-minute samples of WFN infrasound at a sound pressure level of 80dBG. There was a small transient increase in delta activity during the first 5 seconds of noise exposure associated with an increased

probability of a K-complex. However, these EEG changes were relatively small and transient. This transient response to the onset of WFN infrasound also did not translate into increased arousals or awakenings. Therefore, there was no qEEG evidence to suggest that the short periods of WFN infrasound disrupts established sleep. To the authors knowledge this is one of the first studies to specifically assess acute EEG responses to windfarm infrasound exposure during sleep time locked to short exposure periods.

The fourth chapter of the thesis focused on whole night PSG recordings to assess sleep macrostructure and quantitative EEG responses to a range of noise exposure conditions. A total of sixty-eight participants were included from four groups of interest. Two groups lived <10 km from a wind farm, one (N=14) with self-reported WFN related sleep disruption and one (N=18) without WFN related sleep disruption. A further group (N=18) were rural residents without prior WFN exposure, and a fourth group (N=18) were urban residents habitually exposed to road traffic noise (RTN) and with self-reported sleep disruption to RTN. The seven-night protocol started with an adaptation night. Two subsequent conditions were randomised for order across participants and included (1) intermittent 20 seconds noise exposures composed of different types of WFN and RTN at varying intensity levels (30-50 dBA, whole night averaged SPL at ~42dBA); (2) a 3-minute noise exposure night composed of different types of WFN; infrasound and RTN at varying levels (30-35 dBA, whole night averaged SPL ~32 dBA). The last four conditions were randomised for order across participants and included: (1) a quiet control background noise night at 19dBA, (2) a full night of WFN exposure at 25dBA from lights out to lights on; (3) a night of continuing WFN at 25dBA but only during established sleep; and (4) a night of WFN exposure at 25dBA where noise was only present during wake periods from lights out to lights on. WFN exposure at 25dBA was utilised to represent the median level of WFN exposure recorded over the course of year-long measurements at residences 1-3 kilometres from a wind turbine. Full polysomnographic recordings were obtained for all participants for the seven exposure conditions and subsequently scored using traditional sleep assessment methods to extract sleep macrostructure variables. Full night qEEG

power spectral analysis was also used to test if qEEG analysis methods may be more sensitive to sleep disruption than traditional manual sleep scoring methods. There were significant main effects of condition on wake after sleep onset, total sleep time, minutes spent in N3 and REM sleep, with higher levels of sleep disruption compared to the quiet background noise control condition on the adaptation and 20 second noise exposures nights. K-complex density was also significantly increased for these nights and the 3-minute noise exposure night relative to the quiet control night. Whole night power spectral analysis revealed significantly increased beta activity during the adaptation and 20 second noise exposure night compared to the control night but with no other condition or condition by group interaction effects of interest. These findings support the presence of first night effects on sleep in an unfamiliar laboratory setting, and in the presence of intermittent 20-sec and 3-min noise exposures of 30-50 dBA overnight. However, continuous WFN exposure at 25 dBA throughout the night similar to real world exposure levels, or WFN only while awake or only while asleep, does not appear to significantly disrupt traditional sleep metrics or more sensitive measures including spectral power analysis of EEG and K-complex density.

Overall, the work presented in this thesis further demonstrates, in concordance with previous research, that intermittent noise events, particularly at higher exposure levels above 30 dBA are somewhat disruptive to sleep EEG, particularly at noise onset. However, there was no evidence in these studies to support that continuous WFN at an ecologically realistic level produces any objectively measured disruption of sleep either at the macro-structural or qEEG micro-structural level. The studies using higher sound pressure levels of noise in Chapter 2 that found some evidence of acute, short lived qEEG effects during sleep suggest that the onset of higher sound pressure levels of WFN have the potential to disrupt sleep and warrants further research.

These studies make an important contribution to understanding the impact of noise on sleep and the most appropriate methodology and tools to employ when measuring noise induced sleep disruption.

Declaration

I certify that this thesis does not incorporate without acknowledgment any material previously submitted for a degree or diploma in any university; and that to the best of my knowledge and belief it does not contain any material previously published or written by another person except where due reference is made in the text.

Signed: Claire Dunbar

Date: 3/05/2023

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List of Abbreviations

AASM	American Academy for Sleep Medicine
BMI	Body Mass Index
CI	Confidence Interval
dB	Decibel
dBA	A-weighted decibels
dBG	G-weighted decibels
df	Degrees of freedom
DSM-V	Diagnostic and Statistical Manual of Mental Disorders – Fifth Edition
DSST	Digit Symbol Substitution Test
ECG	Electrocardiography
EEG	Electroencephalography
EMG	Electromyography
EOG	Electrooculography
ESS	Epworth Sleepiness Scale
FFT	fast furiour transform
Hrs	Hours
Hz	Hertz
IQR	Interquartile Range
ISI	Insomnia Severity Index
Km	Kilometres
M	Mean
m	Metres
Mins	Minutes

ms	Milliseconds
N	Sample Size / number of participants
N1	Non-Rem /Stage One Sleep
N2	Non-Rem /Stage Two Sleep
N3	Non-REM / Stage Three Sleep
NREM	Non-rapid eye movement sleep
ORP	odd's ratio product
POMS	Profile of Mood States
PSG	Polysomnography
PSQI	Pittsburgh Sleep Quality Index
PVT	Psychomotor Vigilance Task
qEEG	quantitative electroencephalography
REM	Rapid Eye Movement Sleep
RTN	Road Traffic Noise
SD	Standard Deviation
Sec	Seconds
SOL	Sleep Onset Latency
SPL	Sound Pressure Level
SPLs	Sound Pressure Levels
TST	Total Sleep Time
WASO	Wake After Sleep Onset
WFN	Wind Farm Noise
WTN	Wind Turbine Noise

List of Publications From This Thesis

Dunbar, C., Catcheside, P., Lechat, B., Hansen, K., Zajamsek, B., Liebich, T., Nguyen, D. P., Scott, H., Lack, L., Decup, F., Andrew, V., & Gorica, M. (2021). EEG power spectral responses to wind farm compared with road traffic noise during sleep: A laboratory study. *Journal of sleep research*, e13517. https://doi.org/10.1111/jsr.13517

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Dunbar, C., Catcheside, P. G., Vakulin, A., Zajamsek, B., Lechat, B., Decup, F., Lack, L., Liebich, T. & Micic, G. A pilot study investigating the effect of Road Traffic and Wind Farm Noise on Electroencephalogram (EEG) Spectral Power During Stage 2 Sleep. Flinders University DocFest 2020, 8th-11th September 2020, Adelaide: Australia.

Dunbar C., Catcheside P., Lack L., Hansen K., Lechat B., Zajamsek B., Liebich T., Decup F., *Vakulin A., *Micic G. A pilot study investigating the effect of road traffic and wind farm noise on electroencephalogram (EEG) spectral power during sleep. *Adelaide Sleep Retreat 2020*, 12th of November 2020, Adelaide: Australia.

C. Dunbar., P. Catcheside., A. Vakulin., K. Hansen., B. Zajamsek., B. Lechat., F. Decup., T. Liebich., L. Lack & G. Micic. A Pilot Study Investigating the Effect of Road Traffic and Wind Farm Noise on Electroencephalogram (EEG) Spectral Power During Stage 2 Sleep. *The European Sleep Research Society Congress 2020*, 22nd -25th September 2020, Spain: Europe (presented virtually).

CHAPTER 1 : INTRODUCTION TO NOCTURNAL NOISE EXPOSURE AND SLEEP ASSESSMENT METHODS

1.1 Overview

Sleep disruption can have serious health and wellbeing consequences (Medic et al., 2017). Noise exposure, particularly traffic noise, is a well-known cause of sleep disruption (Basner & McGuire, 2018a). Thus, wind farm noise, like other noise types, has the potential to contribute to noise-related sleep disturbance. An ongoing shift towards clean renewable energy sources has led to a rapid increase in wind turbine facilities as part of the shift away from fossil fuel use. In Australia, the cumulative annual installed wind turbine capacity has increased from 1841 megawatts (MW) in 2010 to 9126 MW in 2021, with eight new wind farms commissioned in 2021 alone ("Clean Energy Australia Report 2022," 2022). This rapid expansion warrants investigation to clarify potential health effects of wind farm noise (WFN). This thesis specifically focuses on sleep given that potential sleep disruption is one of the primary mechanisms through which negative health effects could be mediated.

There are clear advantages for generating renewable energy in Australia, however, reports of impaired sleep by impacted communities cannot be ignored (Ageborg Morsing et al., 2018; Nissenbaum et al., 2012; Onakpoya et al., 2015). Debate continues around objective compared to subjective sleep responses to WFN exposure and the possible influence of individual characteristics such as noise sensitivity, annoyance and attitudes toward wind farms on sleep (Bakker et al., 2012; Liebich et al., 2021). Some studies suggest little to no influence of wind farm noise on objectively measured sleep (Liebich, Lack, Hansen, et al., 2022; Michaud, Feder, Keith, Voicescu, Marro, Than, Guay, Denning, Murray, et al., 2016). However, other research supports that nocturnal noise exposure can influence subjective and objective sleep, with impacts dependent on noise sound pressure level and sources of exposure (Basner et al., 2008; Muzet, 2007). Despite recent studies

suggesting no influence of WFN on objectively measured sleep, key questions remain unanswered such as whether traditional objective tools used to measure sleep are sufficiently sensitive to detect potentially more subtle impacts of noise on sleep. An objective sleep measuring tool considered to be more sensitive than traditional measures is quantitative electroencephalography (qEEG). This has been used to assess subtle changes in sleep to external stimuli to more comprehensively understand EEG responses in the potential absence of more traditional manual scoring-based EEG measures of sleep disturbance (Lechat, Hansen, et al., 2022; Scott et al., 2020). Thus, qEEG may be a more sensitive and useful technique to detect sleep disruption to low level noise sources.

The primary aim of the work presented in this thesis was to use qEEG to determine if there are more subtle impacts of noise on sleep than might be apparent with conventional EEG sleep scoring. Objective methods of spectral analysis were used in this thesis to quantify subtle changes in EEG to determine the response of the sleeping brain to traffic noise, a well-established sleep disrupter, compared to noise produced by wind turbines, at a range of relevant sound pressure levels.

Further this thesis work aimed to examine whether differences in past history (via residential location) of noise exposure and noise sensitivity influence the sleep EEG under a range of controlled laboratory conditions including intermittent noise exposure, no noise (control) and continuous wind farm noise exposure at levels similar to average real-world levels. Noise history and individual differences in noise sensitivity may assist in explaining varying reports in sleep disturbance to date and could assist in formulating relevant guidelines towards mitigation strategies for adversely affected individuals.

1.2 Primary Implications

This thesis sought to expand knowledge to assist in the understanding of qualitative reports of significant sleep impairment attributed to wind turbine noise. A recent study that

applied conventional sleep staging analysis failed to find wind farm noise exposure effects on sleep (Liebich et al., 2021). However, this does not rule out more subtle effects on the sleep EEG analysis which may still compromise sleep quality (Martin et al., 1997; Terzano et al., 1988). Thus, more comprehensive and objective EEG analysis of sleep in the presence of noise exposure is needed to determine whether nocturnal noise impacts the sleep EEG beyond traditional analysis of sleep disruption. This work may be helpful to better inform guidelines and potential noise mitigation strategies potentially needed to help protect noise-impacted individuals in the community.

1.3 Human Sleep

1.3.1 Sleep regulation

Sleep is regulated predominantly by two main processes including process C (the circadian system) and process S (the homeostatic system) (Tarokh et al., 2010). Process C is regulated largely by the supra-chiasmatic nucleus in the hypothalamus of the brain which receives light input through retinal melanopsin containing ganglionic cells within the eyes and controls the overall behaviour of the body clock (Borbély, 1978; Tarokh et al., 2010). This includes physiological and behavioural factors that change over the 24-hour sleep/wake cycle. These circadian rhythms are one of the main reasons humans are referred to as diurnal (i.e., we sleep during the night and wake during the day).

Process S describes the homeostatic process underpinning the biological need for sleep which increases "sleep pressure" during wakefulness and decreases while we sleep. "Deep" or slow-wave sleep is specifically thought to be recuperative of normal brain function and reduction of sleep pressure built-up during extended wake (Tarokh et al., 2010). Chronically disrupted slow wave sleep, sleep deprivation or poor-quality slow wave sleep can have significant impact on our wellbeing and daytime function (Williamson & Feyer, 2000).

1.3.1 Sleep stages

Human sleep is recognised to consist of four sleep stages, with each dominated by particular features and frequency characteristics of the electroencephalogram (EEG). In traditional sleep scoring, this divides sleep into Rapid Eye Movement (REM) and Non-REM (NREM) sleep (N1, N2, N3) (Conrad Iber et al., 2007). Figure 1.1 depicts the EEG characteristics which dominate each sleep stage, including wakefulness. In addition to sleep EEG, several other signals are used to help categorise and score sleep into each recognised stage of sleep. These include electromyography (EMG) and electrooculography (EOG), which help to capture muscle relaxation and slow rolling eye-movements around the time of sleep onset. During REM sleep, muscle activity assessed using EMG is normally at its lowest level throughout sleep. REM is also characterised by rapid eye movements observed in EOG. These markers assist in identifying and classifying REM sleep in comparison to NREM sleep where rapid eye movements are absent. Traditional EEG scoring is applied to characterise each 30 second segment of a sleep recording. The criteria for scoring EEG are specified in more detail in section **1.13 Sleep Assessment and Scoring**.

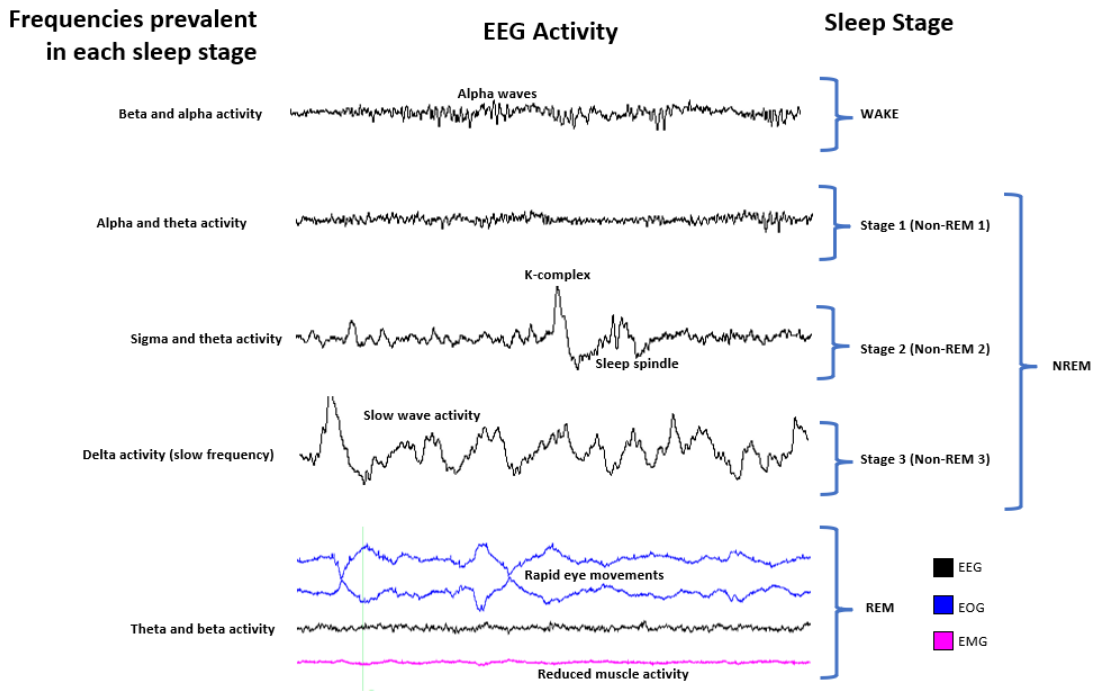


Figure 1.1 Image owned by author: Sleep stages as seen in electroencephalography and defining characteristics. EEG =electroencephalography, EOG=Electrooculography, EMG=Electromyography.

1.3.2 Sleep Cycles

As shown in Figure 1.1, human sleep is characterised by electrophysiological changes in the brain. Each stage of sleep occurs with greater prevalence at certain times in the sleep cycle. Normal nocturnal sleep is made up of sleep cycles that typically last ~90-minutes, and consist of transitions from light, too deep through to REM sleep. Normal adults generally experience around five ~90minute sleep cycles across a 7-8 hour sleep opportunity. Early sleep cycles are dominated by deeper slow wave sleep, and later cycles by more REM sleep. In traditional sleep scoring, this can be shown in a hypnogram as depicted in Figure 1.2.

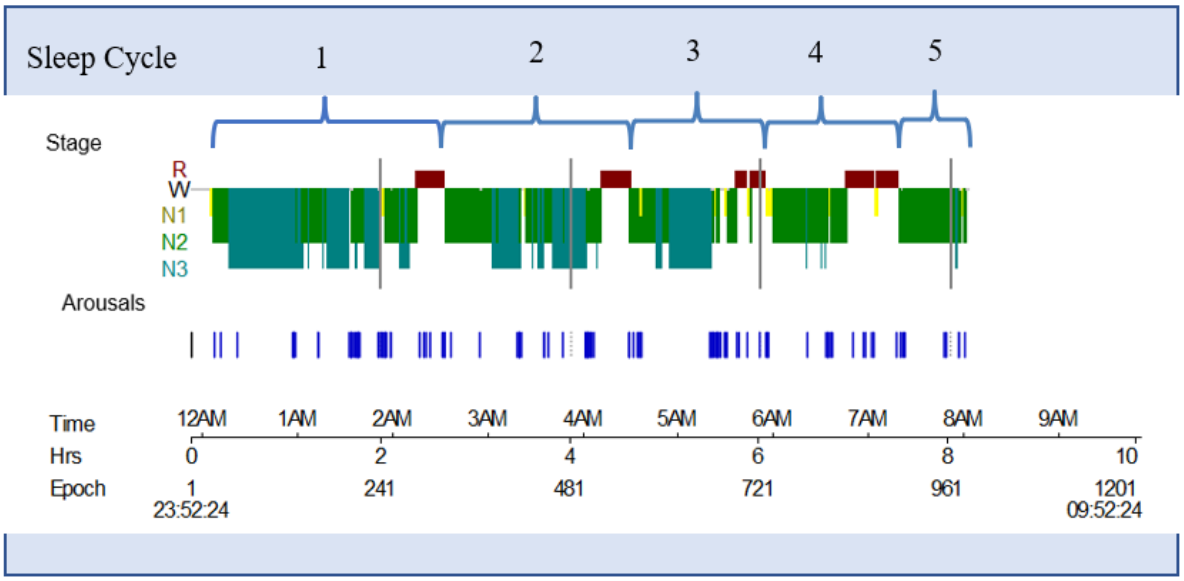


Figure 1.2 A relatively normal sleep across the night showing five sleep cycles of approximately 60-90 minutes from a hypnogram of a sleep study conducted on a human. R=REM, W=Wake, N1=NREM 1, N2=NREM 2, N3=NREM 3.

1.3.3 Sleep Macro and Micro-structure.

For the purpose of this thesis, the term sleep macrostructure is used to refer to traditional sleep measurement outcomes obtained from conventional manual sleep scoring applied to each 30-second epoch of the recording. The most common of these measures are Total Sleep Time (TST), Wake After Sleep Onset (WASO), Time in Bed (TIB), Sleep Onset Latency (SOL), percentage and time spent in sleep stages (N1 min and N1%, N2 min and N2%, N3 min and N3%, REM min and REM%), and the frequencies (count per hour) of awakenings (>15 seconds EEG shifts to faster frequencies) and shorter arousals (>3 second EEG shifts to faster frequencies but <15 sec). Sleep macrostructural parameters largely ignore more subtle short-temporal features and frequency changes within each 30-second period of the recording.

In comparison, the term sleep microstructure refers to more subtle changes and shorter than 30-second events within sleep EEG. These are typically assessed by applying a spectral analysis to quantify EEG power in different frequency bands, and by considering

discrete short time-scale EEG events such as EEG micro arousals (3-15 seconds) and K-complexes, which are a type of EEG slow wave often elicited by sensory stimuli such as noise events, and one of the characteristic features of N2 sleep. Typical EEG frequency bands used to assess sleep, and applied in the studies presented in this thesis, are beta (15-30Hz), sigma (12-15Hz), alpha (8-12Hz), theta (4-8Hz), and delta (0.5-4Hz). These bands cover the full frequency range of EEG most relevant to sleep and allow for a more in-depth analysis of EEG changes in response to noise stimuli than is possible with traditional manual EEG scoring alone.

1.4 Sleep Disruption

Sleep disruption can cause short- and long-term consequences on health and wellbeing (Medic et al., 2017). Even if an individual is otherwise healthy, chronically impaired sleep can lead to increased stress responses in the body, difficulties with memory and task performance and emotional distress. Long term incidental consequences of impaired sleep may include serious health conditions such as hypertension, cardiovascular disease, problems with body weight, increased risk of mental health problems and some forms of cancer (Medic et al., 2017). Persistent sleep disruption resulting in fragmented sleep is also associated with an increased risk of all-cause mortality (Lechat, Hansen, et al., 2022; Medic et al., 2017; Roth, 2007). Although associations may not necessarily indicate causal relationships, given the importance of sleep for normal brain functioning and general health, reports of chronic sleep disruption within the community clearly warrant investigation.

1.5 Noise Induced Sleep Disruption

Despite the loss of conscious awareness and ability to behaviourally respond intelligently to external stimuli during sleep, a degree of auditory sensory processing remains active (Muzet, 2007). Nocturnal noise exposure has previously been shown to measurably disrupt sleep and elicit

physiological activation responses (Bruck et al., 2009; Catcheside et al., 2002, 2006), which could potentially contribute to daytime functional performance and health problems. Environmental noise has been shown to significantly disrupt the micro-and macrostructure of sleep (Griefahn, 2002; Griefahn et al., 2006), and impair daytime cognitive functioning and mood (Muzet, 2007). Noise effects differ based on noise type, sound pressure level (SPL, typically quantified in deciBels dB) and frequency of the noise (typically quantified in Hertz Hz) (Micic et al., 2018). Noise effects on sleep are well established for aircraft, road vehicle, and rail traffic noise, which are all types of mid to high frequency noise known to affect both objective and subjective sleep measurements (Evandt et al., 2017; Griefahn et al., 2008; Griefahn et al., 2006; Micic et al., 2018). However, little is known about the potential for sleep disruption from low frequency noise (LFN), and in particular for wind farm noise (WFN). More research is emerging which supports the need to research LFN and the possible impact on health and well-being (Baliatsas et al., 2016; Erdélyi et al., 2023; Kristina H. Erdélyi et al., 2023). A review of the available literature on LFN conducted by Waye (2004) highlighted the potential for low frequency noise to impact sleep and the need for further consideration of low frequency noise impacts on sleep beyond subjective outcomes. Subjective reports of sleep disruption and health impacts residents in the vicinity of wind farms have attributed to wind farm noise supports the need for sensitive and objective analysis of sleep effects of WFN exposure (Botelho et al., 2017; Carlile et al., 2018; Pedersen, 2011). Mid to high frequency noise typically produced by traffic, air and rail noise and low frequency noise, including those dominant components of WFN, have been shown to influence self-reported annoyance and sleep disruption (Basner et al., 2011; Evandt et al., 2017; Leventhall, 2004; Micic et al., 2018; Zajamšek et al., 2016). For example, several differences in subjective and objective sleep measures with road, rail and air noise exposures appear to depend on the dominant acoustic frequency bands, SPL and acoustic rise time (e.g. sudden onset) (Basner et al., 2011).

1.6 Cortical Responses of the Brain During Noise Disturbed Sleep

The human body continues to respond to the environment around it during sleep through cortical and biophysiological mechanisms during the night (Öhström & Björkman, 1988; Zaharna & Guilleminault, 2010). Noise induced sleep disturbance may occur on both a macro- and microstructure level. Sleep disruption is commonly assessed by quantifying the number of awakenings or arousals in response to a period of noise exposure (Elmenhorst et al., 2019). Additionally, the response of the body to noise exposure can be observed in subtle changes through cardiovascular and electroencephalography changes (Carter, 1996; Griefahn & Jansen, 1978; Lechat, Scott, et al., 2022). The absence of macrostructural sleep changes does not rule out the possibility of more subtle sleep disruption effects and potential consequences such as increased daytime sleepiness and mood impairment (Martin et al., 1997; Terzano et al., 1988). Cortical responses to noise during sleep can be assessed through a spectral analysis of recorded EEG, using Fast Fourier Transform (FFT) methods to quantify brain wave activity in the frequency domain (Prerau et al., 2017).

The method of quantifying sleep through spectral power analysis is not a new technique and has been extensively used previously to analyse sleep disruption in the context of sleep related breathing disorders and insomnia (D'Rozario et al., 2017; Zhao et al., 2021). These studies support the value of quantitative EEG analysis in identifying subtle changes in EEG which is not necessarily observed in traditional sleep scoring techniques. For example, an individual could be diagnosed with paradoxical insomnia (subjective insomnia without objective evidence), when in fact they may be experiencing sleep fragmentation only discernible in subtle EEG features not necessarily evident in conventional sleep macrostructural variables such as total sleep time.

1.7 Auditory Processing of External Noise Events

The human auditory system continues to process external noise stimuli during sleep and thus,

processing of nocturnal noise events can be observed through evoked response potentials (ERP's) in the brain to external auditory stimuli (Atienza et al., 2001; Campbell & Muller-Gass, 2011). These ERP's are most commonly categorized as K complexes, a certain wave form most commonly observed in lighter stages of sleep as a positive peak followed by a negative deflection (see Figure 1.3). K-complexes have previously been shown to be a sign that the brain is processing external auditory information, particularly responses to sudden noise exposure onset. Lechat et al. (2021) provided a comprehensive examination of how the prevalence and odds of a K-complex occurring relate to noise exposure and level.

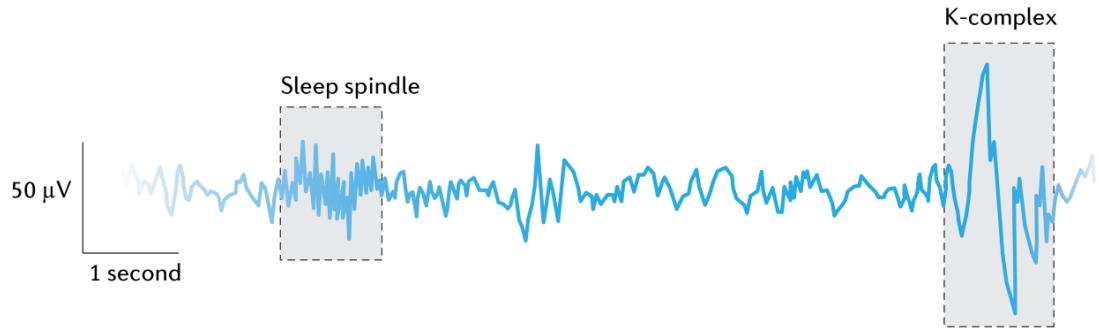


Figure 1.3. K-complex wave formation (the sharp peak and inflection) and a sleep spindle

Lechat et al. (2020) validates and provides the techniques to extract and quantify k-complexes and this technique will be applied in the current thesis to examine the brain response to specific noise events. Specifically, **Chapters 2 and 3** will examine dose-response relationships to short onset noise events and **Chapter 3** examines the potential for elicited responses in the form of K-complexes.

1.8 The Impact and Potential Burden of Noise Disturbed Sleep

There are several mechanisms through which ongoing exposure to noise may disturb sleep, including the potential for hyperarousal.

1.8.1 Hyperarousal

Hyperarousal in the context of insomnia is recognised as the tendency to focus on sleep difficulties and ultimately developing learned sleep preventing habits (Riemann et al., 2010). This hyperfocus may also cause anxiety about the quantity and quality of the individuals sleep and stress leading up to the sleep period subsequently making sleep a stressful and difficult process. Furthermore, a dual relationship may exist between anxiety and sleep, such that anxiety may in itself increase risk of hyperarousal and sleep disturbance. Furthermore, continued and ongoing difficulties with sleep can ultimately lead to an increased likelihood for the development of mental health difficulties including depression and anxiety (Riemann et al., 2010; Wu et al., 2023). When applied in the context of noise induced sleep disturbance, an individual may develop hyperarousal to the presence of nocturnal noise, and therefore become more prone to sleep impairment in the presence of noise (Waye et al., 2003; Zajamsek et al., 2023).

1.8.2 Daytime Impairment

Research supports the impact of noise disturbed sleep on daytime functioning (Hume et al., 2012; Muzet, 2007). Daytime sleepiness is a commonly reported consequence of impaired sleep. Increased noise exposure is positively correlated with subjective daytime sleepiness (Basner, 2008; Joost et al., 2018). Daytime sleepiness levels increase in a dose-response relationship with noise exposure. Daytime sleepiness has many consequences, including an increased risk of daytime impairment with the potential to cause serious detrimental effects on ability to function such as impaired reaction speed, slowed executive function and difficulty with critical decision making (Bioulac et al., 2017). This consideration is of particular importance in the context of investigating sleep impairment for individuals in rural populations, where long stints of driving may be more likely to occur (Probst et al., 2007).

Exposure to WFN has been linked to reported reduced quality of life (Shepherd et al., 2011). The degree to which WFN or noise emitted from wind turbines causes a detrimental impact has in part been attributed to how annoying individuals found the noise or how sensitive they deemed themselves to the noise (Knopper & Ollson, 2011; Pohl et al., 2018).

1.9 Wind Farm Noise

WFN is variable with fluctuating SPL and frequencies making it dynamic in nature (Hansen et al., 2017b). WFN is comprised of low frequency noise (LFN) below 200 Hz, making it unique compared to other forms of environment noise such as traffic noise. The dominant frequency content of RTN depends on the distance from the road. However, within one kilometre, RTN is typically dominated by 100-2000Hz.(Bolin et al., 2011; Hansen & Hansen, 2008). The primary sound produced by wind turbines is an aerodynamic effect as the blades pass through the air and pass the tower (Bolin et al., 2011). Firstly, as the blade passes the tower it produces a noise in the very low frequency range (<30Hz) and generates infrasound which is defined as noise <20Hz. The infrasound produced by wind turbines at realistic exposure levels is typically well below average human hearing acuity curve (Leventhall, 2007) and is thus inaudible. However, it should be noted that much like any other sound, infrasound exposure at sufficiently high sound pressure levels can become audible. Another sound produced by wind turbines is often referred to as the “trailing edge” or swish component and can produce an amplitude modulation (AM) effect. Dissipation of higher frequencies from multiple combined wind turbines creates a “rumbling” sound. The amplitude modulation of this sound is created by the variation in sound pressure level as the blade passes the tower at different frequencies creating changes in frequency and level of the sound hence the modulation (Micic et al., 2018).

The swish or trailing edge component of the sound is often in the higher frequencies (500-1000 Hz) and dissipates to a greater degree over distances (due to being absorbed by the

atmosphere and ground), which leaves the lower frequency components (<200 Hz) including those which contribute to amplitude modulation. In Australia residential dwellings are commonly located > 1 kilometre from a wind turbine, such that noise at the property is comprised to a greater degree of lower frequencies (≤200 Hz) which are less likely to dissipate over distances (Micic et al., 2018). The degree of audibility of LFN depends on factors such as wind turbine type, land topography, wind condition, speed and direction with respect to the listener (Hansen et al., 2017b; Van den Berg, 2005). Atmospheric conditions at night with more stable conditions of lower temperatures, temperature inversions and wind shear, are also typically more favourable to noise propagation, particularly of low frequency components, further increasing the likelihood that that more individuals may be exposed to audible levels of wind farm noise (Hansen & Hansen, 2021). Furthermore, it has been suggested that the amplitude modulated component of WFN may be more discernible at night when other background noise is typically lower (Hansen et al., 2014).

1.10 Infrasound

An ongoing debate around the infrasound component of WFN exists, with some anecdotal reports suggesting this form of sound contributes to health and sleep impairment despite being below the hearing threshold. While some literature states that infrasound cannot be processed by the human sensory system due to being considered inaudible (Leventhall, 2007), theoretically infrasound may be detectable by the inner ear (Salt & Kaltenbach, 2011). The cochlea (Organ of Corti) in the human ear contains sensory hair cells (inner hair cells (IHC) and outer hair cells (OHC)) that transmit auditory information to the brain. The IHC is insensitive to low frequency stimuli. However, the OHC responses rely on a different ear mechanism (i.e., displacement when sound displaces the air and hair cells in the ear rather than velocity where sound waves vibrate and move through the air to the ear) and therefore hearing levels do not reduce at lower frequencies at the same rate as the IHC. Therefore,

although components of WFN may be inaudible, they may still stimulate the OHC enough to elicit a response in the brain. Infrasound audibility is dependent on sound pressure level, where infrasound with sufficiently high SPL will be audible even if it is < 20Hz (Salt & Hullar, 2010; Salt & Kaltenbach, 2011). Furthermore, functional magnetic resonance imaging (fMRI) studies support that sub-audible infrasound (relative to the hearing threshold) can activate areas of the brain during wakefulness showing the potential for a physiological effect (Weichenberger et al., 2017). However, infrasound produced by wind turbines typically falls below the hearing threshold of 85 dB(G) (Broner, 2010). For context, reference to G-weightings or dB (G) refers to G-weightings based on the audibility curve for frequencies <20Hz which are frequencies below the normal audible human hearing range of 20-20,000Hz. In contrast audible frequencies are referred to in the dB(A), or A-weighted frequencies which fall in the audible human hearing range (Salt & Hullar, 2010; Salt & Kaltenbach, 2011). When referring to infrasound in the current thesis it will be referred to in the context of dB(G) and other noise types will be described in dB(A) as per appropriateness for the frequency and audibility of the noise examined.

Despite this, the potential impact of infrasound as a contributing factor of WFN cannot be ruled out as a possible contributing noise component to the reported sleep disruption and individual variability in hearing thresholds should also be considered.

1.11 Road Traffic Noise

Road traffic noise (RTN) is generated by the friction of tires on roads, aerodynamic noise, motor, transmission and exhaust noise of motor cars, trucks, motorbikes and other vehicles. The frequency range of RTN typically includes mid to high frequency components (500-1500Hz), with the higher frequency components (>3 kilohertz (kHz)), (including but not limited to; exhaust noise, lack of mufflers, motorcycles on sharp take offs and brakes from buses and trucks) commonly reported to be the most disruptive at higher sound pressure

levels and short SPL rise times (i.e., sudden onset high SPL events) (Basner et al., 2011). Sufficiently high SPL RTN is established to significantly disrupt sleep. Reports on how high the SPL of RTN needs to be to cause disruption varies depending on noise type and the population of the researched sample but generally falls around a threshold of 40 dB(A) (Basner & McGuire, 2018a; Kawada, 1995). To help further validate the use of qEEG as a marker of sleep disturbance, RTN will be used in the present thesis as a positive control. The inclusion of a positive control is important, particularly in the context of WFN, where the effects of noise on sleep may be small and potentially specific to underlying noise features. RTN is also a useful comparator in the context that existing noise guidelines are predominantly designed around RTN exposure. m

Given the varying forms of WFN elements, and the mix of these separate components on sleep it is important to consider which component may lead to the highest sleep disruption to inform mitigation strategies. This thesis will examine these components separately using sensitive measures of sleep assessment to carefully determine the ability of the sleeping brain to respond to the low frequency noise components of WFN including amplitude modulation, in relation to higher frequency noise components derived from traffic noise. This will be conducted in **Chapter 2**.

1.12 Literature Review on Objective Assessment of Noise and Sleep

This introduction has previously covered and acknowledged the effect of noise exposure in general on sleep. In brief the following section will now concentrate on discussing the literature in relation to the specific types of noise which will be focused on throughout this thesis including RTN, WFN, infrasound and the current literature available around the impact of these noise types on objectively measured sleep.

1.12.1 Traffic Noise (Road Rail and Air) Sleep Disturbance

Road, rail, and air traffic noise are among the most widely studied source of sleep

disruption. The volume of this research in itself speaks to the theory that noise impaired sleep is an ongoing issue for the community (Basner & McGuire, 2018a). Studies show linear associations between sound pressure level and increasing sleep disruption measured by increased awakenings or arousals, and fragmented sleep macrostructure (Basner et al., 2011; Griefahn et al., 2006). Different noise types in the mid to high frequency (M-HF) range appear to influence this dose-response relationship with SPL. Research suggests that the likelihood of disruption has a threshold of 40 dB(A) whereby noise levels below this do not appear to elicit disruption (Kawada, 1995). However, more recent research suggests the threshold for disruption is dependent on noise onset time, type of noise and potentially the familiarity or annoyance rating of the noise (Basner et al., 2011; Elmenhorst et al., 2019; Oswald et al., 1960). Particularly of interest is the variability in threshold for awakening or sleep disruption for lower noise levels, and more conformity at particularly higher noise levels (70 dB(A)) (Elmenhorst et al., 2019). This is relevant for the present thesis where lower SPL's occurring in WFN are typically thought to be too low to elicit sleep disruption.

Polysomnography (PSG) measured sleep macrostructure, in response to road rail and air noise exposure at 50-74 dB(A) compared to control quiet nights (32 dB(A)) in a controlled laboratory environment prolonged slow wave sleep (SWS) onset, reduced TST, reduced SWS in the first cycle of sleep, and resulted in reduced evaluated sleep quality (Griefahn et al., 2006). Type of traffic noise is also shown to impact sleep related disruption, where shorter rise time (i.e., time taken for noise to reach its highest sound pressure level) for rail noise when compared to road and air noise at the same SPL foremost contributed to a greater degree of sleep disruption.

In another study, intermittent RTN exposure across a night with a 6.3 second rise time and an average 20.5 second duration of each discrete noise exposure event was the most disruptive to objective measures of sleep when compared to intermittent rail and air single noise exposures (Basner et al., 2011). It should be noted here that exposure length was shorter

for road and rail noise exposures than air noise exposure (which was on average 66 seconds) (Basner et al., 2011). Short intermittent exposure may be more disruptive due to less opportunity for the sleeping brain to habituate to the noise although research is still emerging to support this theory and further studies are required to understand the mechanism behind differences in intermittent and continuous noise exposure on sleep (Smith et al., 2023). Road traffic noise appears to be most disruptive to objectively measured sleep followed by rail noise with air traffic noise showing the least adverse effects when compared with noise free control night. Furthermore, significant disruptions of sleep parameters were noted when individuals were exposed to more than one noise type during the night with multiple noise exposure events, compared to only one noise type, speaking to the importance of considering constant versus changing noise types on sleep (Basner et al., 2011).

When investigating the impact of environmental noise on sleep, it should be carefully considered not only from the sound pressure level of the noise, but also the onset time, exposure length, variety of noise presentations and the individual's relationship to the noise including their attitude and annoyance towards noise.

The literature to date suggests that RTN is the most disruptive noise source to sleep for the following reasons: a) it affects sleep microstructure b) it impairs sleep above and beyond rail and air (Basner et al., 2011).

Hence, it is the most appropriate positive control comparator to use in this thesis. The onset time of the noise, exposure length of the noise, noise presentations, attitudes, and annoyance (Basner et al., 2011; Shepherd et al., 2010) are also considered to impact sleep and thus will be examined in this work specifically in **Chapters 2, 3 and 4**.

1.12.2 WFN Sleep Disturbance

A less comprehensively studied area of nocturnal noise disruption as previously discussed is the exposure to noise generated by wind farms. However, more recently research

on WFN effects on sleep has noticeably increased (Ageborg Morsing et al., 2018; Jalali, Bigelow, et al., 2016; Liebich, Lack, Hansen, et al., 2022). Reviews of this literature have previously demonstrated that when meta-analysed, objectively measured sleep macrostructure using overnight PSG and actigraphy showed no change in sleep macro structure parameters in the presence versus absence of WFN (Liebich et al., 2021). However, studies do vary in their outcomes and, to date, studies examining measures beyond traditional manually scored sleep macrostructure are limited.

A recent study directly compared WFN noise at 25dBA across four intervention nights with continuous exposure to noise, exposure only during sleep or wake against a quiet control noise night (19 dB(A)) in a controlled laboratory setting considering previous exposure and attitudes to noise source (Liebich, Lack, Hansen, et al., 2022). This study found no significant impact of WFN exposure on sleep macrostructure relative to quite control nights (Liebich, Lack, Hansen, et al., 2022). Despite the level of noise (25 dB(A)) being significantly lower than the quiet control nights in previous traffic noise literature, the WFN exposure in the Liebich, Lack, Hansen, et al. (2022) study was averaged whole night in-field measurements from Australian wind farms. However, 25 dB(A) is significantly lower than the recommended noise guidelines (Basner & McGuire, 2018a) and may not represent the likely fluctuations of WFN across the night due to changing wind speed and condition. Furthermore, this study did not consider changes in sleep microstructure between exposure conditions which may be more sensitive to subtle noise exposures at low levels such as these.

An in laboratory study undertaken by Smith et al. (2020) exposed individuals with and without previous wind farm noise exposure to full night exposure of WTN at 32dB $_{laeq}$ and a control quiet night. The authors found some increases in polysomnographic markers of sleep disturbance during the WTN night, including longer REM latency and reduced REM sleep but no further markers of polysomnographic sleep disruption. However, self-reported sleep disruption was rated worse by individuals previously exposed to wind turbines after

WTN exposure. These findings support the need for an exploration of potentially more sensitive measures of sleep microstructure which could help to explain the discrepancies between traditional polysomnographic sleep scoring outcomes and subjective reports of sleep disruption. Using actigraphic markers of sleep disruption, Michaud, Feder, Keith, Voicescu, Marro, Than, Guay, Denning, Murray, et al. (2016) also found some evidence to support WFN exposure effects on sleep to warrant further qEEG assessments.

A pre and post methods study (Jalali, Bigelow, et al., 2016) conducted in residents homes using ambulatory PSG recorded higher overall night time SPLs (e.g., average of 36.55 dB(A) pre turbine operation and 36.5 dB(A) post operation) compared to the Liebich, Lack, Hansen, et al. (2022) study. However Jalali, Bigelow, et al. (2016) also found no difference in macrostructure sleep parameters, but did find a significant reduction in self-reported sleep quality, and an increase in stress post exposure (Jalali, Bigelow, et al., 2016). This supports the previous finding that sleep macrostructure may not be impacted by the presence of WFN or is too blunt of a measure, not sensitive enough to detect subtle effects. Alternatively, it may be the case that there is no effect on any aspect of EEG during sleep and that participants awareness of the presence of wind farm contributes to self reported annoyance and potentially reporter bias. Individual attitudes and beliefs regarding noise exposure and inter-individual differences in annoyance may play a significant role in subjective outcomes. Such effects are very difficult to control, particularly in real-world environments, and warrant randomised controlled trials in controlled environments with participant and investigator blinding to the extent that is possible in laboratory studies. The authors also performed an event related analysis attempting to look further at EEG responses, and found mixed results on a small sample of EEG segments. Jalali, Bigelow, et al. (2016) were unable to conclude whether exposure to WFN caused distinctive change to EEG. However, this EEG analysis did find value in depicting some EEG response in the form of arousals and shifts to higher frequency EEG not identified in sleep macrostructure. The authors noted the need for more

rigorous analysis of EEG to truly understand the relationship of these EEG events to noise exposure (Jalali, Bigelow, et al., 2016). This study nicely highlights the need and value of looking beyond traditional sleep analysis techniques and despite the small sub analysis shows potential for more comprehensive EEG analysis in evaluating sleep related disturbance and provides an initial insight when subjective and traditional scored objective sleep macro structure are discordant.

There is some evidence that different characteristics of WFN including sound pressure level, amplitude modulation and frequency range of the noise (low or mid frequency) may have different effects on sleep (Ageborg Morsing et al., 2018). In a controlled laboratory setting, Ageborg Morsing et al. (2018) showed that nights with an average SPL of 34 dB(A) resulted in an increased frequency of awakenings relative to quiet control nights. However no other measures of sleep macrostructure were impaired according to Ageborg Morsing et al. (2018). Further sleep disruption in the form of increased wakefulness was observed at 32.8 dB(A) when the noise sample included strong low frequency amplitude modulation, depicting the importance of assessing WFN characteristics on sleep.

1.12.3 Infrasound and Sleep

Finally infrasound and its effects on sleep are still relatively un-explored, predominately as infrasound in the frequencies exhibited in the environment are usually below the audible hearing threshold and therefore viewed as unlikely to affect sleep (Leventhall, 2007). However, as previously discussed, audibility of infrasound is inter-dependent and may still provide potential cause for investigation particularly during night hours when background noise levels are likely to be lower. Infrasound can also be produced not only by wind turbines but also by other environmental sources (e.g., wind turbulence, storms and earthquakes) (Broner, 1978). In a study of infrasound and low frequency exposure

on sleep undertaken by Okada and Inaba (1990), the authors pre-determined the infrasound response thresholds to be at 105 dB for an exposure of 20 Hz. The authors used these responses to inform a possible threshold for any sleep disruption for infrasound at 20Hz level and concluded that for infrasound below 20Hz (i.e., 10 Hz), sound pressure level threshold for any sleep disruption needs to be higher than the 105 dB. As such the authors exposed individuals over 3 nights to varying SPLs ranging from 60-105 dB for 10 and 20 Hz infrasound. The exposure periods were short (30seconds) in each 20 minutes of sleep across the night. Traditional measures of sleep disruption including changes in sleep stage and sleep efficiency appeared to be non-significantly impacted by exposure to infrasound (<=20Hz) or low frequency sound (40 and 60Hz at 95dB and 90dB respectively) when compared with a control night. These results suggest that the infrasound used in this experiment did not significantly disturb objectively measured sleep macrostructure.

More recently the effects of wind farm infrasound on sleep have been explored in a three arm cross over trial comparing infrasound (at an average 90 dB peak infrasound level) against a sham noise and traffic noise (Marshall et al., 2023). This more comprehensive study examined whole night analysis split by REM and NREM sleep for traditional and non-traditional metrics of sleep including spectral power in the common sleep associated frequencies from beta to delta activity (0.5-30Hz). This study concurred with previous research that infrasound exposure did not appear to detrimentally influence sleep or next day functioning. The study did find a small reduction in alpha activity during tests of wakeful infrasound exposure in alpha activity with eyes closed compared to sham.

Another study suggests that the reported impact of infrasound on sleep when measured in the vicinity of wind turbines is possibly related to psychological annoyance rather than a direct effect of the sound itself on sleep physiology (van Kamp & van den Berg, 2018).

A previous pilot study in a small sample showed potential for noise sensitive

individuals to perceive infrasound at a greater level than chance at 20Hz during wakefulness when exposed at 48dBG below the accepted hearing threshold of 85 dB(G) (Leventhall et al., 2003; Nguyen et al., 2019). The potential for sleep disruption at higher dB (G) levels should be considered in the context of the individual's reports of noise sensitivity and ability to perceive the sound during wakefulness, which previous studies have as of yet, not comprehensively conducted. This is examined in **Chapter 3** of the current thesis**.**

1.12.4 Literature Summary

This growing body of research on low frequency noise when considered alongside previous research on traffic noise sleep disruption shows three major considerations when assessing the impact of nocturnal noise exposure on sleep.

- A relationship can be expected between increasing sound pressure level and increasing elements of objective sleep disruption; however;
- Thresholds for sound pressure level dependent sleep disruption must be considered in the context of the frequency of noise, the rise time of noise onset, duration of noise exposure, the nature of the noise and the relationship of the individual to the noise (including reported attitudes and noise sensitivity). Finally;
- Where differences occur between subjective and objective sleep parameters on the macrostructure level, closely examining the sleep EEG microstructure may assist in explaining this disparity.

1.13 Sleep Assessment and Scoring

1.13.1 Polysomnography

Polysomnography (PSG) is the currently accepted gold standard assessment method for measuring sleep (Kapur et al., 2017). PSG encompasses measures of electroencephalography: EEG; electrooculography: EOG; electromyography: EMG; electrocardiogram: ECG; limb movements, body position, pulse oximetry, oxygen saturation,

and airflow and respiratory measurements including snoring and thorax and abdominal respiratory effort. When combined these measures provide a comprehensive assessment of the physiological components allowing for an accurate assessment of sleep (Rundo & Downey III, 2019). PSG has been used extensively and is the preferred method for assessment and diagnosis of sleep related breathing disorders and further used to assist in diagnosis of objective insomnia alongside subjective reports of sleep disruption (Rundo & Downey III, 2019). An illustration of PSG set up on an actor can be viewed in Figure 1.4.

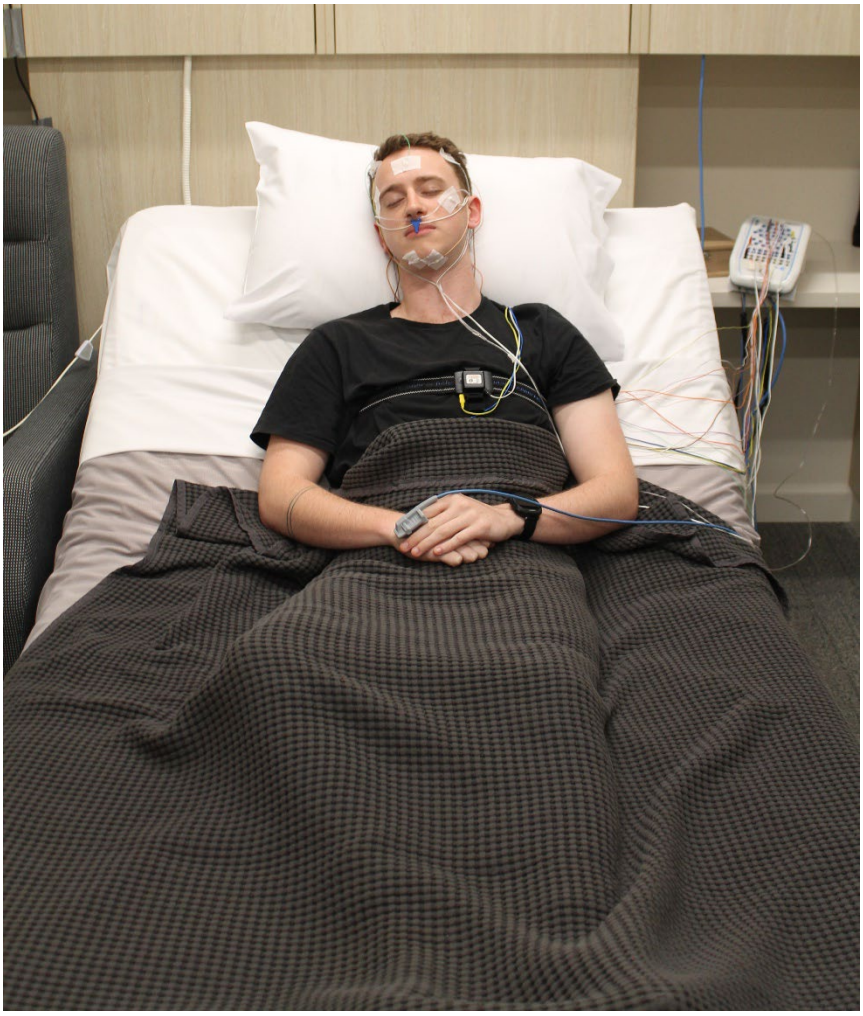


Figure 1.4 A mock set up of actor in full PSG as was completed for the current thesis. Note: Image owned by author, EEG, EOG, EMG, Respiratory bands, oximetry, and body position.

EEG is measured using gold cap electrodes, placed on the surface of the patient's scalp

using conductive gel. EEG measurement sites can be changed depending on research needs, however in typical assessment PSG EEG measures will be taken from frontal (F3, F4), central (C3, C4), and occipital (O1, O2) brain regions encompassing measures across the brain region in accordance with the internationally accepted 10-20 system. These placements are commonly grounded to the mastoid sites (M1 or A1, M2 or A2) to assist with removing muscle artifact. This assessment allows sleep to be scored accurately as the earliest signs of sleep are often observed in the occipital lobes of the brain through alpha activity and deep sleep delta waves are more clearly observed in the central and frontal regions of the brain. For the current thesis an additional electrode site at FZ at the frontal central position was added for research purposes beyond the scope of this thesis.

1.13.2 Scoring Sleep

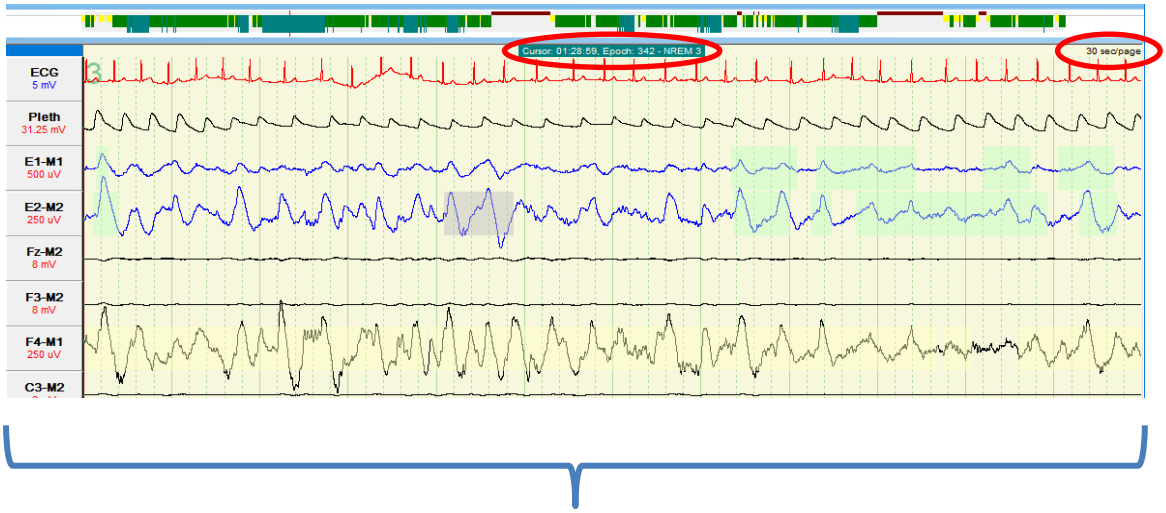
Scoring of the sleep period is undertaken by a trained sleep technician adhering to the American Academy of Sleep Medicine (AASM) guidelines (Berry et al., 2012). In the current study, by traditional convention, sleep recordings were separated and scored in 30 second epochs of each PSG recording. Each 30 second epoch is then examined by a qualified sleep technician to look for EEG patterns that correspond to different stages of sleep according to the AASM scoring criteria. The scorer can define the period as Wake, N1, N2, N3 or REM (see earlier description of sleep stages) depending on the physiological signs observed in the EEG, EMG and EOG according to AASM criteria. Only one judgement can be made for each 30 seconds of EEG data and these can only be classified into one of the five sleep states. This creates an inherent resolution limitation where 30 seconds of data can only be assigned one stage of sleep but may contain subtle and shorter changes in the EEG representative of multiple sleep states. The limitation of applying one state of sleep to a 30 second period of EEG is not relevant to spectral power analysis which quantifies all frequencies within a defined time period capturing a full representation of EEG states across frequencies ranging

from 0.5Hz to ~30Hz. During the time the scorer applies a sleep state to the 30 seconds of EEG, the scorer also looks for sleep architecture abnormalities, respiratory events and arousals or awakenings. These are further scored in the process to generate a report of the entire sleep period. Typically, these reports provide a description of the sleep macrostructure including, time spent in bed, TST and percentage of sleep stages and states (Wake, N1, N2, N3 and REM), sleep onset latency (SOL), wake after sleep onset (WASO), arousals and apnoea hypopnea index (AHI), which reflects the number of complete (apnoea) or partial (hypopnea) upper airway collapse events that last at least 10 seconds per hour of sleep (C. Iber et al., 2007). These measures are used in research as well as providing differential diagnosis of sleep disorders in the sleep community and to assist with formulation of appropriate treatment (Bloom et al., 2009). However, PSG alone is not the only consideration to be taken into account when diagnosing sleep disorders, which requires a comprehensive clinical assessment (Reite et al., 1995).

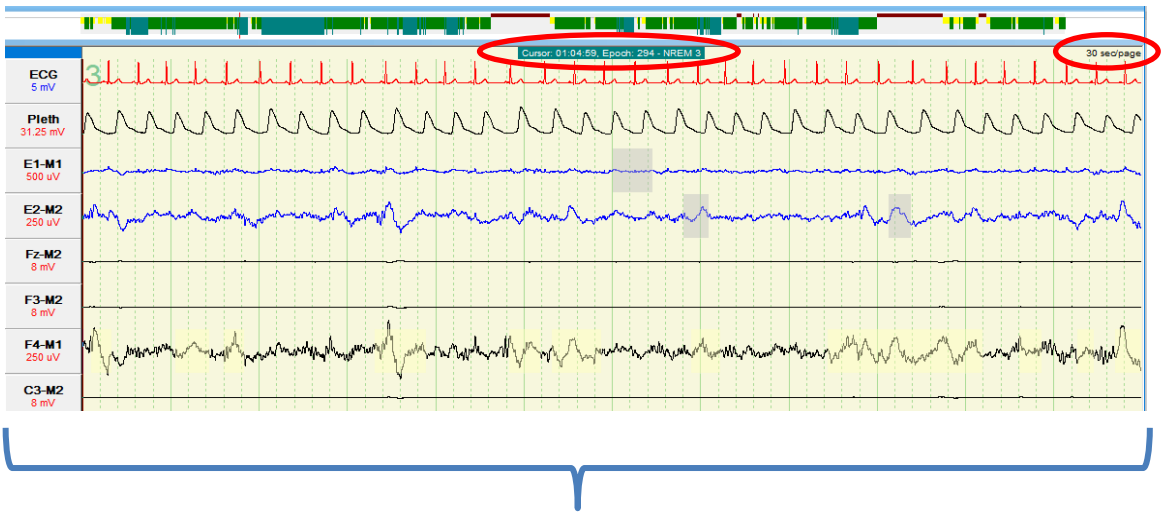
1.13.2.1 Considerations of Traditional Sleep Scoring

As described above, the accurate analysis of PSG is heavily reliant on the accurate interpretation of the sleep scorer who attributes stages to the short epochs of sleep across the night. This method introduces the potential for human error and considerations for interrater reliability problems between scorers. For stages with clear indications of sleep stage (N2 and N3) interrater reliability of scorers is more likely to be higher, whilst scoring of stages which require more clinical judgement and interpretation (N1), interrater reliability is likely to be significantly lower (Lee et al., 2022). Sleep scoring in this traditional sense does not allow for the interpretation of EEG markers which have previously been shown to be valuable in understanding daytime impairment (Vakulin et al., 2016). The primary use of this traditional sleep scoring was to provide sufficient objective EEG information to clinicians for sleep disorder diagnosis. This resulted in the original sleep stage classifications which have been

reduced over the years combining what was traditionally stage 3 and stage 4 sleep into one category of N3 sleep. In analysis of N3 sleep the sleep scoring criteria requires that 20% of a 30-second epoch with 75 microvolts (mV) or greater wave amplitude needed to meet classification of N3 (delta slow wave activity) (Keenan & Hirshkowitz, 2011). However, there is considerable difference in sleep quality between a 30-second epoch that contains 20% delta activity of 75 mV amplitude and one that contains 80% delta activity at 150 mV amplitude. Here lies the problem, that whilst two segments of EEG recording may meet macrostructure criteria to be scored the same, the EEG information contained in the same two epochs (30-second time window) may show very different electrophysiological sleep patterns. Thus, by using traditional methods of sleep scoring with a 30-second resolution there is the potential to miss the subtle elements of sleep physiology at a finer grain level. The difference that can occur but still be scored as N3 sleep is highlighted in the below figure shown in the F4-M1 channel.



One 30 second epoch scored as N3 sleep



One 30 second epoch scored as N3 sleep

Figure 1.5.Two 30 second epochs (circled in red) from the same individual on the same night at different times both traditionally scored as N3 sleep despite significant variation in high amplitude EEG as seen in the F4-M1 channel.

Quantitative analysis of EEG power spectra is now beginning to be acknowledged as helpful in further understanding and identifying sleep reported difficulties in patients beyond traditional sleep scoring alone. It further allows for a greater understanding in differentiating clinical sleep disorder groups such as obstructive sleep apnoea and insomnia (D'Rozario et al., 2017). A good example of the value of EEG analysis can be seen in the analysis of macro

structure reports of sleep difficulties for individuals with subjective symptoms of insomnia compared to individuals with objective sleep impairment (Andrillon et al., 2020). Despite differences in their macro structure reports which showed less objective sleep impairment for subjectively reporting insomnia patients, the study showed a considerable overlap in measurement of their EEG; to the degree that the subjectively reporting insomniacs EEG spectra reflected much more similarity to their objective insomniacs EEG spectra despite their macro structure reflecting more closely a healthy sleepers (Andrillon et al., 2020). Our growing understanding of the usefulness of using EEG sleep spectra when differentiating insomnia phenotypes in conjunction with traditional measures of sleep macrostructure may assist in understanding the disparity of research findings when considering noise induced sleep disruption, particularly given the subjective nature of sleep disruption reports from affected individuals.

Ultimately, this research suggests that individuals who are objectively sleeping the same and when measured using EEG spectra are being diagnosed differently than when assessed using traditional less sensitive measures of sleep macrostructure. This has a high risk of leading to problematic misdiagnosis and false negatives which may prevent sleep impaired individuals from receiving the treatment they require. Therefore, how we measure sleep, the appropriateness of the measure in relation to the sleep impairment report and the source of impairment matters greatly.

1.14 Physiological EEG Measurement of Sleep Beyond Traditional Scoring

Intricate analysis of sleep micro-structure and changes in EEG spectra can be assessed through quantifying the EEG using power spectral analysis by applying a Fast Fourier Transform (FFT) (De Carli et al., 2004; Geering et al., 1993; Vakulin et al., 2016).

1.14.1 Mathematical Methods of Quantifying Sleep EEG Spectra

The Fast Fourier Transform (FFT) is a mathematical process which, when applied to a

specified EEG time segment (for instance 30 seconds of EEG sleep recording), quantifies the amount of EEG within the specified time segment that falls within a specified frequency range. This quantification is precisely mathematical, perfectly reliable in giving the same answer for the same sample of EEG, is not reliant upon subjective judgement, and is transferable between different sleep EEG patterns. It moves the interpretation of the sleep EEG signal from a crude time domain into a precise frequency domain (Achermann, 2009).

Once in the frequency domain there are several methods for analysing the area under the curve. When extracting the area under the curve both variance and resolution should be considered. Variance refers to the variability of the signal. Too much variance could mean increased noise and artifact in the sample, and too little variance means you may be washing out the variance of interest (i.e., those that show an increase/decrease in a specific frequency attributable to a stimulus). Methods for analysing sleep data have previously been compared (Prerau et al., 2017). By comparing methods including different ways of tapering segments of EEG, Prerau et al. (2017) outlines that one of the most effective forms is the multi-taper method, which uses multiple tapers on the chosen time segment of EEG to quantify the area under the curve. Given the dynamic nature of sleep EEG data with changing frequencies between and within sleep stages the multi-taper analysis proved more efficient in discerning differences in the EEG spectra than single taper analysis, providing a balance of resolution and variance (Prerau et al., 2017).

To better demonstrate this, Figure 1.7 and 1.8 express the EEG brain wave extracted from a segment of EEG recorded from a C3 electrode placement across the night and shows the different methodology variations of where the frequencies fall when quantified into the frequency domain.

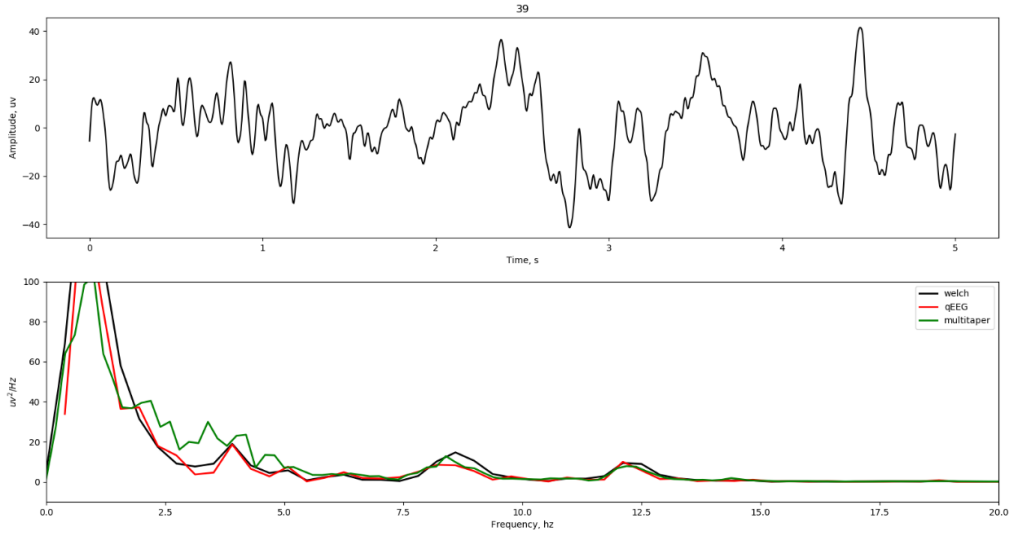


Figure 1.6. Image owned by author. Top: An extracted segment of deeper sleep EEG in the time domain. Bottom: The converted quantified frequency (area under the curve) across multiple analysis methods including welch, qEEG (with a single taper) and Multitaper analysis.

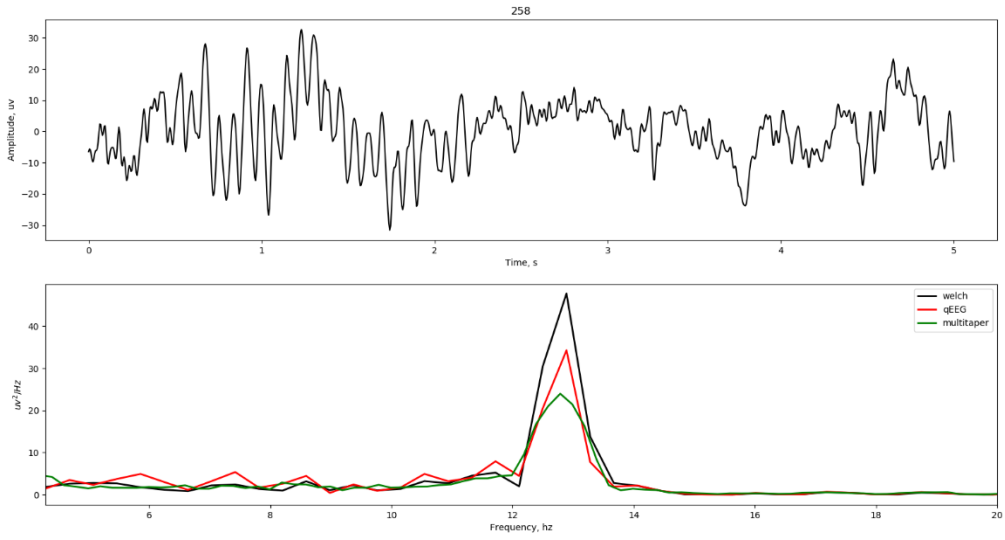


Figure 1.7. Image owned by author. Top: An extracted segment of lighter sleep EEG in the time domain. Bottom: The converted quantified frequency (area under the curve) across multiple analysis methods including welch, qEEG (with a single taper) and Multitaper analysis.

Because of the ability to retain variance of interest and sufficient resolution the multi-taper method was used in the current thesis to quantify the area under the curve during the

FFT and data extraction process.

Unless otherwise specified, EEG spectra in the current thesis is derived as per the processes described above in 5-second time windows. These are then averaged into intervals of interest where appropriate for **Chapters 2 and 3** and averaged for whole night comparisons in **Chapter 4**.

1.15 Justification of qEEG use in the Present Thesis

To the knowledge of the researcher, there is no previously published work utilising quantitative EEG (qEEG) to examine the effect of full spectrum WFN on overall sleep micro-structure in humans. However, qEEG derived via Fast Fourier Transform (FFT) power spectral analysis has been used in various clinical aspects to quantify sleep disruption for clinical patients with obstructive sleep apnoea (OSA), insomnia, and explored environmental noise impacts in a rat model and through an odds ratio product analyses in humans (Cervena et al., 2004; Krystal et al., 2002; Rabat et al., 2004; Smith et al., 2019; Vakulin et al., 2016).

In a rat model, adjusted spectral analysis showed decreased and fragmented slow wave sleep (SWS) during the first four hours of exposure to environmental noise (French aircraft carrier) and fragmented and decreased paradoxical sleep (Rabat et al., 2004). The nature of noise events evident in the environmental noise (i.e. low frequency spectrum adjusted to the rat model, and unpredictable noise events) prevented the establishment of paradoxical sleep, and furthermore the unpredictable nature did not allow for habituation and sleep pressure to take over resulting in fragmented SWS even during natural nocturnal times (Rabat et al., 2004). This study suggests that it is the combination of intermittent unpredictable noise exposure and potentially the low frequency spectral composition of the noise together have the most detrimental effects of the establishment and maintenance of sleep in a rat model. However, these characteristics of environmental noise need further investigation in humans.

The impact of traffic noise on sleep depth was further explored by Smith et al. (2019). The authors used an odds ratio product (ORP), based on alpha, theta, beta, and delta activity in EEG. Changes in the ORP reflect changes in EEG activity indicative of varying levels of alertness, ranging from zero (comatose) through to highly alert wakefulness. ORP was used to assess sleep depth in the presence of traffic noise events in 72 participants. Over a period of 10 nights, participants were exposed to varying types of traffic noise (Aircraft, road, rail, or combinations of the three) at 45-65dB comparative to a control 30dB night. Results show a significant effect of traffic noise, in particular road and rail noise, which increased the ORP, concluding a reduction in SWS across traffic noise exposures but to a greater extent during road and rail exposure relative to control. The authors further argue the odds ratio derived from spectral power analysis is a superior method of assessing noise related events on sleep (Smith et al., 2019).

Whilst spectral power analysis is a relatively novel approach to quantify the impact of noise related events on sleep, it has been used extensively and successfully for the objective measurement of sleep in various clinical populations such as insomnia and obstructive sleep apnoea research (Cervena et al., 2004; Krystal et al., 2002; Vakulin et al., 2016).

Differences in EEG power spectra has been documented in sleep onset insomniacs (Freedman, 1986), objective, subjective and primary insomniacs compared to controls (Cervena et al., 2004; Krystal et al., 2002). In a comparison of 12 self-report sleep onset insomniacs and 12 healthy controls in a laboratory-controlled environment, EEG power spectral frequency analysis was conducted on the first minute of defined sleep stages W, N1, N2, N3 and REM. The frequencies were processed using FFT from 0.5-30.5 Hz with 1Hz resolution. The insomniacs and normal sleepers did not differ in simple comparisons of sleep stage distribution based on their macrostructure. However, insomniacs had significantly less alpha power and increased beta power in a wake (eyes closed) condition. An increase in beta power was also evident during N1 sleep, and the beginning of the first REM cycle

(Freedman, 1986). Further differences between subjective and objective persistent primary insomniacs compared to controls yielded similar findings (Krystal et al., 2002). NREM EEG delta power was decreased and alpha, sigma, beta power was significantly increased in subjectively reporting insomniacs, where conventional PSG recordings of sleep stages did not show any differences. This study further found that in subjective insomniac's sleep, subjective sleep impairment complaints were related to reduced delta power and increased alpha, sigma, and beta, but showed no relationship with PSG sleep stage macrostructure (Krystal et al., 2002). On the other hand, in objective insomniacs there was a relationship between sleep complaints and conventional PSG measures, but no relationship with relative EEG power frequencies. The study suggests that EEG power spectral analysis (diminished low frequency and increased high frequency) may be important in the assessment of individuals who report subjective claims of insomnia symptoms that is not reflected in conventional PSG readings (Krystal et al., 2002).

The potential for disparity between macrostructure sleep reports and differences in EEG analysis should be considered in the present study given the subjective sleep complaints from residents in wind farm communities. Ultimately, these studies suggest that future research should not discount reports of sleep disruption based on macrostructure analysis of sleep alone. In particular, traditionally scored sleep metrics may not be sufficiently sensitive to detect noise-induced EEG changes that may nevertheless impair sleep and next day cognitive functions. Thus, qEEG and other objective markers of physiological changes during sleep warrant further investigation to test for noise exposure effects.

Quantitative EEG has also been examined in relation to driving simulator performance in individuals with obstructive sleep apnoea (OSA) (Vakulin, et al, 2016). This study used quantitative EEG analysis from overnight clinical PSG recordings to assess the relationship between EEG power during REM and NREM sleep with simulated driving performance (AusEd driving simulator). Regression models showed that greater beta EEG

power in NREM sleep and greater delta power during REM sleep, as well as extended sleep onset latency were associated with steering deviation in patients with OSA. Ultimately the quantitative EEG allowed for an intricate analysis of the PSG recording in addition to macrostructure analysis to inform associations between EEG power frequencies and daytime vigilance and performance tasks. This supports the use of spectral power analysis for objective assessment of subtle EEG changes during sleep, which may predict significant impairments in daytime alertness.

1.15.1 Quantitative EEG as a Sensitive Measure of Sleep Deprivation

In addition to its uses in clinical sleep studies, quantitative EEG has been used to examine the effects of sleep deprivation in healthy individuals (Tucker et al., 2007). In healthy individuals who were sleep deprived for varying lengths of time (e.g., 36 hours between 1am to 6am), EEG power spectra was a sensitive measure of sleep deprivation. EEG spectra was robust at distinguishing between normally rested individuals and sleep restricted individuals and showed significant shifts in EEG spectra toward increased slow wave delta activity (a potential marker of sleep homeostasis or sleep drive) during sleep onset relative to controls (Tucker et al., 2007). Furthermore, differences in wake alpha activity (alpha bursts) as well as changes to sleep onset in frequencies associated with arousal and sleepiness (delta, alpha and beta activity) could be seen as markers of sleep deprivation (Gibbings et al., 2021). The sensitive markers of states of EEG were beneficial in understanding reaction time for healthy but sleep deprived individuals. The use of the qEEG as a means of detecting mild sleep deprivation is becoming more common in research as it is considered to be sensitive enough to measure even mild changes in sleep states (Gibbings et al., 2021). Therefore the applications of EEG spectra assessment during and prior to sleep are becoming more readily used as predictors of scenarios requiring sustained attention such as driving simulation and in understanding the effects of sleep deprivation on recovery sleep (Gibbings et al., 2022;

Tucker et al., 2007).

Because qEEG allows for a greater understanding of the microstructure (beyond conventional and subjective measures) of sleep through power spectral analysis of the delta, theta, sigma, alpha, and beta frequency bands, it has been logically chosen as the objective measure of sleep in the current thesis. However objective sleep alone is not the only consideration when dealing with impaired sleep microstructure and daytime subjective self-reports of sleep disruption should also be considered (Martin et al., 1997).

1.16 Post Sleep Daytime Assessment Measures

Self-reported daytime impact of noise exposure is an important subjective dimension in addition to objective physiology measures. As such the impact of noise exposure on individual's reports of daytime symptomology and daytime sleepiness after noise exposure are commonly investigated and reported.

1.16.1 Negative Symptomology

Daytime physiological impacts of noise exposure including nausea, headaches, dizziness and tinnitus are commonly reported from individuals experiencing noise disturbance (Havas & Colling, 2011). This may be attributed to noise sensitivity, however evidence around these reports of negative symptoms is unclear. Daytime subjective reports of physiological symptoms were measured each morning after awakening in the study reported in **Chapter 4** through the administration of a self-report symptomology questionnaire which requested individuals to rate symptoms they may be experiencing from 0 (not affected at all) to -100 (extremely affected) on a sliding visual analogue scale (VAS) for commonly reported symptoms. The symptoms included headaches, vertigo, dizziness, nausea and more based on previously published work with additional items specific to our study (Tonin et al., 2017) (See Appendix F). Scores on individual items were compared across noise exposure conditions to discern any change in symptomology.

1.16.2 Daytime Sleepiness

Nocturnal noise exposure to both traffic and WFN is associated with daytime sleepiness (Abbasi et al., 2015; Basner, 2008; Schapkin et al., 2006; Tassi et al., 2013). However, what objective sleep measures and mechanism might relate to the subjective daytime sleepiness remains unclear (Liebich et al., 2021). EEG power spectra may in part help explain the link between noise exposure and daytime subjective sleepiness (Gaudreau et al., 2001). The study presented in **Chapter 4** of this thesis includes state daytime sleepiness assessed using the Karolinska Sleepiness Scale (KSS) each morning post awakening (Åkerstedt & Gillberg, 1990; Shahid et al., 2011). This is a nine-point self-report scale and asks the patient to report their level of sleepiness by rating from 1 (extremely alert) to 10 (extremely sleepy, can't keep awake). A score of 5 indicates neither feeling alert nor sleepy. Individual scores were compared across study mornings to determine any change in state levels of sleepiness post night-time noise exposure in **Chapter 4**.

1.17 Addressing the Knowledge Gap

The effect of nocturnal exposure to road, rail and air noise is well established for disruptions in sleep macrostructure (Basner & McGuire, 2018a; Griefahn et al., 2006; Griefahn et al., 2000). A growing body of evidence shows the value of delving deeper into analysis of the EEG beyond traditional macrostructure methods including evoked sleep responses, spindle activity and EEG spectra analysis (Basner et al., 2008; Rudzik et al., 2018; Tassi et al., 2013). These emerging studies show the use of spectral power analysis as a sensitive tool when distinguishing between differences in subjective and objective assessments of sleep disturbance particularly in the presence of subtle stimuli which may warrant more sensitive measures of sleep (Andrillon et al., 2020; Scott et al., 2020). Spectral power analysis allows us to look for changes in the sleep EEG not obtainable through traditional methods of sleep scoring. Given the sensitive nature of spectral analysis to pick up

variations within the EEG, it is well suited to the analysis of sleep in response to stimuli which may require more sensitive methods. This is particularly relevant as we have seen the successful use of spectral power in the analysis of nocturnal noise exposure for frequencies well within the audible hearing range such as traffic, road and rail noise, but evidence is mixed and continuing to emerge around noise exposure to lower intensities and lower frequency ranges (Liebich, Lack, Hansen, et al., 2022). Evidence to date suggests that WFN is unlikely to impact sleep macrostructure, however particular forms and components of this noise source may be more likely to cause disruption and degree of disruption may be mediated by the individuals sensitivity and relationship with the noise (Ageborg Morsing et al., 2018; Nguyen et al., 2019).

Given the serious adverse effects of long-term sleep disruption, research on the impact of noise on sleep needs to use methods that are sensitive to subtle impacts of noise. Solely concentrating on traditionally scored sleep macrostructure may result in missing changes in sleep physiology and quality and furthermore may underestimate the potential sleep disrupting effects of noise sources. It is also important that as new sources of sleep disruption begin to emerge, the tools we use to measure these sources are deemed appropriate. Therefore, the following thesis provides multiple opportunities to not only comprehensively assess the impact of noise on sleep, including subtle noise exposure at low frequency levels which may require more sensitive assessment methods by using quantitative EE; it also provides a unique opportunity to increase the knowledge and use of this new form of sleep assessment for wider use in assessing sleep disruption alongside previously established sleep assessment methods.

1.18 Thesis Aims

The aims of this thesis are, for the first time, to apply a novel measure of sensitive and mathematically precise sleep quantitative electroencephalography (qEEG) to assess the

impact of noise on sleep across infrasonic, low frequency and mid to high frequency ranges in carefully controlled laboratory settings. The primary aim of this thesis, which carries through in each chapter, was to apply and examine the impact of various characteristics of WFN on sleep qEEG to aid in investigating possible aspects of sleep EEG disruption in the presence or absence of primary sleep disruption typically assessed through sleep macrostructure. Furthermore, the thesis aimed to assess sleep disruption under different noise characteristics and sound pressure levels including mid to high frequency traffic noise compared with amplitude modulated wind turbine noise directly to determine possible dose-relationship responses of the sleeping EEG and how this differs depending on noise frequency and type. Furthermore, infrasound derived from WFN in the <20 Hz frequency range at subaudible levels against quiet background noise is also assessed taking into consideration, for any effects measured, the potential for influence of inter-individual participant noise sensitivity and daytime perception. Finally, the thesis will compare exposure to full spectra WFN at 25 dB(A) (including amplitude modulation and infrasound components), for three nights of study; under continuous exposure, exposure only during sleep (established N2, N3 and REM sleep) and exposure only during wake periods to ascertain the potential for this noise to influence sleep EEG in the presence or absence of conventionally measured macrostructural sleep disruption. In addition to these three study nights, the same participants will be studied on two nights of intermittent noise exposures with short exposures (20 seconds) on one night and longer (3 minutes) on the other night. Noise exposures will vary in intensity (30 dB(A) to 50 dB(A) in increasing increments) and type (WFN or RTN). These five noise exposure nights will be compared to a quiet no noise control night (19dB (A)). When combined these nights allow us to comprehensively assess intermittent and continuous noise exposure and SPL variation, sudden onset noise events compared to control and continuous noise exposure with stable SPL. Comprehensively these six nights of noise exposure provide an insight into how noise may disrupt the microstructure

of sleep under various exposure conditions.

1.18.1 Justification of Methodology for Sleep Studies

As previously discussed, there is significant variation in currently available research to assist in understanding the response of the sleeping brain on nocturnal noise exposure, particularly WFN and low frequency noise exposure. In part this can be attributed to the difficulty in studying this form of noise exposure. To be studied accurately, the WFN components must first be accurately reproduced requiring professionally trained personnel to measure, quantify and reproduce not only the full spectrum of the noise, but further isolate and administer the amplitude modulation and infrasonic components of interest. Furthermore, reproducing low frequency and subaudible noise in a laboratory setting requires specialist acoustic equipment with the capacity to handle frequencies in the sub-audible range which cannot simply be commercially purchased.

For these reasons, the laboratory studies presented in this thesis employed specially trained acoustic engineers with adequate training in the measurement and reproduction of low frequency noise including WFN. These valuable personnel were responsible for ensuring faithful reproduction of the noise stimuli for both laboratory trials. Yearlong measurements of WFN at residential dwellings within 3 kilometres from a wind turbine were comprehensively assessed and showed an average SPL of 26 dB(A) with variation in noise components (Nguyen et al., 2021). These yearlong results were not published at the commencement of this thesis; however, measurements were taken to inform the use of SPL at similar distances (approximately 3 kilometres) and were used to create a base level for dose dependent relationships starting at 25 dB(A) on the quietest noise exposure nights and increasing in SPL.

Current noise guidelines as assessed by the World Health Organisation (WHO) (1999) report acceptable continuous noise limits should not exceed 30 dB(A) for background noise

or 40 dB(A) at night as this has the potential to disrupt sleep. Therefore, it is important to examine a range of SPLs. The chosen SPL comparisons in this thesis reflect the current noise guidelines and the yearlong measurements taken by the trained acoustic engineers.

The noise stimuli were delivered via acoustically altered speakers to increase low frequency handling capacity in sound attenuated bedrooms. In the final chapter of the thesis to confirm faithful noise reproduction, the sound was further measured as it was produced by the speaker to ensure replicability and ensure the absence of noise artefacts.

Finally, the sample of individuals chosen to partake in these experiments was carefully considered. Firstly, **Chapters 2 and 3** of this thesis, during the process of establishing dose dependent and possible noise characteristic differences of nocturnal noise exposure, young healthy sleepers with minimal comorbidities were chosen to reduce confounding factors. For the final study presented in **Chapter 4,** careful consideration was taken to assess individual differences in noise exposure including noise sensitivity and relationship to the noise (i.e., whether the noise has an impact or benefit on their lives). As such participants were recruited from four pre-determined groups including; (1) RTN exposed individuals (residing within proximity (~<1km) to a busy road) who reported RTN related sleep disturbance; (2) WFN exposed individuals (residing <10km from a wind turbine) who reported WFN related sleep disturbance; (3) WFN exposed individuals (residing <10km from a wind turbine) who *did not* report WFN related sleep disturbance, and; (4) a quiet rural control group comprised of individuals who lived >10km from any wind turbines. The purpose of recruiting from these four different sample groups allows for group comparisons and assisted in answering whether previous exposure history and noise sensitivity are key contributing factors when assessing sleep disruption.

This thesis compiles and presents one of the first controlled laboratory examinations of sleep using the novel qEEG method of sleep assessment to examine noise exposure on sleep including both RTN and WFN with associated disruptive components, going beyond

traditional sleep metrics. As a research community, appropriate sleep assessment must be considered and regularly updated to reflect the emerging claims of sleep disruption to more subtle stimuli to inform appropriate mitigating strategies to prevent the potential for harm to the community. This thesis provides a first step at the possibilities and usefulness of expanding sleep assessment measurement looking at EEG in more depth and provides a premise for why this should be considered in standard research practice. The remaining four chapters of the thesis are presented in brief below.

1.18.2 Chapter 2

This shorter laboratory-based study focused on establishing the sleep disruption characteristics of audible WFN with amplitude modulation relative to a known disruptor road traffic noise (RTN). It utilised a laboratory within-subject's design administering short three-minute samples of amplitude modulated WFN against mid to high frequency RTN at three increasing sound pressure levels across established sleep (N2, N3) for young healthy individuals. The primary purpose of the study was to determine whether WFN relative to RTN showed differences in EEG and whether this was reflected in sleep macrostructure.

1.18.3 Chapter 3

This chapter follows on from the previous study utilising the same dataset to comprehensively distinguish the potential for EEG changes during high level (80dBG) low frequency infrasound exposure derived from WFN, comparative to quiet control background noise (23 dB(A)) periods during established sleep (N2, N3, REM) for young healthy sleepers. Results of differences within the EEG are further examined against ability to perceive infrasound during the day and noise sensitivity. The primary purpose of this chapter was to clearly distinguish whether the infrasonic component of low frequency WFN has the potential to change the sleeping brain EEG despite being classed as subaudible.

1.18.4 Chapter 4

This chapter consists of a comprehensive laboratory within and between-subject seven-night study consisting of intermittent variable SPL exposure nights (~30-50 dB(A)) and continuous full spectra (including low frequency components infrasound and AM) WFN exposure nights (25 dB(A)) compared to a quiet control background noise night (19 dB(A)). Here the thesis comprehensively assessed whether the short interval changing noise types with short onset times and varying length (20 seconds compared with 3-minute exposures) was more or less disruptive to sleep EEG comparative to low level continuous exposure or intermittent low-level exposure during the night compared with a quiet control noise exposure night. This study used ecologically representative samples of individuals from four exposure groups as previously described (See 1.18.1: Justification of Methodology for Sleep Studies) to determine the potential impact of self-reported noise sensitivity and previous exposure history on EEG outcomes. Whilst not a primary focus of the thesis work, the comparison of different groups in this chapter sought evidence to guide if individuals who may be more likely to be sensitised or potentially to have habituated to wind farm noise, or have different attitudes and beliefs toward a given noise source may exhibit corresponding differences in their sleep EEG.

Subsequent daytime sleepiness and daytime reports of negative symptoms because of noise exposure was further compared between groups and within individual night exposures. This chapter supports multiple purposes, the main one allowing for the comparison of traditional sleep scoring methods against the novel use of qEEG in the analysis of subtle and more evident sleep disruptive stimuli. Secondly, this chapter comprehensively assess the sleep disruption characteristics of WFN exposure at real world exposure levels compared to intermittent high noise exposure nights and control quiet noise exposure nights.

1.18.5 Chapter 5

The final chapter of this thesis will combine the chapters previously discussed to comprehensively address and answer the aims of this thesis and discuss avenues for further research and possible limitations.

CHAPTER 2 : EEG POWER SPECTRAL RESPONSES TO WIND FARM COMPARED TO ROAD TRAFFIC NOISE DURING SLEEP: A LABORATORY STUDY

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Abstract

Wind turbine noise (WTN) is dominated by low frequencies for which effects on sleep relative to more common environmental noise sources such as road traffic noise (RTN) remain unknown. This study examined the effect of WTN compared to RTN on sleep using quantitative electroencephalogram (EEG) power spectral analysis.

Twenty-three participants were exposed to three-minute samples of WTN and RTN at three sound pressure levels (SPL; 33, 38 and 43 dBA) in randomised order during established sleep. Acute [0-30 seconds] and more sustained [30-180 seconds] effects of noise presentations during N2 and N3 sleep were examined using spectral analysis of changes in EEG power frequency ranges across time in 5-second intervals.

Both noise types produced time and SPL dependent increases in EEG power, but with significant noise type by SPL interactions in beta, alpha, theta and delta frequency bands (all $p<0.05$). WTN showed significantly lower delta, theta and beta activity immediately following noise onset compared to RTN (all $p<0.05$). However, alpha activity was higher for WTN played at lower sound pressure levels (33 dBA ($p=0.001$) and 38 dBA ($p=0.003$)) compared to traffic noise during N2 sleep.

These findings support that spectral analyses show subtle effects of noise on sleep and that EEG changes following WTN and RTN onset differ depending on SPLs, however these effects were mostly transient and had little impact on conventionally scored sleep. Further studies are needed to establish if EEG changes associated with modest environmental noise exposures have significant impacts on sleep quality and next-day functioning.

Keywords

Sleep, wind farm noise, wind turbines, traffic noise, qEEG, spectral power.

2.1 Introduction

Ongoing construction and operation of renewable energy sources, including wind farms in rural areas, is likely to continue to expand towards reduced carbon emissions and reliance on fossil fuel energy sources. Thus, clarification of potential noise impacts on sleep remains important for community acceptance and evidence-based policy and decision-making around wind turbine use and placement; including noise and building regulations and potential noise abatement and remediation strategies that appropriately consider the wellbeing of nearby communities.

Environmental noise is known to disrupt sleep and to impair daytime cognition and mood (Muzet, 2007). Sound pressure level (SPL) and frequency (Hz) both influence perceptual responses to noise including the degree of subjective annoyance (Lee et al., 2011) and impact on sleep (Basner et al., 2011). Whilst sleep disturbance with increased arousals and awakenings from air, road and rail traffic noises are well-established, the relative impact of wind farm noise (WTN) on sleep remains largely unknown.

Unlike traffic noise, which consists mainly of sporadic mid- to high-frequency (500Hz to <4kHz) noises from a range of sources, WTN typically contains more prominent and time-varying low frequency components. These noises are predominantly from variable air flow over the wind turbine blades and their passage past the supporting tower, along with gear box, nacelle movement, tower vibration and transformer noise (Zajamšek et al., 2016). Aerodynamic sources produce audible noise fluctuations such as “swishing” (reflecting temporal variations in SPLs in the region of 500-1000Hz) and similar noise features characterised by amplitude modulation, particularly at low frequencies. Noise characteristics at nearby residences strongly depend on environmental factors such as wind direction, speed, temperature, humidity, land topography and the nature of intervening building materials. Due to their time-varying nature, these noise fluctuations may make WTN more intrusive,

annoying and difficult to habituate to than other noises of equivalent SPLs (Leventhall, 2006; Micic et al., 2018; Van den Berg, 2005). Thus, wind turbine noise clearly has the potential to disturb sleep (Leventhall, 2006; Van den Berg, 2005).

In carefully-controlled experimental studies, traffic noise has been shown to disrupt sleep through reduced total sleep time and time spent in slow wave sleep, and through more frequent arousals compared to quiet control conditions (Okada & Inaba, 1990; Smith et al., 2019). Sleep disruption effects also depend on the noise intensity and sleep stage at noise onset, with a greater likelihood of arousals and self reported sleep disturbance with rising SPL from 30 dB to 55 dBA (Basner et al., 2014) and a smaller likelihood of sleep disruption in deep compared to light sleep (Okada & Inaba, 1990).

Although noise effects on conventional sleep measures remain important, electroencephalographic (EEG) power spectral analysis may be more useful for evaluating EEG responses to different noise types than traditional sleep staging and arousal scoring alone (Martin et al., 1997), particularly over short time scales following noise onset. Quantitative power spectral analysis of EEG (qEEG) in the form of odds ratio product responses to aircraft, road and rail noise between 45-65 dBA shows reduced sleep depth in the presence of road or rail noise compared to a quiet (30dBA) control night (Smith et al., 2019). However, qEEG has not previously been used to investigate the impact of wind turbine compared to road traffic noise on sleep.

This study aimed to use qEEG analysis to directly compare EEG responses to WTN and RTN exposure during N2 and N3 sleep. Given existing evidence to support that low frequency noise components of WTN may be more annoying and sleep disruptive compared to higher frequency noise components within RTN, we hypothesised that WTN would result in more prominent qEEG changes than RTN.

2.2 Methods

2.2.1 Participants

This study was approved by the Flinders University Social and Behavioural Research Ethics Committee. Study participants were recruited through advertising on University noticeboards, word of mouth and social media. Interested individuals were screened for study eligibility via an online questionnaire (Qualtrics Pty Ltd, Utah, USA) and were provided with comprehensive study information prior to providing informed written consent. Participants were reimbursed AUD$300 for study completion. Subjects were informed that the purpose of the study was to examine wind farm and traffic noise effects on sleep.

Inclusion criteria included body mass index (BMI) <30 kg/m^2; Pittsburgh sleep quality index (PSQI) <6 (Backhaus et al., 2002; Buysse et al., 1989; Carpenter & Andrykowski, 1998); PSQI-based self-reported sleep efficiency >85%; insomnia severity index (ISI) <8 (Morin et al., 2011); Epworth sleepiness scale (ESS) <10 (Johns, 1991; Johns, 1992); and <2hr discrepancy in self-reported weekday vs. weekend sleep onset and offset times. Exclusion criteria included current smoking; trans-meridian travel within the last two months; pregnancy/lactating; and any comorbidity that could affect sleep (e.g. sleep apnoea). Participants were also screened for normal hearing (<20dB(HL) difference below normal hearing threshold between 125 - 8000Hz (Schaette & McAlpine, 2011)) by trained personnel using a calibrated audiometer (Entomed, Sweden, Type: SA 201) in the quiet sleep laboratory (< 23 dB(A)). Normal hearing was subsequently confirmed via more comprehensive hearing assessments undertaken by a qualified audiologist in an audiology testing facility.

Following participant screening and consent, participants completed a seven-day sleep diary (Carney et al., 2012) and actigraphy (Actiwatch 2, Philips Respironics, USA) for one week to assess usual sleep patterns and habitual lights out time. Participants then attended the sleep laboratory two hours prior to their habitual bedtime for the experiment.

2.2.2 Polysomnography sleep study setup

Overnight polysomnography (PSG) was recorded using Grael 4K hardware and Profusion 4 acquisition software (Compumedics Ltd., Abbotsford, Victoria, Australia). PSG setup included gold-cup electroencephalography (F3, Fz, F4, C3, C4, O1 and O2 referenced to linked M1 and M2), electrooculography (EOG), electrocardiography (ECG), chin electromyography (EMG), leg movement, nasal cannula, oro-nasal thermistor, chest and abdominal motion and finger oximetry.

2.2.3 Noise reproduction

For faithful reproduction of pre-recorded WTN and RTN samples, a Krix Pheonix V2.1 speaker with flat frequency response from 35Hz to 40kHz was placed alongside the bedroom wall approximately one metre from the foot of the participants' bed. Three-minute noise samples were reproduced from prior recordings inside residences with windows closed. WTN was recorded approximately 3.3km from a wind farm in rural South Australia. RTN was recorded around 20m from a busy main road (~44,000 cars per day) in metropolitan Adelaide, Australia. WTN samples contained prominent tonal amplitude modulation at 46 Hz, typical of worst-case conditions near a wind farm (Hansen et al., 2019). Three sound pressure levels (33, 38 and 43 dBA SPL) were selected to span real-world exposure levels (Hansen et al., 2019), commencing from approximately 10 dBA above background noise in the sleep laboratory and ranging up to 43dBA; which represents the upper range of WTN exposure levels in the field and substantially exceeds indoor recommended night-time noise limits in most countries (Alamir et al., 2021). 33dBA is also relatively high, but is more realistic for indoor WTN recordings in the field, where background noise levels are typically lower and indoor and outdoor SPLs in a year-long study were 26 and 32 dBA respectively (Nguyen et al., 2021). Prior to the experiment, noise reproduction equipment was calibrated using a SVANTEK 958 sound level meter. Noise randomisation, reproduction and synchronisation timing signal output was controlled by custom software implemented in MATLAB (Version 2018a/b 9.4/9.5, Mathworks,

Natick, Massachusetts). This also maintained technical staff blinding of noise types and SPLs.

Laboratory temperature was controlled at around 23 °C using a low background noise air-conditioning system. The absence of windows and the use of acoustically sealed doors helped to further reduce external noise to achieve low background noise (23 dBA). Light levels were <10 lux prior to and following sleep, and <1 lux during the sleep period.

2.2.4 Study protocol

Participants were instructed that they may or may not hear a range of noises during the study night. However, noise exposures for this study only commenced following lights-out and at least five minutes of N2 or deeper sleep using AASM scoring criteria (AASM; (Conrad Iber et al., 2007). WTN and RTN samples were then played throughout the night in randomised sequences, each consisting of all noise samples at each SPL, including quiet control periods (23dB(A)) and infrasound (80dB(G)) to be reported elsewhere. Each noise sample was separated by a 20-second quiet period designed to minimise potential carryover effects between noise samples, but with as much noise sample replication as practical across the night. In the event of an awakening (>15 second EEG frequency shift), overnight technicians were instructed to use the custom noise-replay interface to cease noise presentations at the end of any ongoing noise sample and to only recommence noise replay after at least five consecutive 30-second epochs of N2 or deeper sleep. These procedures were designed to ensure that noise samples continued through brief arousals or shifts to wake until the end of each sample for full evaluation of EEG power spectral changes to noise, whilst also facilitating the re-initiation of sleep without ongoing noise disturbances after awakenings.

2.3 Data analysis

PSG sleep staging and arousal scoring were conducted by a qualified sleep technician blind to

the acoustic intervention and aims of the study, using American Academy of Sleep Medicine scoring criteria (AASM; (Conrad Iber et al., 2007)).

2.3.1 EEG power spectral analysis

All spectral analysis was based on Fast-Fourier Transform (FFT) of C3-recorded EEG in 5-second sequential epochs, accurately aligned to noise onset. The C3 electrode was chosen in this instance as a multi-channel qEEG analysis was considered too impractical and given that C3 was the primary channel used to score sleep. Furthermore, the K-complex detection algorithm also investigated in this study was only developed and validated using C3. Prior to FFT, EEG was band-pass filtered from 0.3 to 35Hz and any 5-second epochs containing large amplitude deflections associated with motion artifacts excluded using customised artefact rejection software based on previous reports (A. L. D'Rozario et al., 2015). This process only removed 1.7 [1.0 to 2.3]% of qEEG analysis epochs from further analysis. Filtered EEG was then subjected to FFT, beginning 15-seconds before to 180-seconds after noise onset, using a custom multi-taper approach based on previously published work (Prerau et al., 2017; Scott et al., 2020). EEG power was then calculated in the delta (0.5-4Hz), theta (4-8Hz), alpha (8-12Hz), sigma (12-15Hz) and beta (15-30Hz) frequency ranges using open source software (Python version 3.7, Python Core Team (2015), Python Foundation) including the MNE-python software package. This multitaper method reduces window-related artifacts associated with single window (taper) analyses (Prerau et al., 2017). Given the study focus on EEG changes in response to noise presentation, and to help account for substantial variability in EEG power between individuals and over time, absolute EEG power (μV^2) values were expressed as a percentage of the average EEG power 15-seconds prior to noise onset (i.e. 100% equates to no change in power from the 15-seconds pre-noise onset). Changes following noise onset were examined in two phases; the acute phase (in 5-second

intervals, from 0-30 seconds after noise onset) to capture transient changes such as brief arousals associated with noise onset, and a sustained phase (in 30-second averages, from 30-180 seconds after noise onset) to test for more persistent EEG changes.

The probability of arousal (shifts to faster EEG frequencies lasting > 3 seconds) was also determined based on the proportion of noise samples of each type, SPL (33, 38, 43 dBA) and in each sleep stage (N2 and N3 sleep), that were associated with at least one EEG defined arousal. Arousal durations and the percentage of individuals who remained or returned to sleep following an arousal (or awakening) within each ongoing three-minute noise sample were also calculated.

2.3.2 Statistical analysis

All statistical analyses were conducted using the Statistical Package for Social Sciences (IBM SPSS, version 25). Percent changes in EEG power from pre-noise onset were non-normal and required log transformation prior to inferential statistical tests. Differences in the transformed percent change in EEG power between noise type (RTN, WTN), SPL (33, 38, 43 dBA), time (within acute and sustained phases) and sleep stage (N2, N3), were examined using linear mixed model analyses. An autoregressive covariance structure was applied to account for correlation over time and subject was included as a random effect, each with their own intercept, to account for expected variability between participants. In the event of a significant interaction or main effect, relevant post-hoc comparisons were conducted using Bonferroni adjusted pairwise contrasts.

Several additional outcomes were non-normally distributed so either Mann-Whitney U tests or General Linear Models were used to examine noise and sleep stage effects on arousal probability, arousal duration and percentage of sleep during noise exposure.

Given several non-normal outcomes, descriptive data are presented as the median

[IQR] or median difference [IQR] unless otherwise indicated. P-values and effect sizes were calculated from transformed normalised data based on two-tailed $\alpha < 0.05$ and effect sizes interpreted according to Cohen's d (< 0.2 small, 0.5 medium, > 0.8 large; (Cohen, 1992) respectively).

2.4 Results

2.4.1 Participant Demographics

Twenty five individuals completed the study. None reported current health problems, but three reported a history of health issues in childhood (tympanostomy tubes, asthma symptoms, recurrent ear infection). During the study, one participant showed <2 hrs of sleep and another took a sleep medication and were excluded from analysis. The characteristics of the remaining 23 participants are presented in Table 2.1. The study sample was comprised of relatively young (age range 18-29 years) healthy individuals with normal noise sensitivity scores and hearing.

Table 2.1 *Participant characteristics including subjective sleep quality, formal hearing assessments and noise sensitivity.*

Demographics	Mean ± SD
Age (years)	21.7±2.1
Body mass index (kg/m²)	20.4±2.9
Insomnia severity index[2]	3.9±2.5
Epworth sleepiness scale[1]	4.0±2.3
Pittsburgh sleep quality index[3]	4.1±2.0
Sleep efficiency (PSQI)	91.5±9.1
Functional outcomes of sleep quality[4]	17.9±2.1
Weinstein noise sensitivity scale[5]	51.0±11.1
Perceived stress	12.9±5.3
Fatigue scale	6.8±5.9
Dysfunction beliefs about sleep	4.0±1.5
Sleep anticipatory anxiety	17.0±6.0
Formal Hearing Assessment	
Right ear hearing 125Hz-8000Hz (dB(HL))[6]	5.2±4.5
Left ear hearing 125Hz-8000Hz(dB(HL))[6]	4.0±4.7
Weekly Sleep Diary	
Habitual sleep onset latency (minutes)	15.0±12.5
Awakenings (N)	0.6±0.6
Habitual wake after sleep onset (minutes)	7.4±6.0
Habitual total sleep time (minutes)	475.2±54.8
Habitual total sleep time (hrs)	7.8±0.9

Note. All data are Mean ± Standard Deviation, N=23.

[1] range of scores: 0-21, normal sleepiness <10 cut off.
[2] range of scores: 0-28, healthy sleeper <8 cut off.
[3] range of scores: 0-24, <10 healthy sleeper cut off.
[4] range of scores: 0-40, lower scores indicate better functional outcomes of sleep.
[5] range of scores: 0 = least sensitive; 105 = most sensitive.
[6] Normal hearing range <20dB(hearing level (HL))

2.4.2 Sleep Parameters

Whole night sleep parameters from standard sleep scoring are presented in Table 2.2.

Despite noise presentations throughout the night, sleep efficiency remained above 85% (a

widely accepted threshold for characterising good sleep (Ohayon et al., 2017)). Participants spent the majority of the night in N2 sleep. There was around 25% of sleep time in N3 available for comparison, but too few noise presentations during rapid eye movement (REM) sleep for meaningful comparisons with REM.

Table 2.2 *Overall sleep study parameters on the study night.*

	Mean [95% CI]
Time available for sleep (min)	520.2 [500.7 to 539.8]
Total Sleep Time (min)	444.7 [415.8 to 473.5]
N1 (%)	9.0 [6.9 to 11.2]
N2 (%)	45.1 [43.2 to 47.1]
N3 (%)	26.8 [23.9 to 29.6]
REM (%)	19.1 [16.7 to 21.4]
Sleep onset latency (min)	26.4 [7.3 to 45.5]
Wake after sleep onset (min)	49.1 [28.5 to 69.7]
Sleep efficiency (%)	85.7 [80.7 to 90.6]

$N = 23$

Table 2.3 shows descriptive statistics for noise presentations and arousal-related outcomes, separately by noise type, SPL, and N2 vs N3 sleep. Despite inevitable variability in sleep and noise presentation distributions, most participants received several replicates of both noise types at each SPL in both N2 and N3 sleep. Each participant received (mean±SD) 3.9±1.8 noise samples of each noise type and SPL in N2 sleep and 3.1±1.4 in N3 sleep. More noise samples were presented in N2 than N3 ($F(1,44.5)=7.30$, $p=0.010$), but the number of noise presentations of each noise type did not differ between sleep stages or SPLs ($F(2,174.54)=2.22$, $p=0.111$).

2.4.3 Arousal responses and percentage of sleep during noise exposure

Arousals occurred relatively infrequently with low probabilities during noise exposures (Table 2.3). There was a significant main effect of sleep stage on arousal probability, with fewer arousals to noise presentations in N3 compared to N2 sleep (general linear model x(1)=28.2, $p < 0.001$, N3 Mean [95% CI], 0.20 [0.16 to 0.24] versus N2

0.36[0.32 to 0.40], *d*=1.62). When arousals did occur, they were typically of short duration, and in the majority of cases sleep resumed before the noise sample ended (Table 2.3). Nonetheless, there was a significant interaction between SPL and sleep stage on arousal duration, $F(2,87.3)=3.5$, $p=0.033$, where the lowest sound pressure level (33 dBA) produced substantially longer arousals during N2 sleep (Mean [95% CI], 21.2 [16.6 to 25.8]) compared to N3 sleep (11.6 [6.8 to 16.3] sec, $p < 0.001$), but with no significant differences at 38 and 43 dBA. However, there was no significant interaction or main effects involving noise type to support any systematic differences between arousal probability or duration between WTN and RTN.

Although percentage of sleep remained high during all noise presentations, there was a significant main effect of noise type, with significantly lower percentage of sleep during RTN compared to WTN (97.9[96.6 to 98.4]% versus 98.3[97.6 to 99.2]%, Mann-Whitney $U=173.0$, $N=23$, $p=0.044$).

Table 2.3 *Number of noise trials, arousal probability, arousal duration, percentage of sleep during noise exposure and the proportion of arousal events associated with resumption of sleep within 3-min exposures to road traffic and wind farm noise at each sound pressure level (33, 38 ,43 dBA) in N2 and N3 sleep. Values are median [IQR] and the number (N) of participants contributing data.*

	Road Traffic Noise		**Wind Turbine Noise**	
	Median[IQR]	*N*	Median[IQR]	*N*
Sound Pressure Level	**33dBA**			
N2				
N Trials	3.0 [2.0 to 4.0]	23	3.5 [3.0 to 5.0]	22
Arousal Probability	0.20 [0.00 to 0.42]	23	0.29 [0.04 to 0.38]	22
Arousal duration (sec)	13.4 [10.4 to 17.6]	13	13.4 [8.3 to 21.4]	16
%Sleep during noise	99.0 [96.5 to 100.0]	23	98.3 [96.2 to 99.9]	22
%Resumed Sleep	100.0 [100.0 to 100.0]	13	100.0 [100.0 to 100.0]	16
Arousal Onset Latency	69.8 [41.1 to 130.4]	13	50.1 [33.2 to 79.5]	16
N3				
N Trials	3.0 [2.0 to 4.0]	23	3.0 [2.0 to 4.0]	22
Arousal Probability	0.25 [0.00 to 0.29]	23	0.17 [0.00 to 0.31]	22
Arousal duration (sec)	9.0 [7.5 to 10.4]	12	11.3 [7.2 to 15.6]	12
%Sleep during noise	99.2 [98.3 to 100.0]	23	99.3 [97.9 to 100.0]	22
%Resumed Sleep	100.00 [100.00 to 100.00]	12	100.0 [100.0 to 100.0]	12
Arousal Onset Latency	112.99 [72.27 to 130.32]	12	104.1 [36.6 to 119.0]	12

	Road Traffic Noise		**Wind Turbine Noise**	
	Median[IQR]	*N*	Median[IQR]	*N*
	38dBA			
N2				
N Trials	4.5 [3.0 to 5.0]	22	3.0 [2.0 to 5.0]	22
Arousal Probability	0.40 [0.27 to 0.50]	22	0.18 [0.00 to 0.50]	22
Arousal duration (sec)	12.3 [8.0 to 18.5]	21	12.1 [8.3 to 13.4]	13
%Sleep during noise	97.1 [95.2 to 98.5]	22	98.7 [96.8 to 100.0]	22
%Resumed Sleep	100.0 [100.0 to 100.0]	21	100.0 [100.0 to 100.0]	13
Arousal Onset Latency	54.9 [14.8 to 64.0]	21	44.6 [28.4 to 56.5]	13
N3				
N Trials	3.0 [2.0 to 3.0]	22	3.0 [2.0 to 4.0]	23
Arousal Probability	0.00 [0.00 to 0.33]	22	0.00 [0.00 to 0.33]	23
Arousal duration (sec)	12.1 [9.9 to 15.5]	8	14.5 [11.1 to 16.4]	9
%Sleep during noise	100.0 [98.0 to 100.0]	22	100.0 [98.0 to 100.0]	23
%Resumed Sleep	100.0 [100.0 to 100.0]	8	100.0 [50.0 to 100.0]	9
Arousal Onset Latency	81.5 [57.4 to 105.8]	8	107.3 [89.7 to 150.9]	9
	43dBA			
N2				
N Trials	3.0 [3.0 to 4.0]	23	4.0 [3.0 to 6.0]	21
Arousal Probability	0.50 [0.33 to 0.67]	23	0.38 [0.25 to 0.50]	21
Arousal duration (sec)	14.7 [11.8 to 16.9]	21	11.9 [9.1 to 14.4]	16
%Sleep during noise	96.0 [94.7 to 97.7]	23	97.0 [96.5 to 98.9]	21
%Resumed Sleep	100.0 [100.0 to 100.0]	21	100.0 [100.0 to 100.0]	16
Arousal Onset Latency	44.9 [29.1 to 62.8]	21	64.8 [36.9 to 80.4]	16
N3				
N Trials	3.0 [2.0 to 4.0]	22	3.0 [2.0 to 4.0]	21
Arousal Probability	0.27 [0.00 to 0.50]	22	0.00 [0.00 to 0.33]	21
Arousal duration (sec)	13.3 [8.8 to 16.8]	12	7.7 [6.0 to 11.1]	7
%Sleep during noise	98.6 [97.3 to 100.0]	22	100.0 [98.5 to 100.0]	21
%Resumed Sleep	100.0 [100.0 to 100.0]	12	100.0 [100.0 to 100.0]	7
Arousal Onset Latency	58.3 [24.0 to 90.5]	12	55.9 [30.3 to 86.3]	7

2.4.4 Power Spectral Changes in EEG in response to noise

Figure 2.1 shows the change in EEG power, relative to EEG power in the 15-seconds immediately prior to noise onset within each frequency band, for each SPL in the acute (0-30 sec) and more sustained (30-180 sec) periods of noise exposure to WTN and RTN. There were similar rapid changes in EEG spectral power immediately following both WTN and RTN onset in delta, theta and alpha frequency bands. At 5-seconds post-noise onset, delta (SPL by time, $F(10,984.92)=2.18$, $p=0.017$) and alpha (SPL by time $F(10,1013.59)=2.54$,

p=0.005) activity peaked followed by a rapid return to pre-noise onset levels by 30 seconds post-noise onset. The peak change in delta power at five-seconds post-noise onset was significantly higher, irrespective of noise type, when the noise was played at 43 dBA (median [IQR], 269.4[164.2 to 335.5] %baseline) compared to 33 dBA (153.7[112.7 to 216.7]%baseline, *p*<0.001, *d*=1.7), and 38 dBA (171.1[144.1 to 227.1] %baseline, *p*=0.021, *d*=1.2). Similarly, the peak change in alpha activity at 5-seconds post-noise onset was significantly higher at 43 dBA (208.6 [146.5 to 246.2]%baseline) compared to 38 dBA (127.6 [105.9 to 176.1] %baseline, *p*<0.001, *d*=1.1), and 33 dBA (119.2 [102.2 to 143.6] %baseline, *p*<0.001, *d*=1.7). These large effects and patterns of response were consistent across both noise types.

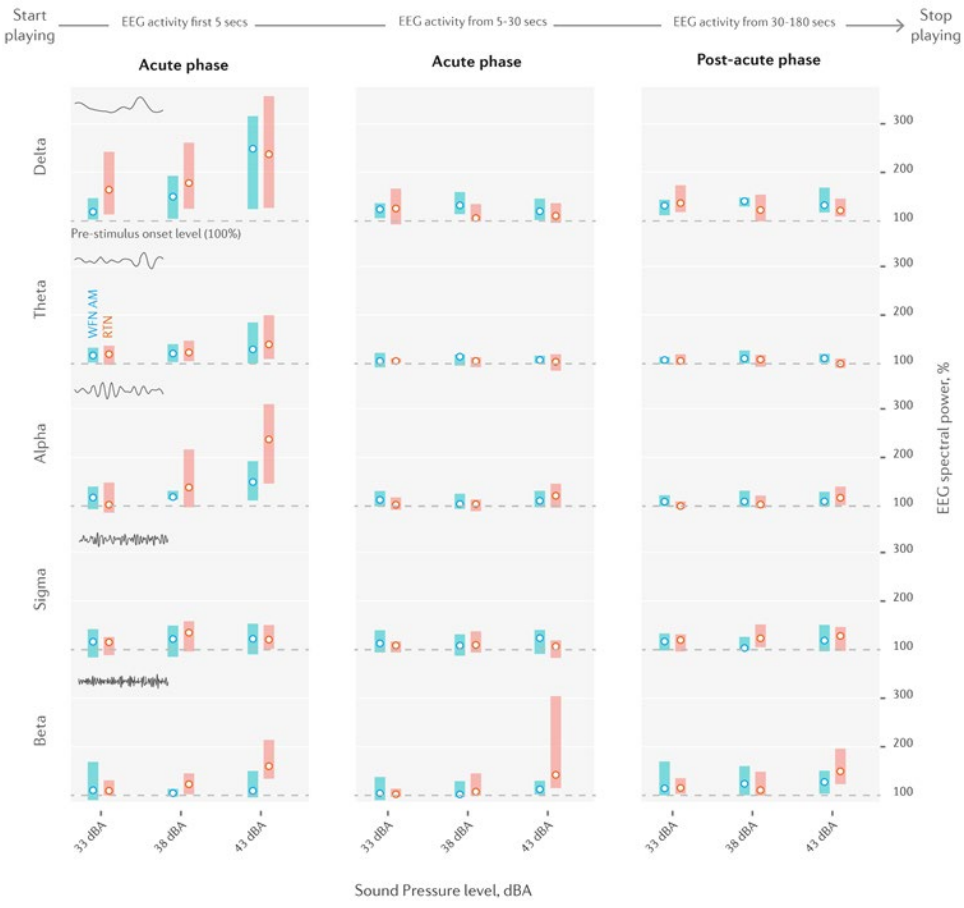

Figure 2.1 EEG spectral power responses to WFN and RTN over time (acute (0-30 seconds from noise onset) and sustained (30-180 seconds from noise onset) at 33,38 and 43 dBA. Values are median and IQR relative to baseline EEG power in 15-seconds prior to noise onset. N=23 participants.

There was a significant interaction between noise type, SPL and time on theta activity in the acute phase ($F(10,1013.38)=1.98$, $p=0.033$). At 10-15 and 25-30 seconds, theta activity was significantly lower after RTN at 38 dBA SPL compared to WTN at the same SPL (10-15 seconds: (RTN-WTN median difference [IQR], -19.3[-27.9 to -5.0] %baseline, $p=0.003$, $d=1.0$; 25-30 seconds: -15.0[-33.1 to 5.6] %baseline, $p=0.038$, $d=0.6$). At 20-25 and 25-30 seconds, theta activity was also lower after RTN at 43 dBA compared to WTN at the same time points and SPLs (difference at 20-25 seconds: -10.6[-42.8 to 2.4] %baseline, $p=0.009$, $d=0.8$; 25-30 seconds: -18.4[-34.4 to 10.6] %baseline, $p=0.015$, $d=0.7$). Beta activity also showed significant noise type by SPL effects in the acute phase ($F(2,514.45) = 13.77$, $p<0.001$). During acute exposure to 43 dBA RTN, beta activity was higher compared to WTN (difference 34.5[18.6 to 180.1] %baseline, $p<0.001$, $d = 1.5$).

In the acute phase, there was a significant main effect of noise type on sigma activity ($F(1,526.38)=7.3$, $p=0.007$), where WTN produced a significantly greater increase in sigma (121.1[108.0 to 125.8] compared to RTN (105.6[97.6 to 127.3] %baseline, $p=0.007$, $d=0.5$). However, there were no interactions involving noise type during the acute phase following noise onset.

In the sustained phase (30-180 seconds), there were several significant noise type by SPL interaction effects on EEG power, including in delta ($F(2,461.46)=7.53$, $p=0.001$), theta ($F(2,472.37)=3.70$, $p=0.026$) and beta activity ($F(2,482.02)=3.36$, $p=0.036$). Compared to WTN, changes in delta activity were lower during RTN at 38 dBA (RTN-WTN difference -8.1 [-37.3 to 6.6] %baseline, $p=0.001$, $d=0.8$) and at 43 dBA (-21.3[-48.8 to 22.9] %baseline, $p=0.020$, $d=0.6$). Theta activity after 30-180 seconds of RTN exposure was significantly lower at 38 dBA (difference -9.9[-15.4 to 8.8], $p=0.001$, $d=0.9$) and 43 dBA (-12.5[-21.4 to 8.6] %baseline, $p<0.001$, $d=1.0$), compared to WTN. Beta activity remained elevated beyond

the acute phase and remained significantly higher during RTN exposure at 43 dBA compared to WTN at the same SPL (difference 15.4[-14.4 to 54.8] %baseline, p=0.003, d=0.8).

2.4.5 Sleep stage effects on EEG power spectral changes with noise

Alpha and sigma activity showed significant three-way interactions between noise type, SPL and sleep stage during the sustained phase (30-180 sec; alpha F(2,506.54)=13.12, p<0.001; sigma F(2,486.16)=7.72, p<0.001, Figure 2.2). During RTN exposure, alpha activity was significantly higher compared to WTN during N2 sleep at 43 dBA (Figure 2.2 A, median difference [IQR], 6.9[-7.9 to 33.4] %baseline, p=0.002, d=0.9) and N3 sleep at 38 dBA (Figure 2.2 B, 3.5[-9.6 to 25.2] %baseline, p=0.031, d=0.5). In contrast, during WTN exposure, alpha activity was significantly higher in N2 sleep at *lower* SPLs compared to RTN (Figure 2.2 A). Alpha activity was higher with WTN at 33 dBA (median [IQR] difference, 11.1 [-4.8 to 25.1] %baseline, p=0.001, d=0.9) and 38 dBA (19.7[-34.8 to 54.3] %baseline, p=0.003, d=0.8) compared to RTN at the same SPLs during N2 sleep (Figure 2.2 A). This was the only statistically significant difference between noise types at 33 dBA. At 38 dBA, sigma activity was significantly higher with RTN compared to WTN SPL during N3 sleep (Figure 2.2 D, 15.7[-13.3 to 48.4] %baseline, p<0.001, d=1.0).

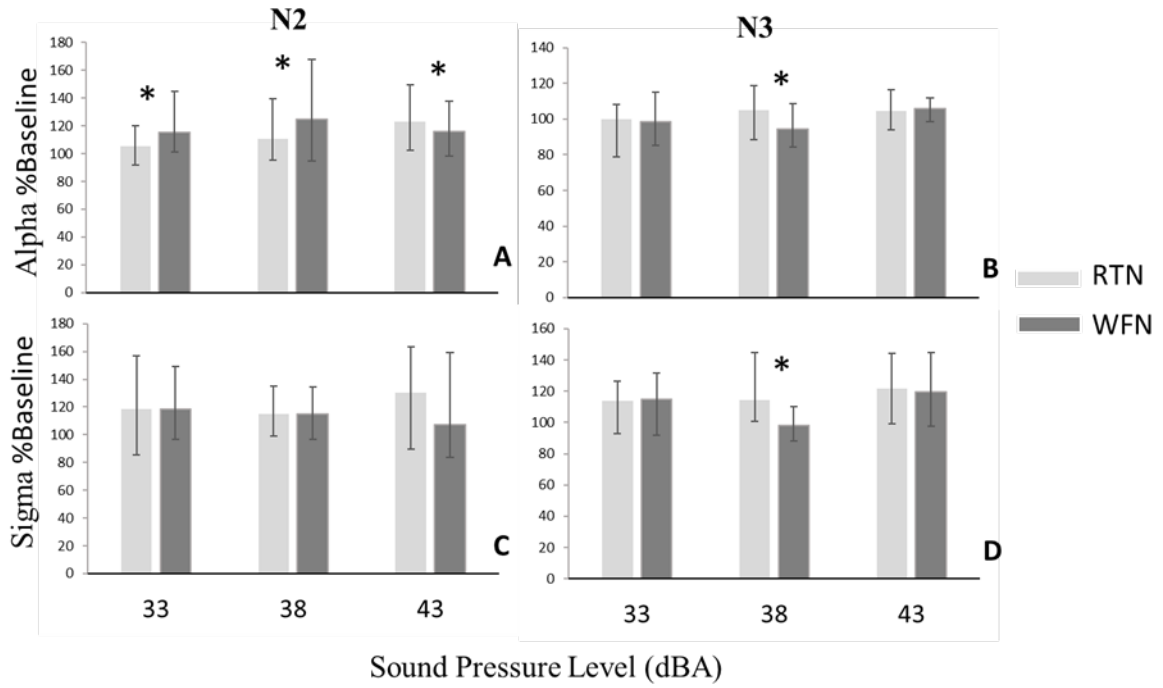

Figure 2.2 EEG alpha (top) and sigma (bottom) activity changes, expressed as a percentage of baseline EEG power in the 15-sec pre-noise onset period, in the 30-180 second (sustained) period following the onset of 3-minute WFN and RTN exposures at 33, 38 and 43 dBA in N2 (left) and N3 (right) sleep. Values are median and IQR. N=23 participants

* Noise type by stage by SPL $p<0.05$

2.5 Sensitivity Analysis

Following removal of 36.1% of noise sample trials containing conventionally scored >3-second arousals, with the potential to dominate EEG frequency changes in response to noise, interactions between noise type and SPL during the acute phase remained significant for delta ($F(2,520.58)=6.08$, $p=0.002$) but were no longer significant for alpha ($F(2,586.33)=2.4$, $p=0.092$) or beta activity ($F(2,568.52)=1.67$, $p=0.189$). Theta activity also no-longer showed a significant interaction between noise type, SPL and time ($F(10,986.0)=1.73$, $p=0.069$).

However, during the acute phase, removal of arousals revealed a significant interaction between noise type, SPL and sleep stage in beta ($F(2,606.54)=3.1$, $p=0.047$) and

theta activity (F(2,542.6)=4.43, p=0.012), where beta activity was higher during RTN compared to WTN at 43 dBA during N3 sleep (median [IQR] difference, 8.3[-2.0 to 17.3] %baseline, p<0.001, d=0.9). During N2 sleep, theta activity was also higher during RTN exposure relative to WTN at 33 dBA (difference 12.2 [-11.9 to 21.3] %baseline, p=0.013, d=0.7), but was lower at 43 dBA (difference -19.6 [-61.7 to 12.9] %baseline, p<0.001, d=1.4). During N3 sleep, theta activity was also lower during RTN exposure relative to WTN at 38 dBA sleep (difference -15.7[-40.5 to 5.9] %baseline, p=0.042, d=0.6).

Following removal of noise samples associated with arousals, significant interactions between noise type and SPL remained during the sustained phase for delta (F(2,463.8)=4.22, p=0.015) and theta activity (F(2,469.0)=13.74, p<0.001), but were no longer significant for alpha (F(2,514.12)=0.25, p=0.779) or beta activity (F(2,553.58)=0.78, p=0.461). Furthermore, new interactions emerged between noise type and sleep stage in alpha (F(1,469.33)=7.87, p=0.005) and beta activity (F(1,452.22)=17.18, p<0.001), with greater increases in alpha during WTN compared to RTN in N2 sleep (median [IQR] difference, 12.2[1.7 to 34.0] %baseline, p<0.001, d=1.0), but greater increases with RTN exposure compared to WTN exposure in N3 sleep (7.8 [-1.8 to 14.8] %baseline, p=0.001, d=0.8).

The significant main effect of noise type on sigma activity in the acute phase also remained following removal of arousals (F(1,556.33)=9.78, p=0.002), and an additional noise type by sleep stage interaction emerged (F(1,464.10)=4.56, p=0.033), with higher sigma activity during WTN exposure compared to RTN exposure during N2 sleep (7.5 [-1.9 to 23.1] %baseline, p<0.001, d=0.8).

2.6 Discussion

This study examined changes in EEG spectral power in response to WTN compared to RTN during sleep whilst controlling for known SPL and sleep stage dependent effects on

sleep EEG and arousal propensity. Given more prominent low frequency components and time-varying features of WTN already established to be associated with greater annoyance compared to RTN during wake, we hypothesised that WTN would be more sleep disruptive compared to RTN during sleep. This hypothesis was partly supported at lower SPLs where WTN consistently produced more substantial power spectral changes compared to RTN. However, at 43 dBA, which substantially exceeds indoor recommended night-time noise limits in most countries (Alamir et al., 2021), RTN consistently produced greater EEG changes across most frequency bands relevant to sleep. However, it should also be noted that these effects were relatively small and mostly transient, with the greatest changes occurring within the first 5-seconds of noise onset, and most EEG changes returning to pre-noise onset levels within the 3-minutes of ongoing noise exposures examined in this study. Compared to WTN, RTN also produced a greater decrement in the percentage of sleep during noise exposures commenced during established sleep, although these percentages remained remarkably high. Nevertheless, both noise types consistently produced SPL dependent increases in arousals and more subtle EEG frequency shifts consistent with some degree of sleep depth-dependent sleep disruption. Both noise types also produced an acute increase in delta activity within the first 5-seconds of noise onset, a response most likely reflecting K-complex activity (Lechat et al., 2021). What impacts low level noise effects on the sleep EEG may have on overall sleep quality and next day and longer-term performance and health outcomes remain unclear and warrants further investigation.

This study was specifically designed to compare acute WTN versus RTN exposure effects on sleep using both spectral and more conventional sleep disruption measures of arousals and percentage of sleep during noise exposure. Spectral power analysis is clearly more sensitive to noise impacts on sleep compared to conventional manual sleep staging and arousal scoring which largely failed to detect a range of noise type, SPL and sleep stage dependent effects of noise detected using power spectral analysis in this study. Previous

studies using manually-scored sleep staging and arousal indices have largely failed to detect systematic sleep disruption effects of WTN (Jalali, Bigelow, et al., 2016). Smith et al. (2020) found increased REM sleep latency and reduced REM sleep in the presence of wind turbine noise coinciding with subjective reports of sleep impairment in a larger laboratory study including a control no noise condition. However no further sleep macrostructure changes were observed. Somewhat discrepant findings may well reflect key methodological differences between studies, including the use of short-noise events in the current study, where transient EEG effects may not necessarily translate into sleep macrostructural changes Smith et al. (2020). Furthermore using odds ratio product (ORP) a novel EEG spectral analysis based marker of wake versus sleep depth, Smith et al. (2019) clearly showed dose-dependent effects of RTN exposure consistent with both spectral EEG changes and increased arousal probability effects of noise exposure in this study. SPL-dependent effects on K-complex response probability to 20 second noise stimuli during sleep further demonstrate the clear sensory disturbance potential for noise of any type to disrupt sleep (Lechat et al., 2021). Thus, sleep EEG responses to RTN and WTN are clearly detectable, but are relatively small at realistic environmental exposure levels, where WTN may be more sleep disruptive at low sound pressure levels but less sleep disruptive than RTN at higher exposure levels. These findings appear likely to help explain previous studies showing no detectable effects of WTN on conventional markers of sleep macro-structure (Jalali, Bigelow, et al., 2016; Michaud, Feder, Keith, Voicescu, Marro, Than, Guay, Denning, Murray, et al., 2016), and recent findings of short-time scale sleep disruption effects of WTN inferred based on actigraphic measures of movement rather than direct EEG measures (Michaud et al., 2021).

At lower SPLs (33 dBA) that are more representative of outdoor-measured levels in the field, WTN significantly increased alpha activity during the post-acute exposure phase relative to RTN during N2 sleep. Although this could reflect a Type I error, a transient increase in alpha is consistent with brief arousal and other significant noise, SPL and stage

dependent effects suggestive of transient EEG disturbances in response to noise. These findings support that WTN may be more sleep disruptive at lower SPLs compared to RTN during lighter sleep. WTN and RTN noise samples presented at 33 dBA during wake are clearly noticeable and elicit similar levels of awareness, annoyance and perceived impacts on daily activities (Hansen et al., 2020), although amplitude modulation of WTN is one characteristic that appears to be more disruptive compared to other noise types at the same SPL (Leventhall, 2006; Micic et al., 2018; Van den Berg, 2005). Amplitude modulation may be more discernible at lower SPLs during N2 sleep compared to higher SPLs and may, in part, explain this finding. However, further studies are needed to establish if this is a replicable and consistent effect.

A key remaining question is to what extent observed changes in EEG power in response to noise exposure impact on the restorative nature of sleep and next-day functioning. Previous studies support that noise disrupted sleep significantly impacts self-reported sleep quality and next day mood (Martin et al., 1997). However, the magnitude of sleep disruption assessed using either EEG power spectral analyses or more conventional sleep quality metrics necessary to negatively impact central nervous system restorative effects of sleep and on daytime functioning is unclear. Although effect sizes of detected EEG changes in this study were moderate to large, absolute differences between noise types were relatively small and of short duration. Future research using sensitive markers of sleep disruption and measures of next day effects clearly remain needed to investigate the levels of noise disruption that measureably impacts daytime functioning and well-being. In addition, future research should also consider the potential for adaptation and sensitisation effects, within and between noise exposure nights and the potential for variables such as noise sensitivity and attitudes to noise to influence individual responses to noise exposure.

2.6.1 Limitations

Several limitations warrant consideration in the interpretation of this study. The specific RTN stimulus, measured close to a busy road, and WTN stimulus containing amplitude modulated noise measured 3.3km from a turbine are only single noise samples. By design discrete noise events are likely to be more representative of wind speed or direction changes compared to more stable wind and noise conditions overnight, both of which can clearly occur during natural WTN exposure. Whilst specifically selected to represent the potentially most disruptive features of RTN and WTN, how representative these specific noise samples are of real-world environmental noise exposure to RTN and WTN remains unclear and different stimuli may well produce different effects. On the other hand, our findings of differential SPL and sleep stage dependent effects of RTN versus WTN support that noise spectral features do influence sensory responses to noise exposure during established sleep. In addition, this study recruited healthy, young, good sleepers with no previous exposure to WTN. Older individuals with more fragmented sleep and reduced N3 would be expected to show increased vulnerability to brief arousals to noise exposure. Future research in more representative residents living close to a wind farm would help to determine if prior exposure effects may influence noise exposure responses during sleep. Furthermore, this study was specifically designed to test for potential SPL-dependent acute effects on sleep itself during established sleep. Thus, further studies are clearly needed to establish if noise type and SPL-dependent differences may also exist across full-night noise exposures, what impacts noise exposures may have during wake, and potential noise effects on next day functioning. Future studies in larger samples should also consider application of a range of signal processing techniques beyond power spectral analysis alone, such as K-complex detection and ORP methods to help evaluate potential relationships with next day functional impacts of noise-induced sleep disruption.

2.7 Conclusion

This study demonstrates subtle but significant differential SPL and sleep stage dependent effects of WTN compared to RTN on sleep EEG power spectral responses to three minute noise exposures. These effects were mostly transient and relatively small, including brief increases in delta and faster EEG activity most notably in alpha during N2 sleep at 33 dBA not evident in manually scored arousals. Whether such effects are sufficient to impact overall sleep quality and next-day functioning is unclear and warrants further studies applying both EEG power spectral analysis and conventional sleep staging and arousal scoring in response to realistic environmental noise exposure during sleep on next day outcomes. Further research in larger samples more representative of populations residing in areas of WTN and RTN exposure is also needed to further elucidate environmental noise impacts on sleep.

2.8 Acknowledgements

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2.9 Disclosure Statement

2.9.1 Financial Disclosure:

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2.9.2 Nonfinancial Disclosure:

This was an investigator led study with no industry support

CHAPTER 3 : THE EFFECT OF WIND FARM INFRASOUND ON SLEEP ELECTROENCEPHALOGRAPHIC POWER SPECTRA IN HEALTHY GOOD SLEEPERS – A PILOT STUDY

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Conflict of interest: None

A B S T R A C T

Study Objectives: To compare the impact of the presence versus absence of infrasound on sleep EEG power spectra during overnight polysomnography in a controlled sleep laboratory environment.

Methods: Twenty-three healthy volunteers were exposed throughout the night to noise samples including wind farm noise infrasound at 80 dB(G), below the generally accepted hearing threshold for noise <20 Hz, and control background noise (23 dB(A)) after at least 5 minutes of established N2 sleep. A multi-taper Fast Fourier Transform approach was applied to quantify electroencephalogram (EEG) spectral power in the delta to beta frequencies (0.5-30Hz) in 5-second epochs from 15 seconds before and throughout the remainder of each noise sample. K-complex responses to infrasound during sleep, and each participant's ability to discriminate infrasound from background noise during wake were also investigated. Infrasound versus quiet control exposure, time and sleep stage effects were examined using linear mixed effects model analysis.

Results: There were significant increases in delta and theta power during the first 5 seconds of infrasound relative to control background noise exposure, which coincided with a significantly higher probability of K-complex occurrence in N2 sleep in the first five seconds of infrasound exposure (mean [95% CI] 37 [47 to 28] vs 19 [24 to 14]%, $p<0.001$). Immediately following these initial transient responses, delta and theta power were significantly reduced relative to control 10-15 seconds following noise onset, which also coincided with a significantly lower probability of K-complex occurrence (10 [15 to 5] vs 25 [31 to 18]%, $p=0.011$). During wake, all participants could detect infrasound at 110 dB(G), but only 9 of 17 (53%) could detect infrasound at 100 dB(G). Responses to 80 dB(G) infrasound during sleep were no different between participants who could versus could not detect 100 dB(G) infrasound during wake.

Summary: These findings support that abrupt infrasound onset at sound pressure levels 20 dB(G) below levels audible to around 50% of study participants consistently elicit sensory responses during sleep. However, these effects were small and short-lived and were no-longer evident beyond the initial 20 seconds of noise exposure. Further work to confirm these findings, rule out potential for noise reproduction artefact effects, and test for transient effects at lower and more realistic real-world exposure levels of around 70 dB(G) remains warranted.

3.1 Introduction

Environmental noise, such as traffic noise, can disrupt the micro- and macro-structure of sleep (Micic et al., 2018). Sleep disruption is well established for mid to high frequency noise (500-10,000Hz) such as aircraft, road traffic and rail noise (Evandt et al., 2017; Griefahn et al., 2008). However, the effects on sleep of wind farm noise, which has particularly prominent low frequency components including infrasound, is still under investigation, with currently available literature unable to clarify if there are wind farm specific sleep impacts (Ageborg Morsing et al., 2018; Carlile et al., 2018; Jalali, Nezhad-Ahmadi, et al., 2016; Smith et al., 2020). Berglund et al. (1999) noted the need for research addressing *"Knowledge on the health effects of low-frequency components in noise and vibration"* in the published Guidelines for Community Noise. More recently Micic et al. (2018) again described the need for research on low frequency noise based on the report from the Australian Senate inquiry (2015) (*Senate Report: Select Committee on Wind Turbines.*, 2015). Infrasound, which is typically defined as sound below 20 Hz (Leventhall, 2004), has been proposed as a potential source of annoyance, and negative sleep and health impacts from wind farms where noise emissions are dominated by low frequency components including infrasound. However, supporting evidence remains lacking (Bolin et al., 2011; Tonin, 2018; Tonin et al., 2016). Although the term infrasound implies sub-audible noise, noise with sufficiently high sound pressure level below 20 Hz can still be audible to individuals with sufficiently high hearing acuity at low frequencies. Examination of hearing thresholds suggest that 20 Hz tones become audible to approximately half the general population at a sound pressure level of 85 dBG (Leventhall et al., 2003; Møller & Pedersen, 2004). However, there is considerable inter-individual variability in low frequency hearing acuity and noise sensitivity (Møller & Pedersen, 2004) so infrasound at 85 dBG may be inaudible to some and audible and annoying to others.

Typical wind farm noise infrasound levels measured indoors at nearby residences fall

below the conventionally recognised hearing threshold below 20 Hz of 85 dBG, although some infrasound frequencies may exceed the hearing threshold of at least some individuals (Zajamšek et al., 2016). Individual noise sensitivity has also been shown to affect infrasound detection, where some noise sensitive individuals can detect infrasound above chance in listening tests (Nguyen et al., 2019). Thus, both low frequency hearing acuity and noise sensitivity may importantly influence responses to infrasound exposure during sleep.

One of the potentially most sensitive approaches to examine the impact of infrasound on sleep is through power spectral analysis of brain electroencephalographic (EEG) responses following noise onset. However, power spectral analysis of EEG responses to infrasound exposure have shown mixed findings and are particularly limited in sleep. Kasprzak (2014b) found reduced wake EEG alpha (8-10 Hz) activity during 20 minutes of exposure to 7Hz, 120 dB and 4-8Hz 110 dB(HP) infrasound produced by 6 amplifiers installed in the ceiling of a Hungarian-type pressure chamber (Kasprzak, 2014b). However, alpha reductions were not replicated during exposure to wind turbine infrasound at 91.6 dB (linear unweighted) after filtering to remove >20Hz noise (Kasprzak, 2014a). Inagaki et al. (2015) also tested the effect of aerodynamic noise across a range of high, low and infrasonic frequencies on EEG and found that exposure to infrasound reduced wake EEG alpha power and increased EEG beta (13-20Hz) power relative to higher frequency (30Hz - 300Hz (at 55dBA [92dBG])) noise exposure. Alpha power was decreased with all noise stimuli, but the greatest reduction occurred with infrasound, consistent with an alerting effect on brain activity (Inagaki et al., 2015). To the authors knowledge only two previous studies appear to have specifically tested infrasound compared to higher frequency noise effects on sleep. Using sleep stage changes and body movements to define a reaction to noise exposures during sleep in 18 healthy volunteers Okada and Inaba (1990) found no detectable responses to 10 Hz infrasound presented up to 105 dB, but more frequent reactions to 105 dB 20 Hz noise and with around 90 dB 40 and 63 Hz noise. In a double blind triple arm cross over trial Marshall et al. (2023)

found that whole night analysis of objective measures of sleep and spectral power in delta, theta alpha, sigma and beta (0.5-32Hz) and traditional sleep metrics including wake after sleep onset were not affected when exposed to wind turbine simulated infrasound at approximately a 90 dB infrasound peak (1.6-20Hz, measurable but described as inaudible) compared to sham control noise. The study separated spectral power analysis to examine REM and NREM sleep stage effects and found no significant difference in average spectral power or sleep traditional sleep metrics. However, potentially more sensitive power spectral assessments of EEG responses time locked to noise onset were not evaluated in either study.

Subjective reports of sleep disruption and negative health impacts that some residents specifically attribute to wind farm infrasound clearly warrants investigation (Botelho et al., 2017; Carlile et al., 2018; Micic et al., 2018; Pedersen, 2011). Furthermore, the potential for misattribution of negative symptoms to infrasound and nocebo effects should be considered (Rubin et al., 2014). The nocebo effect, known as the expectation of negative symptoms resulting in the occurrence of impairment may assist in explaining subjective reports of symptoms attributed to infrasound. This is potentially the result of conflicting information sources being provided to individuals in the community which contribute to a nocebo effect and increase the likelihood of misattribution of new but unrelated symptoms to infrasound, audible wind farm noise or potentially visual cues associated with wind (Rubin et al., 2014). Thus, the purpose of this study was to apply quantitative power spectral analyses (qEEG) to previously recorded EEG responses to a range of noises presented during established sleep (Dunbar et al., 2021), that included infrasound and no noise controls, to specifically test for WFN infrasound effects on sleep.

3.2 METHOD

3.2.1 Study participants

Further details of the study from which these data were derived have been previously

reported in Chapter 2 (Dunbar et al., 2021). Briefly, following ethics approval from the Flinders University Social and Behavioural Research Ethics Committee, the study sought healthy good sleepers via university flyers, word of mouth, and online social media (Facebook). An online survey was used to screen for eligibility, after which more detailed information provided to participants via email and face-to-face. Those who remained eligible and agreed to participant provided written informed consent and were reimbursed AU$300 for study completion.

Eligibility required a body mass index (BMI) <30 kg/m², and no smoking; trans-meridian travel in the last two months; pregnancy or lactating; comorbidities that affect sleep. Normal hearing was also required, confirmed by a comprehensive hearing assessment by a qualified audiologist undertaken at the Flinders Medical Centre (<20dB(HL) at 125 - 8000Hz (Schaette & McAlpine, 2011). Participants were considered healthy sleepers if they scored <6 on the Pittsburgh Sleep Quality Index (PSQI) (Buysse et al., 1989), reported an average of >85% sleep efficiency (i.e., portion of total sleep time as a factor of total time in bed), scored <8 on the Insomnia Severity Index (ISI) (Morin et al., 2011), <10 on the Epworth Sleepiness Scale (ESS) (Johns, 1992), and had minimal circadian misalignment (<2 hr discrepancy in self-reported weekday vs. weekend sleep onset and offset times).

3.2.2 Participant Bedrooms

Participants were studied in a sound attenuated bedroom in the Flinders University College of Education, Psychology and Social Work Sleep Laboratory. The room temperature was set to 23 degrees Celsius and on low fan. The background noise level at night was 23dB(A). Two hours prior to lights out and two hours post awakening, light exposure was <10 lux.

3.2.3 Polysomnography

Polysomnography (PSG) signals included gold-cup electroencephalography (EEG at F3, Fz, F4, C3, C4, O1 and O2 referenced to linked M1 and M2), electrooculography (EOG),

electrocardiography (ECG), chin electromyography (EMG), leg movement, nasal cannula, oro-nasal thermistor, chest and abdominal motion and finger oximetry. Data were recorded using Grael 4K hardware and Profusion 4 EEG software (Compumedics Ltd., Abbotsford, Vic). All sleep studies were scored by an independent qualified sleep technician, who remained blinded to the conditions and aims of the study, using American Academy of Sleep Medicine (AASM, (C. Iber et al., 2007)).

3.2.4 Noise reproduction system and infrasound stimulus

The noise reproduction system consisted of an RME BabyFace Pro sound card, modified (without vent) Krix KX4010S commercial cinema subwoofer (dimensions 118 x 670 x 410 cm) with 10-inch driver and Crown DC-300 power amplifier with a flat frequency response down to 0 Hz. The speaker was placed along the bedroom wall approximately one metre from the foot of the participants' bed.

To test for wind farm noise infrasound effects on sleep, participants were exposed to three-minute blocks of wind farm noise infrasound at 80 dB(G) or quiet control background noise at 23dB(A). The noise spectrum of the infrasound stimulus and full spectrum from which it was derived are shown in Figure 3.1. The infrasound noise sample was derived from pre-recorded wind farm noise by applying a low-pass filter at cut-off frequency of 15 Hz, resulting in no other wind farm noise characteristics above this frequency being audible to participants with normal hearing threshold according to ISO 226:2003. The stimulus contained a 0.8Hz blade-tower infrasound pulse replayed at a relatively high sound pressure level of 80dB(G) (Zajamšek et al., 2016; Zajamsek, Yauwenas, et al., 2019)below the generally accepted audibility threshold of 85 dB(G) for infrasound (Møller & Pedersen, 2004). Prior to each experiment, noise reproduction equipment was calibrated using a SVANTEK 958 sound level meter which is a digital, four channel 0.5 Hz to 20 kHz signal analyser, type 1 sound level meter (meeting IEC 61672-1:2002) and vibration meter (meeting ISO 8041:2005)

with capacity to calibrate down to 1Hz frequency range.

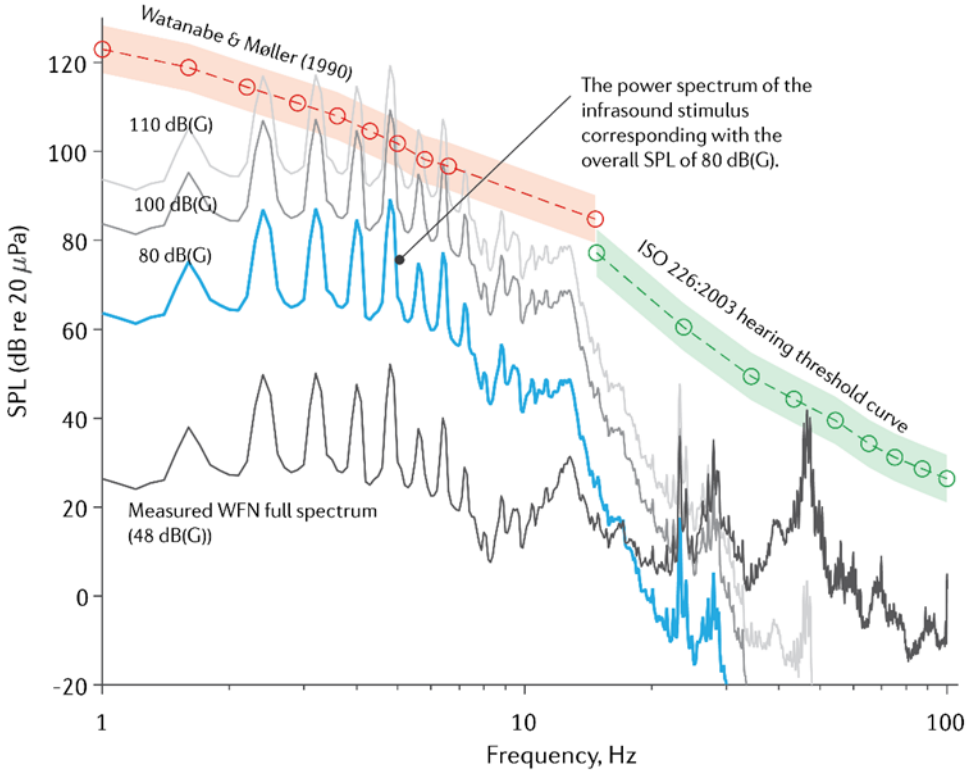

Figure 3.1. The wind farm noise sample full spectrum and filtered spectra with infrasound components only used in this study. The filtered signal was amplified to the overall SPL of 80 dB(G) to create the stimulus used for the sleep study while the stimuli corresponding with 100 and 110 dB(G) were used in the daytime infrasound detection tests. The background noise of the sleep laboratory at night was 23dB(A). The stimulus used during sleep is well below the infrasound hearing threshold curve (shading indicates ±1 SD) reported by Watanabe and Møller (1990) with no wind farm noise characteristics above 20 Hz within the audible range according to the ISO 226:2003 hearing threshold curve (shading indicates ±1 SD).

3.2.5 Experimental Procedure

Participants attended the sleep laboratory two hours prior to their estimated habitual bedtime, determined from sleep diaries from the prior week, for PSG setup then relaxed in their bedroom undertaking a quiet activity (e.g., reading) until their set lights out time. Overnight technicians used a custom MATLAB (Version 2018a/b 9.4/9.5, Mathworks, Natick, Massachusetts) interface to commence noise exposure once participants had established at least 5 minutes (10 epochs of 30 seconds) of N2 sleep or deeper sleep. The interface system maintained technician blinding of noise types and sound pressure levels, and controlled noise

sample randomisation and replay, and the output of a noise sample onset timing signal for millisecond accurate time synchronisation against sleep recordings. Quiet control noise and infrasound samples were then played throughout the night in randomised sequences, each consisting of all noise samples at each SPL, including WFN and RTN reported elsewhere (Dunbar et al., 2021). Each 3-minute noise sample was separated by a 20 second quiet (23dB(A)) period. At observed awakenings (>15 second shift to faster frequency EEG), overnight technicians were instructed to cease noise presentations at the end of the currently playing sample. These procedures were designed to ensure that noise samples continued through brief arousals or shifts to wake until the end of each sample for full evaluation of EEG power spectral changes to noise, whilst also facilitating the re-initiation of sleep without ongoing noise disturbances after awakenings.

3.2.6 Infrasound detection tests

Infrasound detection was tested during the day in a separate laboratory inside a Faraday cage to minimise electrical noise interference of EEG recordings to assess for potential auditory brainstem responses for a separate study. The infrasound detection test was conducted separately to the overnight sleep study and took approximately four hours to complete. As a result, only 17 of the 23 participants agreed to and were able to fully complete the daytime detection tests with 6 participants choosing to withdraw from this additional test. Each participant was presented with two sequences of 20 second periods of infrasound and 20 equivalent periods of silence in a counter balanced and randomised order via inner ear insert headphones (E-A-Tone, Cabot Safety Corporation/Auditory Systems Division, Indianapolis, USA). The stimulus was generated using a custom-made loudspeaker housed inside a box to constrain the speaker cone and direct volume displacement via a 4.3 meter long and 2-millimetre inner diameter silicone tube to deliver sound to the participants ears. Care was taken to pre-calibrate this system to ensure the SPL of the reproduced noise was within safe

limits. Participant instructions were as follows: *"We would like to know if you can hear, detect or perceive any obvious changes in the audio character that indicates to you that infrasound is being played. For example, you may perceive it as a change in pitch or intensity or feel another physiological sensation"*. A red cross was displayed on a black screen when the stimulus, infrasound, or silence, was presented. One sequence contained infrasound presented at 110 dB(G) and the other at 100 dB(G). During both sequences, participants were asked to respond via a handheld button to indicate if they could detect the infrasound stimulus.

3.3 Data Analysis

Power spectral analysis of electroencephalography (EEG) recordings was performed on C3 channel using methodology described previously (Dunbar et al., 2021; Lechat, Hansen, et al., 2022). Briefly, EEG from all noise samples that commenced from N2, N3 or REM sleep were filtered to remove frequencies above 35 Hz then analysed in consecutive 5 seconds non-overlapping epochs, from 15-seconds prior to stimulus onset through to stimulus offset regardless of any stage changes, although epochs with EEG amplitude changes >400 μV were excluded to avoid movement artefacts (Sweetman et al., 2021). Absolute power in the delta (0.5-4Hz), theta (4-8Hz), alpha (8-12Hz), sigma (12-15Hz) and beta (15-30Hz) frequency ranges were then calculated using a multi-taper based fast fourier transform. Absolute power within each frequency band was presented as a percentage of each individuals' average baseline (15 seconds prior to stimulus onset) to show changes relative to the pre-stimulus baseline and log transformed for normalisation during analysis. The ratio of absolute power within each frequency band relative to power in all other bands was also evaluated in a similar manner. Similar to the prior analysis of wind farm compared to traffic noise responses (Dunbar et al., 2021), qEEG responses were examined in 5-sec intervals over the first 30 seconds after stimulus onset and separately in the subsequent 30-180 seconds of

noise exposure to test for acute and more sustained effects of infrasound versus quiet control exposures.

3.3.1 K-complex responses

A previously reported K-complex detection algorithm (Lechat et al., 2020), with a probability threshold of 50%, was used to identify the presence or absence of K-complexes (KCs) during each 5 second time window before and after each noise sample onset. Any 5-second epoch containing at least fifty percent of a KC was classified as containing a K-complex, otherwise epochs were classified as without a KC.

3.4 Statistical analysis

Statistical analysis of spectral power outcomes was conducted in the IBM statistical package for social sciences (SPSS©, version 25). Differences in absolute power and ratios over time and between quiet control and infrasound noise exposures were examined using linear mixed model analyses. Fixed factors were specified as noise type (infrasound, silence), sleep stage (N2, N3, REM) and time (5 second windows for acute analysis and 30 second windows for sustained analysis). Subjects were specified as a random effect, each with their own intercept. For the daytime infrasound detection test, receiver operating characteristic (ROC) curve analysis was used to establish if each individual could discriminate infrasound from silence above chance, based on a ROC area under curve (AUC) statistically significantly greater than 0.5.

Changes in K-complex probabilities over time from 15 seconds before to 35 seconds after noise onset were examined using logistic mixed effects regression models in the lme4, glmer and car open-source packages from the computing environment R, with noise type (infrasound versus control), and time (in 5 second epochs from -15 to 35 seconds) as fixed factors with subject as a random factor, each with a separate intercept. Effects of infrasound detection acuity, from the daytime listening test, and self-reported noise sensitivity on K-complex responses to infrasound presentations during N2 sleep were also examined. All data are reported as Mean±SD unless

otherwise specified. $p<0.05$ was considered statistically significant.

3.5 Results

The characteristics of the twenty-three young healthy normal good sleepers with normal hearing who participated in the study and were included in the analysis are summarised in Table 3.1.

Table 3.1 *Baseline participant characteristics*

Demographics	
N (%) males:females	10:13 (43:57)
Age (years)	21.7 ± 2.1
Body Mass Index (kilograms/metres²)	20.4 ± 2.9
Weinstein Noise Sensitivity Scale[1]	51.0 ± 11.1
Formal Hearing Assessment	
Right Ear Hearing 125Hz-8000Hz (dB(HL))[2]	5.2 ± 4.5
Left Ear Hearing 125Hz-8000Hz(dB(HL))[2]	4.0 ± 4.7
Weekly Sleep Diary[3]	
Habitual Sleep Onset Latency (minutes)	15.0 ± 12.5
Awakenings (N)	0.6 ± 0.6
Habitual Wake After Sleep Onset (minutes)	7.4 ± 6.0
Habitual Total Sleep Time (minutes)	475.2 ± 54.8

Note. All data are Mean ± Standard Deviation, N=23.
BMI=Body Mass Index, Epworth Sleepiness Scale, N=number, min=minute.
[1] range of scores: 0 = least sensitive; 105 = most sensitive (Weinstein, 1978).
[2] Normal hearing range <20dB(hearing level (HL).
[3] Sleep Diary (Carney et al., 2012).

3.5.1 Sleep parameters and noise stimuli

Electrode impedances were 3.3±0.9 kOhms when attached. Overall sleep characteristics and the number of noise stimuli available for analysis are presented in Table 3.2. Further details regarding sleep in this study sample are presented previously Dunbar et al. (2021). Overall participants slept relatively well despite noise presentations throughout the night and achieved around 7.5 hours of sleep with around 85% sleep efficiency. There were a similar number of infrasound versus no noise stimulus presentations overnight, but with fewer in REM compared to N2 and N3 sleep, consistent with reduced REM compared to N2 and N3

sleep.

Table 3.2 *Sleep study parameters and noise stimuli presentation counts*

Overall sleep study parameters on the study night.	**Mean ± Standard Deviation**
Time available for sleep (min)	520.2 ± 45.2
Total Sleep Time (min)	444.7 ± 66.7
N1 (%)	9.0 ± 5.0
N2 (%)	45.1 ± 4.6
N3 (%)	26.8 ± 6.7
REM (%)	19.1 ± 5.5
Sleep onset latency (min)	26.4 ± 44.2
Wake after sleep onset (min)	49.1 ± 47.7
Sleep efficiency (%)	85.7 ± 11.5
Arousal index (/h)	9.8 ± 2.5

Average noise stimulus presentations per night		
	Infrasound (Median [IQR])	Quiet Control (Median [IQR])
N2	11.0 [7.0 to 14.0]	11.0 [8.5-13.5]
N3	10.0 [8.0 to 11.0]	8.0 [6.5 to 11.0]
REM	3.0 [1.0 to 5.5]	5.0 [0.5 to 6]

Note: N = 23

3.5.2 Power spectral changes in EEG in response to infrasound exposure

Figure 3.2 shows the change in EEG power relative to 15 seconds immediately prior to noise onset within each frequency band in the acute (0-30 seconds) and more sustained (30-180 seconds) exposure to infrasound and quiet control noise. There were no significant noise type by time by stage interaction effects on qEEG outcomes in either the acute (0-30sec) or sustained (30-180sec) analysis. There was a rapid change in qEEG activity immediately following infrasound onset in delta (noise type-by-time interaction ($F(5,152.04)=7.346$, $p<0.001$) and theta (noise type-by-time $F(2,259.568)=2.859$, $p=0.019$) frequency bands, which peaked in the first 5 seconds. Compared to control, the median [IQR] acute increase in delta (infrasound-control) was 93.6 [42.4 to 147.1]% of baseline ($p<0.001$) and in theta was 16.1 [-0.6 to 35.9]% of baseline ($p=0.007$), after which delta and theta activity were transiently reduced at 10-15 seconds (delta -28.8[-47.4 to 13.6]% baseline,

p<0.001), theta -10.0[-28.1 to -0.5]% baseline, p=0.021). Although the difference compared to control was small, delta activity remained reduced throughout the 30-180 second infrasound exposure period (noise type F(1,370.154)=4.579, p=0.033, -4.5[-22.6 to 26.2]% baseline). Beta activity was also higher than control in the first 30 sec of infrasound onset ((noise type effect F(1,253.693)=13.79, p<0.001, 23.4[0.3 to 53.0]% baseline), but this effect was not sustained beyond 30 seconds.

Although there was no significant noise type by time by stage interaction effects on the primary qEEG responses there were some significant stage dependent effects of infrasound onset. Delta power was lower during the first averaged 30 seconds of infrasound exposure and the more sustained 30-180 seconds of noise exposure relative to control during N2 sleep (noise type by stage effect, (Acute) F(2,326.77)=6.35, p=0.002, Median difference [IQR]%baseline, -3.3[-27.1 to 14.1], p=0.015), (Sustained) F(2, 255.37)=25.90, p<0.001), -10.2[-26.8 to 20.6]% baseline, p=0.008). During N3 sleep delta power was increased during the first 30 seconds of infrasound exposure relative to control (17.4[-6.3 to 35.5]% baseline, p=0.012), but this was not sustained over time. During N2 sleep sigma power initially reduced during the first 30 seconds of infrasound exposure (noise type by stage, F(2,219.60)=3.57, p=0.030), Median difference[IQR]%baseline, -10.2[-22.6 to 6.7]%baseline, p=0.008) and then increased relative to control from 30-180 seconds (noise type by stage, F(2, 173.43)=3.88, p=0.022, 4.1[-9.2 to 23.7]% baseline, p=0.003). During N3 sleep sigma power initially reduced during the first 30 seconds of infrasound exposure (-18.8[-40.9 to 18.5]% baseline, p<0.001) before returning to comparable baseline EEG levels from 30-180 seconds of exposure.

There were no further noise type-by-time or noise type effects over the first 30 seconds or subsequent 30-180 seconds following noise onset in any other frequency bands.

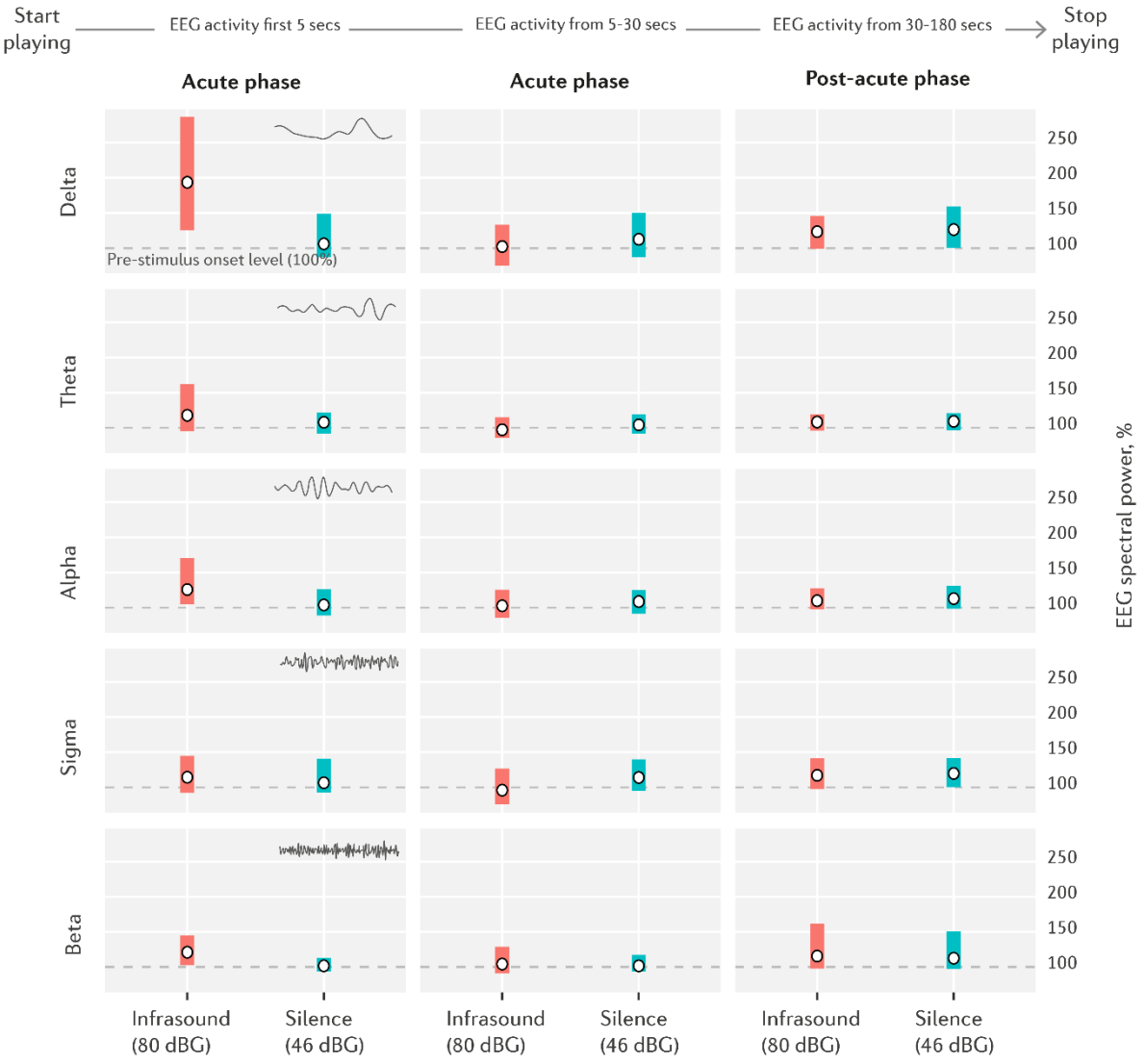

Figure 3.2 EEG spectral power changes during 180 second exposure to infrasound (80dB(G)) and control background noise (46 dB(G) (for comparison) which converts to 23dB(A)) in stage 2, 3 and REM relative to power exhibited 15 seconds prior to noise onset. Values are median and IQR relative to baseline EEG power in 15-seconds prior to noise onset. N=23 participants

3.5.3 K-complex and arousal responses

K-complex and arousal probabilities and the odds of responses relative to the pre-stimulus onset baseline are shown in Figure 3.3. K-complex probability (χ^2=42.42, df=9, p<0.001, Figure 3.3 A) and the corresponding odds of a KC response (Figure 3.3 B) were increased in the first five seconds of infrasound onset, followed by a reduction 10-15 seconds post infrasound exposure (p=0.011). However, arousal response probabilities were low and remained unchanged following infrasound onset compared to control (Figure 3.3 C-D).

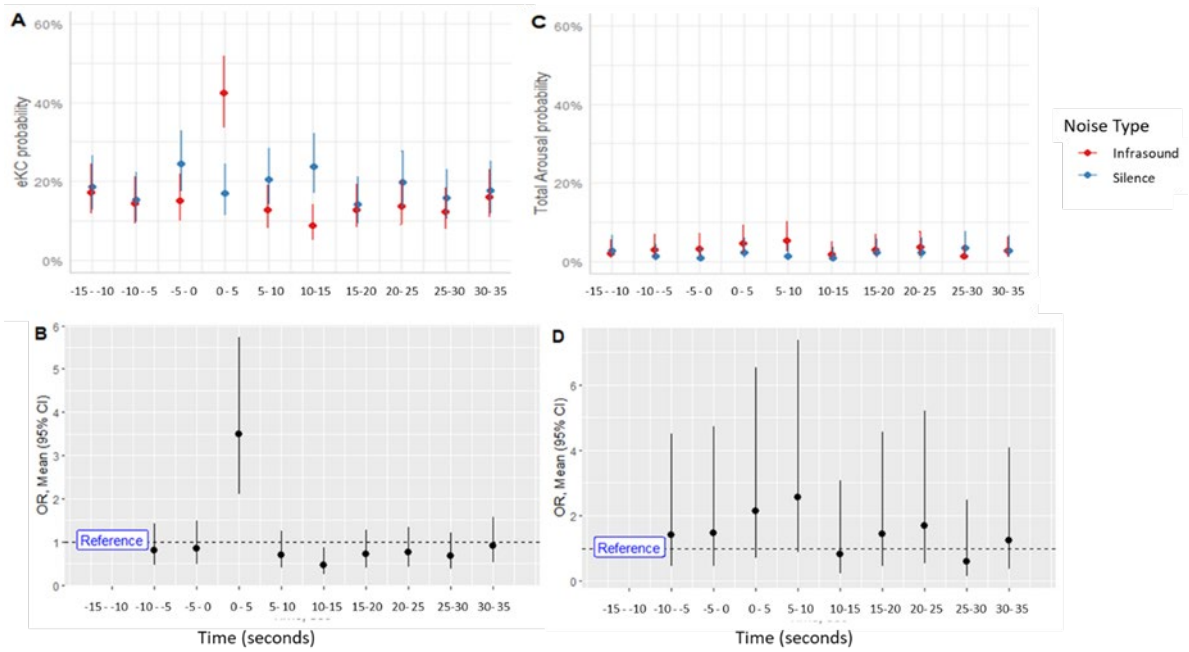

Figure 3.3 Probability and 95% CI of K-complex (A) and arousal (<15seconds) (C) occurrence at a given time (sec). Odds ratio (95% CI) of evoking a K-complex (B) and arousal (D) at a given time window, relative to pre-noise onset.

3.5.4 Infrasound detection acuity and noise sensitivity

Results of the daytime infrasound detection test completed by 17 of the 23 study participants are summarised in Table 3.3. All 17 could correctly discriminate 110 dBG infrasound from silence above chance, and with high sensitivity and specificity. Nine of the 17 could also correctly discriminate 100 dBG infrasound from background noise above chance. During N2 sleep, there was a significant group (able versus unable to detect 100 dBG infrasound) by time interaction effect on KC response probability following infrasound onset (χ^2=18.80, df=9, p=0.027). Although both groups showed an acute increase in KC response probability following infrasound onset, individuals able to detect 100 dBG infrasound during the day showed a reduced K-complex probability 10-15 seconds post stimulus onset (Figure 3.4). However, there was no significant interaction effect of self-reported noise sensitivity on KC responses to infrasound during N2 sleep (χ^2=7.46, df=9, *p*=0.589, Figure 3.5).

Table 3.3 *Daytime infrasound detection test*

	110 dBG Infrasound		100 dBG Infrasound	
	Detectors	Non-detectors	Detectors	Non-detectors
N	17 (100%)	0 (0%)	9 (53%)	8 (47%)
AUC	0.99 [0.98 to 1.00]	-	0.93 [0.87 to 0.99]	0.52 [0.49 to 0.55]
Sensitivity	1.00 [0.99 to 1.00]	-	0.88 [0.78 to 0.98]	0.11 [0.01 to 0.20]
Specificity	0.99 [0.97 to 1.00]	-	0.98 [0.94 to 1.00]	0.93 [0.87 to 1.00]

Note. Values are N (%) and mean [95% CI] from 17 participants who completed both the daytime infrasound detection test and overnight sleep study. AUC indicates the area under the receiver operator characteristic (ROC) curve constructed for each participant. Detectors were defined as those individuals who could correctly discriminate infrasound from silence statistically significantly above chance (AUC significantly greater than 0.5).

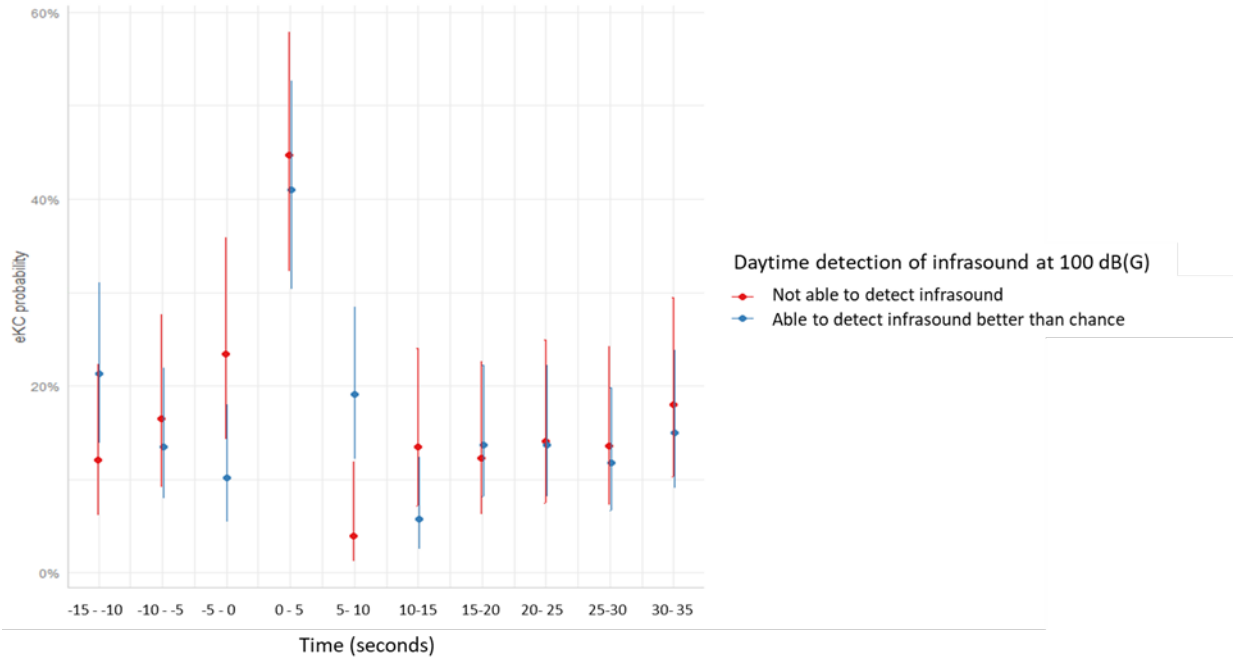

Figure 3.4 Probability and 95% CI of K-complex occurrence at a given time (sec) following infrasound onset in N2 sleep in individuals able versus not able to detect infrasound above chance during a daytime listening tests.

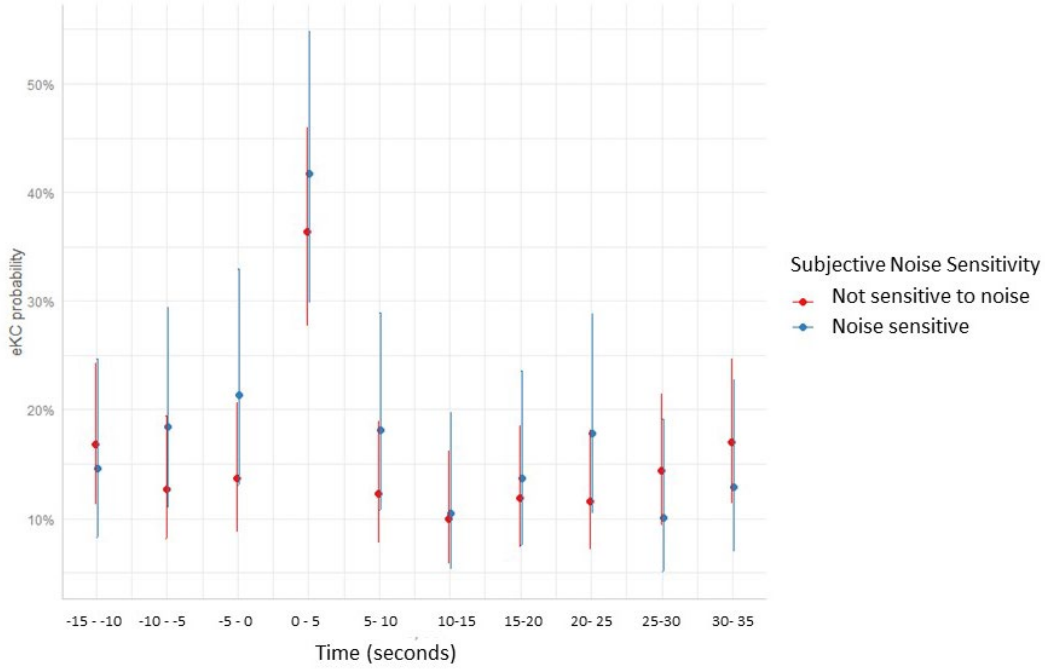

Figure 3.5 Probability and 95% CI of K-complex occurrence at a given time (sec) following infrasound onset in N2 sleep in individuals with low versus high self-reported noise sensitivity.

3.6 Discussion

This study sought to determine if relatively high (80 dB(G)) but sub-audible levels of wind farm noise infrasound presented during sleep elicit any measurable EEG changes in quantitative EEG and K-complex responses. The major infrasound components arising from the 0.8Hz blade pass frequency were faithfully reproduced in a controlled laboratory environment following low-pass filtering to remove non-infrasonic wind farm noise components above 20 Hz. Based on wake EEG findings of reduced delta EEG frequencies to pure-tone infrasound exposure (Inagaki et al., 2015), we hypothesised that wind farm infrasound would also decrease delta power and increase higher frequency power during sleep. Although the main findings did show a small but significant sustained reduction in in delta power from around 10 seconds post-stimulus onset, the main finding was an acute

increase in delta power and K-complex probability within the first five seconds after infrasound onset.

K-complexes are a transient EEG delta wave during sleep that can occur spontaneously, as a prominent feature of N2 sleep, and in response to auditory and other sensory disturbances during sleep. K-complexes are thought to reflect thalamic sensory processing (Colrain, 2005; Naitoh et al., 1982; Pirrera et al., 2010) that may predominantly serve to sustain sleep following relatively mild sensory disturbances not warranting stronger physiological activation responses towards awakening (Colrain, 2005). An acute transient increase then decrease in K-complex response probability with corresponding changes in delta and theta activity show that the abrupt onset of infrasound consistently elicits a cortical sensory response, despite sound pressure levels well below the generally accepted wake audibility threshold. Even participants who were unable during wake to discriminate background noise from infrasound reproduced at 100 dB(G) (around the average hearing threshold below 20 Hz (Watanabe & Møller, 1990) showed increased K-complex probability in the first 5-seconds of 80 dB(G) infrasound onset during established sleep. 80 dB(G) infrasound is well below the lower limit of average human hearing acuity under 20 Hz, but substantially higher than more realistic infrasound exposure levels of around 60-70 dB(G) (Jakobsen, 2005). These findings suggest that sensory processes during sleep are more sensitive to low frequency noise onset than would be anticipated for noise unlikely to be perceived as audible during wake.

On the consideration of detectable vibration, Nguyen et al. (2020) investigated wind farm noise generated vibration at residences (including infrasonic components) and showed the levels at residences unlikely to cause discomfort to a sleeper. However, given the higher level of infrasound in the present study, and speaker reproduction comparative to Nguyen et al. (2020) field study of full spectrum wind farm noise with infrasound (30dB below the human auditory threshold) it is difficult to compare the possible vibration response. Future

studies may consider measuring vibro-tactile responses to high level infrasound which may influence detectability (Hansen et al., 2017a). Although non-auditory vibro-tactile sensory pathways could potentially be relevant, the ear seems most likely to explain cortical responses to infrasound exposure during sleep.

Although there were clearly discernible changes in K-complex and qEEG responses to noise onset and some more sustained effects on some qEEG features, these effects were small and did not translate into increased transient arousal or awakening events. Consistent with higher frequency and sound pressure level noise onset events, the most potent sleep disruption features of noise appear to cluster around noise onset (Carter, 1996; Dunbar et al., 2021; Eberhardt et al., 1987) and the overall impact of infrasound exposure on sleep was small and only discernible through detailed qEEG analysis of the sleep EEG. Potential impacts on sleep and next day functional outcomes remain unclear, but in a related study with more prolonged wind farm noise exposures reproduced in the laboratory at median levels recorded in the field, there was no evidence of disruption to traditional measures of sleep time and quality or impacts on next day function (Liebich, Lack, Hansen, et al., 2022).

This study was specifically designed to test for acute effects of infrasound exposure on sleep EEG responses to help inform the potential future need to consider infrasound exposure effects on sleep and next day functional outcomes. Given clearly discernible but small and transient EEG impacts, a key remaining question is to what extent transient EEG changes in response to infrasound may impact on overall sleep and next day functioning. A recent study released by Marshall et al. (2023) demonstrates that whole night exposures to high level infrasound exposure (~90dB peak) does not appear to influence next day behavioural measures of reaction time, or processing speed, whilst other studies using frequent higher frequency audible noise stimuli suggest that nocturnal noise events do impact next day functioning and mood (Martin et al., 1997; Muzet, 2007). This may indicate that audibility and sound pressure level of the sound is most likely to influence sleep and next day

functioning. Infrasound hearing acuity and sensory responses are challenging to assess and differentiating low level infrasound from higher frequency and intensity noise disruption effects on sleep is also difficult. Nevertheless, the findings from this study support that infrasound effects warrant consideration in future studies. On the other hand, evidence to support any substantial sleep disruption effects from overnight wind farm noise including infrasound at realistic exposure levels are lacking (Liebich, Lack, Hansen, et al., 2022; Marshall et al., 2023; Smith et al., 2020).

3.6.1 Limitations

The infrasonic stimuli used in this study, whilst below the widely accepted audibility threshold of >85dB(G) below 20 Hz (Leventhall et al., 2003; Møller & Pedersen, 2004), was higher than 60-70dB(G) previously recorded at residences nearby to wind turbines (Jakobsen, 2005). Further studies are clearly warranted to test if more realistic levels of wind farm generated infrasound also elicit a discernible sensory response during sleep and wake. Although the sound reproduction system was calibrated prior to each study and infrasound exposures were compared to control periods without additional noise, full overnight recordings were not obtained. Overnight recordings would have been useful to confirm faithful noise reproduction and to help rule out potential effects from extraneous noise. Commercially available speakers are not made to reproduce high level infrasound at 80 dB(G) and the potential for noise artefacts (i.e., crackles and clicks because of hardware limitations) is high and should be considered in the context of the study outcomes. This study was conducted primarily to be a pilot study to inform practice for future research. Based on the findings of this study clear overnight simultaneous acoustic recordings are warranted to rule out potential noise artifact.

Participants were healthy good sleepers recruited from built up urban areas and

therefore likely to be habitually exposed to higher levels of background noise during sleep than individuals from rural areas exposed to wind farm noise. Responses in individuals habitually exposed to wind farm noise might be different and would be worth investigating to test for potential relationships between K-complex and qEEG responses and self-reported daytime noise annoyance and impacts. Individuals who are habitually exposed to wind farm noise may experience different reactions to the noise itself. Knowledge, beliefs or expectations around noise exposure potentially including inaudible noise could also influence responses, depending on prior experiences and expectations regarding wind turbines and individual tolerance and sensitivity to noise and real or expected annoyance. These factors may contribute to both physiological and psychological reactions to noise that may differ within and between individuals and noise exposure contexts.

3.6.2 Conclusion

Overall, this study found transient EEG responses to relatively high levels of wind farm infrasound reproduced in a controlled laboratory setting. The clearest effects were a transient increase in delta activity and K-complexes in the first five seconds of exposure, followed by a smaller sustained reduction in delta power over the remainder of the stimulus. However, these effects were relatively small and there was no evidence of increased EEG micro-arousals or awakenings compared to control background noise exposures. Thus, wind farm noise infrasound exposure during sleep at levels below the conventionally accepted lower limit of hearing supports that infrasound warrants further consideration as a potential source of sleep disruption.

3.7 Acknowledgements

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CHAPTER 4 : A QUANTITATIVE EEG ANALYSIS OF OVERNIGHT NOISE EXPOSURE EFFECTS ON SLEEP – A LABORATORY STUDY

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Abstract

Introduction: Nocturnal noise exposure can disrupt sleep, but conventional sleep assessment methods may not be sufficiently sensitive to detect potentially subtle noise exposure effects on sleep such as from low frequency dominated wind farm noise (WFN). The aim of this study was to determine whether quantitative electroencephalography (qEEG) may be more sensitive and useful for assessing WFN noise effects on sleep and to further examine whether the effect of noise on sleep and to test for potential between group differences in individuals with and without habitual WFN exposure and self-reported noise-related sleep complaints.

Methods: After an acclimation night in a sleep laboratory, 68 individuals (Males: Females; 30:38) aged (Median[IQR]) 55.5yrs [31.3 to 66.3], from four groups (rural residents (a) with and (b) without WFN-related complaints, (c) urban traffic-noise-exposed residents and (d) quiet rural area controls) underwent polysomnography recordings during six different noise exposure nights in random order. These included; 20 second intermittent noise exposure (overnight average sound pressure level (SPL) 42 dBA), 3 minute intermittent noise exposure (overnight average SPL 32 dBA), continuous WFN at 25 dBA from lights out to lights on, WFN exposure only during sleep, WFN only during wake, and a quiet control night (background noise, 19 dBA). Likelihood ratio tests were used to examine noise condition, group and sleep stage (Wake,N1,N2,N3, REM) effects on qEEG power in delta (0.5-4 Hz) to beta (32 Hz) frequencies, K-complex density, spectral entropy of delta activity and traditional sleep metrics obtained from polysomnography recordings.

Results: Wake after sleep onset, time spent in N1 sleep and relative beta activity were higher, whilst time spent in N3 reduced on the 20 second noise exposure night when compared to the control night (all $p<0.05$). K-complex density was significantly increased during both 20-sec and 3-min intermittent noise exposure conditions comparative to control ($p<0.001$). No significant differences in qEEG outcomes were observed between groups.

Conclusion: Whole night average qEEG did not appear to be more sensitive to noise related

sleep disturbance relative to traditional methods and were not different between groups. However, K-complex density was increased with intermittent noise exposure so may be particularly useful in future studies.

4.1 Introduction

Noise related sleep disruption is a commonly reported problem within the community and underpins World Health Organisation (WHO) recommended guidelines for night time noise exposure levels (Basner & McGuire, 2018a). The gold standard for assessing sleep disturbance, including from noise, is polysomnography (PSG). PSG has been used to definitively assess noise impacts on sleep as a result of mid to high frequency noise sources including road, rail and air traffic noise (Basner et al., 2011; Griefahn et al., 2006). However, very few studies have specifically evaluated wind farm noise (WFN) effects on sleep using PSG, despite significant community concerns and complaints regarding WFN annoyance and sleep disturbance. WFN typically occurs in rural and remote areas with much lower nocturnal background noise levels than in urban areas. WFN is also dominated by low frequency components, with much longer transmission distances and greater penetration into building structures compared to higher frequency dominated noise. Given that rapid and ongoing growth in wind turbine power generation is exposing more individuals to WFN, it is important to definitively evaluate WFN effects on sleep using appropriately sensitive assessment tools. To date, literature reporting gold-standard PSG and actigraphy assessments of sleep with wind farm noise exposure at representative noise levels have shown mixed results (Ageborg Morsing et al., 2018; Liebich, Lack, Hansen, et al., 2022; Liebich, Lack, Micic, et al., 2022; Michaud, Feder, Keith, Voicescu, Marro, Than, Guay, Denning, Murray, et al., 2016; Smith et al., 2020). The most relevant study to date was conducted by Smith et al. (2020) which compared exposed and non-exposed individuals to WTN at 32 dB_{leaq} and found REM related differences between WTN exposure and quiet control exposure but no other instances of polysomnographic changes. Smith et al. (2020) also noted significantly worse subjective reports of sleep for the previously WTN exposed group during WTN exposure conditions. This discrepancy between the subjective and objective reports of sleep disruption under WTN exposure may be explained by the objective measurement techniques

which may not be sufficiently sensitive to capture slight changes in the EEG. This work supports the need for further detailed analysis of EEG to examine potential effects of noise on sleep not captured by traditional sleep scoring techniques.

Further additional studies suggest the need for more in depth analysis of sleep electroencephalography (EEG) to determine analysis of sleep microstructure (Jalali, Bigelow, et al., 2016; Lechat et al., 2021). Jalali, Bigelow, et al. (2016) showed no difference pre and post wind turbine operation in an analysis of sleep macrostructure, however they did find mixed results when looking at evoked changes on noise matched EEG segments. This analysis suggests the need for a more sensitive analysis of EEG to reveal any noise related EEG disturbance. Lechat et al. (2021) showed that the odds of experiencing a K-complex wave formation was increased during noise exposure and provided a sensitive measure of noise exposure to low levels of noise at 33dBA beyond analysis of EEG arousals and awakenings which were only associated with noise events above 39 dBA.

The degree of sleep disruption with overnight noise exposure depends on noise level, spectral features and the type and timing of exposure during sleep, where higher level intermittent noise has been shown to be more sleep disruptive than low level continuous noise (Ageborg Morsing et al., 2018; Basner et al., 2011; Griefahn et al., 2006). Therefore, overnight assessments of EEG responses to noise exposure should consider intermittent versus continuous noise exposure effects.

Chapters 2 and 3 of this thesis explored the utility of qEEG analysis, temporally aligned to noise events, to specifically evaluate EEG changes following noise exposure onset (Dunbar et al., 2021). Those analyses supported the utility of qEEG for detecting subtle within night EEG changes largely not discernible with traditional sleep and arousal scoring. However, the evaluation of overnight sleep disturbance, such as for the diagnosis of sleep disorders, or the evaluation of sleep quality more generally, typically relies on analysis of full-night recordings without any acoustic recordings or more detailed information regarding

noise exposure conditions. Hence, the primary aim of this study was to test the utility of qEEG analysis to discriminate EEG frequency differences between nights with, versus without, a range of overnight noise exposure conditions, including WFN simulating realistic real-world exposure levels in a controlled laboratory setting. The main qEEG analysis was based on conventional Fast Fourier Transform (FFT) based methods (A. D'Rozario et al., 2015). Two additional recently developed algorithms were also applied to evaluate noise exposure effects on K-complex density (Lechat et al., 2021b) and spectral entropy of EEG delta power (Lechat, Hansen, et al., 2022). K-complexes are a well-established EEG sensory processing phenomenon commonly elicited by noise (Colrain, 2005; de Zambotti et al., 2016) and are a type of delta wave for which noise exposure could potentially also influence the overnight distribution, and thus spectral entropy, of delta power.

A secondary aim was to examine the potential impact of different forms of noise exposure on EEG frequencies during sleep including more intermittent, varying sound types and levels, and continuous low frequency full-night noise exposures around average real-world levels in a carefully controlled laboratory environment. Part of this aim was to test for potential wake versus sleep dependent effects of WFN exposure on sleep qEEG measures by comparing full-night qEEG measures between nights when noise exposures were restricted to wake, sleep, or played across a full-night compared to a control night without noise exposure.

Given that habitual noise exposure and self-reported sleep difficulty in the presence of noise could also influence noise-related sleep-disruption effects (Liebich et al., 2021; Pedersen, 2011), a further aim was to consider the impact of different prior-exposure groups on qEEG outcomes. An individual's psychological factors, prior experiences and attitudes and beliefs regarding individual noise sources are known to impact annoyance ratings and other responses to noise exposure. These factors could potentially contribute to perceived negative impacts of noise exposure on daytime function and long-term health (Baudin et al., 2021; Gong et al., 2022). Therefore, although not the primary focus, consideration of

potential prior noise exposure and self-reported noise-related sleep disturbance effects were considered as part of this thesis work.

It was hypothesised that qEEG measures derived from full-night recordings would show greater differences compared to a quiet control night on nights with higher and more variable sound pressure level exposures, with shifts towards high EEG frequencies and lighter sleep with reduced qEEG power in slower frequencies associated with deeper sleep. As a secondary aim of the study it was further hypothesised that WFN-disturbed individuals would show greater qEEG disturbances than other groups during night with audible WFN exposure, including nights with continuous WFN exposure presented during periods of wake, sleep and both wake and sleep.

4.2 Method

4.2.1 Participants

This study was approved by the Southern Adelaide Clinical Human Research Ethics Committee (Protocol number 343.18) and was prospectively registered on the Australian and New Zealand Clinical Trials Registry (ACTRN12619000501145, UTN U1111-1229-6126). Study participants were recruited through advertising on university noticeboards, word of mouth, social media and previous inclusion and documentation of interest in the study from a computer assisted telephone survey of individuals in rural, wind farm and urban traffic noise exposure areas conducted as a part of the wider project and beyond the scope of this thesis. Interested individuals were screened for eligibility via an online screening questionnaire administered via Qualtrics software (Utah, USA) and were provided with comprehensive study information prior to providing informed written consent. Participants were reimbursed AUD$800 for full study completion, and either AUD$100 (urban participants) or AUD$400 (rural participants) to help cover travel costs.

Four groups were recruited as follows; (1) individuals living in a quiet rural area (as

classified by Australian government rural and remote area criteria (*Rural, Remote and Metropolitan Area*, 2021) >10km from any wind turbines (Rural control); (2) individuals residing <10km from a wind turbine with mild to more severe WFN related sleep disturbance (WFN sleep disturbed); (3) individuals residing <10km from a wind turbine without WFN sleep disturbance (WFN non-sleep disturbed); and (4) individuals residing < 500m from a busy road (averaging ~40,000 cars per day) and with RTN related sleep disturbance (RTN sleep disturbed). Participants with WFN related sleep disturbance reported >1 and ≤5 on a 6 point Likert scale (1=not at all, 2=mildly, 3=moderately, 4=severely, 5=very severely, 6=unsure, 7= declined to answer) in response to the question *"Thinking about the last 12 months or so, when you are at home does the noise from wind turbines bother, disturb or annoy you while you are in bed trying to sleep?".* The choice of greater than 1 for sleep disturbance reflected the limited sample size from which it was not possible to meaningfully examine responses in those with higher self-reported WFN related sleep disturbance. Participants with RTN related sleep disturbance answered >1 and ≤5 to an equivalent question regarding road traffic noise. All non-noise disturbed participants responded 1 ("not at all") to the same questions.

Post screening and consent, participants were booked for a seven night laboratory stay and provided with a seven-day sleep diary (Carney et al., 2012) and actigraphy (Actiwatch 2, Philips Respironics, USA) for two weeks prior to their scheduled stay to assess usual sleep patterns and habitual sleep timing (to determine appropriate lights out and lights on times for their laboratory stay). All participants also underwent comprehensive audiometry assessment by a qualified audiologist in an audiology testing facility (See Table 4.1).

4.2.1.1 Sleep Laboratory

The study was undertaken at the Adelaide Institute for Sleep Health in the Nick Antic Laboratory equipped with two purpose-built bedrooms (each with adjoining ensuite) with no windows, constructed with double-walls over a vibration isolated

concrete slab, heavy noise-insulated doors and low noise air-conditioning system to achieve low background noise (19 dBA). Laboratory temperature was controlled at around 23 °C. Light levels were <10 lux prior to and following sleep, and <1 lux during the sleep period. A shared living room and meal area was available to participants and all meals were provided. Between study nights participants were free to leave the laboratory.

4.2.2 Polysomnography Sleep Study Setup

Overnight polysomnography (PSG) was recorded using Grael 4K hardware and Profusion 4 acquisition software (Compumedics Ltd., Abbotsford, Victoria, Australia). PSG setup included gold-cup electroencephalography (F3, Fz, F4, C3, C4, O1 and O2 referenced to linked M1 and M2), electrooculography (EOG), electrocardiography (ECG), chin electromyography (EMG), leg movement, nasal cannula, oro-nasal thermistor, chest and abdominal motion and finger oximetry. EEG signals were exported at a sampling rate of 512 and unfiltered.

4.2.3 Noise Samples and Reproduction

Noise samples for in-laboratory reproduction for this study were selected from field recordings. Briefly, wind farm noise samples were obtained from representative samples approximately 3.3km from a nearby wind farm in South Australia (Nguyen et al., 2021). Road traffic noise samples were obtained from overnight recordings obtained inside a residence close to a busy suburban road. Faithful reproduction of road traffic noise samples was via a Krix Pheonik V2.1 speaker (50-200 Watts dimensions; 950 (H) x 195(W) x 295(D) mm) with 6 Ohms input impedance (35Hz to 40kHz frequency response). Wind farm noise sample reproduction, including infrasound, was via an RME BabyFace Pro sound card, and a modified (without vent) Krix KX4010S commercial cinema subwoofer with 10-inch driver and Crown DC-300 power amplifier with a flat frequency response down to 0 Hz. Speakers were placed along the bedroom wall approximately one metre from the foot of the

participants' bed. Room acoustics were considered during set up and calibration of the experiment with all noise samples calibrated at the pillow of the bed to mimic the listener. Noise was equalised until the spectra at the pillow matched the spectra of the noise that was recorded in the field to most accurately represent ecological validity of noise presentation.

Prior to each experiment, noise reproduction equipment was calibrated using a SVANTEK 958 sound level meter. Noise randomisation, reproduction and synchronisation timing signal output was controlled by custom software implemented in MATLAB (Version 2018a/b 9.4/9.5, Mathworks, Natick, Massachusetts). This system also allowed for technical staff blinding of nightly noise exposure conditions and SPLs. Noise exposures were concurrently recorded using a PROSIG P8004 24-bit Data Acquisition System and a GRAS 40AZ microphone placed ~1 metre above the bed-head, to confirm faithful reproduction of the noise sample levels and elements. The microphone can record noise level as low as 17 dBA (dynamic range: 17 to 132 dB) and from 0.5 Hz to 20 kHz (frequency range ± 2dB).

The study nights comprised of seven exposure nights and are as follows: (1) An initial adaptation night (Adaptation) followed in random order by either; (2) a night with frequent intermittent 20-sec noise samples at sound pressure levels varying between 30-50dBA with an average exposure level across the whole night of ~42dBA (20 second night), or; (3) a night with 3-minute RTN and WFN noise exposures at 30 and 35dBA, with an average whole night noise exposure of ~32dBA (3 minute night), followed in random order by; (4) a full exposure night of WFN at 25dBA from lights out to lights on (Continuous WFN); (5) a night with 25dBA WFN exposure administered only during established N2, N3 or REM sleep (WFN-asleep); (6) a night with 25dBA WFN exposure administered only during periods of wake or N1 sleep(WFN-awake); and (7) a night without noise exposure with only background noise at 19 dBA (Control).

4.2.3.1 25 dBA WFN Exposure Nights

Full spectrum wind farm noise including amplitude modulation and infrasound components was reproduced at 25dBA to approximate average indoor year-long measurements of around 26 dBA measured in residences close to a wind farm (Nguyen et al., 2021). This noise sample was played on a continuous 3-minute loop and was selected to represent a near worst case WFN recording with prominent amplitude modulation and infrasound. For the full-exposure night this noise sample was played continuously from lights out to lights on. However, to also help test for potential psychological influences of audible WFN during wake on subsequent sleep, this noise sample was also played on WFN-awake and WFN-asleep nights. The absence of discernible effects of these interventions on conventional sleep measures is presented elsewhere (Liebich, Lack, Hansen, et al., 2022), so the focus of this study was on the exploration of the potential utility of full-night qEEG metrics to detect acoustically-induced sleep disturbance.

4.2.3.2 20 Second Intermittent Noise Exposure Night

This night was used to help evaluate the sensitivity of full-night qEEG analysis for detecting noise-induced sleep disturbance. WFN and RTN noise samples with and without amplitude modulation and with varying mid to high frequency components were reproduced at 30, 40 and 50 dBA using a rapid 250-millisecond ramp up time. This exposure condition was anticipated to be more disruptive to sleep with little chance for noise habituation compared to other noise exposure nights. Average expected overnight noise exposure levels were (Mean [95% CI]), 42.1 [41.9-42.2 dBA]), which represents the upper range of outdoor WFN exposure levels in the field and substantially exceeds indoor recommended night-time noise limits in most countries (Alamir et al., 2021).

4.2.3.3 3 Minute Short Intermittent Noise Exposure Night

This night was used to further evaluate the utility of qEEG analysis to detect noise induced sleep disturbance at noise levels anticipated to be more representative of real world exposure levels in the field. Following a short ramp up time of 250 milliseconds, noise

samples were reproduced at 30 and 35 dBA, corresponding to approximately 72 and 77dBG (with audible components filtered) for samples of WFN infrasound stimulus. Average overnight noise exposure levels were 31.8 (31.2 to 32.3) dBA, close to average outdoor noise measurements from a year-long study of 32 dBA (Nguyen et al., 2021), and World Health Organisation (WHO) recommended acceptable indoor noise limits of 30dBA (Berglund et al., 1999).

4.2.4 Daytime Symptomology and Sleepiness

Subjective reports of daytime symptomology following nocturnal noise exposure were evaluated each morning post awakening via a questionnaire adapted from the visual analogue scale (VAS) for symptoms questionnaire presented by the Woolcock institute (Tonin et al., 2017) with some additional symptoms. Participants were asked to *"please tell us to what extent you are affected by any of the following symptoms right now",* and given the opportunity to use a sliding scale from *0 =(Not at all affected) to 100 =(Extremely affected),* to rate symptoms including headache, nausea, dizziness, pressure in the ears, ringing in the ears, itchy skin, blurred vision, vertigo, tiredness, feeling faint, difficulty concentration, difficulty remembering, irritability, muscle spasms and ear pain.

At forty-five minutes post awakening, participants were asked to rate their current level of subjective sleepiness. This was timed later in the morning to avoid potential impacts of sleep inertia. Subjective sleepiness was assessed using the Karolinska Sleepiness Scale (KSS) which asked the following question *"Please indicate how sleepy you have felt during the preceding 10 mins by selecting the number that corresponds to the words that best describe your state",* on a 9 point scale from *1=(Extremely alert) through 5 =(Neither alert or sleepy) to 9 =(Very sleepy, fighting sleep, great effort to stay awake)* (Shahid et al., 2011*).*

4.2.5 Daytime Cognitive Performance

To evaluate the potential impact of nocturnal noise exposure on next day functioning several

daytime cognitive performance tasks were administered in the hour post awakening.

4.2.5.1 Digit Symbol Substitution Task (DSST) as a Measure of Processing Speed

The DSST is based on a subtest of the Wechsler Intelligence Scale for Children, Fifth Edition (WISC-V) (Wechsler, 2014) and the Wechsler Adult Intelligence Scale, Fourth Edition (WAIS-IV) (Wechsler, 2008) to assess cognitive processing speed and accuracy. A digital version was used to determine the number of digits correctly matched to symbols and the number of errors made. This task asks individuals to view a series of numbers with symbol associations and match as many of the presented symbols to their corresponding number as quickly and accurately as possible within a 2-minute time limit. The DSST shows good sensitivity, validity and high correlations with real world functional outcomes (Jaeger, 2018) and has previously been used to evaluate effects of noise exposure during sleep on next day functional outcomes (Liebich, 2022).

4.2.5.2 Digit Span as a Measure of Short Term Working Memory

Short term working memory was assessed using the Digit Span task based on a subtest of the WISC-V (Wechsler, 2014) and the WAIS -IV tests (Wechsler, 2008). A computerised version was used where individuals were asked to watch numbers appearing one after another on a computer screen in sequences of progressively increasing length (starting with two numbers) and to then recall each sequence correctly. Each correctly recalled sequence was followed by an incrementally longer sequence until the individual failed to correctly recall two series of the same length at which point the test ended. Both forwards and backwards sequence recall were tested. Mean span of digits recorded (i.e., the average count of numbers correctly recalled) and the maximum length of digits correctly recalled (i.e., the maximum number the individual was able to recall correctly) were determined. The Digit Span test has previously been used to examine the effects of noise induced sleep disturbance on next day memory recall (Carter, 1996).

4.2.5.2 Psychomotor Vigilance Task (PVT) Reaction Time and Lapses

The PVT assesses reaction time by asking participants to respond as quickly as possible via a button push, to repeated stimuli presented at a randomised interval between two to ten seconds over 10 minutes of testing. The PVT task has previously shown sensitivity to increasing night time SPLs, where participants are less accurate after night-time noise exposure (Elmenhorst et al., 2010). The variables of interest for the current study were mean and median reaction time (i.e., how long it took in milliseconds for the individual to press the button in response to a stimulus), total false commissions (i.e., pressing the button when no stimulus was present) and lapses (i.e., failing to respond to a stimulus for more than 500 milliseconds).

4.2.5.3 Next Day Mood

Mood following night-time noise exposure was assessed using the Profile of Mood States (POMS) self-report questionnaire. This questionnaire has previously been validated and has shown high discriminate validity, internal consistency and good test-retest reliability (Gibson, 1997; Nyenhuis et al., 1999). The questionnaire is a 65-item scale which asks the participant to indicate on a Likert rating scale from 0 (=not at all) to 4 (=extremely) in response to the question "*Describe how you feel right now by checking one space after each of the words listed below*". Items were words describing moods such as "*tense, angry, sad"*. Total mood disturbance scores were calculated by adding the raw scores from tension, depression, anger, fatigue, and confusion sub-scores and subtracting the vigour score. The total mood disturbance scores range from -32 to 200, with higher scores indicating greater mood disturbance.

4.3 Data analysis

PSG sleep staging and arousal scoring were conducted by an independent qualified sleep technician, who remained blind to the acoustic intervention and aims of the study, and

used the American Academy of Sleep Medicine scoring criteria (AASM; (Conrad Iber et al., 2007)).

4.3.1 EEG Power Spectral Analysis

4.3.1.1 Pre-Data Quality Checks and Cleaning

Prior to analysis raw EEG spectra were examined for quality. Two independent scorers visually examined the C3 (central left) electrode spectra and time series waveforms of each sleep study for artifact and noise. EEG spectra and waveform for the whole night were given a quality score from 0-100 corresponding with the percentage of observed poor quality signal. The most common source of signal artefacts was the electrode falling off during the night. Agreement between scorers was high (See Appendix A, Figure 2), supporting quality control and process reliability. Studies with an average quality rating indicating that 15% or more of the night was corrupted by artefact were excluded from analysis based on previously excepted thresholds for data cleaning (A. D'Rozario et al., 2015). This process removed 43 sleep studies (9.6%, Appendix A) and the majority of outliers. The C3 electrode was chosen for consistency and as a result of sleep scoring being undertaken manually on this channel by the sleep technicians, and for the purpose of comparison to the new methods including K complex analysis which is validated on this channel (Lechat et al., 2020).

4.3.1.2 Spectral Analysis

All spectral analysis was based on Fast-Fourier Transform (FFT) of C3-recorded EEG in 5-second sequential epochs aligned to time in bed from lights out to lights on. Prior to FFT, the signal was bandpass filtered between 0.5 and 35 Hz to reduce electrical noise and muscle artefacts. Epochs with absolute EEG deflections > 400 μv were also considered noise artifact and excluded as previously reported (Sweetman et al., 2021). Filtered EEG was then subjected to FFT, using a custom multi-taper approach based on previously published work (Prerau et al., 2017; Scott et al., 2020). EEG power was then calculated in the delta (0.5-4 Hz), theta (4-8 Hz), alpha (8-12 Hz), sigma (12-15 Hz) and beta (15-30 Hz) frequency ranges

across the whole night, using open-source software (Python version 3.7, Python Core Team (2015), Python Foundation) including the MNE-python software package. This multi-taper method reduces window-related artefacts associated with single window (taper) analyses (Prerau et al., 2017). Both absolute and ratio values (one frequency range divided by the sum of the others) of spectral power were calculated across the whole night as median values.

4.3.2 Additional Measures

Two recently developed algorithms were also applied to examine overnight K-complex density and spectral entropy of EEG delta power. Spectral entropy is a measure derived from the full frequency of delta wave activity across the sleep period and provides a numerical representation of the irregularity of delta power over the night. The entropy measure increases with increased irregularity of the sleep (Lechat, Hansen, et al., 2022). K-complex density was evaluated using an automated algorithm recently developed by Lechat et al (Lechat et al., 2020; Lechat et al., 2021) applied to EEG data recorded from the C3 electrode. Spectral entropy was evaluated from the same signal using another algorithm developed by Lechat et al (Lechat, Hansen, et al., 2022).

4.4 Statistical Analysis

The primary analysis tested for within-subject differences in absolute and relative EEG powers between conditions (nights) using generalised linear mixed models with a gamma distribution and log link function, which was selected on the basis of a non-normal distribution by comparing the Akaike Information Criterion (AIC) between different distribution families (gamma, gaussian) and link function (identity, log) and selecting the model with the lowest AIC value. All models were adjusted for sleep stage as a covariate to test for expected sleep stage effects on EEG power, including a two-way interaction term between condition (night) and sleep stage. Participants were also included as a random effect to account for a repeated measurement of the response variable within-subjects. The overall significance of fixed effects were tested using a likelihood

ratio test. In the event of significant interaction or main effects, relevant post-hoc comparisons were conducted using Wald tests. To address the secondary aims, group was added to the model to test for condition by group interaction effects.

Analysis of traditional sleep macrostructure, daytime symptomology, sleepiness and K-complex density and spectral entropy outcomes were also examined using generalised linear models (glm) from the glmer package in the R statistical software, Version 4.0.1.

Prior night noise exposure condition effects on daytime performance measures of processing speed, memory and reaction time were also examined using similar models. Pearson correlations were also used to determine the strength of potential associations between qEEG markers of noise disrupted sleep with daytime performance outcomes.

Given the non-normal distribution of the data, descriptive data are presented as the median [IQR] or median difference [IQR] unless otherwise indicated. All statistical tests were two-sided, and *p* values of less than 0.05 were considered statistically significant. All analyses were conducted in R statistical software [2], Version 4.0.1, using the lme4 package. Unless otherwise specified, where transforms were necessary to normalise data for statistical analysis, untransformed data are presented in figures and tables to help simplify interpretation of the results. Effect sizes were interpreted according to Cohen's d (< 0.2 small, 0.5 medium, > 0.8 large; (Cohen, 1992) respectively).

Differences in baseline participant demographics were examined using linear mixed modelling (LMM), in the IBM statistical package for social sciences (SPSS©, version 25). Non-normal values were log transformed for normality.

4.5 Results

4.5.1 Participants

Figure 4.1 shows a CONSORT diagram depicting the flow of participants through the study. Sixty-eight participants aged 18-80 years consented to participate and completed the

study. Demographic data for the study sample are summarised in Table 4.1. Further demographic data from study sub-groups have previously been reported elsewhere (Liebich, Lack, Hansen, et al., 2022). Sixty-one participants were of Caucasian decent, 2 were of North East Asian, 1 Sub-Saharan African, 2 South East Asian, 1 South Asian, and 1 of Anglo-Saxon descent. The WFN sleep disturbed group was the smallest as this group was the most difficult to recruit and was further impacted by COVID-19 travel restrictions. The WFN sleep disturbed group were significantly older, and the WFN non-sleep disturbed group had a significantly higher BMI than both the rural control and RTN disturbed group. The WFN disturbed group reported higher symptoms of baseline insomnia and poorer quality of sleep relative to both the rural control and RTN disturbed groups. The WFN sleep disturbed group also reported higher levels of sleepiness than the rural control group. Audiology reports showed the WFN sleep disturbed group also had overall worse hearing relative to the RTN disturbed group. As expected, and by design, the degree of reported WFN related sleep disruption was significantly higher in the WFN sleep disturbed group and the degree of reported RTN sleep disruption was significantly higher in the RTN sleep disturbed group relative to all other groups (Table 4.1).

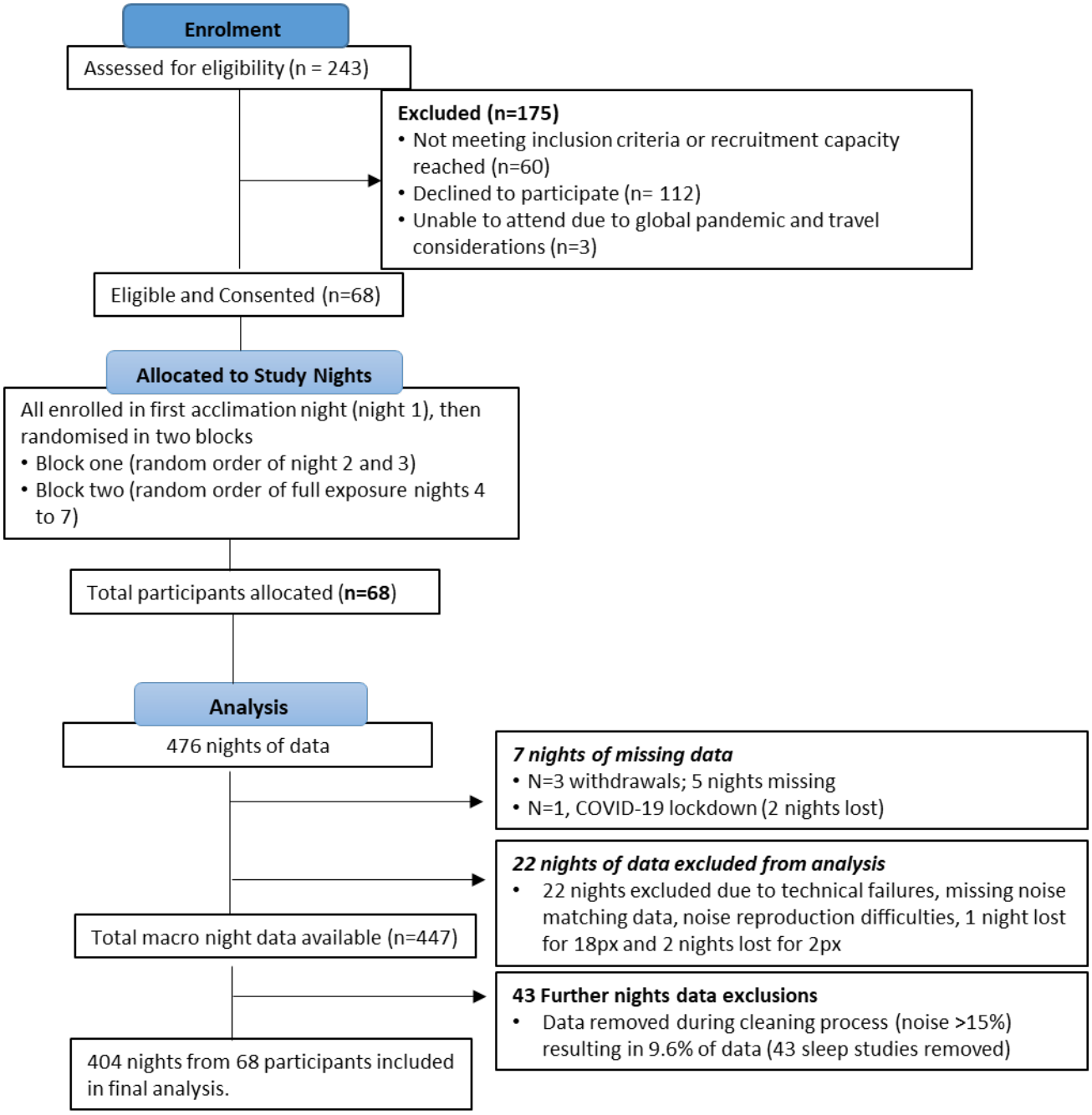


Figure 4.1 CONSORT flow diagram showing the process from expression of interest to analysis for the sample of the study.

Table 4.1 *Participant demographics*

	WFN sleep disturbed	WFN non-sleep disturbed	Rural Controls	RTN sleep disturbed	P -Value	Overall
N	14	18	18	18	-	68
Sex N (Number of Males:Females)	7:7	9:9	4:16	10:8	-	30:38
Age (YEARS)	68.0 [63.3 to 69.8]*≠	58.5 [45.8 to 66.0]┼	46.5 [27.0 to 64.3]≠	27.0 [23.3 to 42.8]*┼	<0.001	55.5 [31.3 to 66.3]
BMI (kg/m²)	27.8 [25.4 to 29.9]	29.3 [26.5 to 33.4]*	26.4 [24.2 to 30.9]	23.1 [21.4 to 25.3]*	0.015	27.0 [23.5 to 30.8]
Weinstein noise sensitivity	65.0 [62.3 to 74.5]*	65.0 [49.0 to 73.0]	55.5 [49.8 to 58.0]*	67.5 [53.0 to 80.8]	0.028	62.5 [53.0 to 71.0]
ISI Global	11.0 [8.3 to 16.5]┼*	7.5 [4.3 to 8.8]	6.5 [3.5 to 7.8]*	5.0 [3.3 to 9.8] ┼	0.006	7.0 [4.0 to 10.3]
ESS Global	8.0 [6.3 to 12.0]*	4.5 [3.0 to 7.0]	4.5 [2.3 to 6.0]*	5.0 [2.5 to 9.5]	0.006	5.0 [3.0 to 8.3]
PSQI Global	11.0 [9.0 to 13.8]┼*	6.5 [5.0 to 9.0]	5.0 [4.0 to 7.8]*	5.0 [4.0 to 7.8]┼	0.001	6.5 [4.0 to 10.0]
Self reported Sleep efficiency (%)	73.0 [55.1 to 85.6]	83.3 [67.7 to 90.7]	87.5 [81.5 to 93.2]	91.9 [85.0 to 94.4]	0.127	87.3 [71.8 to 93.3]
EQ5D health rating	7.5 [5.0 to 9.0]	6.0 [5.3 to 8.8]	6.0 [5.0 to 7.0]	5.0 [5.0 to 6.0]	0.070	6.0 [5.0 to 8.0]
EQ5D self reported percentage of health today	75.0 [67.8 to 89.3]	80.0 [70.0 to 83.5]	85.0 [66.3 to 89.3]	85.5 [76.3 to 90.0]	0.141	80.0 [70.0 to 90.0]
FOSQ Global	14.5 [13.0 to 15.8]	16.7 [15.3 to 18.8]	17.3 [15.5 to 18.6]	15.8 [14.5 to 17.8]	0.070	16.0 [14.7 to 18.1]
DASS-21 (Depression Score)	3.0 [0.0 to 4.8]	0.5 [0.0 to 2.8]	1.0 [0.0 to 2.0]	3.0 [1.3 to 4.8]	0.498	1.5 [0.0 to 3.3]
DASS-21 (Anxiety Score)	2.5 [1.0 to 5.5]	1.0 [1.0 to 2.0]	1.0 [1.0 to 2.0]	2.0 [0.3 to 4.8]	0.196	1.5 [1.0 to 3.3]
DASS-21 (Stress Score)	3.0 [1.3 to 6.5]	1.5 [0.0 to 5.3]	2.0 [0.3 to 3.8]	3.5 [2.3 to 8.8]	0.237	2.5 [0.0 to 6.0]
Average HL 125-1000Hz RIGHT	11.3 [9.4 to 19.4]┼*	8.1 [6.3 to 13.4]	5.0 [3.3 to 8.8]*	5.0 [2.5 to 10.6]┼	0.021	7.5 [3.6 to 12.8]
Average HL 125-1000Hz LEFT	12.5 [5.0 to 25.0]	7.5 [1.3 to 11.3]	3.8 [1.3 to 8.8]	3.8 [0.3 to 6.3]	0.146	5.6 [1.3 to 11.6]
Average HL L&R	11.9 [8.8 to 19.4]┼	8.1 [4.2 to 10.9]	4.2 [1.9 to 8.1]	4.4 [1.4 to 8.0]┼	0.018	7.2 [2.3 to 11.9]
Degree of RTN disruption	1.0 [1.0 to 1.8]*	1.0 [1.0 to 2.0]┼	1.0 [1.0 to 2.0]≠	3.0 [3.0 to 4.0]┼≠*	<0.001	1.5 [1.0 to 3.0]
Degree of WFN disruption	4.0 [2.3 to 5.0]┼≠*	1.0 [1.0 to 1.0]┼	1.0 [1.0 to 1.0]≠	1.0 [1.0 to 1.0]*	<0.001	1.0 [1.0 to 2.0]
Average distance from nearest road traffic noise source (km) [a]	0.6km	0.8km	0.4km	0.3km	-	-
Average distance from nearest turbine (km)[a]	2-4km	4-6km	>10km	>10km	-	-

Note. * and ┼ and ≠ indicates significantly different group pairwise comparisons (p<0.05) based on log transformed normalised values assessed using linear mixed modelling. Values are median[IQR] or n [%], n=68 except for hearing assessments where n=60. BMI=Body Mass Index. dB=decibel. HL=Hearing Level. Normal hearing cut off =<20dBHL. DASS 21 item Depression, anxiety and stress scale (Henry & Crawford, 2005). Depression subscale (0-4=normal, 5-6=mild, 7-10= moderate, 11-13= severe, 14+ = extremely severe symptoms of depression). Anxiety subscale score (0-3 = normal, 4-5 = mild, 6-7 = moderate, 8-9= severe, 10+ = extremely severe symptoms of anxiety). Stress subscale (0-7=normal, 8-9= mild, 10-12= moderate, 13-16=severe, 17+= extremely severe symptoms of stress). EQ5D= Measure of current health quality (max 25; higher scores indicate worse health). EQ5D% todays health: higher percentage scores indicate greater health (Lee et al., 2021). FOSQ-10 (range 5-20, 20 max score, higher scores indicate better functioning) (Chasens et al., 2009). PSQI (range=0-21, >6 = poor sleep quality) (Buysse et al., 1989). PSQI Sleep Efficiency % (<85% clinical cut off for healthy sleep efficiency). ISI (range 0-28, higher scores indicate greater insomnia severity, 0-7= no clinical insomnia, 8-14 = subthreshold insomnia symptoms (>8 indicates clinically relevant insomnia symptoms), 15-21= moderate severity clinical insomnia, 22-28 = severe clinical insomnia (Morin et al., 2011). ESS (range 0-24), scores >=10 indicate excessive daytime sleepiness (Johns, 1991; Johns, 1992). Cut-offs for the Weinstein Noise Sensitivity Scale (range 1-105)= >78 indicates high noise sensitivity, scores <26 indicate low noise sensitivity based on upper and lower quartiles of the original study (Weinstein, 1978). [a] Responses are based on participant self-reports of distance from noise source.

4.5.2 Whole night sleep structure metrics

Table 4.2 shows overnight sleep parameters on each different noise exposure night. There were several significant first (adaptation) night effects, including increased REM latency, time spent in REM sleep, and WASO, and reduced time spent in N3 sleep. In addition, WASO and time spent in N1 sleep were significantly greater, and N3 sleep duration was reduced, on the 20 second noise exposure night compared to control. N3 sleep duration on the 3-minute noise exposure was marginally but significantly greater than control. REM sleep duration was also higher on nights with WFN exposure only presented during sleep than on the quiet control night. K-complex density was significantly greater compared to control on the adaptation night, 20-second and 3-minute noise exposure nights (Table 4.2).

Table 4.2 *Whole night sleep macrostructure metrics*

	Control	Adaptation	20 second	3 minute	WFN full exposure	WFN only asleep	WFN only awake	P-value
N	59	62	59	60	59	47	58	
Noise conditions								
Average SPL (dBA)	19dBA	19dBA	42.1[41.9-42.2]dBA	31.8[31.2-32.3]dBA	25dBA	25dBA	25dBA	-
Number of noise events	-	-	437 [359 to 558]	96 [56 to 114]	-	14 [10 to 19]	17 [12 to 23]	-
Traditional Sleep Metrics								
Sleep latency (min)	11.5 [6.3 to 23.0]	13.0 [8.5 to 21.9]	14.0 [7.8 to 22.5]	10.0 [5.5 to 19.0]	13.5 [6.8 to 20.0]	10.0 [5.5 to 18.8]	12.5 [8.0 to 22.1]	0.389
REM latency (min)	78.0 [64.8 to 107.0]	112.0 [76.0 to 159.0] ***	92.8 [69.6 to 133.1]	77.5 [65.5 to 116.3]	83.0 [64.9 to 104.3]	74.0 [61.3 to 95.8]	76.3 [64.3 to 107.5]	<0.001
WASO (min)	34.0 [16.3 to 76.0]	45.3 [22.6 to 87.6] **	53.0 [21.3 to 73.8]*	36.8 [21.6 to 69.1]	32.0 [15.8 to 79.3]	30.0 [15.5 to 64.3]	31.0 [15.0 to 59.4]	<0.001
N1 (min)	33.5 [25.8 to 56.0]	38.5 [26.6 to 59.4]	41.0 [28.8 to 61.5]*	36.3 [28.0 to 49.0]	33.0 [23.8 to 49.8]	39.5 [23.8 to 48.8]	33.0 [24.8 to 53.3]	0.006
N2 (min)	198.0 [176.8 to 230.5]	192.3 [172.9 to 217.0]	196.0 [165.3 to 237.3]	189.8 [164.3 to 222.6]	202.0 [176.3 to 228.8]	201.0 [177.8 to 226.8]	192.8 [159.8 to 229.0]	0.221
N3 (min)	84.5 [54.5 to 119.5]	81.8 [57.4 to 114.9]**	77.0 [51.0 to 108.5]*	89.3 [68.5 to 122.6]**	95.5 [53.3 to 122.3]	94.0 [60.8 to 116.8]	90.3 [52.1 to 123.0]	<0.001
TST (min)	433.0 [402.0 to 457.3]	400.5 [364.1 to 448.5]*	440.5 [389.8 to 470.0]	424.8 [386.8 to 465.4]	440.5 [391.3 to 478.5]	449.0 [398.8 to 488.0]	425.8 [399.6 to 471.0]	0.002
NREM sleep (min)	331.5 [301.5 to 361.5]	332.3 [286.8 to 361.0]	332.5 [297.8 to 371.0]	324.5 [292.5 to 361.1]	330.0 [299.3 to 365.3]	330.5 [296.0 to 375.5]	330.3 [291.3 to 369.5]	0.476
REM sleep (min)	100.0 [77.5 to 115.0]	75.3 [57.6 to 95.1] ***	91.0 [72.0 to 114.5]	97.8 [81.0 to 108.8]	96.5 [82.8 to 110.5]	108.5 [91.5 to 124.0]*	100.0 [79.6 to 116.3]	<0.001
Sleep efficiency %	86.8 [79.2 to 92.3]	85.8 [78.4 to 89.7]	85.8 [79.8 to 90.7]	87.7 [81.3 to 91.6]	86.2 [79.0 to 92.6]	87.9 [80.5 to 93.2]	86.7 [80.3 to 92.7]	0.149
Total Arousal (/h)	12.0 [8.5 to 16.7]	12.4 [7.4 to 18.4]	10.6 [8.6 to 16.5]	9.9 [7.6 to 15.1]	11.5 [8.1 to 16.2]	10.3 [7.3 to 14.8]	10.8 [8.6 to 14.3]	0.645
Spontaneous Arousal (/h)	4.2 [2.5 to 6.1]	4.3 [2.7 to 8.0]	4.7 [3.1 to 6.7]	4.0 [2.4 to 5.8]	4.2 [2.8 to 6.2]	4.0 [2.8 to 5.8]	3.8 [2.5 to 5.6]	0.0153 a
Non Traditional Sleep Metrics								
K-complex density (/min)	1.9 [1.2 to 2.7]	2.5 [1.6 to 3.7]***	2.6 [1.8 to 3.6]***	2.8 [1.7 to 3.7]***	2.0 [1.3 to 2.8]	2.1 [1.4 to 2.5]	2.0 [1.3 to 2.7]	<0.001
Spectral Entropy (au)	4.0 [3.6 to 4.5]	4.1 [3.7 to 4.5]	4.1 [3.8 to 4.5]	4.1 [3.7 to 4.4]	4.1 [3.8 to 4.4]	4.0 [3.8 to 4.4]	3.9 [3.6 to 4.4]	0.531

Note. Values are Median and IQR unless otherwise specified. *p*-values indicate the condition main effect. ([a] designates a significant main effect with no significant pairwise comparisons). Significant pairwise comparisons are indicated with the following: * indicates p<0.05, ** indicates p<0.01, *** indicates p<0.001 relative to the control condition. au indicates arbitrary units.

4.5.3 Spectral Power Changes During Sleep

As expected for qEEG measures of spectral features relevant to sleep, alpha and beta power were significantly higher during wake than in sleep, and theta and delta activity were significant higher during sleep than in wake (see Figure 4.2). There were also significant group by stage interaction effects in absolute and relative EEG powers. These are presented in more detail in Appendix C and D. However, none of these effects showed any significant interactions with condition.

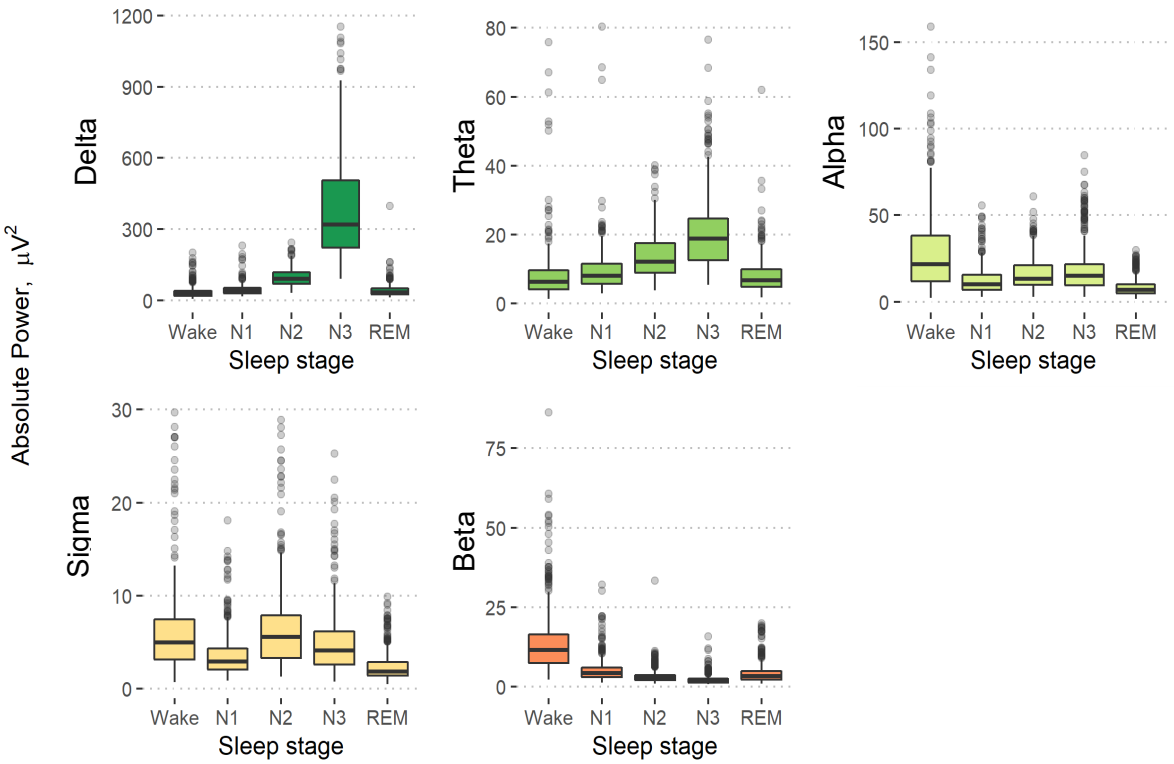


Figure 4.2 *Box plot graphs of QEEG outcomes by sleep stage.*

4.5.4 Noise Condition Effects on Spectral Power

Figure 4.3 shows relative EEG spectral powers from delta to beta frequency across each noise exposure condition night. There were no significant stage-by-condition interaction effects on any qEEG outcome. Effect sizes were very small, but there were several significant main effects of condition, including in absolute beta activity (Likelihood ratio test χ^2 =31.35,

df=6, p<0.001), and in relative beta (χ^2 =39.9, df=6, p<0.001) and theta (χ^2 =19.1, df=6, p=0.004) power expressed as a ratio of power in other frequencies. Compared to the control night, absolute and relative beta power were higher ((Mean difference [95%CI] 0.44[0.21 to 0.72] p<0.001, *Cohens d* = 0.064; 0.004[0.003 to 0.006], p<0.001, *Cohens d*=0.069), and relative theta power was lower (-0.004[-0.002 to -0.006], p=0.001, *Cohens d*=0.131) on the adaptation night (Figure 4.3). Relative beta activity was also higher than control on the 20 second noise exposure night (0.002[0.0002 to 0.004], p=0.035, *Cohens d*=0.031). Absolute EEG data are presented in Appendix B.

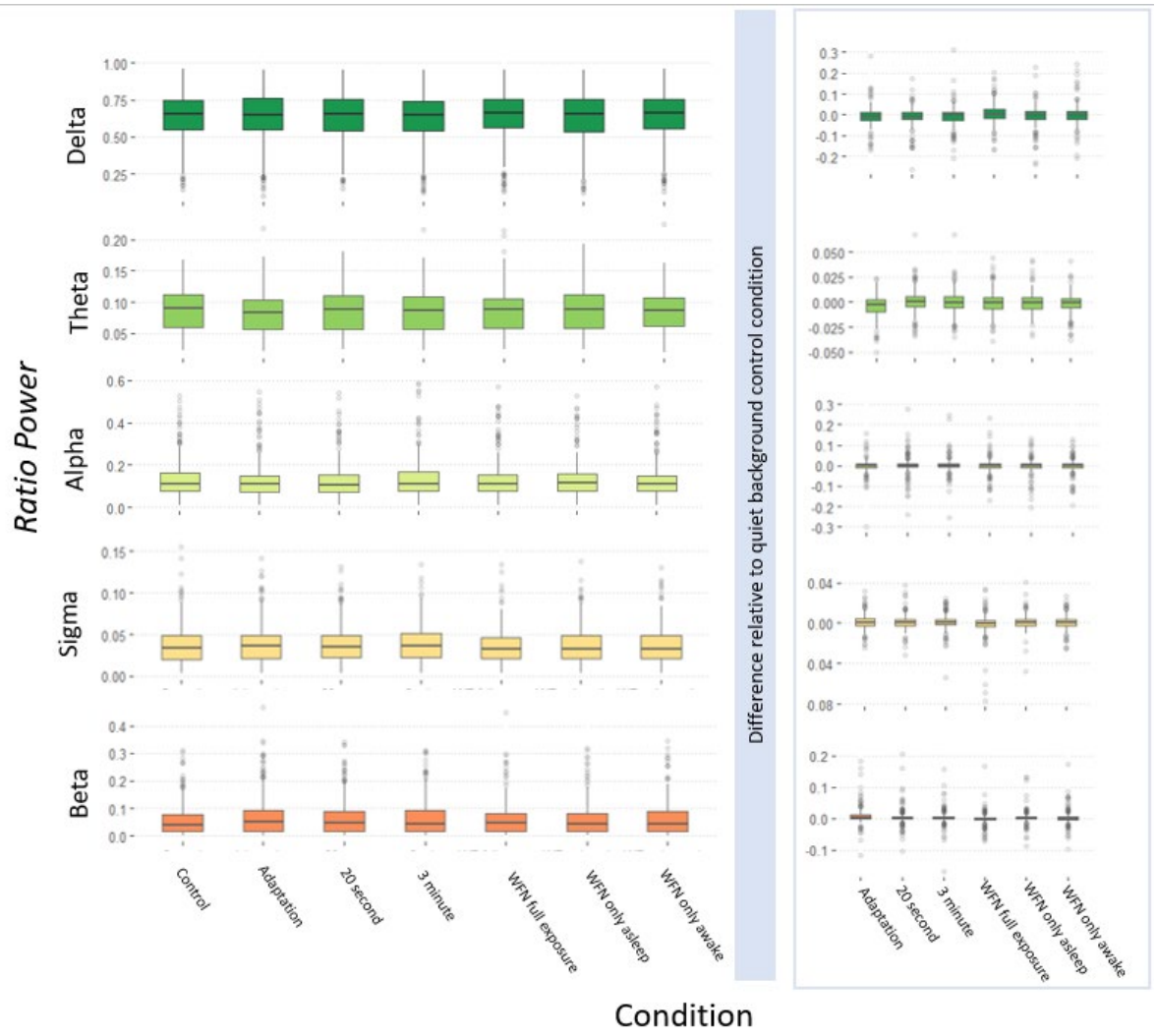


Figure 4.3 Box plots showing (left) median and interquartile range, Tukey-style whiskers (extended to a maximum 1.5 x IQR outside of the box) and outlier values (circles) in delta through to beta power ratios across each different noise condition night, and (right) ratio differences compared the control condition night (19dBA background noise).

Although there were significant group by condition interaction effects in absolute sigma activity (χ^2 =29.6, df=18, p=0.042) and absolute beta activity (χ^2 =30.97, df=18, p=0.029), pairwise comparisons showed no significant differences between any of the groups (RTN sleep disturbed, WFN sleep disturbed or WFN non-sleep disturbed, relative to the rural control group) or between any noise condition versus control background noise exposure (see Figure 4.4).

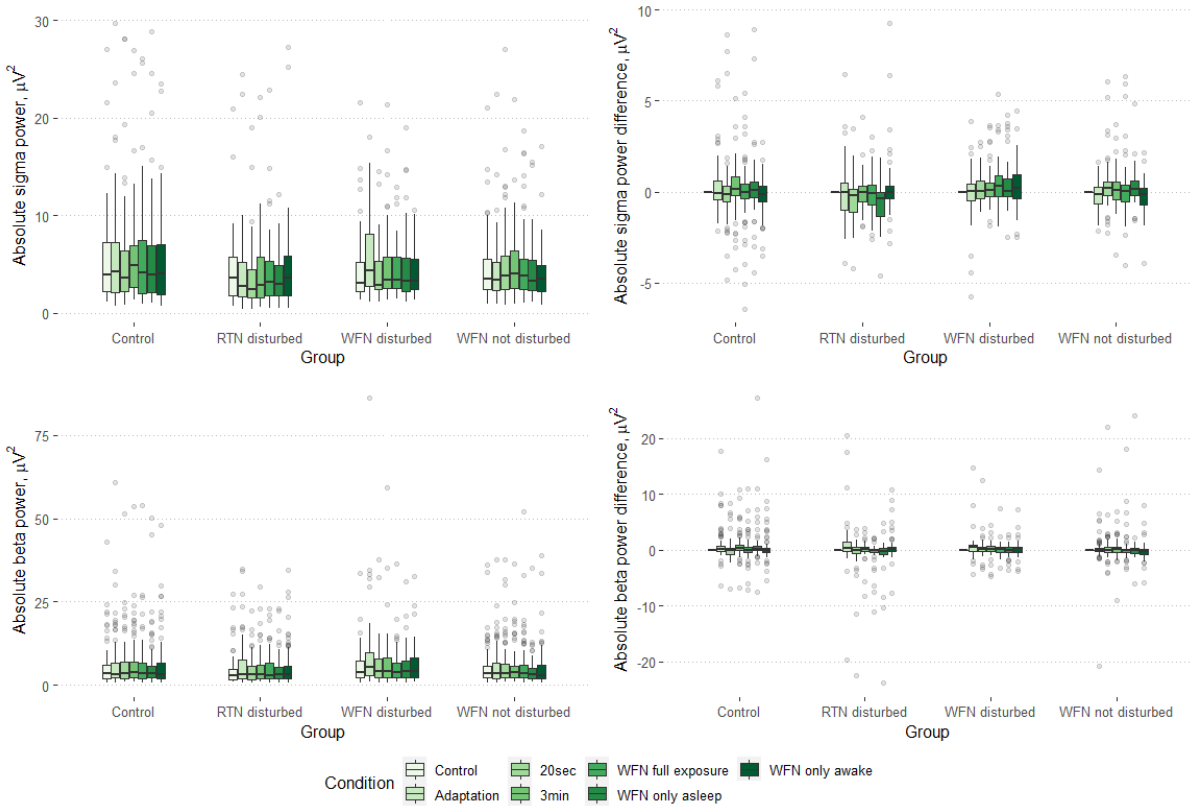


Figure 4.4 (Left) Box plots showing absolute sigma and beta qEEG power relevant to significant group by condition interaction effects. (Right) Box plots showing the relative change in sigma and beta activity compared to the control condition at 0. The box bounds the IQR divided by the median. Whiskers are Tukey-style (extended to a maximum 1.5 x IQR outside of the box) and circles indicate individual data points beyond these ranges (outliers).

4.5.7 Next Day Performance and Symptoms

Table 4.3 summarises the daytime cognitive, reaction time and mood measures. There was a significant condition by group interaction effect in number of values correctly substituted

during the DSST task (χ^2 =32.62, df=18, p=0.019). Pairwise comparisons revealed that post WFN full exposure, the RTN disturbed group correctly substituted significantly more values than the control group (p=0.042). During only asleep exposure and only awake exposure to WFN, the wind farm non-sleep disturbed group correctly substituted more values than the control group (p=0.045, p=0.010 respectively).

There were significant main effects of condition in DSST task performance (χ^2 =122.58, df=6, p<0.001), the maximum length of numbers recalled during the backwards Digit Span task (χ^2 =53.58, df=6, p<0.001), the mean backwards Digit Span recalled (χ^2 =66.80, df=6, p<0.001), the forward Digit Span maximum length of numbers recalled (χ^2 =59.64, df=6, p<0.001) and the mean forward Digit Span recalled (χ^2 =54.91, df=6, p<0.001). These effects were dominated by a first night effect, with significantly fewer correct substitutions on the DSST task (p<0.001), shorter backwards digit length recalled (p=0.016), backwards mean Digit Span (p=0.001), lower forward recall maximum length of numbers recalled (p<0.001) and mean forward span of numbers recalled (p<0.001) following the adaptation night compared to the control condition (Table 4.3). There were also lower values correctly substituted following only awake WFN exposure relative to the control condition (p<0.001). No further between condition effects on daytime performance were observed.

There were no significant effects of prior night noise exposure condition on any subjective daytime symptoms such as headaches, nausea, or vertigo (see Appendix E).

There were no significant main effects or interactions observed for total mood disturbance scores as assessed using the Profile of Mood States for any of the nightly conditions relative to control or between groups (all p>0.05) (see Table 4.3).

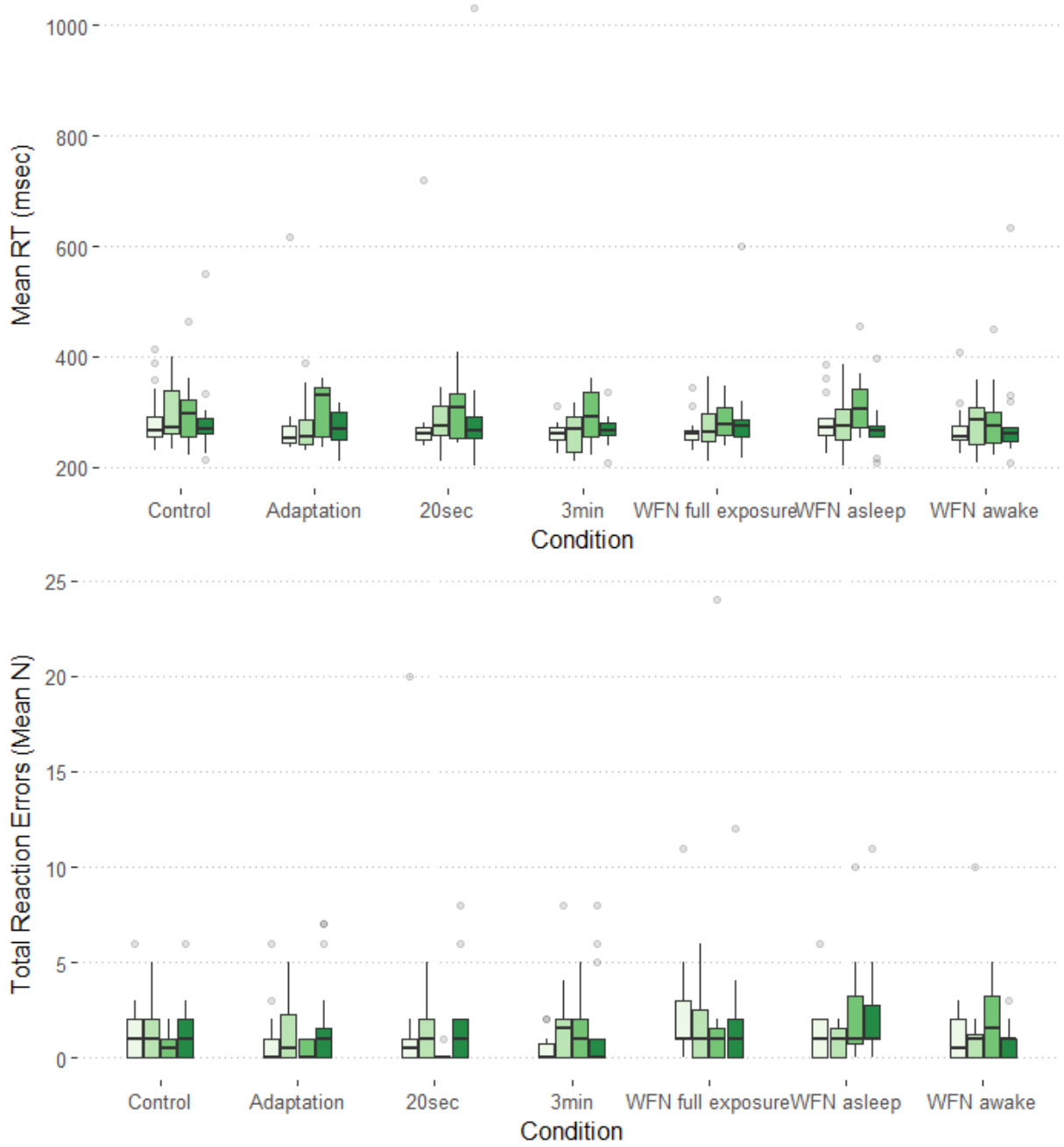
Table 4.3 *Daytime performance measures of mood, cognitive function, and reaction time across conditions for all groups combined.*

Condition	Control	Adaptation	20 sec	3 min	WFN full exposure	WFN only asleep	WFN only awake	*p*-value
N	59	62	59	60	59	47	58	-
Correct Substitutions (DSST)	48.0 [36.0 to 57.0]	40.0 [28.0 to 50.0]***	46.0 [32.5 to 53.5]**	45.0 [33.0 to 58.0]**	48.0 [40.0 to 62.0]	48.0 [34.0 to 57.0]	48.0 [36.0 to 58.0]*	<0.001
Total errors (DSST)	2.0 [1.0 to 3.0]	2.0 [2.0 to 3.0]	2.0 [1.0 to 3.0]	2.0 [2.0 to 3.0]	2.0 [1.0 to 3.0]	2.0 [1.0 to 3.0]	3.0 [1.0 to 4.0]	0.075
POMS	0.0 [-8.5 to 14.5]	1.0 [-8.0 to 16.8]	4.0 [-9.5 to 22.5]	3.0 [-5.0 to 20.3]	1.0 [-7.0 to 14.5]	-3.0 [-10.0 to 13.0]	1.5 [-9.0 to 11.8]	0.428
Mean RT (ms) PVT	272.1 [254.5 to 311.5]	261.0 [245.5 to 298.1]	268.5 [252.0 to 294.4]	267.0 [248.3 to 289.4]	266.3 [247.1 to 285.3]	273.9 [257.5 to 309.9]	261.4 [246.7 to 301.0]	0.267
Median RT (ms) PVT	257.5 [242.6 to 298.8]	244.5 [230.0 to 273.0]	253.0 [235.5 to 270.5]	250.0 [236.0 to 271.0]	255.0 [237.0 to 272.0]	257.5 [244.0 to 294.0]	253.0 [238.0 to 286.0]	0.260
Errors (PVT) (N)	1.0 [0.0 to 2.0]	1.0 [0.0 to 1.0]	1.0 [0.0 to 1.0]	0.0 [0.0 to 2.0]	1.0 [0.0 to 2.0]	1.0 [0.0 to 2.0]	1.0 [0.0 to 2.0]	0.224
Lapses RT>500msec (PVT) (N)	1.0 [0.0 to 3.0]	1.0 [0.0 to 3.0]	1.0 [0.0 to 3.0]	1.0 [0.0 to 2.0]	1.0 [0.0 to 2.0]	1.0 [0.0 to 3.8]	1.0 [0.0 to 2.0]	0.201
Backward maximum length DS	7.0 [6.0 to 8.0]	6.0 [5.0 to 7.0]*	6.0 [5.0 to 8.0]	6.0 [5.0 to 7.0]**	7.0 [6.0 to 8.0]	7.0 [6.0 to 8.0]	7.0 [6.0 to 8.0]	<0.001
Backward mean span DS	6.3 [5.1 to 7.2]	5.3 [4.4 to 6.6]**	5.7 [4.8 to 7.3]	5.8 [4.7 to 6.6]*	6.4 [5.2 to 7.8]	6.3 [5.4 to 7.3]	6.4 [5.5 to 7.4]	<0.001
Forward maximum length DS	7.0 [6.0 to 8.0]	7.0 [6.0 to 8.0]***	7.0 [6.0 to 8.0]	7.0 [6.0 to 8.0]*	8.0 [7.0 to 9.0]	7.0 [6.0 to 8.0]	8.0 [7.0 to 9.0]	<0.001
Forward mean span DS	6.9 [5.8 to 7.7]	6.2 [5.4 to 7.0]***	6.2 [5.8 to 7.5]	6.5 [5.8 to 7.3]*	7.1 [6.3 to 8.4]	7.0 [5.6 to 7.6]	7.1 [6.1 to 7.9]	<0.001

Note. Values are Median and IQR unless otherwise specified. p-values indicate the condition main effect. ([a] designates a significant main effect with no significant pairwise comparisons). Significant pairwise comparisons are indicated with the following: * indicates p<0.05, ** indicates p<0.01, *** indicates p<0.001 relative to the control condition. N=Number of individuals contributing to data, DSST (Digit Symbol Substitution Task), RT (Reaction Time), ms (milliseconds), PVT (Psychomotor Vigilance Task), DS (Digit Span), POMS (Profile of Mood States).

4.5.7.2 Psycho-motor Vigilance Task (PVT) Reaction Time

There were no significant differences observed for mean or median reaction time, errors of commission (false starts) or lapses in reaction (>500msec) for any of the nightly conditions relative to control or between groups (see Figure 4.5).



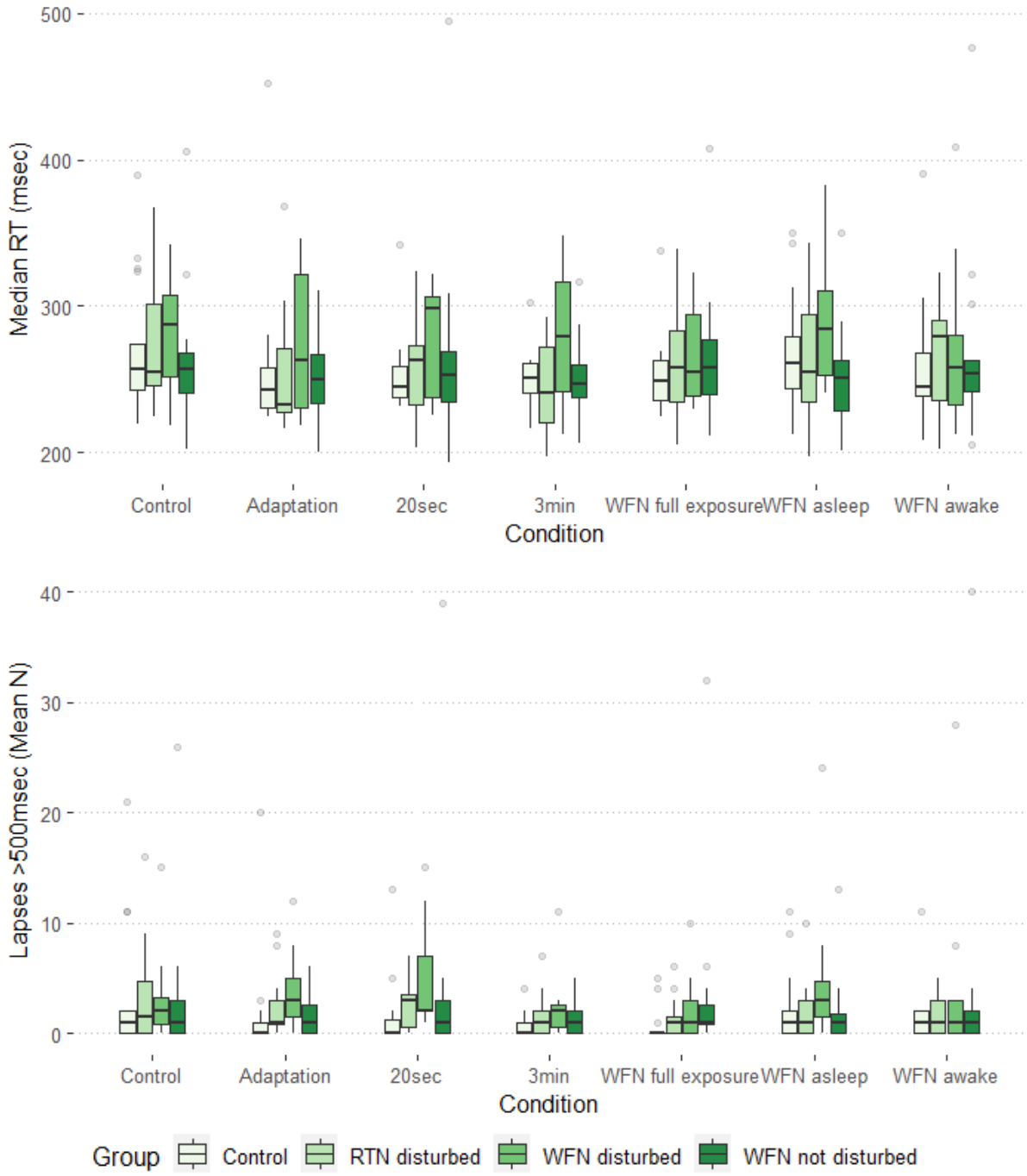


Figure 4.5 Box and whisker plot of reaction time variables across nightly condition (x-axis) and between groups (legend). The box bounds the IQR divided by the median. Whiskers are Tukey-style (extended to a maximum 1.5 x IQR outside of the box). Note. Total reaction errors refers to error of commission (false starts).

4.5.7.3 Next Day Processing Speed (Digit Symbol Substitution)

Overall condition effects further revealed that mean values correctly substituted were

significantly lower following the 20-second and 3-minute noise exposure nights compared to control (p=0.002, p=0.010 respectively) (See Figure 4.6).

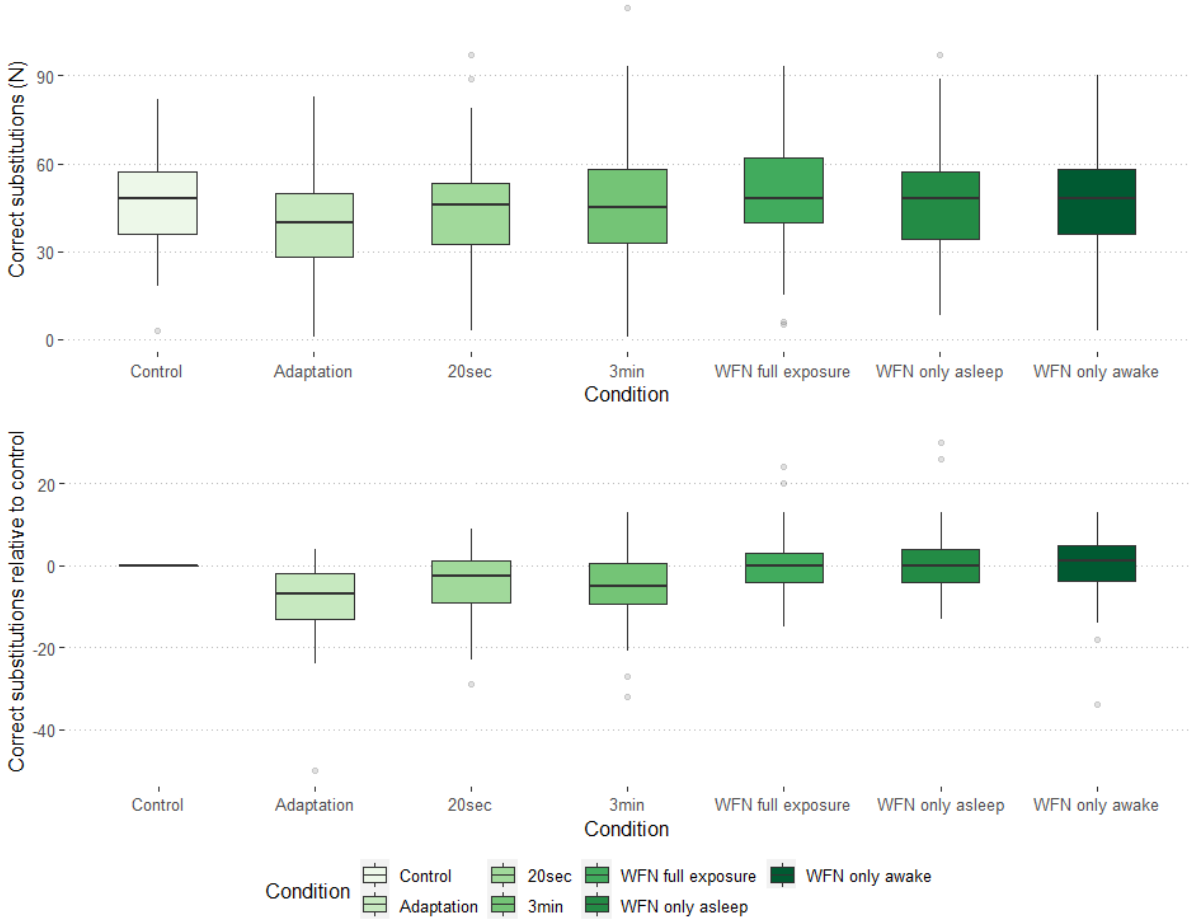


Figure 4.6 (Top) Box and whisker plot of correctly substituted values in the digit symbol substitution task across nightly condition (x-axis), (Bottom) Box and whisker plot of difference scores for correctly substituted values relative to the control condition.

4.5.7.4 Immediate Next Day and Extended Working Memory (Digit Span)

There were no significant group by condition interactions on any digit span recall variables including maximum length of digits recalled (backwards: χ^2 =13.79, df=18, p=0.743, or forwards: χ^2 =13.072, df=18, p=0.787) or mean span of digits recalled (backwards: χ^2 =8.56, df=18, p=0.969, or forwards: χ^2 =14.80, df=18, p=0.676).

However significant overall effects of condition in backwards and forwards length of digits recalled, and mean span of digits recalled were observed (backwards maximum length

χ^2 =53.58, df=6, p<0.001; backwards mean span χ^2 =66.80, df=6, p<0.001; forwards maximum length, χ^2 =59.64, df=6, p<0.001; forwards mean digit span; χ^2 =54.91, df=6, p<0.001).

Beyond first night effects already discussed, Figure 4.7 depicts the differences across nightly conditions. Pairwise comparisons revealed significantly lower backward digit maximum length (p=0.007) and mean Digit Span (p=0.033) and forward maximum digit length (p=0.020) and mean Digit Span (p=0.035) was achieved post overnight exposure to the 3 minute exposure condition compared to the control condition.

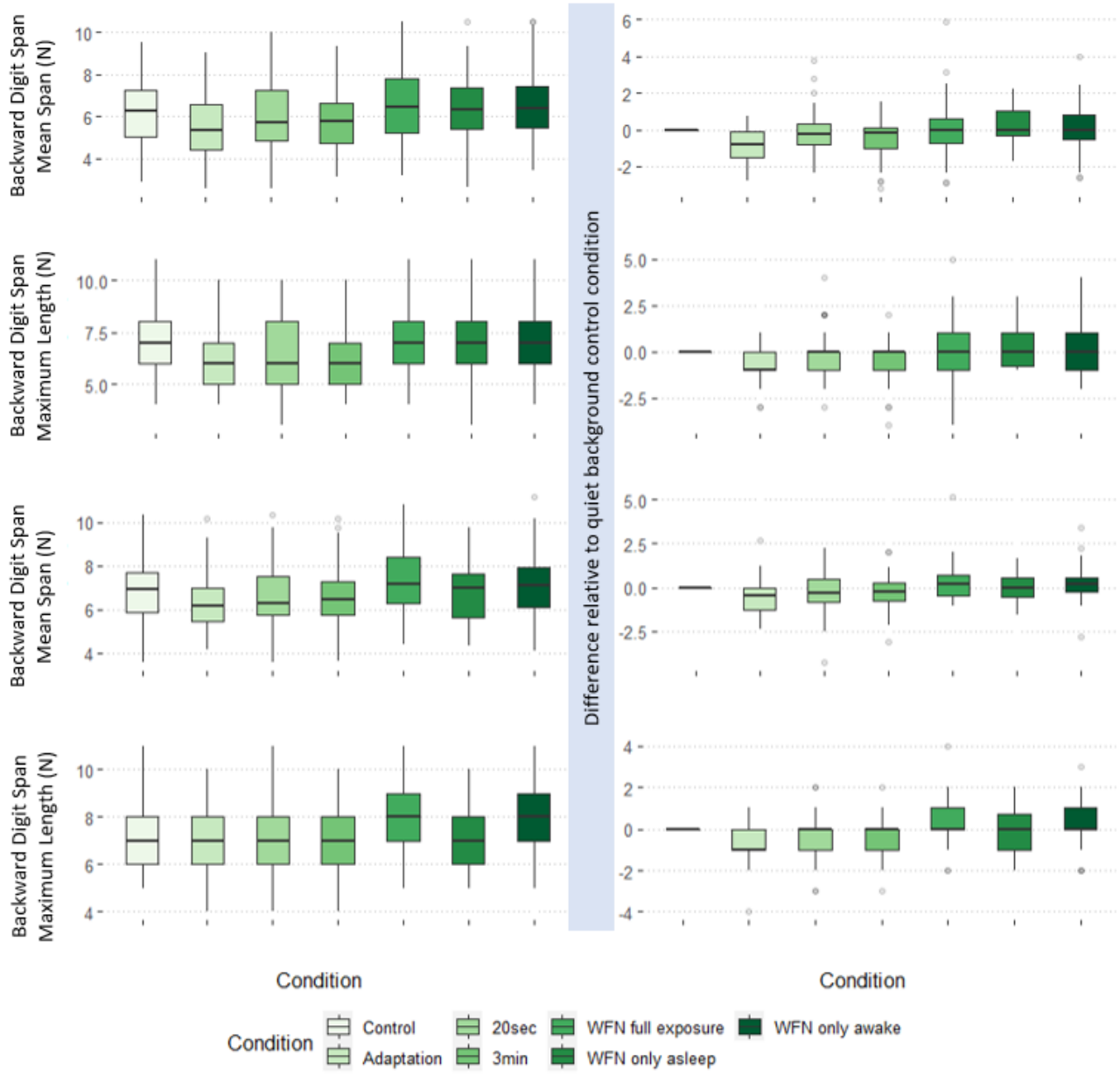


Figure 4.7 Box and whisker plot of; (RIGHT) digit span backwards and forwards recall, maximum length (ML) of recall and mean span (MS) of recall across nightly conditions (x-axis), (LEFT) relative difference scores in relation to the control night.

Figure 4.8 depicts the significant overall effects of group for backwards maximum length (ML) and mean span (MS) of digits recalled (χ^2 =16.90, df=3, p=0.001, χ^2 =17.89, df=3, p=0.001 respectively). This shows that the wind farm sleep disturbed group demonstrated significantly lower mean backwards recall span (p=0.002) and lower maximum

length of digits recalled (p=0.017) compared to the rural control group.

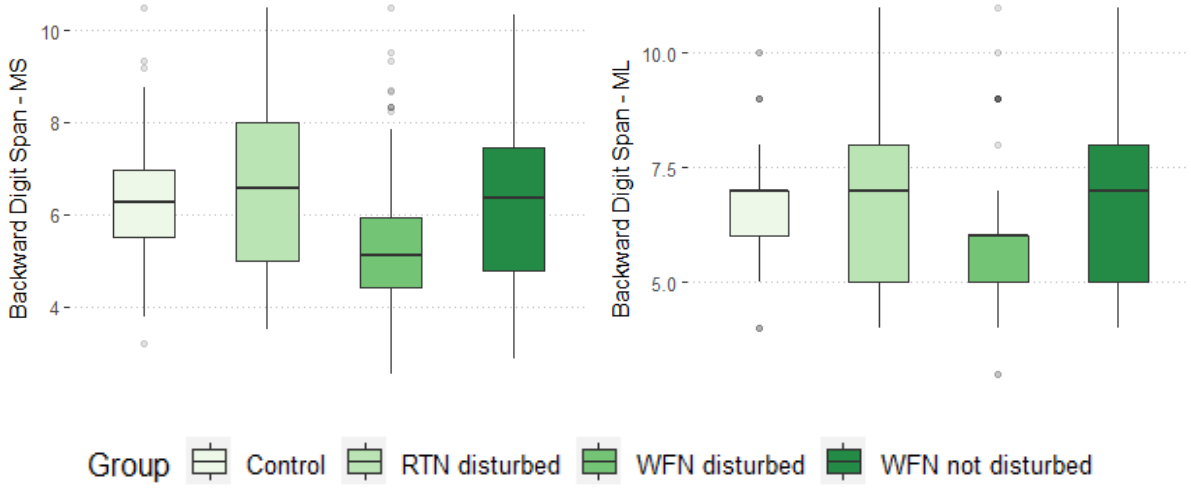


Figure 4.8 Box and whisker plot of digit span backwards maximum length of recall and mean span of recall across groups (x-axis).

4.5.7.5 Correlating Daytime Processing Speed with Sleep Variables

Figure 4.9 shows the strength of correlations between daytime performance outcomes and whole night sleep metrics, including traditional and qEEG measures that showed significant differences from the control condition during noise disturbed sleep. K-complex density and N3 sleep duration were positively correlated with DSST performance. However, other metrics were not.

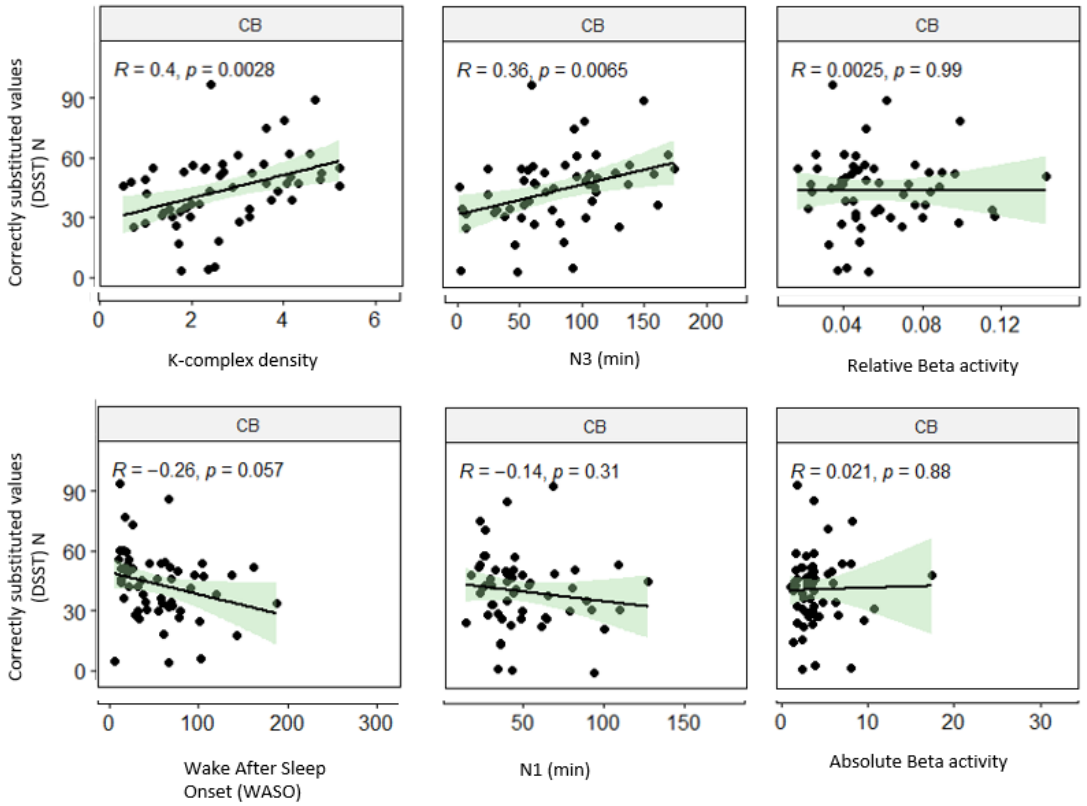


Figure 4.9 Correlations between significant whole night sleep metrics and daytime processing speed assessed by the values correct on the DSST.

4.6 Discussion

This study tested for differences in whole night EEG spectral power, K-complex density and traditional sleep metrics with a quiet control night compared to intermittent and continuous noise exposure conditions in a controlled laboratory environment. Given the sensitivity of qEEG to detect short noise exposure effects on EEG reported earlier in this thesis (**Chapter 2** (Dunbar et al., 2021)), it was anticipated that whole night qEEG metrics may be a useful tool for detecting and evaluating overnight noise exposure effects on sleep.It was hypothesised that qEEG outcomes would be more sensitive to sleep disruption from overnight environmental noise exposure than traditional sleep macrostructure metrics. This hypothesis was partially supported in that beta activity and K-complex density were elevated

on both the 3 minute and 20 second intermittent noise exposure nights compared to the control night. K-complexes are known to be frequently elicited by noise, and particularly with noise onset events (Lechat et al., 2021; Niiyama et al., 1995) so increased K-complexes density supports the potential utility of this EEG assessment approach for detecting noise-related sleep disturbance. However, other potential non-noise related sources of sleep disturbance also need consideration, particularly given that absolute beta and theta activity as well as K-complex density in the present study also appeared to be sensitive to first night effects.

Spectral entropy of slow wave activity, a novel marker of sleep disturbance previously shown to be associated with all-cause mortality (Lechat, Hansen, et al., 2022), showed no significant differences between nights in this study. Therefore, spectral entropy does not appear to be sensitive to noise exposure conditions examined in this study. Similar to other EEG measures this could reflect that many full-night summary metrics are insensitive to transient EEG features that may be more reflective of noise disruption effects, such as arousals and K-complexes. On the other hand spectral entropy was designed as a marker though likely to be sensitive to fragmented sleep (Lechat, Hansen, et al., 2022). Thus, we hypothesised that spectral entropy would be more sensitive to noise induced sleep disturbance than traditional sleep scoring. This hypothesis was not supported indicating that spectral entropy is not sensitive to noise exposures used in this study.

Overall, absolute and relative qEEG measures were also largely insensitive to noise exposure conditions. Whilst there were some statistically significant effects, the magnitude of noise condition effects were very small and highly unlikely to be useful for detecting noise-related sleep disruption. Previous work presented in **Chapter 2 and 3** of this thesis showed that qEEG is sensitive to noise exposure effects when evaluated using time series analysis time-matched relative to noise onset. This approach clearly showed acute qEEG changes

occurring immediately after stimulus onset and lasting for approximately five seconds, consistent with a K-complex response and infrequent noise-induced arousals. Increased K-complex density with intermittent noise exposures across the night in this study is also consistent with the noise-onset induced sleep disruption. Other research has used spectral power analysis to detect sleep disruption from sleep apnoea and daytime sleepiness in drowsy drivers (D'Rozario et al., 2017; Gibbings et al., 2022). In combination, these findings support more strategic use of qEEG analysis temporally aligned, where possible, to sensory stimuli with sleep disruption potential, including noise and overnight breathing disturbance events. However, the finding that K-complex density appears to retain sensitivity to intermittent noise exposure, even when evaluated across full night recordings without temporal alignment to noise exposure events, supports that K-complexes may be a particularly useful EEG feature for evaluating noise disrupted sleep.

Clear sleep stage effects also support that qEEG is sensitive to frequency shift between sleep stages. Although this is an expected outcome, it supports that largely automated EEG analysis, in combination with other EEG metrics, such as K-complexes, particularly when evaluated in combination with acoustic recordings, could be a particularly useful systematic approach for evaluating noise exposure effects on human sleep in future studies.

In traditional sleep metrics, there were significant first night effects on the adaptation night across multiple variables, including increased REM latency and duration, increased WASO, and reduced N3 sleep duration. First night effects were also apparent in several qEEG outcomes and in K-complex density. Poorer sleep with instrumentation for sleep study measurements on the first night in an unfamiliar laboratory is consistent with previous research (Agnew Jr et al., 1966). However, there were also evidence of more disrupted sleep with 20-second and 3-minute noise exposure conditions compared to control that were not

evident on other nights. Noise exposures on the 20-second night reached up to 50 dBA and are likely to have caused transient EEG changes, as previously demonstrated (Dunbar et al., 2021). Confirmatory EEG analyses temporally aligned to noise events was beyond the scope of this study, so this remains to be demonstrated in future analyses. However, it also appears likely that relatively short transient EEG changes associated with noise onset events may not reliably be detectable in whole night analyses time weighted to much longer non-transient features.

On the other hand, the 20-second intermittent noise exposure condition with an average whole night SPL of ~42 dBA [ranging 30-50 dBA] was associated with increased WASO and time spent in N1, and reduced N3 sleep duration, relative to the quiet control background night. This whole night averaged noise level was well above the World Health Organisation recommended guidelines for *outdoor* noise levels (45 dBA) (Basner & McGuire, 2018a; World Health Organization, 2018), which would be expected to translate to an indoor SPL in the order of 30-35 dBA given a 10-15 dBA noise attenuation from outdoor-to-indoor noise levels for open windows (Hansen et al., 2015; World Health Organization, 2018). On the 3-minute intermittent exposure night, where the average whole night SPL was around 32dBA, N3 sleep duration and K-complex density were both increased relative to control, much closer to WHO noise guideline recommended indoor noise limits for mid to high frequency noise exposure (Hurtley, 2009), given outdoor-to-indoor noise attenuation. Whilst there were some significant first night effects and EEG changes associated with intermittent noise exposure, there was no evidence to support that WFN presented at 25dBA across the whole sleep period, or restricted to wake or sleep periods alone, has any discernible effect on sleep macro structure, qEEG outcomes, K-complex density or next day impairments. This is in line with majority of research in the field which has found small or no evidence of wind farm noise impacts on sleep (Jalali, Bigelow, et al., 2016; Smith et al.,

2020). The only study to the authors knowledge that examined first night effects also found similar first night effects (Liebich, Lack, Hansen, et al., 2022). However, most studies simply remove the first night from analysis to avoid potential first night effects without testing for them. This clearly does not rule out effects at higher noise levels, particularly given evidence of sleep disturbance with intermittent noise exposures at higher sound pressure levels. However, 25 dBA WFN exposure is similar to average noise levels recorded in real-world exposure levels so the absence of significant effects at 25 dBA, and findings of only small differences with intermittent noise exposures at substantially higher levels, suggests that sleep impacts are relatively small.

Whilst there were some statistically significant differences in macrostructural and qEEG outcomes between nights, beyond first-night effects, the magnitude of further between night differences were relatively small, such as a ~20 minute increase in WASO on the 20-sec noise exposure night compared to control, still below the 60 minutes which is classically considered as meeting criteria for maintenance insomnia for single night data (Lineberger et al., 2006). It remains unclear if this effect may be clinically meaningful in the context of chronic noise exposure. Overall sleep efficiency also remained relatively high across all noise exposure conditions (>85%) although was close to the generally accepted normal cut-off of 85% (Buysse et al., 1989). Only the 20 second noise exposure night showed consistent evidence of increased WASO, beta activity and next day performance impairment in short-term memory as assessed using the DSST. These findings are consistent with prior studies supporting that intermittent noise disrupted sleep causes some next day impairments in performance (Carter, 1996; Martin et al., 1997).

Several sleep variables which showed differences between conditions, particularly on the 20-sec noise exposure night also showed significant correlations with next day cognitive processing speed. These results support that variables such as K-complex may be particularly

useful markers of noise disrupted sleep and next day functional impacts.

The lack of between group differences in sleep related outcomes was not anticipated as the WFN sensitive group was expected to show greater noise-disturbance responses. These findings most likely reflect the lack of clearly discernible noise-disruption effects with the majority of noise conditions utilized in this study. Thus, despite groups with prior habitual exposure the exposures used in this study did not appear to substantially impact sleep compared to the control night. Thus, higher noise levels may well be required to detect or rule out potential between-group effects. The highest SPL on the 20 second and 3 minute exposure nights was 50 dBA, which is well above the recommended outdoor noise limit of 45dBA. These were transient events, so the time-weighted overall overnight exposure SPL was more modest, but still relatively high compared to recommended outdoor nighttime noise limits. In this context, more substantial noise exposure effects were anticipated for which between-group effects were also expected. Overall, the lack of clearly discernible sleep disruption effects between nights or groups does not support that overnight noise exposures at levels relevant to recommended noise guidelines has any marked effects on sleep.

4.6.1 Study Limitations

This study utilised a six-night protocol of noise exposure conditions block randomised in two stages following an adaptation night. This approach was designed to control for potential order and carry-over effects from one night to the next, and to help ensure that the primary dose-response study nights (20 second and 3-minute intermittent noise exposures) were randomised and completed together prior to potential study withdrawals. Nevertheless, the potential for carryover and order effects remains.

Given the majority of statistically significant differences between noise condition nights were relatively small, study power warrants specific consideration. By study design, the primary comparisons between noise condition nights were conducted within-subjects,

with substantially greater power to detect noise condition effects than for secondary analyses involving between group comparisons. The overall sample was clearly sufficient to detect a range of small first-night effects, supporting that similar magnitude effects between other study nights should also have been detected. However, the study may well have been underpowered to detect between group effects, which may also have been confounded to some extent by a significant age difference between groups. Some of the significant findings could be spurious Type 1 errors. Mixed model analyses applied Bonferroni corrections for all relevant multiple pairwise comparisons to ensure the Type 1 error risk remained at 0.05 for each outcome. However, further adjustments across independent variables was not conducted and would not normally be recommended for independent secondary exploratory outcome variables. These exploratory outcomes clearly warrant cautious interpretation, particularly given small effect sizes.

Noise exposure levels for the study nights ranged from no additional noise (19 dBA background noise) on the adaptation and control nights to 20 second discrete noise events up to a maximum of 50 dBA which, with substantial noise repetition, produced an overnight average noise exposure level of around 42 dBA. Given outdoor-to-indoor noise attenuation effects, this level of noise exposure is above WHO recommended average overnight outdoor night noise levels of 45 dBA (World Health Organization, 2018). Thus, a degree of sleep disruption is expected and consistent with extensive literature from which WHO guidelines were derived. Previous studies of traffic noise exposure with overnight noise levels ranging from 50-70 dBA found higher likelihood to awaken with increasing sound pressure level. Research also shows a higher likelihood of awakenings to noise events with more high frequency components than those with high frequencies removed, when presented at the same comparative SPL (Elmenhorst et al., 2019; Griefahn et al., 2006). Small effects on conventional and qEEG markers determined over the whole sleep period with relatively low

noise exposure levels in the current study are consistent with these studies and support that higher overnight exposure levels are more likely to be problematic.

Onset and offset rise times of the intermittent noise exposure nights were short (250ms) and thus represented sudden noise onset events which clearly may not be representative of more natural noise exposures in real-world environments where both transient noise events (e.g. from wind gusts or direction changes) and slow noise transitions from slow changes in environmental factors would be expected. A more comprehensive within night analysis on these study nights, similar to that applied in Chapter's 2 and 3 but beyond the time and scope of this study, clearly remains warranted to test for changes in EEG spectral power immediately following noise onset compared to later during ongoing noise exposure events. Based on prior work described in Chapter 2 and 3, it appears most likely that abrupt noise onset is the most sleep disrupting component of noise exposure, such that frequently changing noise environments, particularly those with high sound pressure level noise events, are most likely to be sleep disruptive.

The use of a 3-minute stimulus on repeat throughout the night was chosen to represent near worst case scenario exposures to a combination of wind farm and road traffic noise. Stimuli were selected to ensure noise samples contained acoustic features representative of those considered most relevant and likely to be disruptive to sleep, which for wind farm noise specifically included prominent amplitude modulation, but also included infrasound. Three-minute samples were chosen to enable assessment of acute noise onset effects and more sustained exposures over several minutes. Three minutes also allowed for substantial noise sample replication a across the nights. Wind farm noise samples were selected from field recordings from which we were able to subsequently determine AM was close to worst case AM exposures during year-long measurements in the field. Clearly 3-minute noise samples are not representative of full-night WFN exposures, which future studies should consider.

However, the advantage of short controlled exposures during established sleep is much greater control over multiple variables that may confound responses. Within night habituation effects were not examined in the current study, but warrant further investigation given some evidence of habituation in vasoconstrictor responses to repeated overnight exposure to short noise events (Zajamsek, Micic, et al., 2019).

Power spectral analysis of EEG in the current study was averaged across the entire night to compare with traditional sleep metrics. This may well have masked transient EEG changes associated with noise onset events through time-weighted averaging towards undisturbed EEG. Nevertheless, the primary purpose of this study was to specifically examine if whole night qEEG metrics are more sensitive to overnight noise exposure compared to traditional macro-sleep metrics, without necessarily additional and more complex analyses temporally aligned to noise recordings. Within-night qEEG analyses temporally aligned to noise could potentially be automated but may not be practical for clinical and basic research settings or field measurements. Had whole night qEEG markers been shown to be sensitive to different noise exposure conditions, sleep disruption assessment from qEEG alone would have been an attractive and pragmatic approach. However, given the absence of clear differences in overall qEEG measures between nights, with no clear advantages over more traditional sleep scoring methods, it appears that qEEG is likely to be much more useful when used in combination with acoustic recordings to more specifically examine noise related impacts on EEG measures. A notable exception was K-complex density, which was significantly elevated on both intermittent noise exposure nights compared to control, supporting the potential utility of this approach for assessing intermittent noise disrupted sleep.

Age differed between the study groups, which is likely to have been problematic for comparisons of noise effects between groups given well established effects of aging on sleep

(Feinberg, 1974; Landolt & Borbély, 2001). Age matching between groups is inherently difficult, particularly for a study of this type where age demographic differences could exist between different noise exposure regions, such as in regional areas exposed to wind farm noise compared to urban traffic noise exposure and quiet rural areas. Age adjustments were examined in statistical analyses but were removed to simplify the model to key terms of interest, and age adjustment alone may not be adequate to control for age as a potential confounder.

Furthermore, the overall self-reported WFN related sleep disruption in the WFN disturbed group was rated to be moderate, so it is possible that noise disrupted individuals were under-represented. By study design the intention was to capture individuals living within 10 km of wind turbines who reported sleep disruption. However, this sample proved to be particularly difficult to recruit, likely reflecting the limited number of residents living within 10km of wind turbines who could be contacted and were also willing to be involved.

The WFN sleep disturbed group self-reported distance to the nearest wind farm (2-4km) was closer than for the WFN non-sleep disturbed group (4-6km). This may well help to explain differences in self-reported noise disturbances given that distance from a noise source plays a key role in determining noise exposure levels and features. However, determination of causation is inherently difficult in the field as many other factors could potentially also play a role. However, there were also relatively few between group differences of note in this study. Future studies would clearly benefit from direct measurements of noise exposure levels across groups, acknowledging that noise features and levels are amongst many factors that could contribute to differences in responses between groups.

Further recruitment challenges included travel distances, individual's ability to leave rural properties unattended for travel to participate in a 7-day laboratory protocol, and COVID-19 related travel restrictions. The COVID-19 pandemic itself could potentially also

influenced study findings to some extent, although the primary analyses were within-subject comparisons between nights over a 7-day period for which major confounding from pandemic effects appear unlikely.

4.6.2 Further Research Areas

Future research directions suggested by this work support that EEG spectral power analysis is likely to be most useful to evaluate transient effects of specific noise events. Thus, a similar analysis as was applied in Chapter 2 and 3 may be particularly useful.

Given the findings of increased K-complex density and some changes in sleep macrostructure with intermittent noise exposure, further studies using K-complex analyses to examine potential relationships with next-day cognitive and physical functioning and mood may also be useful. Recent evidence also supports that K-complexes are sleep protective (Parrino & Vaudano, 2018). Given the lack of changes in arousals in this study, K-complex density could potentially be a more sensitive and useful marker of sleep disruption for which next day functional impacts would likely be useful to explore. Our study further showed that increased K-complex density on high noise exposure nights was positively associated with increased DSST which potentially supports that K-complexes may be sleep protective in the presence of intermittent noise exposure events (Wauquier et al., 1995). This is in line with previous research in the field which outlines the potential for K-complexes to promote thalamocortical gating of sensory neuronal traffic to help protect sleep (Colrain, 2005).

Next-day symptomology reported to be associated with noise induced sleep disruption and daytime subjective sleepiness showed no evidence of prior night noise impacts and were on average low (See Appendix E). It is possible that symptoms and complaints associated with noise disrupted sleep may not be adequately captured with the visual analogue scales used in this and other studies. Thus, further qualitative work could potentially be useful towards more comprehensive assessments of next-day impacts.

4.7 Conclusion

This study does not support that qEEG analyses applied to whole night recordings are sufficiently sensitive to detect differences in overnight noise exposure conditions to warrant their use over traditional sleep measures.

However, K-complex density was sensitive to intermittent noise exposure during sleep and may be a useful tool for future research into sleep disruption. Furthermore, intermittent whole night averaged noise levels at ~42 dBA appears to show mild sleep disruption and next day impairment relative to quiet control night at 19 dBA, while continuous noise levels around 30-35 dBA showed little to no effect on overall sleep macrostructure. Although qEEG outcomes determined as averages across full-night sleep recordings did not appear to be any more sensitive to noise exposure effects than conventional sleep macrostructural parameters, this does not preclude that qEEG changes may nevertheless be discernible to discrete noise events, such as was shown in the work presented in Chapter 2. Thus, further research remains warranted to test if qEEG effects are discernible over the course of discrete noise exposure events that may have been masked by averaging across the whole night of noise exposure. However, since the only apparent effects on sleep EEG were very briefly at the onset of bursts of WFN or RTN observed in Chapter 2, and that this is not the typical way in which these noise sources occur in the environment, the present study found no objective evidence of sleep EEG disruption from continuous WFN presentations at least at the SPL of 25 dBA for the WFN in this study. Since this SPL is typical of WFN from wind turbines at a distance of approximately 2-3km further research using higher SPL values could potentially find sleep EEG effects and thus provide more definitive guidelines for wind turbine placement distances to residences.

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CHAPTER 5 : GENERAL DISCUSSION AND CONCLUSION

5.1 Discussion of Thesis Aims and Chapter Findings.

Broadly, the aim of this thesis was to use a sensitive and objective measure of EEG activity to investigate changes in sleep under nocturnal noise exposure including traffic noise and wind farm noise. In addition to conventional macrostructural EEG sleep measures the thesis examined sleep using quantitative EEG assessment. Understanding possible sleep EEG micro architecture changes with WFN exposure was hypothesized to help explain the disparity between previous research in the area of low frequency dominated noise exposure to wind farm noise. A greater understanding of the extent of hypothesized noise related sleep disruption could help to guide appropriate recommendations for affected individuals, such as decision making around relevant supports for affected individuals including psychological support interventions and noise mitigation strategies. The following Chapter summarizes the primary findings from Chapters 2-4 and the implications these have for further study in the area of sleep related noise exposure.

5.2 Chapter Summaries and the Original Contribution to Knowledge

Chapter 1 introduced nocturnal noise exposure, sleep and current methodologies for sleep assessment. This chapter outlined the theoretical framework supporting the need for sensitive assessments of sleep during wind farm noise exposure. Previous studies had already shown that mid to high frequency noise induced sleep disruption can be measured using traditional sleep measurements (Basner et al., 2010; Basner et al., 2011; Eberhardt et al., 1987; Griefahn et al., 2008; Griefahn et al., 2006; Tonin et al., 2017). However, recent studies showing no discernible sleep related disturbance to wind farm noise, which is dominated by low frequency noise at relatively low levels, raises the possibility that conventional sleep assessment methods may not be sufficiently sensitive to capture potentially more subtle EEG

features of noise-induced sleep disturbance (Liebich, Lack, Hansen, et al., 2022). This concept is supported by previous studies showing that noise-induced sleep microstructural disturbances in the EEG not captured via traditional sleep staging and scoring cause measurable physiological disturbances and next day impacts on mood (Basner et al., 2008; Martin et al., 1997). Therefore, more sensitive measures of EEG may be required to more definitively determine if wind farm noise exposure disrupts sleep compared to road traffic noise (RTN), a better understood source of noise-related sleep disruption.

In Chapter 2, dose response relationships between noise exposure levels during established N2, N3 sleep with quantitative EEG measures, in relevant sleep related EEG frequency bands, was examined in response to 3-minute exposures to RTN and WFN in 23 healthy young sleepers. The primary finding was a clear dose response relationship, where increasing sound pressure levels resulted in increased EEG high frequency activity and reduced low frequency activity following noise event onsets. These findings are largely consistent with previous literature (Basner et al., 2008). However, there were some subtle differences between noise types at low noise exposure levels (33dBA) where alpha activity was increased during acute exposure to WFN relative to RTN. These findings could potentially reflect Type 1 error due to the relatively small number of noise samples acquired across the night. Noise effects were also short-lasting, with EEG frequency shifts returning to baseline levels after 30 seconds of noise exposure. This study used healthy young individuals who may not have been sufficiently representative of rural and remote residents more typically exposed to wind farm noise. Thus, differences in hearing acuity, overall sleep structure and sensitivity to noise remains areas warranting further consideration and examination.

Chapter 3 utilized the same study sample as Chapter 2 and specifically examined the effect of exposure to relatively high intensity infrasound at 80dBG on quantitative EEG measures. Infrasound, a term used to describe sound below 20 Hz, is typically not audible to humans. Nevertheless, wind farm infrasound has been implicated as a factor potentially contributing to negative impacts on humans, including sleep disturbance. Thus, this pilot study was designed to establish experimental and analytical methods needed to specifically test for sleep disruption effects of infrasound. Pre-recorded wind farm noise containing infrasound was filtered to remove frequencies >20 Hz and was reproduced at 80dBG, which remains below the human hearing threshold of 85dBG <20 Hz, but has a substantially greater SPL than infrasound levels of 60-70 dBG typically recorded from wind farms at nearby residences. When 3-minute exposures of infrasound were compared to control background noise exposure at 23dBA, infrasound was found to elicit an acute increase in delta activity corresponding to an increase in K-complex probability within the first 5 seconds of noise exposure. EEG changes then rapidly returned to pre-noise exposure levels and showed no lasting effects over the last 150 seconds of noise exposures. Although these results suggest that the sleeping brain can detect and respond to an infrasound stimulus, it was not possible to rule out an audible noise reproduction artefact, such as a click associated with large speaker cone travel, since independent noise recordings to help rule out extraneous noise events were unfortunately not conducted. Further research using in-room recordings to help confirm faithful infrasound reproduction and rule out artefact effects and with more ecologically representative 60-70dBG levels of WFN infrasound are warranted to determine if the EEG response observed in this study are replicable at more realistic noise exposure levels.

The work presented in Chapter 2 and 3 focused on establishing qEEG responses to RTN, a noise source well-known to have sleep disruptive effects, compared to much less well

understood WFN, which has more prominent low frequency characteristics including audible and sub-audible (Infrasound) components. When qEEG analyses were applied to discrete noise events, qEEG measures were a sensitive to noise-induced sleep disturbance effects not discernible with traditional sleep macrostructure outcomes which were not different between noise types. However, such detailed analyses require careful time-matching to known noise onset events, which would require detailed synchronous acoustic recordings rarely utilized and unlikely to be practical in real-world noise exposure settings. Therefore, further research was needed to determine if qEEG analysis applied to whole night sleep recordings are sufficiently sensitive to detect the presence of noise-induced sleep disruption effects.

In a larger separate study, involving a seven-night laboratory protocol, the work presented in Chapter 4 tested if qEEG measures are more sensitive than traditional sleep metrics to the presence versus absence of overnight noise exposure. Following an acclimation night, a range of noise conditions were examined. These included intermittent noise exposure nights with RTN and WFN (20 second noise battery with an average whole night SPL of 42dBA and a 3-minute noise battery with an average SPL of 32dBA), a full-night of continuous WFN exposure (at 25dBA similar to average WFN levels recorded in field studies), the same WFN stimulus (25 dBA) applied only during established sleep, only during wake, and a quiet night without noise exposure (background noise 19dBA). A secondary aim was to compare responses between four different groups of individuals with different prior histories of noise exposure. These included two groups of individuals living within 10 km of a windfarm, one group with and the other without WFN related sleep disturbance, a group of urban residents reporting RTN related sleep disturbance, and a control group of rural residents without prior WFN exposure. This was one of the largest controlled laboratory experiments to date to have examined WFN compared to RTN effects on direct

measurements of sleep, and the first to comprehensively evaluate noise effects on qEEG compared to traditional sleep macrostructure markers of sleep disturbance. Despite qEEG showing significant promise as a sensitive marker of acute EEG responses to noise onset, in Chapters 2 and 3, qEEG analysis applied across full night recordings was no more sensitive than traditional sleep metrics at detecting EEG differences between different noise condition nights. Nevertheless, there were small but statistically significant main effects of condition, with increased beta activity, WASO and time spent in N1 sleep for the 20 second noise battery night compared to the quiet control condition. However, these effects were relatively small changes, and relatively high sleep efficiency (>85%) was maintained across all study nights so overall impacts on sleep appeared to be minimal. Increased K-complex density on intermittent noise exposure compared to the control night, supports that K-complexes may be a particularly sensitive and useful marker of transient noise event disrupted sleep.

Thus, this study did not support that intermittent noise exposures averaging ~32 to ~42dBA, or continuous WFN exposure at 25 dBA presented all night or only during sleep or only awake substantially disrupted conventional measures of sleep macrostructure or potentially more sensitive qEEG microstructural markers of sleep disruption compared to background noise. Whilst some measures in traditional (WASO) and new metrics (qEEG and K-complex density) showed differences between noise exposure conditions these were predominantly small effects. Increased WASO was predominantly seen in the averaged ~42dBA intermittent noise exposure night compared to control and is consistent with previous research and current nighttime allowable noise limits (Basner & McGuire, 2018a). Although qEEG analysis applied to full-night recordings did not appear to be more sensitive to noise effects on sleep than conventional sleep macrostructural measures, this likely reflects effects of averaging EEG frequencies across the whole night, which likely masks more acute

and transient effects of noise, such as increased K-complex density effects. Future research should focus more specifically on the application of qEEG analyses time-locked to noise stimulus onset, such as was applied in Chapters 2 and 3, which appears likely to be needed to help clarify potential differences in WFN compared to RTN on sleep in this larger sample. Although originally planned, this more detailed analysis was beyond the scope and time available for this thesis work.

5.3 Theoretical Implications: Does WFN Impact Sleep More Than RTN?

Together, the overall results of this thesis work do not support that WFN disturbs sleep to a greater extent than RTN. Sleep disturbance appears to be primarily sound pressure level dependent such that louder and intermittent rather than continuous noise exposure appears to cause more disruption to sleep regardless of noise type. These findings are in line with other research in the field (Brink et al., 2019; Carter, 1996; Eberhardt et al., 1987). Carter (1996) describes in their review of the literature that slow wave sleep is particularly reduced during exposure to traffic noise sources with intermittent components (i.e., the passing individual trucks and cars causing noise peaks at 55dB $L_{Amax,}$ or low flying aircraft noise events). A study by Brink et al. (2019) further supports that intermittent noise is disruptive and may increase noise annoyance ratings and led Brink et al. (2019) to develop the intermittency ratio. Noise intermittency could potentially help to explain the differences between our extended 3 minute exposure and shorter 20 second exposure conditions which align with differences in response to highly intermittent exposures (Wunderli et al., 2016). We did not determine noise intermittency measures, but this would likely be useful in future studies and analyses to help further examine relationships between noise exposure features and sleep and next day disruption outcomes. Comparatively continuous traffic noise was not described to be detrimental to slow wave sleep. Therefore previous research suggests the relevance of considering the maximum volume of discrete noise events which may disrupt

sleep (Eberhardt et al., 1987). Some findings in Chapter 2 suggest that exposure to short sudden onset WFN at 33 dBA may cause increased alpha activity comparative to RTN at the same SPL. This result was not observed at 43 dBA and supports the need for a similar qEEG analysis of the work presented in Chapter 4 to more specifically test for differences between RTN compared to WFN at indoor levels both above and below those recommended for night-time noise limits in this larger sample, which also included individuals impacted by WFN exposure. The effect of noise exposure on sleep seen in levels above 30 dBA in Chapters 2 and 4 is in line with previous studies of environmental noise exposure on sleep (Elmenhorst et al., 2019; Pirrera et al., 2010). These studies show clear sound pressure level dependent effects on sleep disruption, such that sufficiently loud noise of any type is likely to disturb sleep. Thus, it is important to clarify if WFN exposure has greater effects on sleep than other noise types such as RTN, and if so, what levels might be considered equivalent to RTN effects and thus potentially an acceptable level of sleep disruption for individuals impacted within the community. The original intent was to apply a time-series analysis as in Chapter 2 to directly compare WFN versus RTN effects on qEEG outcomes in a larger study sample, including WFN impacted members of the community. However, this was not possible within the time-constraints of this PhD work, so the analysis presented is somewhat limited and not sufficient to fully address the original study aims. Nevertheless, if qEEG outcomes from full-night analysis had shown differences between different noise conditions nights, this would have supported the value of qEEG analysis for helping to quantify noise-disrupted sleep.

In Chapter 4, no significant differences were found between WFN presented at 25dBA compared to background control noise at 19dBA, supporting that WFN at 25dBA, similar to average noise levels in year-long recordings in the field (Nguyen et al., 2021), does not measurably alter sleep qEEG or conventional sleep macrostructure parameters. This is an important finding and is consistent with other research in the field (Jalali, Bigelow, et al.,

2016; Liebich, Lack, Hansen, et al., 2022). However, the work presented in Chapter 4 also demonstrated that intermittent noise exposures resulting in average whole night sound pressure levels of ~42dBA produced significantly less N3 sleep, more time spent awake across the night, increased beta EEG activity and reduced total sleep time. These results support the presence of sleep disruption at sound pressure levels greater than 25dBA. However, the observations that the few EEG sleep disruptions that did occur at higher sound pressure levels were only transient, and did not extend beyond 30 seconds of abrupt noise stimulus onset, suggests that even if higher intensity WFN is presented, if it is continuous, it may show relatively minimal sleep disruption overall. However, this is a question needing further experimental research, including noise stimuli above relatively average WFN levels of 25 dBA more representative of worst-case WFN exposure scenarios.

The results of Chapter 4 clearly support that intermittent and variable noise types and sound pressure levels are more disruptive to sleep than low level continuous WFN. Due to the study design and research aims serving multiple study purposes, including several beyond the scope of this thesis, the 20 second and 3-minute noise exposure nights contained a combination of both WFN and RTN. However, there was no evidence of sleep disruption overall on the 3-minute noise exposure night, where the average whole night SPL was 32dBA, compared to the control of 19dBA. This supports that overall noise exposure of around 32dBA at night does not have a sufficiently high enough SPL to disrupt overall sleep macrostructure or EEG spectral power across whole night sleep EEG frequency bands relevant to sleep.

Whilst continuous WFN exposure at 25dBA was chosen to represent median levels observed in year-long WFN recordings in the field, it should be noted that WFN can vary considerably over the night according to variable wind, temperature and other environmental variables that influence noise generation and transmission. The current study utilized a 3-

minute loop of WFN at 25dBA that was selected as likely to be representative of near worst–case amplitude modulation and infrasound conditions. However, the sound pressure level of WFN in the field will vary depending on wind speed, proximity of the wind farm and direction of the wind in relation to residences. Thus, real-world WFN exposure is expected to be more variable with changing sound pressure levels, including occasional relatively sudden onset intermittent noise events when wind direction changes (Nissenbaum et al., 2012; Romero-Sanz & Matesanz, 2008). Sudden noise onset is clearly a potent stimulus for eliciting transient sleep disruption, particularly with higher SPLs. Further work, using a whole night WFN samples, more representative of worst-case WFN exposure conditions is warranted given consistent findings that intermittent noise transiently disrupts sleep, and thus appears likely to be more sleep disruptive than more extended but consistent noise exposure.

Given the findings of Chapter 2, which demonstrated the potential for WFN to increase alpha activity at 33dBA when compared to RTN at the same SPL, further research is needed to consider whether noise guidelines may warrant some adjustment for WFN.

5.4 Is qEEG a Sufficiently Sensitive Tool to Detect Sleep Disturbance to WFN and its Low Frequency Components?

A key aim of this thesis was to apply qEEG methods to explore the sleep disruption characteristics of overnight wind farm noise exposures in a carefully controlled laboratory setting. Chapters 2 and 3, showed that application of qEEG analysis time-locked to the onset of discrete noise events was able to detect subtle EEG frequency changes in response to nocturnal noise exposure to both audible WFN, RTN, and sub-audible WFN infrasound. These Chapters strongly support the utility of qEEG methods for a more detailed and objective evaluation of sleep disruption characteristics of noise exposures than is possible with traditional manual sleep scoring methods. qEEG was able to detect significant noise type and SPL differences in the absence of manually discernible arousal and awakening events or

sleep stage transitions. Thus, qEEG analyses may ultimately help to detect and explain subjective reports of WFN related sleep disruption for which traditionally scored sleep macrostructure parameters may not be sufficiently sensitive to noise-related sleep disruption.

In Chapter 4, qEEG analyses were applied to whole night recordings, which is a more practical analysis approach when synchronous acoustic measures may not be available or practical to record. However, this approach was largely unable to detect differences in the sleep EEG beyond those captured in more conventional sleep macrostructure analysis. Whilst a more detailed analysis of these data, similar to that applied in Chapters 2 and 3, clearly remains warranted, this appears likely to reflect that full-night analysis is insensitive to short-transient EEG changes associated with discrete noise events.

This thesis also examined sleep disruption effects of noise in several different study populations, including young healthy good sleepers, a group of urban residents with road traffic noise related sleep complaints, and groups more representative of those living nearby to wind farms, including groups with and without reports of WFN related sleep disruption. Chapters 2 and 3 studied responses in the young healthy good sleeper group. These chapters showed that qEEG analysis was sensitive to transient EEG changes following discrete noise onset events. Although qEEG analysis applied across full night recordings did not appear to be any more sensitive to noise effects than traditional manual sleep scoring methods, further analysis to replicate that of Chapters 2 and 3 remains warranted to more specifically test for qEEG differences between WFN and RTN in the more representative study samples studied in Chapter 4.

Together these results suggest that qEEG is clearly a sensitive and useful analysis tool to more specifically test for acute effects of noise events on sleep beyond conventional manual sleep scoring methods. However, application across full-night recordings without more careful consideration of variable acoustic effects appears to be of limited utility. Noise

events clearly can and do elicit transient changes in the EEG, predominantly according to sound pressure level above background noise. Thus, EEG analysis methods that are sensitive to transient events, such as EEG spectral changes and K-complex detection analysis are clearly useful approaches needed to test for potential differences between noise exposure types. Future work applying machine-learning methods could be particularly useful to systematically screen for EEG features most discriminatory of noise effects, and to identify noise features most disruptive to sleep.

5.5 Impact of the Current Work on World Health Organisation Endorsed Noise Guidelines.

This thesis examined the effect of RTN and WFN on sleep EEG. Chapter 2 suggested that WFN may cause increased alpha activity compared to RTN at 33dBA, however differences between noise types were not observed at 43dBA. These findings could indicate differential effects of low frequency dominant noise produced by WFN compared to RTN at lower but not at higher sound pressure levels. To help confirm these findings and rule out the potential for a Type I error (false positive inference), these findings warrant replication in a future analysis of data presented in Chapter 4, where full-night, but not more specific and likely sensitive noise-onset related qEEG analysis was employed. Although the work presented in Chapter 4 does not support that continuous WFN presented at 25dBA has any discernible effect on sleep macrostructure or microstructural parameters compared to quiet background noise control, this does not rule out noise-onset related effects, or effects at higher noise levels. On the other hand, transient noise onset effects may be relatively infrequent events in the context of real-world wind farm noise exposure. Furthermore, the absence of discernible effects at sound pressure levels similar to average levels in year-long recordings in the field suggest that wind farm noise impacts on sleep are relatively minimal, and are unlikely to be worse than those from other noise types at equivalent sound pressure

levels.

The results of Chapter 3 suggested discernible EEG responses to the onset of WFN infrasound reproduced at 80dBG, which is below the average human hearing threshold for infrasound. However, these pilot study data were collected without confirmatory noise recordings needed to confirm faithful noise reproduction to rule out potential audible noise onset effects. Nevertheless, even with discernible noise onset related effects, whole night sleep macrostructure parameters showed no evidence of significant sleep disturbance with conventional manual scoring of sleep EEG.

Whole night analysis of WFN reproduced at 25dBA also showed no impact on whole night sleep macrostructure compared to control. Daytime cognitive and reaction time functioning following 25dBA WFN exposure has been previously presented from the same sample and showed no differences from control (Liebich, Lack, Hansen, et al., 2022). The evidence presented in this thesis further supports no discernible effect of WFN at 25dBA on the investigated qEEG outcomes or any significant associations between qEEG outcomes and markers of next day functioning. Given the differences in ages across the group it is interesting that no differences were observed in reaction speed or cognitive performance. This could potentially reflect a highly motivated self-selected participant sample that may not be representative of the target sample. Type 2 error is also possible. Participants practiced the cognitive tasks on their acclimation morning in the laboratory and particular care was taken to assist in instructing participants unfamiliar with using computers to help ensure all participants could complete each task to the best of their ability.

Findings from full night exposure to WFN at ~31dBA previously studied in the field (Jalali, Bigelow, et al., 2016) showing some sleep microstructural changes but without discernible changes in sleep macrostructure largely concur with the results from this thesis. Given World Health Organization (WHO) (1999) (Berglund et al., 1999) recommendations

for indoor noise levels <30dBA for continuous noise to avoid sleep disruption, these results support that these recommendations may be appropriate even for low frequency noise produced by WFN. However, further work remains warranted to more specifically test for qEEG effects with WFN compared to RTN and other noise types at higher sound pressure levels at and above WHO recommended noise limits. Chapters 2, 3, and 4 all showed some evidence to support discernible EEG response to intermittent noise onset events. Thus, rapid changes in wind speed or direction may well be associated with transient sleep disruption events.

To help inform national guidelines, full night variable WFN to approximate noise levels and features experienced at residents in closer proximity to wind turbines should be conducted. Replication of the continuous WFN conditions of Chapter 4 but at higher moderate (30dBA) and more intense (35dBA) WFN levels more likely to approximate worst-case WFN exposure would be useful to help better inform guidelines for wind turbine placements with respect to adjacent residences. In regard to current noise guidelines the present work supports that the typical recommended outdoor noise level of 45dBA (Basner & McGuire, 2018b) based primarily on traffic noise exposure data, may be appropriate for residences exposed to nocturnal wind farm noise. However, this study was not specifically designed to test full night noise exposures at any specific noise exposure level, and was instead primarily focused on qEEG markers of noise-related sleep disruption, and predominantly to transient noise onset events. Thus, extrapolating these findings to noise guidelines is somewhat problematic.

5.6 Methodology and Process Limitations and Considerations

The studies conducted in this thesis are amongst the largest to date in the examination of WFN exposure on sleep, and among the first to directly compare RTN and WFN at variable

sound pressure levels. To the authors knowledge it is also among the first study to date to use qEEG analysis to examine the potential for WFN noise induced sleep disruption on a microstructure sleep level.

A major strength of this study was the comprehensive assessment of sleep, using novel and sensitive measures to examine the potential for subtle changes in EEG beyond traditional sleep assessment. All studies presented in this thesis were also conducted in carefully controlled laboratory conditions, where background noise was low and noise samples could be carefully controlled and faithfully reproduced. This study design also reduced the likelihood of extraneous noise from confounding noise interventions, overcoming a significant challenge of field based experiments where noise levels inevitably vary according to wind conditions and other variables that likely make noise-related sleep impacts difficult to detect and specifically attribute to noise (Jalali, Bigelow, et al., 2016). Nevertheless, field studies are also likely to be useful for establishing how noise exposure may affect sleep in the home environment, but are likely to require simultaneous acoustic recordings and appropriately designed analytical techniques to help separate acoustic from non-acoustic effects on sleep outcomes. The studies presented in this thesis provide a basis to further examine noise exposure in the field using sensitive measures of qEEG. As outlined in Chapter 4, qEEG requires sufficiently ‘clean’ EEG data to ensure high quality EEG measurements can be obtained. Reliable EEG data collection is more difficult outside of the laboratory environment where EEG can be carefully monitored overnight and electrodes replaced when signal quality is poor, such as with dropped electrodes. Further research is needed to determine the effectiveness of using qEEG on EEG data obtained in the field. Furthermore, specific analysis of within-night noise effects on the sleep EEG requires carefully matched time-synchronous EEG and noise recording data, which is technically quite difficult and more practical in a laboratory compared to field environment. These additional

barriers to field studies make the analysis of within-night EEG effects more difficult than in laboratory-based studies such as were used in this thesis.

A significant strength of qEEG is the objective nature of the analysis which largely avoids significant intra- and inter-rater differences with traditional sleep scoring, which requires trained individuals to allocate a stage of sleep to a 30 second section of EEG recording. The American academy of sleep medicine (AASM) (C. Iber et al., 2007) provide guidelines with specific criteria that seek to direct sleep scorers how to consistently evaluate sleep stages and discrete EEG arousal and awakening events. However, despite the use of these guidelines, intra- and inter-scorer differences remain and represents an inherent weakness in sleep macro-structural analysis dependent on traditional sleep scoring (Collop, 2002; Norman et al., 2000). In contrast, qEEG applies a more systematic spectral based analysis approach, typically over shorter periods (e.g. 5 sec as applied in this thesis) of EEG recording than is evaluated with traditional manual sleep staging in 30-sec epochs. This process facilitates a more systematic, consistent, and finer-grained analysis of the data even when run by multiple researchers. This is a significant strength of the tool utilized throughout this thesis as it enables greater confidence in the consistent application of the technique across multiple sleep studies and removes the potential for experimenter bias and error in traditional scoring.

Despite the relatively large sample of individuals utilized in Chapter 4, the lack of discernible EEG differences between the control night and study nights which used 25dBA WFN exposure could potentially reflect a Type II error. A more detailed analysis of acute effects of noise on qEEG outcomes, similar to that applied in Chapters 2 and 3, would be very useful to help rule-in versus out subtle effects of noise on sleep, particularly at lower SPLs most relevant to WFN exposure. Significant effects of higher SPL intermittent noise conditions on sleep macrostructure outcomes further support the need for a more detailed

evaluation of qEEG changes with different noise types and sound pressure levels. Chapter 2, showed WFN differences in qEEG at 33dBA relative to RTN, consistent with a need in future studies to evaluate WFN exposure effects above and below recommended night-time noise limits.

Part of the work presented in Chapter 4 sought to test for potential group differences in individuals who did versus did not report sleep disruption to noise exposure. These secondary analyses found no group dependent differences in EEG variables between the groups of interest (WFN sleep disturbed, WFN not sleep disturbed and RTN sleep disturbed) compared to rural controls for the control noise condition relative to the five nights of noise exposure. Given reduced sample size and thus power for between group analyses, a more detailed qEEG analysis of noise onset affects within and between groups would likely be useful to help evaluate what group sizes may be needed in future studies of group by condition effects. The study presented in Chapter 4 initially intended to achieve a larger sample size of 80 individuals, however study time constraints, bushfire weather (where rural residents were unwilling to leave their properties) and travel limitations as a result of COVID-19 all impacted on the ability to recruit the intended sample. The WFN sleep disturbed group in particular was smaller than the three other groups recruited. Future research should consider balancing the length of the study (reducing the time commitment (<1 week) or splitting the study into more achievable blocks of time commitment (two study lab attendances)) against rural and remote individuals' commitment to their rural properties (i.e., working around harvesting and bushfire seasons). These measures may make recruitment easier and commitment to the study more achievable. Furthermore, the WFN sleep disturbed group was older than the RTN disturbed group and a decline in slow wave EEG, shorter sleep duration and more time spent in lighter states of sleep with aging are well established (Carrier et al., 2001; Landolt & Borbély, 2001). Whilst these are all normal

aspects and expected changes of sleep patterns during ageing, if not appropriately controlled, these natural changes may be misattributed to noise-related sleep disruption. Other research also supports that older adults may be more sensitive to noise induced sleep disruption due to more 'fragile' sleep architecture and greater time spent in lighter states of sleep (Terzano et al., 1993). Within subject analyses are expected to be more robust to potential confounding from age effects, as the sleep of each participant is compared across different conditions, compared to group by condition interaction effects which could be influenced by differences in age. However, given limited time, resources and population samples to recruit from, age matching across groups was not practically possible in the current study. Future studies of potential group effects should consider age matching between groups to the extent that it is practical and possible to help avoid the potential for age to influence EEG outcomes.

5.7 Future Directions and Use of qEEG

This thesis work had two primary aims; firstly to establish whether WFN at variable sound pressure levels would disrupt sleep to a greater degree than RTN and quiet background control noise; and secondly to explore if sleep qEEG analysis could detect effects of nocturnal noise in the absence of effects on traditional macro-structural measures.

The work presented in this thesis, which shows clearly discernible but relatively small and short-lasting effects of relatively quiet noise samples on sleep, supports the need for further work to more definitively establish if currently accepted nocturnal noise guidelines (World Health Organization, 2018)) remain appropriate for WFN compared to RTN exposure.

The work in this thesis also supports that qEEG is sensitive to noise-induced EEG changes not captured in full night sleep macrostructure analyses. New tools to examine sleep beyond traditional sleep metrics, including qEEG and K-complex density metrics examined in this thesis, may be particularly useful for identifying noise effects on sleep. This thesis

work provides new insight into the application of these tools and supports that qEEG frequency analysis is particularly useful for examining short time-scale noise effects (Chapter 2 and 3) but is unable to discriminate between noise exposure at low levels and quiet control when applied in full night analysis (Chapter 4). This may well be because the noise levels used in Chapter 4 for full night exposure WFN were too low to produce discernible responses. However, averaging of qEEG power spectral outcomes over extended recordings is unlikely to be sensitive to noise effects given the findings of relatively small and transient effects on qEEG outcomes, largely dominated by increased K-complex probability and transient shifts to higher frequencies following noise onset, with no evidence of prolonged effects. Thus, even on the noisiest night with an average overnight SPL of ~42dBA, qEEG measures were no more sensitive to the presence of overnight noise than traditional sleep macrostructure assessments when evaluated as overnight spectral power averages.

In contrast, K-complex density was increased on the 20-sec and 3-min noise exposure nights compared to the quiet control night. Given the work in Chapters 2 and 3 showed a clear increase in K-complex probability within the first 5-seconds of noise onset events, these findings support that K-complex analyses may be a particularly useful and sensitive marker of overnight exposure to sudden onset noise events.

Several traditional measures of sleep macrostructure (TST, WASO, N3 sleep and REM sleep), as well as qEEG and K-complex density measures evaluated as full-night averages showed first night effects. It is well known that the first night spent in an unfamiliar laboratory for a sleep study can disrupt sleep (Agnew Jr et al., 1966). The findings of first night effects in this study are important and support the use of an acclimation night to avoid potential confounding of experimental intervention effects, such noise exposures, particularly when noise intervention effects may be subtle. Nevertheless, qEEG measures determined from full night recordings have previously been shown to be sensitive to sleep disruption

from obstructive sleep apnea (Vakulin et al., 2016), and sleep deprivation (Gibbings et al., 2021). Thus, although full-night qEEG analysis without any temporal alignment to noise events is clearly insensitive to low level noise exposures, full-night qEEG metrics are clearly able to detect more substantial noise-related sleep disruption, and could be particularly useful for evaluating snoring-related sleep disruption where noise levels can frequently exceed 50-60 dBA (Chirakalwasan et al., 2013; Sowho et al., 2020). However, given the findings of this thesis, qEEG appears likely to be most useful for evaluating noise-related sleep disturbances via more strategic noise-feature alignment with simultaneous noise recordings.

5.8 Clinical Implications, Options for Disturbed Populations

The results of the present study do not discount previous subjective reports of sleep impairment as a result of WFN exposure from exposed residents (Bakker et al., 2012). It is possible that adverse effects from WFN could arise via psychosocial factors related to the presence of noise, such as annoyance related-sleep disruption, stress and reduced quality of life that individuals may attribute to the presence of wind farms and noise (Michaud, Feder, Keith, Voicescu, Marro, Than, Guay, Denning, Bower, et al., 2016; Pohl et al., 2018). In Chapter 4 the research design tried to address potential contributions of physiological as distinct from 'psychological' variables to the disruption of sleep from WFN. By applying low-level but audible WFN only when participants were already asleep, any sleep disruption effects would most likely reflect a direct physiological sensory disturbance effect on sleep. However, when WFN was only present when participants were awake and then stopped at sleep onset, any disruption of sleep EEG would be mainly of psychological origin. Continuous WFN throughout the sleep period would combine both factors. It was also anticipated that the participants who previously complained of sleep disruption from WFN would show the strongest 'psychological' effect. However, having found no sleep effects, even with continuous 25dBA WFN in any condition, it was not possible to differentiate

between contributing factors as there was no effect from 25dBA WFN and no significant interaction effect between groups. Research conducted by Welch et al. (2013) shows noise impairs quality of life and may result in anxiety or noise sensitivity. Leventhall et al. (2012) and Aazh and Allott (2016) suggest that where this occurs for noise from wind farms and other forms of noise sensitivity causing distress, cognitive behavioral therapy may be useful in reducing psychological activation or response to disruptive noise types and provide healthy strategies to combat negative effects of noise sensitivity (Bernstein et al., 2013; Reid et al., 2016).

Individuals who are affected by noise-disrupted sleep are likely to benefit from noise mitigating strategies, such as noise abatement (e.g. reduced noise at the source, improved noise insulation), masking noise (e.g. added masking noise or via increased surrounding vegetation to generate local wind-related masking noise), and the use of earplugs or noise cancelling headphones. Masking strategies have been successfully used in other high noise environments to protect sleep and could relatively easily be translated to individuals who experience noise disruption as a result of wind farm noise exposure (Karimi et al., 2021; Xie et al., 2009). For example these strategies are common place in high noise environments such as hospitals and have been shown to assist with alleviating sleep disruption as a result of nocturnal noise exposure (Scotto et al., 2009; Wallace et al., 1999).

Another interesting possibility is to mask WFN through other environmental factors. This could involve more strategic selection of areas to build wind farms, or potentially rooftop wind generators that have sufficient background noise to mask wind turbine noise. This might be possible in areas with a naturally higher background noise, such as near built up cities with road traffic where residents may also be more habituated to nocturnal noise exposure (Pedersen et al., 2010).

Noise masking has been shown to be an effective strategy when aiming to reduce the

negative impact of noise induced sleep disruption in hospitals and as a result of road traffic noise (Xie et al., 2009). The strategy of masking wind farm noise using naturally occurring noises in the local environment (such as wind in trees and sea waves) has also shown promise to partially mask WFN and reduce perceived sound pressure level of the noise for listeners during the day and may be usefully applied to nocturnal WFN exposure (Bolin et al., 2010). These considerations may only be relevant where these natural masking noises occur. For residences with little natural wind or no surrounding trees to create masking noise naturally, synthetically reproduced palatable masking noise may be a viable option to assist with WFN related concerns.

Chronic sleep disruption can have serious health implications, and warrants strategic investigation and interventions to assist. Sleep disruption caused by psychological distress without a significant isolated cause can lead to severe consequences and the development of serious health concerns and psychological difficulties such as depression, anxiety and insomnia. Insomnia symptoms in the absence of any objective sleep disruption can cause clinical distress and is subsequently a diagnosable sleep difficulty in the current classification of sleep disorders, coined paradoxical insomnia (*The International Classification of Sleep Disorders:(ICSD-3)*, 2014). Regardless of cause and presence or absence of objective sleep disruption, subjective reports of sleep disturbance warrant treatment to reduce distress. Options including noise mitigation strategies, psycho-therapy or cognitive behaviour therapy and psycho-education on noise disturbance and sleep health, may all be helpful in alleviating individual and community concerns related to noise, sleep disruption and insomnia (Aazh & Allott, 2016; Leventhall et al., 2012).

5.9 Conclusions

The findings of this thesis support that qEEG is a useful and sensitive measure of EEG response to noise onset effects when noise interventions or synchronous noise

measurements are available to evaluate acute effects on the EEG. However, other measures including K-complex density and more traditional sleep scoring methods may be more practical for evaluating overnight noise onset exposure effects on sleep. Wind farm noise (WFN) reproduced in a sleep laboratory at 25dBA, similar to yearlong median levels from field recordings, did not significantly alter overall sleep macrostructure or qEEG parameters. However, acute onset of WFN at 33dBA produced increased alpha activity compared to road traffic noise (RTN), supporting the need for a more detailed qEEG analysis than was possible within the time-constraints of this thesis. Further research, perhaps ideally utilizing multiple nights of WFN versus RTN exposure at currently recommended indoor noise limits of around 30dBA, would also be useful to help determine if current noise limits are appropriate for WFN exposure, where low frequency dominated noise components could potentially be more problematic for sleep compared to high frequency dominated noise. This work concurs with other research in the field of nocturnal noise exposure which shows that more intermittent and variable noise exposure is likely to be more disruptive to sleep than continuous noise exposure at consistent noise levels. Appropriate noise mitigation strategies for residents reporting noise-related sleep disruption may be beneficial to offer strategies to assist with and potentially help to prevent the development of stress and insomnia. This thesis work provides a significant new contribution to knowledge demonstrating the utility of qEEG for evaluating noise induced changes in sleep EEG and contributes to the understanding of potential low frequency WFN, sub audible infrasound and road traffic noise effects on sleep. This thesis is consistent with an earlier evaluation of data from the same larger trial, which failed to find any evidence of changes in objective sleep macro-structural variables from 25dBA continuous WFN(Liebich, Lack, Hansen, et al., 2022). Through application of qEEG analyses, this thesis work extends these findings by also demonstrating no evidence of qEEG effects from continuous 25dBA WFN

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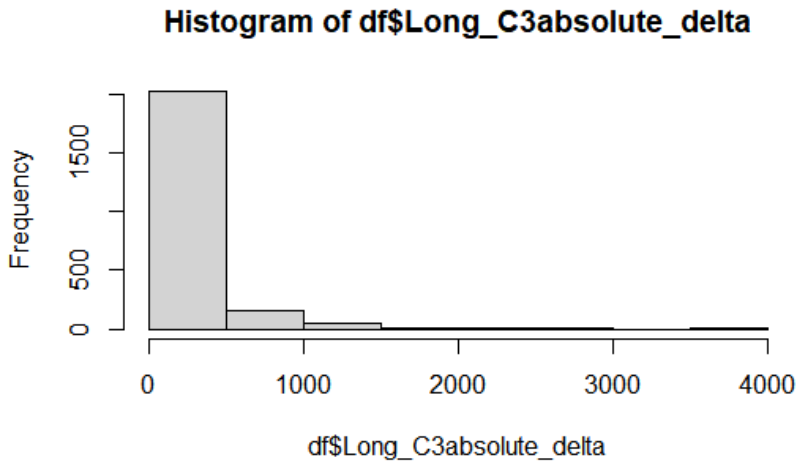
APPENDICES

Appendix A: Artifact removal process showing the visual workflow undertaken during the data cleaning process with two independent scorers in Chapter 4.

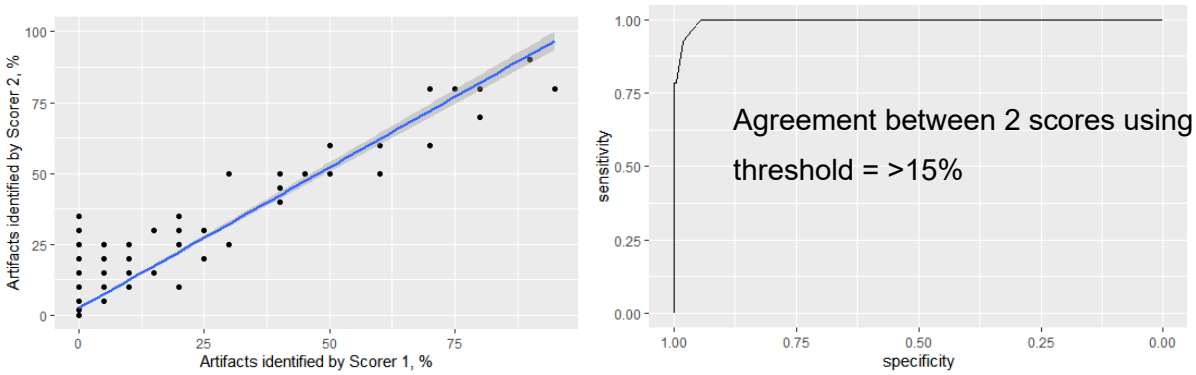
Data visualisation of absolute delta activity prior to artifact removal can be observed below.



Appendix A: Figure 1: Histogram Visualisation of raw absolute delta activity prior to data cleaning.

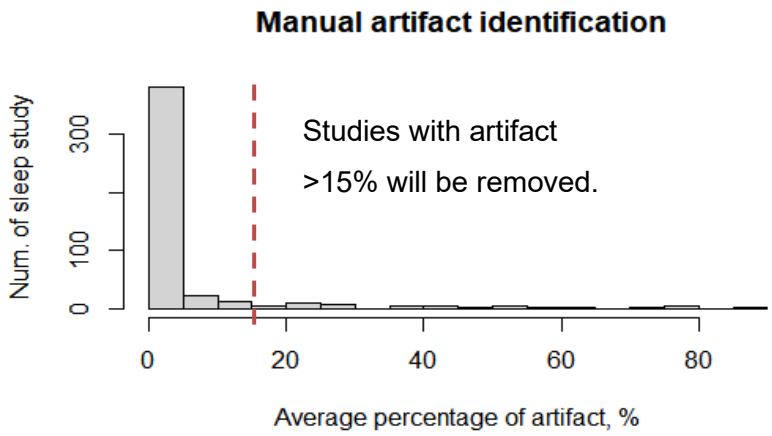
Two independent scorers visually inspected and rated each sleep study for artifact.

Agreement between scorers was high.



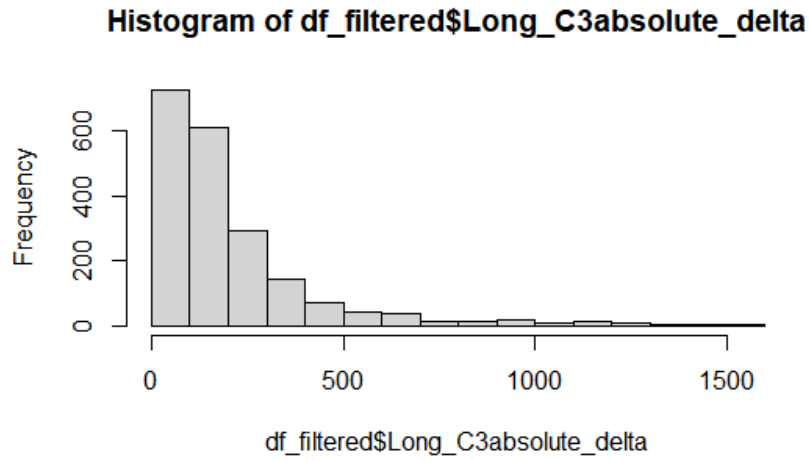
Appendix A: Figure 2: Left: Scatterplot of scorer ratings. Right: ROC curve depicting the specificity and sensitivity of scorer agreement on visual artifact.

Manual Artifact identification process identified sleep studies as containing greater than 15% artifact and these will be removed.



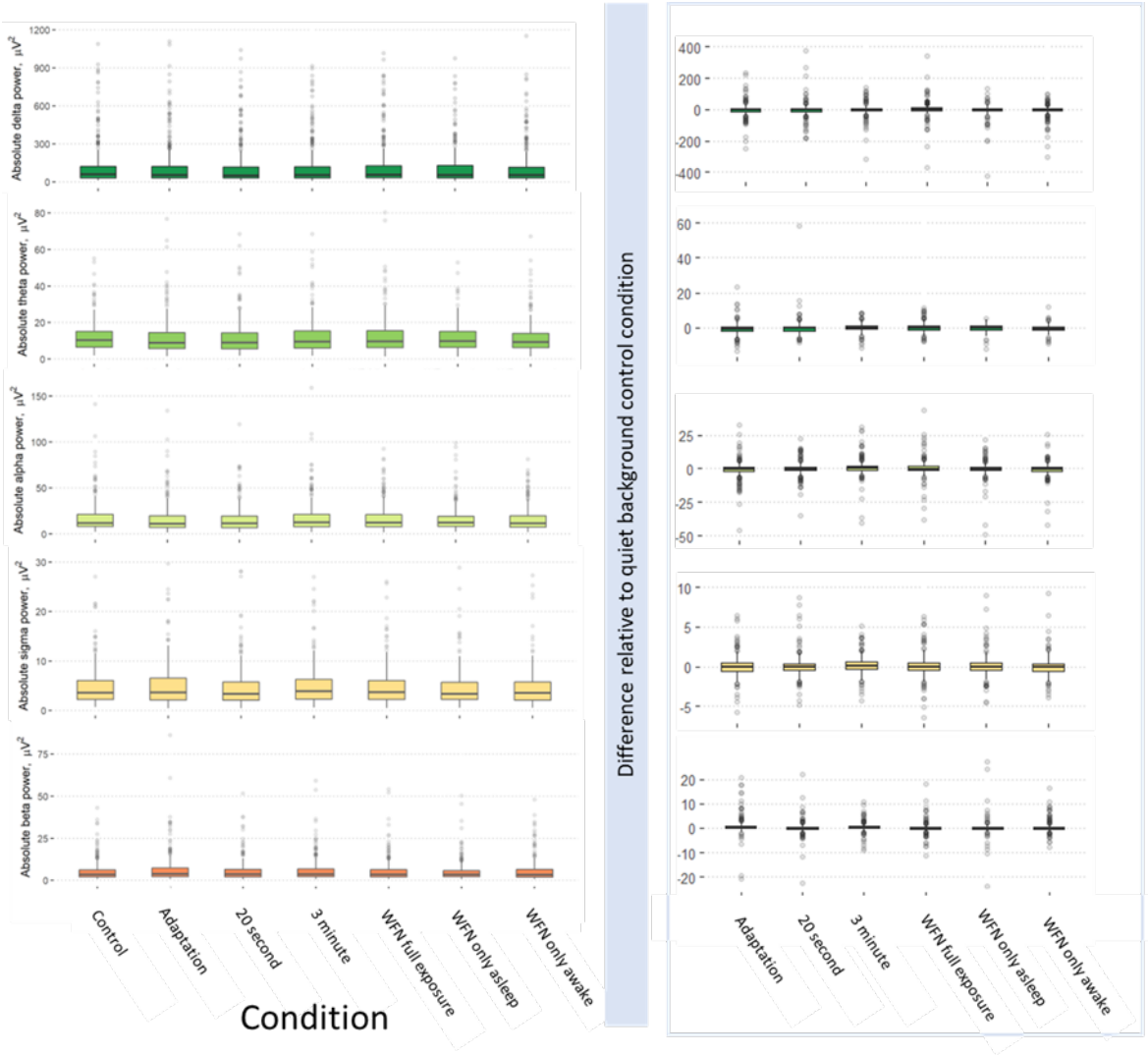
Appendix A: Figure 3: Left: Number of studies removed above visually scored extraneous noise cut off at 15% of identified artifact.

Visual depiction of data post artifact removal process showing significant removal of outlying data for absolute delta activity.



Appendix A: Figure 4: Left: Histogram depiction of absolute delta activity post artifact removal process.

Appendix B: Box plot of absolute quantitative EEG outcomes by nightly condition.



Appendix B: Figure 1: Box plots showing (left) median and interquartile range, Tukey-style whiskers (extended to a maximum 1.5 x IQR outside of the box) and outlier values (circles) in absolute delta through to beta power across each different noise condition night, and (right) absolute differences compared the control condition night (19dBA background noise).

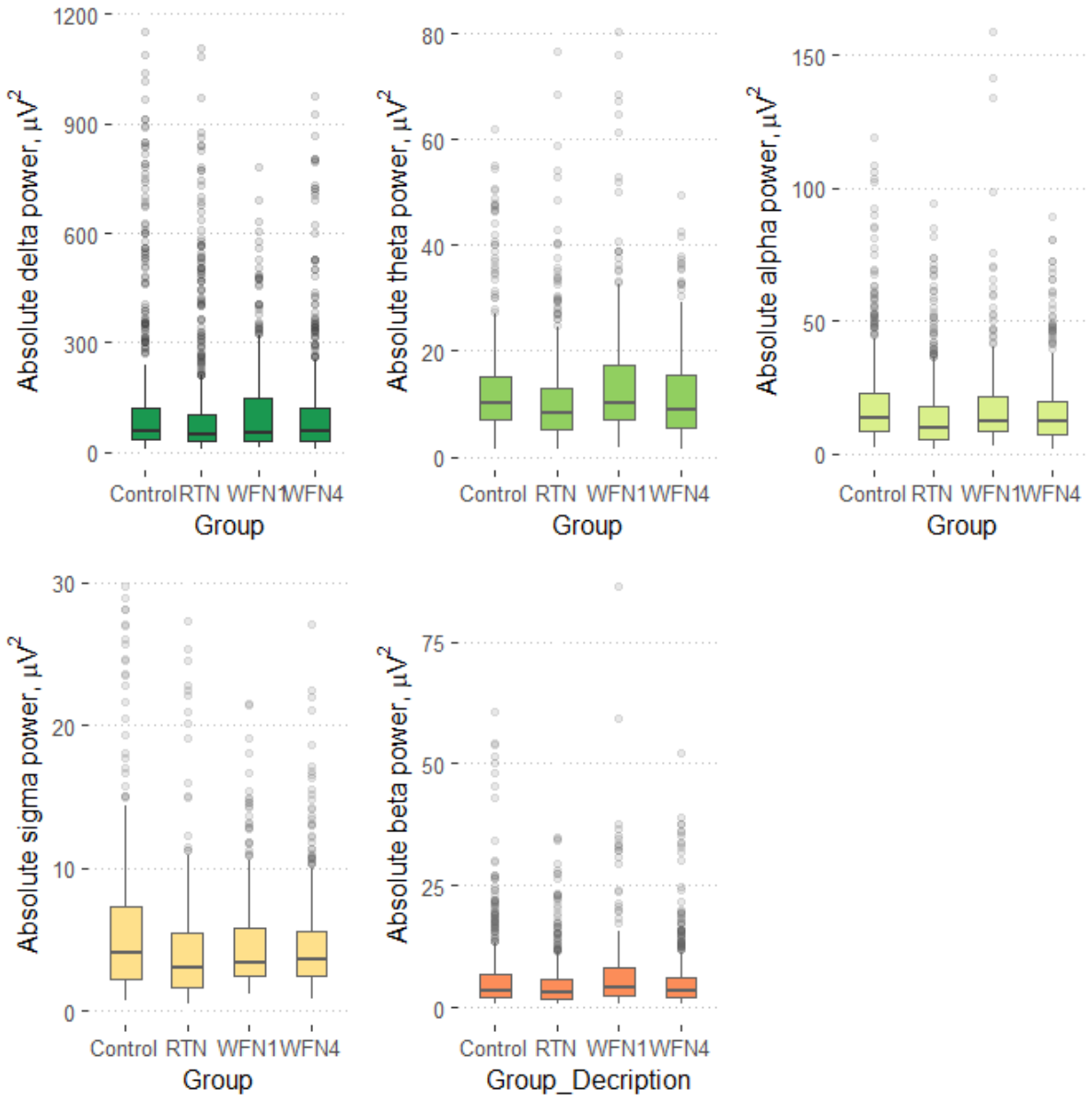
Appendix C: Group by stage interaction effects on spectral power

Interaction inferential statistics for group by stage interactions.

	Chi-square		
	χ^2	**df**	***p-value***
Absolute Beta Activity	37.5777	12	<0.001
Absolute Alpha Activity	65.9713	12	<0.001
Absolute Sigma Activity	315.1624	12	<0.001
Absolute theta Activity	62.7193	12	<0.001
Absolute delta Activity	83.467	12	<0.001
Delta Ratio	109.0086	12	<0.001
Theta Ratio	116.618	12	<0.001
Alpha Ratio	34.6136	12	0.001
Sigma Ratio	231.9753	12	<0.001
Beta Ratio	51.4109	12	<0.001

Appendix D: Main effects of group spectral power

Significant main effects of group where observed in the absence of higher order group by condition interaction effects for absolute theta power (x^2 =19.28, df=3, p<0.001), where the RTN group was seen to have borderline significantly less absolute theta activity when compared to the control rural group (p=0.04993) (See figure D.1).

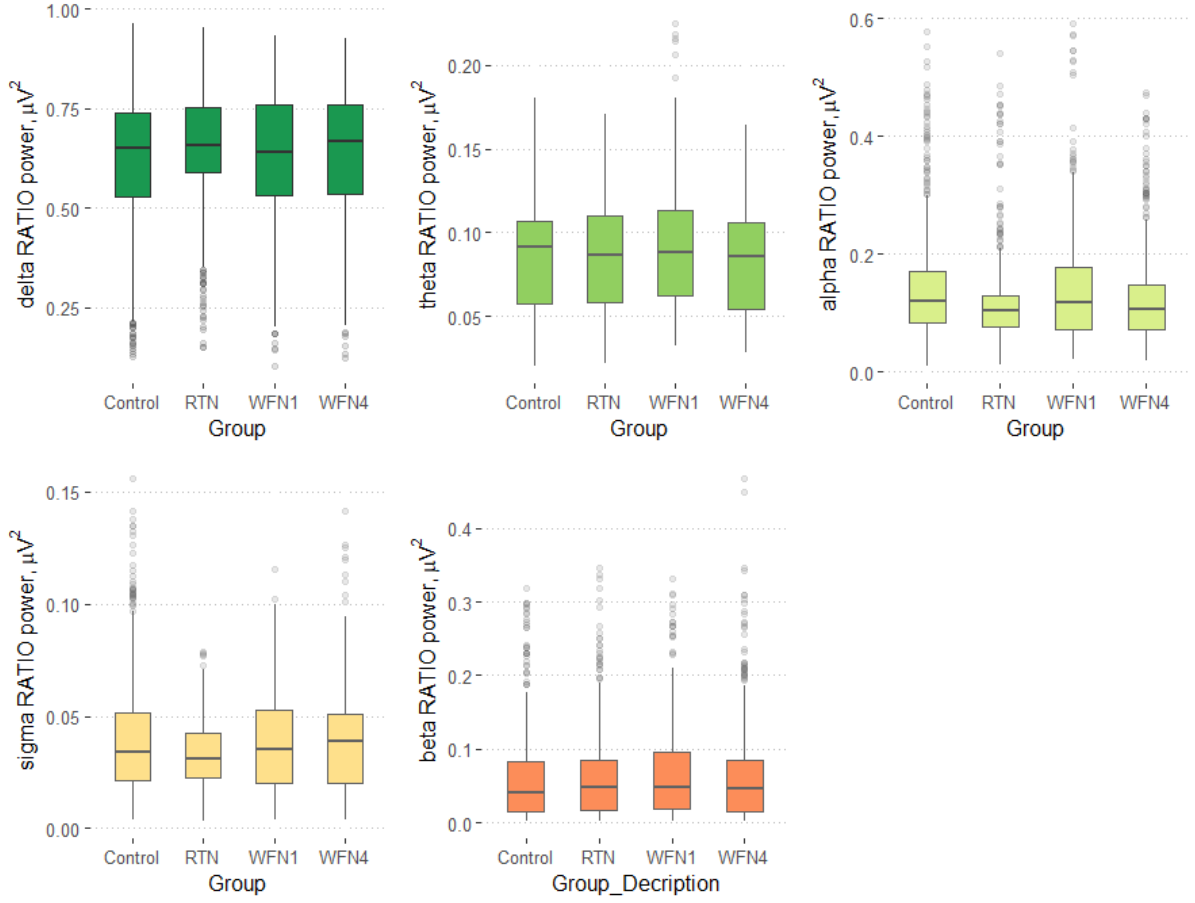


Appendix D Figure.1. Absolute spectral power values by group

For spectral power ratio activity all frequency bands (bar relative beta activity) observed a significant main effect of group in the absence of group by condition higher order interactions (delta ratio: χ^2 =16.38, df=3, p=0.001; theta ratio: χ^2 =14.37, df=3, p=0.002; alpha ratio: χ^2 =15.46, df=3, p=0.001; sigma ratio: χ^2 =8.56, df=3, p=0.036).

Relative delta activity was higher for the RTN group compared to control (p<0.001) and lower for the WFN sleep disturbed group relative to control (p=0.002).

Relative theta activity was significantly higher for the WFN sleep disturbed group (p=0.039) and relative alpha and sigma activity was lower for the RTN group (p=0.026, p=0.006 respectively) when compared to the control group (See figure D.2).



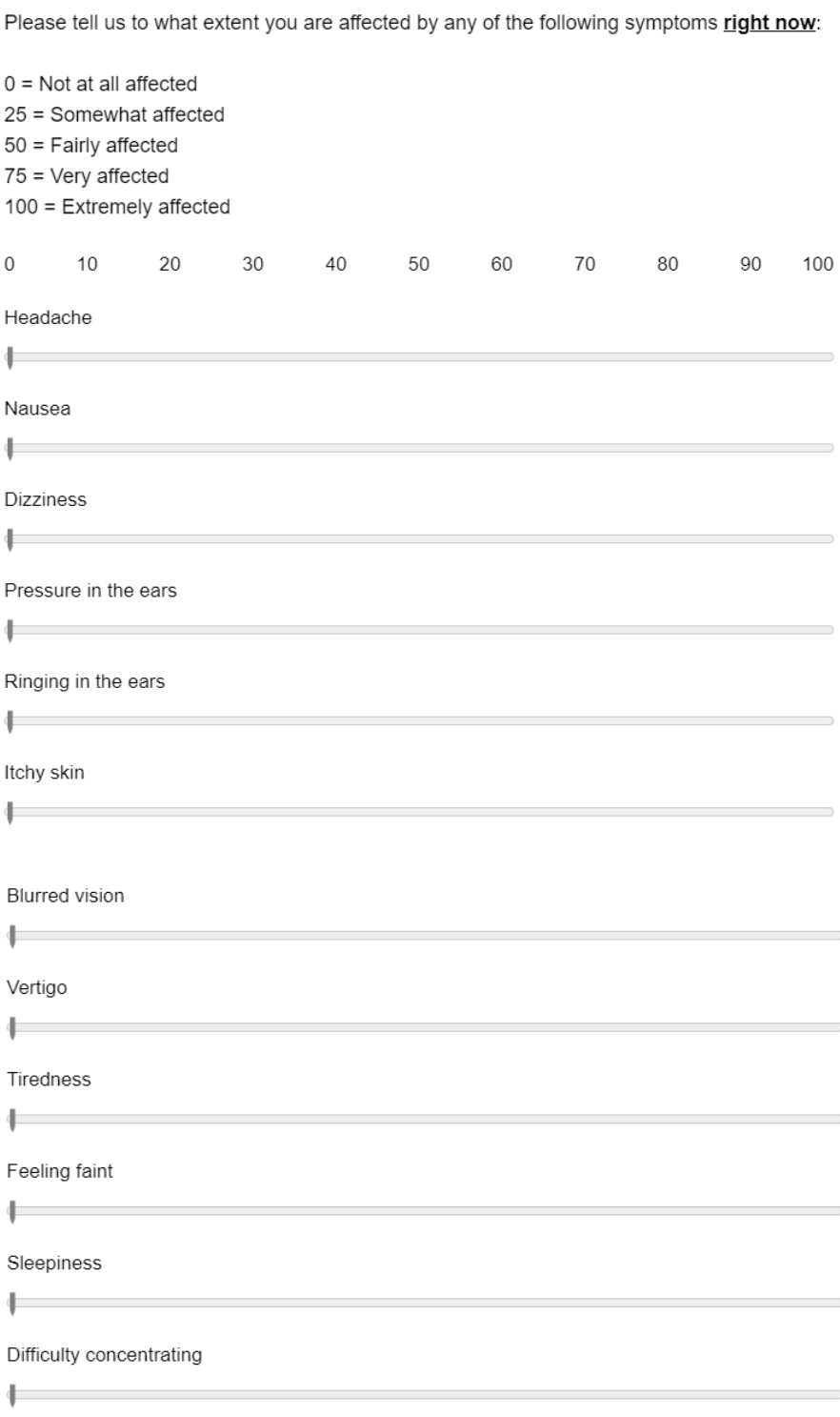
Appendix D, Figure 2. Spectral power ratio values by group

Appendix E: Daytime symptomology reported the morning after noise exposure using the visual analogue scale

	Control	Adaptation	20 second	3 minute	WFN full exposure	WFN only asleep	WFN only awake	P-Value
Headache	4.4 [1.2 to 7.7] (51)	2.0 [-0.2 to 4.1] (48)	4.3 [2.0 to 6.5] (53)	5.0 [0.8 to 9.2] (51)	4.3 [0.5 to 8.1] (42)	1.7 [-0.4 to 3.8] (40)	4.9 [0.4 to 9.3] (47)	0.584
Nausea	2.3 [-0.1 to 4.8] (50)	0.2 [0.1 to 0.3] (47)	2.0 [0.5 to 3.5] (52)	2.1 [-1.1 to 5.3] (51)	2.8 [-0.2 to 5.7] (42)	0.3 [0.0 to 0.5] (40)	3.1 [-0.6 to 6.9] (48)	0.531
Dizziness	1.1 [-0.8 to 2.9] (50)	1.5 [-0.2 to 3.2] (49)	1.3 [0.0 to 2.6] (51)	2.4 [-1.0 to 5.8] (50)	2.3 [-0.6 to 5.2] (42)	0.6 [0.0 to 1.2] (40)	2.8 [0.1 to 5.4] (48)	0.501
Pressure in ears	2.8 [-0.2 to 5.7] (50)	0.5 [0.0 to 0.9] (48)	1.1 [-0.2 to 2.4] (51)	3.2 [-0.9 to 7.3] (50)	2.7 [-1.4 to 6.8] (42)	1.7 [-0.3 to 3.7] (40)	3.1 [-0.4 to 6.5] (48)	0.790
Ringing in ears	14.8 [8.3 to 21.3] (52)	9.9 [4.0 to 15.8] (49)	10.2 [3.7 to 16.7] (52)	11.9 [5.7 to 18.1] (51)	8.8 [2.5 to 15.0] (43)	12.5 [5.7 to 19.2] (40)	14.0 [7.2 to 20.7] (48)	0.807
Vertigo	2.5 [-0.8 to 5.7] (50)	0.4 [0.1 to 0.7] (47)	0.7 [-0.2 to 1.6] (51)	3.3 [-1.2 to 7.8] (50)	1.7 [-1.1 to 4.5] (42)	1.8 [-1.2 to 4.7] (40)	3.4 [0.4 to 6.4] (47)	0.390
Feeling Faint	1.5 [-0.5 to 3.5] (51)	0.6 [-0.3 to 1.5] (48)	0.5 [-0.1 to 1.0] (51)	1.8 [-1.3 to 5.0] (51)	1.6 [-0.3 to 3.5] (42)	0.4 [-0.1 to 0.9] (40)	1.2 [-0.3 to 2.7] (47)	0.922
Difficulty concentrating	5.7 [0.9 to 10.5] (51)	4.7 [1.6 to 7.9] (49)	6.9 [3.3 to 10.6] (52)	7.0 [2.2 to 11.8] (53)	8.5 [3.2 to 13.7] (42)	4.5 [0.1 to 8.9] (40)	9.7 [3.3 to 16.1] (47)	0.302
Difficulty remembering	5.0 [1.1 to 8.9] (50)	2.9 [0.4 to 5.3] (47)	3.5 [1.1 to 5.8] (51)	3.8 [-0.3 to 7.8] (50)	4.4 [0.7 to 8.0] (42)	3.5 [-0.4 to 7.4] (40)	5.9 [2.0 to 9.9] (47)	0.796
Irritability	5.2 [0.5 to 9.9] (51)	2.0 [-0.2 to 4.1] (47)	4.9 [1.3 to 8.5] (52)	4.5 [0.2 to 8.8] (51)	4.2 [0.1 to 8.3] (42)	3.1 [0.6 to 5.6] (40)	4.2 [0.5 to 8.0] (48)	0.449
Muscle Spasms	1.3 [-1.1 to 3.7] (50)	1.2 [-0.9 to 3.4] (47)	0.3 [0.0 to 0.7] (51)	1.7 [-0.9 to 4.3] (50)	2.2 [-0.5 to 4.9] (42)	0.5 [-0.1 to 1.1] (40)	1.8 [-0.6 to 4.2] (47)	0.886
Ear pain	3.0 [-0.8 to 6.9] (50)	0.3 [0.1 to 0.5] (49)	0.3 [0.0 to 0.5] (52)	2.1 [-1.8 to 6.0] (51)	2.4 [-2.0 to 6.9] (42)	0.8 [-0.1 to 1.7] (40)	2.6 [-1.5 to 6.7] (47)	0.210
KSS morning sleepiness	4.4 [4.0 to 4.9] (53)	4.1 [3.7 to 4.6] (57)	4.4 [3.9 to 4.8] (54)	4.6 [4.1 to 5.1] (54)	4.6 [4.1 to 5.0] (47)	4.3 [3.8 to 4.8] (41)	4.3 [3.8 to 4.8] (50)	0.331

Note. Values are Mean [95%CI] (N) unless otherwise specified. p-values indicate the condition main effect. The symptom scale ranges from 0 (not at all affected)-100 (extremely effected). **Note.* Due to participant completion rates some data are missing as expressed by the N reported next to each value.

Appendix F: Visual Analogue Scale (VAS) for symptoms used to measure morning symptom reports following noise exposure



Difficulty remembering

Fatigue

Irritability

Muscle spasms

Anxiety

Ear pain

→