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.