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Original Article

ORIGINAL ARTICLE

An experimental investigation on the impact of wind turbine noise on polysomnography-measured and sleep diary-determined sleep outcomes

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Abstract

Study Objectives: Carefully controlled studies of wind turbine noise (WTN) and sleep are lacking, despite anecdotal complaints from some residents in wind farm areas and known detrimental effects of other noises on sleep. This laboratory-based study investigated the impact of overnight WTN exposure on objective and self-reported sleep outcomes.

Methods: Sixty-eight participants (38 females) aged (mean ± *SD*) 49.2 ± 19.5 were recruited from four groups; *N* = 14, living <10 km from a wind farm and reporting WTN related sleep disruption; *N* = 18, living <10 km from a wind farm and reporting no WTN sleep disruption; *N* = 18, reporting road traffic noise-related sleep disruption; and *N* = 18 control participants living in a quiet rural area. All participants underwent in-laboratory polysomnography during four full-night noise exposure conditions in random order: a quiet control night (19 dB(A) background laboratory noise), continuous WTN (25 dB(A)) throughout the night; WTN (25 dB(A)) only during periods of established sleep; and WTN (25 dB(A)) only during periods of wake or light N1 sleep. Group, noise condition, and interaction effects on measures of sleep quantity and quality were examined via linear mixed model analyses.

Results: There were no significant noise condition or group-by-noise condition interaction effects on polysomnographic or sleep diary determined sleep outcomes (all *ps* > .05).

Conclusions: These results do not support that WTN at 25 dB(A) impacts sleep outcomes in participants with or without prior WTN exposure or self-reported habitual noise-related sleep disruption. These findings do not rule out effects at higher noise exposure levels or potential effects of WTN on more sensitive markers of sleep disruption.

Clinical Trial Registration: ACTRN12619000501145, UTN U1111-1229-6126. Establishing the physiological and sleep disruption characteristics of noise disturbances in sleep. https://www.anzctr.org.au/. This study was prospectively registered on the Australian and New Zealand Clinical Trial Registry.

Statement of Significance

Carefully controlled laboratory studies to investigate the effect of wind turbine noise (WTN) on polysomnographically and sleep diary determined sleep outcomes are limited. This study found no evidence to support that overnight WTN exposure levels similar to average year-long indoor WTN levels significantly impact key objective or subjective sleep outcomes, including in residents habitually exposed to WTN. However, sleep disturbance effects at higher worst-case noise exposure levels or more subtle microstructural sleep effects cannot be ruled out so further studies remain warranted.

Key words: sleep disruption; sleep disturbance; sleep quality; wind farm; wind turbine; environmental noise

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Introduction

A rapid ongoing shift away from fossil fuels to renewable energy generation includes the expansion of wind turbines in reliable wind exposure areas, often with existing near-by residences. Therefore, it is important to clarify whether wind turbine noise (WTN) has detrimental health effects and through what mechanisms to help inform the need for and design of potential mitigation strategies.

Chronic exposure to environmental noises (eg, road, rail, and aircraft noise), of sufficient sound pressure levels (SPLs which govern the overall amplitude/intensity of the noise) are known to negatively impact sleep [1–3]. However, only a limited number of studies have examined the impact of WTN on sleep, and these have shown inconsistent and inconclusive findings. This potentially reflects a combination of factors such as more modest sound levels, lower frequency content, variable exposure levels, and a reliance on self-report and cross-sectional study designs, which may make WTN effects difficult to reliably detect.

WTN has several prominent and acoustically unusual features compared to other environmental noises known to affect sleep, such as road traffic noise (RTN). Unlike RTN, which typically reduces in SPL at night because of reduced road traffic, WTN SPLs and acoustic characteristics are largely dependent on atmospheric and wind conditions, which are often more stable and favor more prominent noise at night when background noise levels are typically lowest; especially in rural areas where wind turbines are typically located. Consequently, residents living near wind turbines, who are likely to be accustomed to very low background noise levels at night, may be susceptible to WTN disruption when attempting sleep at night. Despite limited high-quality evidence, consistent reports of sleep complaints support that sleep disruption is problematic for some residents living close to wind turbines [4–7]. Therefore, it is possible that WTN has direct physiologically disruptive effects on sleep and on subsequent daytime functioning and health. In that context, the most effective mitigation strategies would likely be to limit the proximity of wind farms to residences, to promote more effective noise abatement through improved WTN locations and residential building design or potentially to mask WTN noise.

However, sleep disruption can also manifest as self-reported difficulties initiating and maintaining sleep and/or experiencing un-restorative sleep without necessarily a specific sleep disruption trigger, such as what occurs with insomnia [8]. Thus, a combination of WTN and other factors including knowledge, attitudes, noise sensitivity, and beliefs around WTN exposure could also produce psychological responses with indirect detrimental effects on sleep. If residents attribute sleep disruption to WTN and symptoms are left untreated, individuals could develop chronic insomnia, via conditioned responses when attempting sleep including maladaptive sleep behaviors and cognitions, which may subsequently impact on daily function, well-being, and potentially health [8–15]. In this context, reliable evidence-based information and education along with psychological therapies would likely be indicated. Therefore, experimental investigations to help clarify the effects of WTN on sleep through direct sleep disruption and indirect psychological effects are important.

To date, three studies have utilized experimental designs in carefully controlled laboratory settings to investigate the impact of WTN on polysomnography (PSG)-assessed sleep [16–18]. Ageborg Morsing *et al.* [16]. conducted two pilot studies ($N = 6$ in both) where participants were exposed to three WTN exposure nights with varying frequencies, amplitude modulation characteristics, and dB L_{AEq} SPLs (L_{AEq} refers to A-weighted equivalent continuous SPLs; See Bergland, Lindvall [19] for more details). SPLs used in Ageborg Morsing *et al.* [16]. ranged from 29.5 to 34.1 dB L_{AEq} in one study and 30.4 to 32.8 dB L_{AEq} in another study, with a quiet control night (18 dB L_{AEq}) for comparison in both. There were no significant effects of noise exposure level on self-report or PSG sleep outcomes, including total sleep time, sleep latency, sleep efficiency, or wake after sleep onset. However, given the small sample sizes, and associated type II error risk, these findings warrant cautious interpretation. However, a recent larger study in 23 urban residents without habitual WTN exposure also found no significant effects of WTN exposure effects during sleep at 33 dB(A) on one night compared to background noise at 23 dB(A) on another night on PSG-derived latency to N1 or N2 sleep or self-reported sleep latency [20]. However, these urban residents may have been more tolerant of higher noise levels, (eg, road traffic), during the sleep period, particularly as these urban residents were also not overly noise sensitive, were healthy sleepers, and did not report RTN related sleep disruption at home [20].

In the largest experimental study to date, Smith *et al.* [18] studied 50 participants, including a group of individuals living near wind farms and another group without prior WTN exposure, during a control background noise only night at 13 dB and a night of indoor WTN exposure at 32 dB L_{AEq}. Rapid eye movement (REM) sleep latency was significantly increased, and REM duration reduced on the WTN noise exposure compared to the control night, but no other PSG-derived sleep parameters changed, including sleep latency. However, self-reported sleep quality, measured on a 5-point scale from “very good” to “very bad” was significantly reduced on the WTN night compared to the control night. This effect was larger for participants previously exposed to WTN versus participants previously unexposed to WTN. Potential participant awareness of WTN exposure prior to falling asleep and during night-time awakenings with continuous noise exposures from lights out until lights on has the potential to influence and bias self-reported responses, particularly in participants with strongly established attitudes, beliefs, and expectations regarding WTN effects on sleep [20]. Therefore, further studies to test for effects of WTN exposure more specifically during periods of wake versus sleep are needed to help separate potential wake-dependent psychological effects from sleep-dependent effects of WTN on objective and subjective measures of sleep difficulties and quality.

Accordingly, the primary aim of this study was to examine the impact of audible WTN, including prominent infrasound and low-frequency amplitude-modulated tones, on conventional PSG (objective)- and sleep diary-determined (subjective) sleep parameters in a carefully controlled laboratory environment. This study was part of a larger study that included two separate study nights for evaluating dose-response characteristics of WTN compared to road traffic noise presented during established sleep. However, the current study, which involved four separate study nights, was specifically designed to examine the effect of realistic levels of audible WTN, including prominent amplitude-modulated tones and infrasound (which are often inaccurately characterized using A-weighted SPL [21, 22]), on the ability of participants to initiate and maintain sleep. We

reasoned that if audible WTN including prominent amplitude modulation and infrasound is problematic for initiating and/or returning to sleep following awakenings, then these effects should be wake-dependent and apparent with audible WTN above background noise, particularly in individuals reporting WTN-related sleep difficulties.

To investigate potential wake-dependent psychological effects versus sleep-dependent WTN effects, four different noise exposure conditions were examined in a randomized order across separate nights, including continuous WTN exposure during wake and sleep (WTN-Continuous); WTN exposure only during established N2, N3, and REM sleep (WTN-Sleep); WTN exposure only during periods of Wake and transitional stages of N1 sleep (WTN-Wake); and no WTN exposure (ie, quiet background noise [control]). A further aim was to elucidate possible effects of prior noise exposure on WTN responses, by recruiting participants living near wind farms who did and did not report WTN related sleep disruption, as well as two control groups: residents from rural communities with no wind farms nearby and participants reporting road traffic noise-related sleep disruption.

It was hypothesized that PSG and sleep diary outcomes would be more disrupted (ie, more wake and less sleep) on the WTN-Continuous night compared to the control night due to direct sleep-dependent WTN effects, indirect wake-dependent psychological effects, or both. If only wake-dependent psychological effects were operating, then WTN-Wake and WTN-Continuous nights would be expected to show reduced sleep compared to the WTN-Sleep and control nights. In the presence of sleep-specific WTN disruption effects, WTN-Sleep and WTN-Continuous nights would be expected to show greater sleep disruption than both the WTN-Wake and Control nights. Furthermore, in the presence of prior exposure and potential noise habituation and noise sensitivity effects, greater levels of sleep disruption were anticipated in residents living near wind turbines and reporting WTN-related sleep disruption compared to other groups.

Methods

Study setting and design

The study was conducted at the Adelaide Institute for Sleep Health, Nick Antic Sleep Laboratory. For seven consecutive nights, study participants spent the night in one of two fully private, heavily sound-attenuated bedrooms (background noise level 19 dB(A)) with their own ensuite and a shared lounge area. Bedroom temperatures were set to 23°C and participants were provided with light bed covering and additional bed covering if requested.

A four-group (WTN-sleep disturbed, WTN-non-sleep disturbed, rural control, RTN-sleep disturbed) by four noise conditions (WTN-Sleep, WTN-Wake, WTN-Continuous, Control) single-blind study design was used to investigate the effect of WTN exposure on PSG and sleep diary outcomes. The first night was an acclimatization night, after which participants were randomized to six different noise exposure conditions over the remaining nights, of which, only four noise exposure nights are relevant and reported in this study.

The primary outcomes were PSG measured sleep efficiency (ie, the total amount of sleep time divided by total time spent in bed), the most widely used objective measure of overall sleep quality, and sleep diary determined sleep efficiency [23]. Secondary outcomes were PSG and sleep diary derived sleep latency, total sleep time, wake after sleep onset, number of awakenings, and time spent in bed, as well as PSG derived total wake time, time spent in each sleep stage, and latency to N2, N3, and REM sleep. Prior to lights out, participants were only instructed that "they may or may not hear noise during the night" and that noise exposures could include a range of noise samples including WTN. Thus, participants remained unaware of specific noise conditions each night, but by study design and use of audible noise, were most likely aware of noise presentations during wake.

Sleep technicians manually commenced and paused noise play-back according to allocation night and observed sleep stage across each study night (sleep, wake, or played continuously) so could not be blinded to noise condition. However, an independent sleep scientist undertook all subsequent sleep staging and arousal scoring analysis blinded to noise exposure conditions.

Participants

Potential participants were recruited via print advertising on community noticeboards, word of mouth, and online social media advertising (Facebook and Gumtree) (see Supplement A in the Supplementary Materials for the recruitment poster used). 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). All participants provided written and informed consent and were financially compensated for study participation and travel costs (total reimbursement: $1300 AUD for rural participants and $1000 AUD for urban participants).

Inclusion and exclusion criteria

Study participants were adults recruited from different noise exposure areas and were considered for inclusion based on residential location and questionnaire responses indicating either the presence or absence of self-reported WTN or RTN-related sleep disruption. Participants in the WTN-non-sleep disturbed and WTN-sleep disturbed group lived <10 km from a wind turbine and reported 1 and >1 respectively on a 5-point Likert scale (1 = not at all, 2 = mildly, 3 = moderately, 4 = severely, 5 = very severely) which involved one item asking, "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?" based largely on the ISO-15666-2003 standard, but with a nonstandard "very severely" instead of "extremely" highest response option [24]. Participants in the RTN-sleep disturbed group reported >1 on an equivalent question regarding RTN-related sleep disruption. Rural control participants lived in a rural or remote area classified by the Rural, Remote, and Metropolitan Area Classification [25] and scored 1 to both WTN and RTN related sleep disruption items.

Study exclusion criteria included age <18 years; any use of sedative medications; any history of substance use in the past six months; night shift work within the last 2 months (ie, any shift between 22:00 and 08:00 hours); or traveled across ≥2 timezones within the past 2 months.

Intervention

Noise reproduction. Experimental noise stimuli were faithfully reproduced via an RME BabyFace Pro sound card, a Krix KX-4010s non-vented subwoofer speaker with a 25 cm driver positioned approximately 3 m from the foot of the participant's bed and a Crown DC-300 power amplifier with a flat frequency response down to 0 Hz [26–27].

Noise stimulus. The WTN stimulus was recorded indoors at a residence located 3.3 km from a wind farm in South Australia. A 3-minute sample was then extracted from the measured data and was played on a repetitive loop (see Hansen *et al*. [28] for further details regarding wind farm layout, properties, and measurement setup). The temporal profile of the WTN included a ramp in of approximately 2.5 s and a very minimal ramp out (approximately 300 ms) to ensure abrupt WTN cessation in the event of awakenings on WTN-Sleep only nights.

The measured recordings generated WTN at an indoor SPL of 25 dB(A) (dB(A) referring to A-weighted decibels, which involves a linearized logarithmic scale of frequencies and SPL over the normal range of human hearing from 20 to 20 000 Hz) and included an amplitude-modulated tone at multiple frequencies in 1/3-octave bands centered at 31.5 and 63 Hz and infrasound at the blade-pass frequency of 0.8 Hz and harmonics. Due to limitations with the loudspeaker, the spectral contents below 1.6 Hz could not be reproduced as shown in Figure 1A. The selection of 25 dB(A) was based on the results of a year-long measurement of WTN that showed that the median indoor SPL at night was between 25 and 30 dB(A) for distances from 1 to 3 km (Figure 1B) [29]. Furthermore, the WHO guidelines [22] (p. xiii) also state that "when noise is continuous, the equivalent SPL should not exceed 30 dB(A) indoors if negative effects on sleep are to be avoided and for noise with a large proportion of low frequency sound, a lower guideline value is recommended". Therefore, choosing an SPL based on median SPLs measured over a year-long period was considered to be more representative of long-term WTN exposure rather than exposure to louder and less common events. Also, the reproduced noise level was approximately six dB(A) above the background noise level in the sleep laboratory, which is clearly perceivable by normal hearing subjects [30–31].

Noise intervention. In the control condition, the only noise present was background noise at 19 dB(A). In the WTN-Continuous condition, 25 dB(A) WTN was played continuously from lights out time to lights on time to investigate the combination of potential sleep- and wake-dependent WTN effects. In the WTN-Sleep condition, 25 dB(A) WTN was played during established sleep periods (N2, N3, REM sleep) and paused during wake and light/transitional sleep periods (N1), to test for potential sleep-specific effects of WTN, with reduced opportunity for noise awareness when participants were attempting to initiate and return to sleep following an awakening. Finally, in the WTN-Wake condition, 25 dB(A) WTN was played during wake and light/transitional sleep periods (N1) and paused during established sleep periods (N2, N3, REM sleep) to test for potential wake-dependent psychological effects of WTN exposure while participants attempted to initiate and return to sleep. The sleep technicians continuously monitored sleep stage throughout each night so that WTN could be stopped and started as appropriate and in accordance with the American Academy of Sleep Medicine scoring criteria [32].

Measures

PSG. Objective sleep parameters were assessed via PSG (Grael 4K, Compumedics Ltd., Abbotsford, VIC, Australia) and scored by a single trained scorer blinded to noise exposure conditions and acoustic data, which were recorded separately. PSG signals included electroencephalograms recorded from gold-plated electrodes placed at Fz, F3, F4, C3, C4, O1, and O2 sites referenced to contralateral mastoids (M1 and M2), and ground and reference electrodes on the clavicle and forehead respectively. Electromyography, electrooculography, electrocardiography, pulse oximeter, and leg movement signals were also recorded.

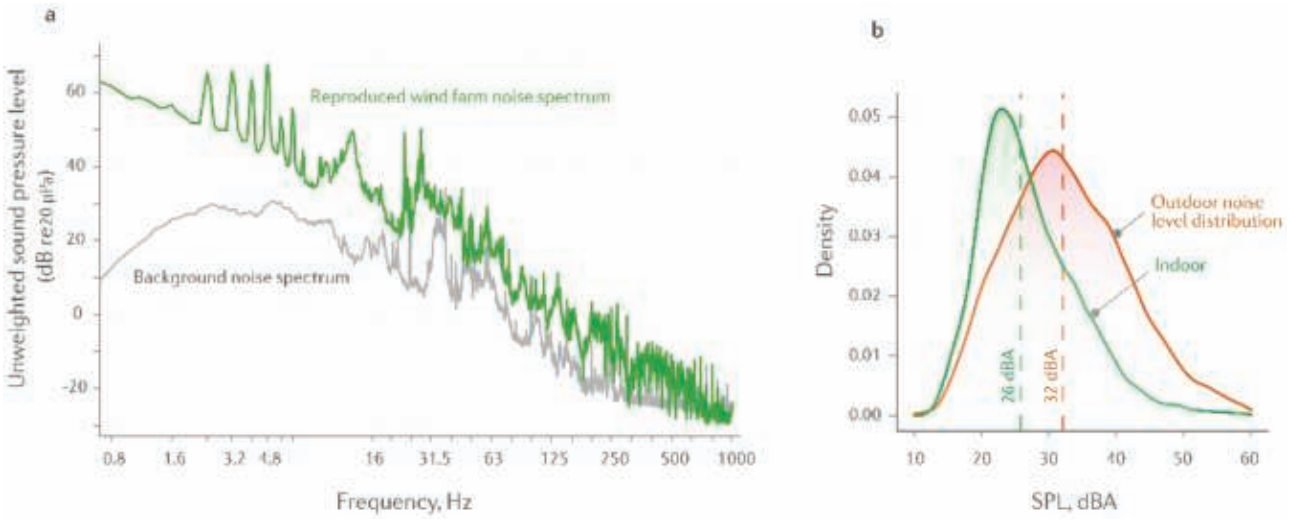

Figure 1. Selected noise stimulus frequency and SPL characteristics. (A) Bedroom background noise and reproduced full spectrum WTN measured in the current study. (B) Density distributions of outdoor and indoor noise levels during a separate year-long study [29]. Dashed lines in (B) indicate median indoor and outdoor SPLs from Nguyen *et al*. [29] This shows that the WTN level used in the current study (25 dB(A)) was similar to median indoor SPLs measured over a year-long study (26 dB(A)). WTN, wind turbine noise; SPL, sound pressure level.

Sleep and daytime questionnaires. Subjective sleep parameters were assessed using an online morning sleep diary based on the Consensus Sleep Diary [33]. The Consensus Sleep Diary asks questions pertaining to time in/out of bed and minutes awake/asleep in bed per night, enabling the calculation of time in bed, sleep latency, number, and duration of awakenings, wake up time, and total sleep time (see Supplement B in the Supplementary Materials for the 22-item online sleep diary used in the current study) [33]. The Consensus Sleep Diary is well-validated and shows high agreement compared to PSG (kappa = 0.87) and high sensitivity (92.3%) and specificity (95.6%) [34].

Participants also completed several questionnaires regarding their usual sleep and noise sensitivity prior to their laboratory visit, including insomnia symptoms (ISI) [35], sleep quality (PSQI) [36], daytime sleepiness (ESS) [37], and noise sensitivity (Weinstein Noise Sensitivity Scale) [38].

Procedure

For 2 weeks prior to their scheduled 7-night stay, participants completed a paper-based version of the Consensus Sleep Diary. Upon arrival to the sleep laboratory, participants were given a tour and reminded of the study procedures. On all nights following dinner (approximately 6:30 pm), participants were set up for PSG recording. Prior to bed, participants were reminded that they may or may not hear noises during the night and to try to sleep as normal. Lights-out time was their habitual bedtime (average bedtime during baseline reported on the sleep diary).

Wake-up times were self-selected by participants prior to lights-out time on each night. Following morning awakening at the prescribed time, participants completed the online sleep diary and responded to questions about noise-related sleep disruption, which took on average 5–10 min to complete. Participants were then free to have breakfast and leave the laboratory (around 9:00–10:00 am) until 5:30 pm that evening for the next study night. On one occasion during the 7-night laboratory stay, participants also underwent an extensive hearing assessment by an independent audiologist to assess hearing thresholds via pure tone audiometry between 125 and 8000 Hz in each ear in an audiology booth.

Data and statistical analysis

Statistical analyses were conducted with IBM Statistical Package for Social Sciences (SPSS; Version 25). Based on the primary outcome of sleep efficiency and previous reports of relatively low between- (*SD* approximately 10% [39]) and within-subject variability over consecutive nights (approximately 3% [40]), we estimated that a repeated measures design with four groups of approximately 17 participants would have approximately 80% power to detect an absolute difference in sleep efficiency in the order of 4.5% between groups and 1.8% between noise conditions. Thus, a target sample size of 20 participants per group was selected to allow for some study technical failures and attrition.

Variables that failed normality tests were transformed ($\log_{10}$ or a Box-Cox selected transform if required) prior to further statistical analyses and *p*-values indicated with an * indicate results based on transformed data.

Group differences in demographics and baseline sleep characteristics were analyzed firstly, using linear mixed model analyses with a first-order autoregressive covariance structure and subject specified as a random effect, each with their own intercept. Given statistically significant age differences between groups, age was subsequently included as a covariate. Statistically significant group effects were examined using Sidak adjusted pairwise comparisons.

For all linear mixed model analyses, the acclimatization night (night 1) was initially included in the analysis to test for potential "first-night effects" and then excluded to control for such effects in follow-up analyses. For all primary and secondary outcomes, effects of noise condition, group, and prior night noise exposure condition were analyzed using linear mixed model analyses using a first-order autoregressive covariance structure, noise condition, and prior night condition specified as a repeated measure within-subjects, and subject ID as a random effect, each with their own intercept. Alternative covariance structures including (scaled identity, unstructured, and diagonal) were examined using Akaike's Information Criterion and for the most part demonstrated that AR(1) consistently provided the best model fit. Given statistically significant age and hearing threshold differences between groups, age and hearing threshold were included as a covariate and random effect. The prior night noise condition was included to test for potential carry-over/order effects between nights and adjusted for when significant order effects were present. Statistically significant main or interaction effects were examined using Sidak adjusted pairwise comparisons within each linear model following adjustment for significant order effects when present.

Spearman's rank correlations (r_s) were used to explore associations between perceived noise sensitivity and changes in PSG and sleep diary determined sleep efficiency in the presence of WTN-Continuous versus control conditions. Spearman's rank correlations (r_s) were also used to explore associations between noise sensitivity and sleep efficiency in the control condition alone. These analyses were also carried out for PSG and sleep diary determined total sleep time, wake after sleep onset, sleep latency, number of awakenings, time in bed as well as PSG sleep stage outcomes.

Bland-Altman analyses were also conducted to assess for potential bias between the primary and secondary PSG and sleep diary parameters listed above. Pearson chi-square tests were used to test for differences in proportions of individuals within each group with different characteristics, including sleep efficiencies <85% and sleep latencies of >30 and >20 min in the WTN-Continuous and control conditions. This was to allow for comparisons with previous studies using commonly used cut-offs for discriminating good sleep from poor sleep [41–44]. Pearson chi-square tests were also used to test for differences in the proportion of individuals within each group who had high perceived noise sensitivity scores (>78) [38]. In a secondary analysis, perceived noise sensitivity was also included as a covariate along with age to test and adjust for potential effects on PSG and sleep diary determined sleep efficiency.

Finally, paired-samples *t*-tests were used to determine if participant's self-reported sleep efficiency, sleep latency, total sleep time, and wake after sleep onset 2 weeks prior to their sleep study at home differed compared to self-reported sleep in the laboratory during the control and WTN-Continuous conditions, and for each participant group separately. All data are presented as median and interquartile range unless otherwise specified. p values < .05 were considered statistically significant.

Results

Study participants

Figure 2 shows a CONSORT diagram of the number of individuals screened from which 68 participants aged 18–80 years participated in the study. From 240 individuals responding to study advertisements, 172 were excluded (104 declined to participate, 65 did not meet the study criteria, and 3 resided interstate and were unable to travel given extended COVID-19 border restrictions). Further reasons for exclusion included urban residents not reporting RTN-related sleep disruption and faster recruitment into the RTN-sleep disturbed group risking group imbalance away from the primary WTN exposure groups of interest.

Demographics and baseline sleep characteristics of the study participants are presented in Table 1. The majority of participants (61/68 or 89.7% of the overall sample) were of Caucasian/European descent, with no differences in proportions between groups. On average, participants in the WTN-sleep disturbed and WTN-non-sleep disturbed groups lived between 2–4 and 4–6 km from the nearest wind turbine respectively. All rural and RTN-sleep disturbed participants indicated living > 10 km from a wind turbine. The WTN-sleep disturbed group lived on average 0.9 km from the nearest road traffic noise source compared to 0.4 km, 0.4 km, and 0.2 km for the WTN-non-sleep disturbed group, rural control, and RTN-sleep disturbed group, respectively.

There were significant age differences between groups, where the WTN-sleep disturbed group was significantly older than the rural control (mean [95% CI] difference of 19.6 [4.1 to 35.0] years, $p = .006$) and RTN-sleep disturbed group (32.8 [17.4 to 48.2] years, $p < .001$) and the WTN-non sleep disturbed group was significantly older than the RTN-sleep disturbed group (20.7 [6.3 to 35.2] years, $p = .001$) (Table 1). Given age differences, all further analyses were adjusted for age. After age adjustment, the WTN-sleep disturbed group showed significantly higher ESS, ISI, PSQI, perceived noise sensitivity scores, and hearing thresholds for frequencies 125–1000 Hz compared to the rural control group, and higher ISI and PSQI scores than the RTN-sleep disturbed group (Table 1). The WTN-non-sleep disturbed group had significantly greater body mass index scores compared to the RTN-sleep disturbed group. Noise sensitivity scores were also higher in the WTN-sleep disturbed group versus the rural control group (Table 1).

By participant selection design, there was significantly greater self-reported WTN-related sleep disruption in the WTN-sleep disturbed group, in the moderate-severe disruption range, versus the three other groups. There was also significantly greater, and moderate-to-severe, self-reported RTN-related sleep disruption in the RTN-sleep disturbed versus the three other groups who reported no or mild disruption. However, there were no further differences in baseline measures of sleep time or quality between groups (Table 1).

First-night effects

There were significant differences between nights for PSG total sleep time ($p = .005$), time in bed ($p = .039$), time spent in REM sleep ($p < .001$), N1 % ($p = .003$), N2% ($p < .001$), N3% ($p < .001$), REM latency ($p = .016$), and total wake time ($p = .034$).

Pairwise comparisons revealed significantly lower PSG total sleep time in the acclimatization night compared to the control night ($p = .019$), the WTN-sleep night ($p = .005$), and the WTN-Continuous night ($p = .037$). Furthermore, there was significantly lower PSG time in bed in the acclimatization night compared to the WTN-Sleep ($p = .033$) night and significantly lower time spent in REM, N1%, N2%, and N3% in the acclimatization night compared to all four noise exposure conditions (all $ps < .05$). REM latency was also significantly longer on the acclimatization night compared to the control night ($p = .010$). Total wake time was also significantly greater on the acclimatization night compared to the WTN-Sleep night ($p = .044$).

There were no other significant differences between nights for any other PSG or sleep diary determined sleep parameters. First night effects were controlled by acclimatization night inclusion and randomization of subsequent nights, so the acclimatization night (night 1) was excluded in further analyses.

Group-by-noise condition interaction effects

Figure 3A shows PSG and sleep diary determined sleep efficiency during the background noise (control), WTN-Continuous, WTN-Sleep, and WTN-Wake exposure conditions within each group as well as the overall group effect irrespective of noise condition (combined) and shaded plots that indicate the overall noise condition effect irrespective of group. Figure 3B shows change in PSG sleep efficiency and sleep diary sleep efficiency from the control condition for each noise condition including a combined noise condition effect within each group and the overall noise condition effect irrespective of group (shaded plots). Tables 2 and 3 show the descriptive statistics for PSG and sleep diary determined sleep outcomes for each group and across each noise condition respectively and Table 4 shows the descriptive statistics for PSG sleep stage outcomes for each group and across each noise condition. As indicated by Figure 3, A and B, there were no significant group-by-noise condition interaction effects on PSG or sleep diary determined sleep efficiency. Furthermore, Tables 2–4 show no significant group-by-noise condition interaction effects on PSG or sleep diary determined sleep latency, wake after sleep onset, total sleep time, time in bed, number of awakenings, or any PSG determined sleep stage outcomes (see Supplementary Tables S1 and S2 and Supplementary Figures S1 and S2 or further details).

There were also no significant differences in the proportions of participants with PSG or sleep diary sleep efficiencies <85% across the four participant groups during the WTN-Continuous condition (PSG: WTN-sleep disturbed 9/14, WTN-non sleep disturbed 9/18, rural control 6/18, RTN-sleep disturbed 7/16, $p = .367$; Sleep diary: WTN-sleep disturbed 8/12, WTN-non sleep disturbed 7/18, rural control 8/18, RTN-sleep disturbed 10/16, $p = .336$), control condition (PSG: WTN-sleep disturbed 9/14, WTN-non sleep disturbed 8/18, rural control 7/18, RTN-sleep disturbed 3/16, $p = .088$; Sleep diary: WTN-sleep disturbed 8/14, WTN-non sleep disturbed 8/17, rural control 7/18, RTN-sleep disturbed 10/16, $p = .528$). There were also no significant differences in the proportion of participants with sleep latencies >30 or >20 min between groups or conditions.

Prior nights noise condition main effects

There were no statistically significant main effects of prior night's noise condition, apart from PSG time in bed ($p < .01$). However, no further PSG time in bed effects were apparent following adjustment for prior night condition effects.

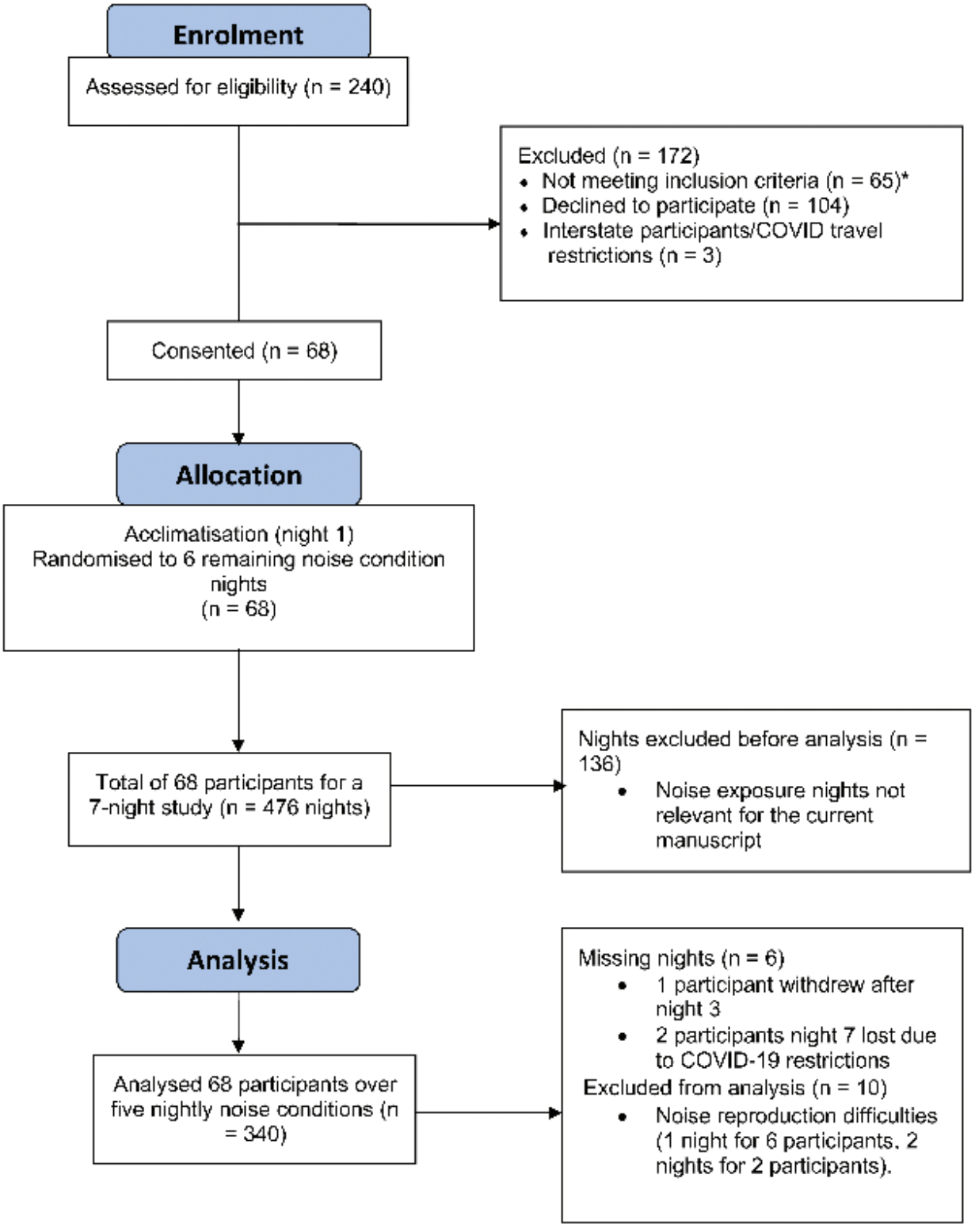

Figure 2. CONSORT flow diagram showing the process from enrolment into the study to analysis. *Reasons for exclusion included urban residents not reporting RTN related sleep disruption or recruitment capacity reached for the RTN-sleep disturbed group. RTN, road traffic noise.

Noise condition main effects

There were no statistically significant main effects of noise condition for Box-Cox transformed PSG or sleep diary determined sleep efficiency. The untransformed mean (95% CI) PSG determined sleep efficiency for the control, WTN-Sleep, WTN-Continuous, and WTN-Wake conditions was 86.4 (83.1 to 89.7), 85.5 (82.7 to 88.4), 85.3 (82.1 to 88.5), and 84.7 (81.9 to 87.6] % respectively. For the sleep diary determined sleep efficiency,

Table 1. Participant demographics and baseline at-home sleep monitoring

Characteristic	WTN-sleep disturbed	WTN-non sleep disturbed	Rural control	RTN-sleep disturbed	*P*
Demographics					
Females:males *N* (%)	7:7 (50:50)	9:9 (50:50)	14:4 (78:22)	8:10 (44:56)	.092
Age (years)	66.3 ± 6.9	54.2 ± 16.3	46.7 ± 20.7*	33.5 ± 15.1*,‡	<.001
BMI (kg/m^2)	27.7 ± 3.4	29.9 ± 5.0	27.9 ± 5.8	24.8 ± 5.9‡	.039
ESS	9.4 ± 4.2	5.5 ± 4.0	4.6 ± 2.8*	6.3 ± 5.2	.014
ISI	12.6 ± 5.9	7.8 ± 5.1	6.3 ± 3.2*	6.5 ± 4.3*	.003
PSQI	10.9 ± 3.5	7.5 ± 3.7	6.2 ± 3.1*	6.2 ± 3.2*	.001
Weinstein noise sensitivity[a]	70.9 ± 14.1	61.6 ± 18.8	52.9 ± 13.6*	65.4 ± 17.4	.019
Degree of WTN related sleep disruption[b]	3.7 ± 1.3	1.0 ± 0.0*	1.0 ± 0.0*	1.0 ± 0.0*	<.001
Degree of RTN related sleep disruption[b]	1.6 ± 1.2	1.5 ± 0.9	1.3 ± 0.5	3.5 ± 0.8*,‡,†	<.001
Hearing Level 125–1000 Hz (dB HL)[c]	15.7 ± 14.5	10.0 ± 10.1	8.3 ± 11.2	6.1 ± 6.3*	.031
Baseline at-home self-reported sleep monitoring					
Habitual bed time (hrs:mins)	22:42 ± 1:30	22:36 ± 3:42	22.48 ± 2:36	23:18 ± 2:24	.193
Habitual wake time (hrs:mins)	6:30 ± 1:0	7:18 ± 1:30	7:06 ± 1:18	7:30 ± 1:54	.208
Total sleep time (hrs)	7.0 ± 1.5	7.2 ± 1.4	7.3 ± 1.3	7.6 ± 1.6	.109
Sleep latency (min)	17.2 ± 25.7	24.7 ± 30.7	19.3 ± 20.4	23.0 ± 25.6	.740
Sleep efficiency (%)	95.6 ± 21.6	93.6 ± 20.1	199.0 ± 465.4*	96.7 ± 20.8	.364
Wake after sleep onset (min)	39.2 ± 44.7	30.9 ± 45.5	27.9 ± 40.0	23.1 ± 30.8	.304
Number of awakenings	2.1 ± 1.4	2.1 ± 1.8	1.7 ± 1.5	1.8 ± 1.6	.474

N = 68. Values are M ± *SD*. All *P* values reflect untransformed data.

[a]Cut-offs for the Weinstein Noise Sensitivity Scale ≥ 78 indicates high noise sensitivity, scores < 26 indicate low noise sensitivity based on upper and lower quartiles of the original study [35].

[b]Scored on a 5-point Likert scale (1 = not at all, 2 = mildly, 3 = moderately, 4 = severely, 5 = very severely) regarding "noise from wind turbines/road traffic bother, disturb or annoy you while you are in bed trying to sleep within the last 12 months".

[c]Normal hearing range <20 dB HL.

P < .05 *versus WTN-sleep disturbed group, ‡versus the WTN-non-sleep disturbed group, †versus the rural control group. *Sleep efficiency calculation illustrates participants inaccurate reporting time in bed and total sleep time.

WTN, Wind Turbine Noise; RTN, Road Traffic Noise; BMI, body mass index; ESS, Epworth Sleepiness Scale; ISI, Insomnia Severity Index; PSQI, Pittsburgh Sleep Quality Index; dB, decibel; HL, hearing level.

the untransformed mean (95% CI) for the control, WTN-Sleep, WTN-Continuous, and WTN-Wake conditions was 79.9 (73.5 to 86.3), 83.2 (77.7 to 88.6), 75.1 (68.9 to 81.2), and 75.3 (69.9 to 80.6) % respectively. There were no other statistically significant main effects of noise condition for PSG or sleep diary determined sleep latency, wake after sleep onset, total sleep time, number of awakenings, time spent in bed, or any of the PSG sleep stage outcomes.

Group main effects

There were no statistically significant group main effects on Box-Cox transformed PSG or sleep diary determined sleep efficiency. The untransformed mean (95% CI) PSG determined sleep efficiency for the WTN-sleep disturbed group, WTN-non-sleep disturbed group, the rural group, and RTN-sleep disturbed group was 79.3 (73.6 to 85.0), 85.7 (81.4 to 90.1), 87.7 (83.8 to 91.5), and 88.7 (85.1 to 92.3) % respectively. For sleep diary determined sleep efficiency, the untransformed mean (95% CI) for the WTN-sleep disturbed group, WTN-non sleep disturbed group, the rural group, and RTN-sleep disturbed group was 73.2 (64.8 to 81.5), 81.2 (73.7 to 88.7), 81.4 (73.8 to 89.0), and 78.5 (70.8 to 86.1) % respectively.

There was a significant main effect of group on PSG determined wake after sleep onset (p = .004), which was higher in the WTN-sleep disturbed group (Mean [95% CI] 98.3 [65.4 to 131.2] min) than the rural group (38.1 [18.3 to 57.9] min, p = .016) and the RTN-sleep disturbed group (32.0 [15.5 to 48.6] min, p = .004), but there were no further main effects of group in sleep diary determined wake after sleep onset (see Supplemental Figure S3 for more details).

Although there was a statistically significant main effect of group (p = .040) on total time spent in REM sleep (WTN-sleep disturbed group 82.4 [63.5 to 101.3] minutes, WTN-non sleep disturbed group 98.3 [85.8 to 110.9] minutes, rural group 105.7 [93.1 to 118.3] minutes, and RTN-sleep disturbed group 111.8 [100.5 to 123.1] minutes) there were no significant post-hoc pairwise differences between groups (see Supplemental Figure S3 for more details). Furthermore, there were no other statistically significant main effects of group for PSG or sleep diary determined sleep latency, total sleep time, number of awakenings, time spent in bed, or any other PSG sleep stage outcomes.

PSG versus sleep diary parameters

Across all participants (all groups combined), all PSG sleep parameters were significantly positively correlated with their sleep diary determined counterparts under each of the noise exposure conditions (Supplementary Table S3). Furthermore, Bland-Altman analysis showed no evidence to support systematic bias between PSG versus sleep diary determined sleep efficiency, sleep latency, total sleep time, wake after sleep onset, or number of awakenings (all *p*s > .05).

At home monitoring versus in laboratory self-reported sleep outcomes

Self-reported wake after sleep onset in the WTN-Continuous condition was greater in the laboratory compared to at home in the WTN-sleep disturbed group (mean difference [95% CI] 122.6 [32.2 to 213.0] minutes, p = .013), the rural control group (90.3 [12.6 to 167.9] minutes, p = .025), and the RTN-sleep disturbed

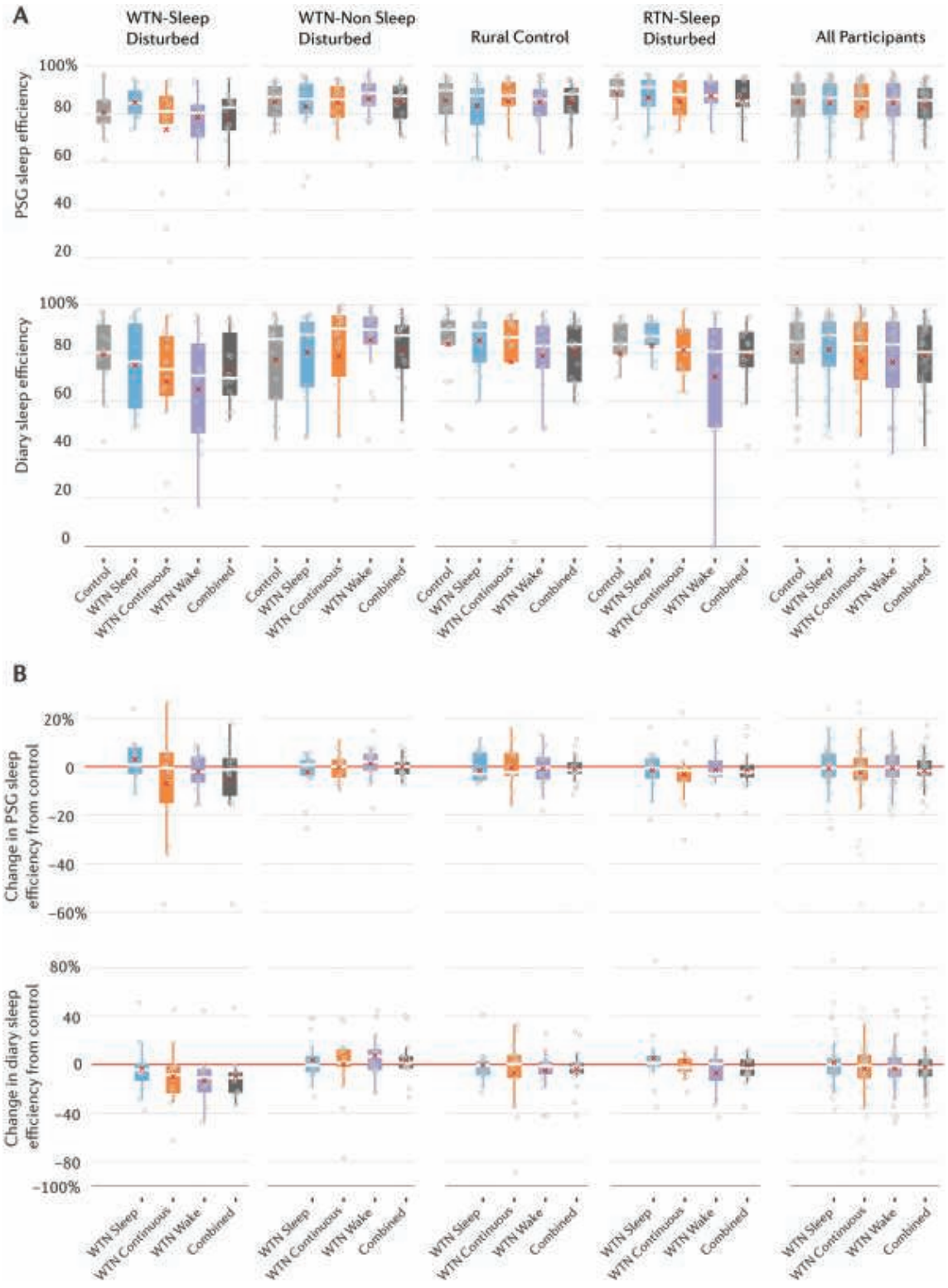

Figure 3. Box and whisker plots showing (A) PSG sleep efficiency (%) (upper panel) and sleep diary determined sleep efficiency (%) (second panel) across groups and noise conditions and (B) difference scores from control (for each WTN condition) for PSG and sleep diary determined sleep efficiency for each group. Plots depict group mean (X) and median (gaps) for each group (WTN-sleep disturbed, $n = 14$; WTN-non sleep disturbed, $n = 18$; rural control, $n = 18$; RTN-sleep disturbed, $n = 18$; and across all participants, $n = 68$) and under each noise condition (control background noise, WTN-continuously across the night, WTN-only during sleep periods, WTN-only during wake period exposures, and combined across all noise conditions). The box bounds the IQR divided by the median. Whiskers are Tukey-style (extend to a maximum 1.5 × IQR beyond the box as described in Krzywinski and Altman [62]. Circles indicate individual data points. PSG, polysomnography; WTN, wind turbine noise; RTN, road traffic noise.

Table 2. Median [IQR] PSG sleep outcomes for each group and noise condition

Group	Noise condition	Sleep efficiency (%)	Sleep latency (mins)	Total sleep time (mins)	Wake after sleep onset (mins)	Time spent in bed (mins)	Number of awakenings (n)
WTN-sleep disturbed	Control	79.8 [76.3 to 85.5]	5.3 [3.1 to 11.9]	430.3 [398.3 to 439.3]	97.8 [66.8 to 116.9]	509.3 [488.1 to 535.4]	31.5 [24.5 to 44.0]
	WTN-Continuous	81.1 [76.2 to 87.1]	7.3 [6.1 to 13.6]	407.3 [339.6 to 452.5]	85.3 [48.8 to 126.6]	494.8 [455.1 to 548.0]	34.0 [19.0 to 40.3]
	WTN-Sleep	84.4 [80.2 to 89.4]	6.0 [3.5 to 10.9]	438.0 [414.6 to 458.5]	67.5 [55.0 to 89.4]	510.8 [480.3 to 539.9]	30.5 [23.8 to 35.3]
	WTN-Wake	80.4 [70.5 to 83.5]	10.0 [6.5 to 16.5]	418.0 [383.5 to 428.0]	79.0 [52.0 to 143.0]	526.5 [467.0 to 539.0]	27.0 [26.0 to 32.0]
WTN-non-sleep disturbed	Control	87.1 [79.1 to 91.2]	10.8 [9.3 to 19.9]	439.0 [396.9 to 466.5]	46.3 [31.5 to 80.8]	513.0 [473.0 to 541.3]	31.0 [27.3 to 45.0]
	WTN-Continuous	85.9 [78.6 to 91.2]	13.3 [5.5 to 17.0]	440.3 [394.9 to 477.0]	60.5 [26.3 to 82.8]	518.8 [473.0 to 531.8]	35.0 [22.3 to 47.3]
	WTN-Sleep	86.2 [80.4 to 92.2]	10.0 [6.5 to 19.0]	430.5 [384.8 to 482.0]	51.0 [22.8 to 92.8]	506.5 [462.3 to 537.5]	29.0 [24.0 to 31.0]
	WTN-Wake	88.6 [83.5 to 92.5]	11.8 [8.8 to 16.9]	444.8 [403.0 to 476.1]	38.5 [29.5 to 68.0]	509.5 [471.0 to 543.5]	33.5 [23.0 to 39.8]
Rural control	Control	89.8 [80.1 to 92.5]	15.5 [8.6 to 34.1]	446.3 [411.5 to 467.9]	33.5 [19.5 to 64.9]	510.5 [494.8 to 555.6]	30.5 [20.5 to 38.8]
	WTN-Continuous	87.6 [83.6 to 92.8]	18.0 [8.9 to 22.5]	439.3 [396.0 to 497.4]	52.0 [18.9 to 70.4]	513.8 [467.9 to 539.4]	31.5 [18.3 to 42.0]
	WTN-Sleep	87.6 [75.7 to 90.4]	12.8 [8.8 to 22.0]	425.8 [386.1 to 478.0]	49.0 [23.1 to 125.8]	512.3 [476.5 to 542.6]	30.0 [20.8 to 40.0]
	WTN-Wake	86.0 [79.4 to 89.9]	17.5 [7.9 to 22.9]	431.5 [416.0 to 474.3]	48.5 [20.0 to 75.4]	506.3 [482.1 to 534.8]	30.0 [21.3 to 37.5]
RTN-sleep disturbed	Control	90.7 [87.2 to 94.2]	16.3 [9.8 to 41.0]	410.5 [391.6 to 452.4]	19.5 [11.3 to 32.8]	477.3 [430.0 to 527.8]	22.0 [20.0 to 24.0]
	WTN-Continuous	88.0 [79.7 to 93.6]	17.3 [10.1 to 21.5]	447.5 [405.3 to 493.5]	37.3 [15.4 to 56.1]	525.5 [469.6 to 569.1]	31.5 [20.8 to 37.5]
	WTN-Sleep	91.1 [83.2 to 93.8]	15.0 [7.9 to 24.1]	447.5 [372.8 to 492.0]	24.3 [15.3 to 58.1]	513.0 [444.6 to 566.9]	27.5 [26.8 to 30.3]
	WTN-Wake	87.6 [84.8 to 93.3]	19.0 [9.0 to 37.0]	427.5 [392.0 to 468.0]	20.5 [12.0 to 48.0]	509.0 [420.0 to 527.5]	27.0 [17.0 to 35.0]

Values represent Median [IQR] for each participant group (WTN-sleep disturbed, n = 14; WTN-non-sleep disturbed, n = 18; rural control, n = 18; and RTN-sleep disturbed, n = 18) under each noise condition.
WTN, wind turbine noise; RTN, road traffic noise; mins, minutes; n, number; IQR, interquartile range.

group (31.8 [8.1 to 55.5] minutes, p = .012). Self-reported wake after sleep onset was also higher in the laboratory compared to home during the control condition in the WTN-non sleep disturbed group (83.4 [10.3 to 156.5] minutes, p = .028) and the RTN-sleep disturbed group (33.9 [10.7 to 48.2] minutes, p=0.005); who also self-reported reduced total sleep time in the laboratory compared to at home (–1.6 [–3.1 to 0.09] hours, p = .040) (See Supplementary Table S4 for further details).

Perceived noise sensitivity

Twelve participants (17.6%) in total were classified as highly noise-sensitive (>78 in top quartile of the Weinstein Noise Sensitivity Scale). However, there were no significant differences in the proportion of noise-sensitive individuals between groups (p = .070; 3/14 (21.4%) in the WTN-sleep disturbed group, 3/18 (16.7%) in the WTN-non-sleep disturbed group, 6/18 (33.3%) in the RTN-sleep disturbed group, and none in the rural control group). After adjusting for age and noise sensitivity there remained no significant group, noise exposure condition or interaction effects on PSG or sleep diary determined sleep efficiency.

There was a significant negative correlation between noise sensitivity and PSG sleep efficiency in the control condition ($r_s(63) = -0.400$, $p < .001$), but not for sleep diary determined sleep efficiency. However, there were no significant correlations between perceived noise sensitivity and the control minus WTN-Continuous condition sleep efficiency difference with either PSG sleep efficiency ($r_s(63) = -0.012$, p = .923) or sleep diary determined sleep efficiency ($r_s(61) = 0.140$, p = .276), or any other objective or subjective sleep parameters.

Discussion

This is the largest laboratory study reported to date that has investigated the impact of WTN on PSG and sleep diary parameters. It was hypothesized that both objective PSG and sleep diary derived sleep parameters would be more disrupted, with more wake and less sleep, on the three WTN nights (WTN-Continuous, WTN-Sleep, WTN-Wake) compared to the quiet control night, with a greater difference in residents reporting WTN-related sleep disruption versus undisturbed residents. After adjusting for age and hearing thresholds, the WTN-sleep disturbed group showed significantly greater PSG wake after sleep onset than the rural and RTN-sleep disturbed group, but with no differences between noise conditions suggestive of poorer sleep overall. Wake after sleep onset increases with age, and in the oldest group, was perhaps greater than expected age-related normal values for healthy older adults (WTN-sleep disturbed: mean [95% CI] 98.3 [65.4 to 131.2] minutes compared to approximately 70 min for healthy individuals between 66 and 83 years [45]). Furthermore, ISI scores were higher in the WTN-sleep disturbed group compared to the rural control and RTN exposure groups and suggestive of subthreshold insomnia (ISI 8–14) rather than clinical insomnia (ISI ≥ 15 [46]). ESS scores also tended to be higher, but remained below the standard clinical cut off >10 for defining significant sleepiness [47]. Furthermore, all groups showed PSQI global scores >5 suggestive of relatively poor self-reported sleep quality [48], particularly in the WTN-sleep disturbed group. Therefore, despite no consistent in-laboratory WTN effects on sleep, the WTN-sleep disturbed group showed consistent evidence of poorer sleep overall compared to the remaining groups.

Failure to demonstrate significant effects of WTN exposure on the primary sleep efficiency outcome could potentially reflect Type II error. Based on previously reported data we estimated that this sample size had 80% power to detect an absolute difference in sleep efficiency in the order of 4.5% for group-by-noise condition comparisons and 1.8% between noise conditions. Based on average total sleep time of around 7 and 8.4 h of time in bed (sleep efficiency 83%), this would equate to around a 19- and 7.5-minute difference in sleep time between groups and conditions respectively. However, the overall findings do

Table 3. Median [IQR] sleep diary determined sleep outcomes for each group and noise condition

Group	Noise condition	Sleep efficiency (%)	Sleep latency (mins)	Total sleep time (mins)	Wake after sleep onset (mins)	Time spent in bed (mins)	Number of awakenings (*n*)
WTN-sleep disturbed	Control	80.2 [73.4 to 91.3]	20.0 [11.3 to 27.5]	405.0 [360.0 to 448.8]	75.0 [18.8 to 112.5]	517.5 [482.5 to 558.8]	3.0 [1.3 to 3.0]
	WTN-Continuous	73.2 [62.5 to 86.6]	30.0 [18.8 to 60.0]	420.0 [285.0 to 431.8]	120.0 [48.8 to 165.0]	540.0 [507.3 to 577.5]	2.5 [2.0 to 3.8]
	WTN-Sleep	76.4 [57.3 to91.8]	30.0 [10.0 to 60.0]	420.0 [330.0 to 457.5]	60.0 [18.8 to 135.0]	550.0 [495.0 to 576.0]	3.0 [2.0 to 5.0]
	WTN-Wake	70.6 [47.1 to 83.6]	30.0 [15.0 to 70.0]	365.0 [262.5 to 426.3]	120.0 [30.0 to 180.0]	550.0 [472.5 to 566.3]	3.0 [2.0 to 5.0]
WTN-non-sleep disturbed	Control	85.7 [61.1 to 91.1]	20.0 [15.0 to 35.0]	450.0 [360.0 to 490.0]	60.0 [20.0 to 150.0]	540.0 [520.0 to 585.0]	2.0 [1.0 to 4.0]
	WTN-Continuous	90.1 [70.6 to 95.2]	20.0 [11.3 to 56.3]	425.0 [360.0 to 480.0]	30.0 [11.3 to 103.8]	517.5 [490.0 to 543.8]	2.0 [1.0 to 4.0]
	WTN-Sleep	88.0 [76.4 to 93.3]	20.0 [15.0 to 30.0]	440.0 [397.5 to 475.0]	30.0 [15.0 to 90.0]	495.0 [465.0 to 540.0]	3.0 [2.0 to 3.0]
	WTN-Wake	89.6 [83.9 to 94.7]	15.0 [10.0 to 30.0]	425.0 [411.3 to 480.0]	22.5 [15.0 to 57.5]	517.5 [476.3 to 547.5]	2.0 [0.3 to 4.0]
Rural control	Control	89.7 [83.5 to 93.0]	20.0 [10.0 to 30.0]	450.0 [425.0 to 475.0]	15.0 [10.0 to 63.8]	500.0 [483.3 to 573.8]	2.5 [1.3 to 4.0]
	WTN-Continuous	86.5 [76.4 to 93.4]	22.5 [15.0 to 30.0]	440.0 [375.0 to 483.8]	45.0 [10.0 to 115.0]	532.5 [490.0 to 550.0]	2.0 [1.0 to 4.8]
	WTN-Sleep	89.8 [76.1 to 92.7]	20.0 [15.0 to 32.5]	465.0 [417.5 to 500.0]	30.0 [17.5 to 67.5]	520.0 [506.3 to 545.0]	2.0 [1.8 to 3.3]
	WTN-Wake	82.9 [73.9 to 90.9]	25.0 [16.3 to 37.5]	462.5 [420.0 to 480.0]	50.0 [22.5 to 92.5]	532.5 [506.3 to 592.5]	3.0 [1.3 to 3.8]
RTN-sleep disturbed	Control	83.6 [79.7 to 91.9]	30.0 [15.0 to 30.0]	410.0 [378.8 to 450.0]	40.0 [28.8 to 60.0]	477.5 [450.0 to 519.0]	2.0 [1.0 to 2.3]
	WTN-Continuous	81.4 [72.9 to 89.7]	25.0 [18.8 to 60.0]	420.0 [356.3 to 482.5]	50.0 [8.8 to 63.8]	513.0 [477.5 to 545.5]	2.0 [1.0 to 3.3]
	WTN-Sleep	86.9 [83.5 to 92.1]	20.0 [18.0 to 30.0]	420.0 [412.5 to 457.7]	35.0 [27.5 to 60.0]	500.0 [454.3 to 562.5]	3.0 [2/0 to 4.0]
	WTN-Wake	80.5 [49.6 to 90.0]	30.0 [18.8 to 32.5]	427.5 [345.0 to 451.3]	60.0 [0.0 to 90.0]	477.5 [420.0 to 555.0]	1.5 [0.8 to 2.3]

Values represent Median [IQR] for each participant group (WTN-sleep disturbed, *n* = 14; WTN-non-sleep disturbed, *n* = 18; rural control, *n* = 18; and RTN-sleep disturbed, *n* = 18) under each noise condition.
WTN, wind turbine noise; RTN, road traffic noise; mins, minutes; *n*, number; IQR, interquartile range.

not support significant group or condition effects on conventional PSG or sleep diary outcomes, or that residents living near a wind farm and reporting WTN-related sleep disruption exhibit a conditioned response to WTN exposure at levels approximating typical levels in the field. Several first-night effects were detected, supporting that WTN-specific exposure effects of a similar magnitude would likely also have been detected with this sample size.

These results are consistent with a previous laboratory study which found no significant differences in PSG or sleep diary measured sleep latency in the presence versus absence of WTN during the sleep onset period in healthy good sleepers not habitually exposed to WTN [20]. The current results are also consistent with the WiTNES study [18], which found no differences in PSG measured sleep outcomes on WTN nights versus control nights in participants both with and without habitual WTN exposure. However, in the WiTNES study, REM latency increased, and REM sleep time was reduced in WTN versus control nights [18], while in the current study there were no significant main effects of noise conditions for REM sleep time. Subjective sleep outcomes were more difficult to compare with the WiTNES study, given the current study used the more widely used and psychometrically validated Consensus Sleep Diary [33], while the WiTNES study used a morning Likert rating scale to assess self-reported sleep disruption [18]. In addition, the WiTNES study used a different noise delivery protocol to the current study, which involved varying synthesized WTN samples that had different noise levels and frequency content, whereas the current study used a real-world recorded WTN sample to approximate median WTN levels measured in the field (25 dB(A)).

The present results were also somewhat different from those reported by Ageborg Morsing *et al.* [16] who found more awakenings and reduced N3 sleep during WTN exposure compared to quiet control nights, but no further significant effects on other objective or self-reported sleep outcomes. However, only six healthy participants without prior WTN exposure were studied and WTN exposures were more representative of outdoor WTN levels and thus worst-case exposure conditions compared to the current study [16, 49]. In the current study, 25 dB(A) is very similar to the median yearly indoor WTN levels of 26 dB(A) recorded from another study [29]. Effects of WTN on sleep are likely to be greatest during worst-case conditions, but the results from this study support that at median noise exposure levels effects on sleep are relatively minimal.

Despite the WTN-sleep disturbed group self-reporting habitual WTN-related sleep disruption, the proportion of insomnia-like symptoms, such as sleep latency >30 min and <85% sleep efficiency, were not significantly different from other groups or impacted by continuous WTN exposure compared to control conditions. There were also no differences in the proportion of participants with sleep latencies >20 min in the WTN-Continuous versus control condition. This finding is similar to a previous field study by Jalali *et al.* [44] which found that 12.5% of participants (*n* = 2/16) showed sleep latencies >20 min post-operational WTN exposure, with no difference compared to preoperational WTN exposure, but also with no detectable change in environmental noise levels pre- versus post-operation suggesting WTN levels were below measurable limits.

ISI scores were higher in the WTN-sleep disturbed group compared to rural control and RTN exposure groups and more suggestive of subthreshold insomnia (ISI 8–14) rather than clinical insomnia (ISI $_{\geq}$ 15 [46]). ESS scores also tended to be higher but below the standard clinical cut-off >10 for defining significant sleepiness [47]. Furthermore, all groups showed PSQI global scores >5 suggestive of relatively poor self-reported sleep quality [48], particularly in the WTN-sleep disturbed group which also showed higher PSG wake after sleep onset than in the rural control and RTN-sleep disturbed groups. Therefore, despite no

Table 4. Median [IQR] PSG sleep stage outcomes for each group and noise condition

Group	Noise condition	N1 (mins)	N2 (mins)	N3 (mins)	REM (mins)	N1 %
WTN-sleep disturbed	Control	32.3 [26.6 to 63.3]	195.3 [171.0 to 208.5]	94.8 [56.1 to 118.3]	86.0 [59.5 to 109.9]	9.9 [6.0 to 14.6]
	WTN-Continuous	29.0 [21.6 to 35.9]	192.8 [157.4 to 214.5]	101.5 [64.1 to 112.6]	84.8 [42.4 to 109.1]	8.6 [5.9 to 10.9]
	WTN-Sleep	37.8 [24.4 to 43.9]	199.8 [179.9 to 212.1]	107.3 [59.5 to 119.4]	101.3 [71.1 to 113.1]	8.2 [5.5 to 11.1]
	WTN-Wake	30.5 [28.0 to 47.5]	186.5 [158.5 to 212.5]	65.5 [39.5 to 122.0]	91.5 [73.0 to 99.0]	7.7 [7.1 to 12.1]
WTN-non-sleep disturbed	Control	41.8 [28.8 to 64.9]	199.5 [169.9 to 220.4]	83.0 [44.6 to 116.4]	96.8 [76.0 to 105.9]	9.5 [7.5 to 16.9]
	WTN-Continuous	42.8 [23.0 to 67.5]	199.3 [178.8 to 216.5]	73.3 [45.3 to 118.6]	92.0 [82.6 to 105.6]	10.9 [5.0 to 17.3]
	WTN-Sleep	41.0 [21.5 to 52.3]	197.5 [160.8 to 215.0]	80.0 [65.3 to 113.8]	106.0 [76.5 to 117.0]	8.7 [5.9 to 14.6]
	WTN-Wake	51.3 [26.4 to 59.3]	197.5 [174.3 to 230.3]	71.8 [45.8 to 110.0]	103.8 [81.0 to 120.5]	11.4 [5.8 to 16.1]
Rural control	Control	31.3 [26.6 to 53.6]	219.8 [184.1 to 242.4]	80.0 [49.0 to 113.6]	108.3 [84.4 to 120.4]	7.3 [5.4 to 12.3]
	WTN-Continuous	33.0 [27.3 to 47.9]	202.3 [190.8 to 236.3]	83.3 [53.6 to 116.0]	97.5 [88.4 to 121.0]	7.7 [5.5 to 14.6]
	WTN-Sleep	36.8 [26.0 to 55.6]	186.5 [163.3 to 218.1]	88.0 [74.0 to 112.3]	96.5 [75.6 to 137.6]	8.5 [5.3 to 14.5]
	WTN-Wake	31.3 [23.8 to 51.6]	215.5 [179.6 to 230.3]	88.5 [63.3 to 122.6]	91.8 [79.8 to 107.9]	7.0 [5.2 to 12.1]
RTN-sleep disturbed	Control	27.3 [22.3 to 35.6]	196.3 [182.9 to 207.3]	90.8 [79.9 to 120.8]	97.8 [80.5 to 121.0]	7.0 [5.3 to 8.7]
	WTN-Continuous	36.8 [25.5 to 50.1]	209.8 [195.5 to 230.8]	95.3 [67.3 to 119.4]	108.3 [97.8 to 120.4]	7.7 [6.1 to 11.9]
	WTN-Sleep	37.8 [21.9 to 48.4]	210.3 [184.0 to 230.6]	92.3 [72.9 to 110.4]	114.3 [91.3 to 134.4]	8.5 [5.6 to 10.5]
	WTN-Wake	33.0 [24.0 to 42.0]	172.5 [146.5 to 199.5]	94.5 [90.0 to 125.5]	107.5 [95.5 to 112.0]	7.3 [5.7 to 8.2]

Values are Median [IQR] for each participant group (WTN-sleep disturbed, *n* = 14; WTN-non-sleep disturbed, *n* = 18; rural control, *n* = 18; and RTN-sleep disturbed, n = 18) under each noise condition.
WTN, wind turbine noise; RTN, road traffic noise; mins, minutes; IQR, interquartile range.

consistent in-laboratory WTN effects on sleep, the WTN-sleep disturbed group showed consistent evidence of poorer sleep overall compared to the remaining groups.

Previous studies have also suggested that perceived noise sensitivity likely influences noise effects on sleep, such that individuals with higher noise sensitivity are more likely to report negative noise, including WTN, effects on sleep than those with lower noise sensitivity [38]. Although there was a significant negative correlation between noise sensitivity and PSG determined sleep efficiency on the control night, this was not the case for sleep diary determined sleep efficiency, differences in PSG or sleep diary determined sleep efficiency, or any other sleep outcomes on control versus WTN-Continuous condition nights. Given no group, noise exposure condition or interaction effects on PSG or sleep diary determined sleep efficiency after controlling for age and noise sensitivity, these results do not support that noise sensitivity influences 25 dB(A) WTN effects on sleep. These results are perhaps not surprising given the absence of WTN effects on sleep outcomes and similar previous findings in a sample of healthy individuals without habitual WTN exposure [20].

Study limitations

The main limitation of this study is that SPL and other WTN characteristics are likely to become measurably sleep disruptive at higher exposure levels not examined in this study. The WTN sample used in this study was comparable to long-term median levels recorded in the field and contained prominent amplitude modulation that was anticipated to impair sleep. However, levels remained below recommended maximum indoor night-time noise limits, so the absence of detectable sleep disruption at the levels used in this study does not preclude the possibility of sleep disruption at higher levels closer to current noise guideline limits.

In these repeated measures, laboratory studies with multiple conditions the testing of more than one WTN level was logistically and financially infeasible. In retrospect, if a higher WTN level had been used and shown either negative or positive results, it would have been more informative. However, 25 dB(A) was chosen to test for possible disruption to sleep as measured by extended sleep latencies and night-time wakeful periods with WTN that were clearly audible while awake, especially in participants reporting WTN-related sleep disruption. The negative results regarding sleep latency and wake after sleep onset measures, even in the WTN sensitive group, are thus informative.

Results from separate night experiments in the same study sample, including groups with different prior exposures and self-reported noise-related sleep difficulties, will be particularly useful to evaluate the sleep disruption characteristics of different levels of WTN compared to RTN exposure during established sleep.

Sleep itself is highly variable with marked changes in sensory acuity which depends on sleep depth. Consequently, the selected WTN sample was played on a 3-minute loop to facilitate tighter control over noise levels than is possible with longer and more variable noise samples. However, there is also the potential for variable annoyance levels during wake and habituation effects over time during wake and sleep to influence sleep propensity. WTN offset/onset could also have an alerting effect on participants due to the temporal profile of the WTN. Previous work supports that noise onset effects on sleep are relatively modest [20, 50–52], particularly at low but audible SPLs. Rapid onset/offset of WTN was considered important for evaluating potential sleep- versus wake-dependent WTN effects utilized in this study, whereas more tapered onsets, have the potential to more variably influence attention towards WTN prior to sleep onset and the return to sleep from overnight wake periods.

Age was significantly different between groups and statistical adjustment using age as a covariate may not adequately control for age as a potential confounder for comparisons between groups. Furthermore, the overall degree of WTN-related sleep disruption in the home environment was reported to be moderate in the WTN-sleep disturbed group. By study design, the intention was to capture residents living near wind turbines with the greatest degree of disturbance attributed to WTN

Table 4. Continued

N2 %	N3 %	REM %	N2 latency (mins)	N3 latency (mins)	REM latency (mins)	Total wake time (mins)
45.6 [42.1 to 52.3]	23.1 [12.9 to 28.3]	21.0 [18.2 to 26.4]	7.0 [4.3 to 15.5]	25.3 [20.0 to 38.8]	68.8 [58.8 to 118.4]	106.3 [73.8 to 130.3]
45.5 [43.3 to 49.8]	24.8 [20.9 to 29.1]	22.7 [17.3 to 24.9]	11.5 [8.8 to 19.9]	26.5 [21.4 to 36.5]	81.3 [66.1 to 97.8]	95.5 [56.0 to 134.0]
45.8 [41.8 to 50.9]	24.2 [14.1 to 28.6]	23.8 [16.7 to 25.9]	11.0 [5.4 to 13.5]	19.5 [12.4 to 28.3]	77.3 [67.8 to 175.5]	75.8 [55.9 to 98.5]
44.4 [40.3 to 52.5]	20.8 [11.4 to 28.8]	23.0 [20.3 to 25.8]	11.5 [9.5 to 17.5]	29.5 [19.5 to 51.5]	110.5 [74.0 to 126.0]	101.0 [85.5 to 155.0]
44.9 [42.2 to 51.3]	20.1 [10.8 to 26.7]	20.2 [18.4 to 25.4]	17.3 [13.6 to 23.5]	40.0 [30.8 to 59.5]	92.8 [79.1 to 124.3]	66.8 [42.6 to 91.4]
45.3 [40.3 to 52.7]	15.8 [12.5 to 26.3]	21.6 [19.8 to 26.3]	16.0 [9.6 to 21.4]	33.0 [26.4 to 46.8]	109.3 [72.0 to 145.3]	75.3 [41.0 to 107.9]
45.6 [39.3 to 49.1]	19.5 [15.9 to 23.9]	23.5 [19.8 to 26.4]	14.0 [10.8 to 21.5]	30.0 [22.0 to 43.8]	91.5 [59.5 to 109.0]	64.5 [41.0 to 99.3]
44.4 [38.7 to 52.1]	14.5 [11.9 to 26.4]	22.9 [19.1 to 28.8]	16.5 [11.3 to 20.3]	32.3 [25.5 to 39.6]	85.8 [76.6 to99.3]	55.3 [40.6 to 85.0]
46.3 [43.0 to 51.3]	18.3 [11.5 to 25.1]	24.4 [19.4 to 27.2]	18.8 [11.5 to 35.8]	31.8 [22.0 to 50.9]	90.0 [79.5 to 113.4]	50.5 [37.5 to 101.5]
48.0 [41.3 to 51.8]	18.9 [15.2 to 27.2]	22.7 [19.3 to 24.9]	21.3 [10.5 to 29.5]	38.3 [28.5 to 47.0]	97.3 [86.4 to 117.4]	60.8 [34.9 to 84.8]
45.5 [41.5 to 49.8]	21.0 [15.8 to 24.0]	24.0 [19.0 to 28.4]	19.3 [14.1 to 33.1]	33.5 [23.5 to 47.6]	100.8 [80.4 to 114.3]	62.0 [43.1 to 135.9]
46.5 [42.2 to 52.6]	21.0 [14.7 to 26.4]	21.6 [18.7 to 24.8]	20.3 [10.4 to 24.8]	33.5 [28.5 to 48.3]	93.5 [77.4 to 103.1]	69.5 [52.0 to 98.9]
47.9 [41.1 to 51.6]	23.1 [17.9 to 26.6]	23.6 [20.7 to 29.5]	20.5 [13.5 to 44.3]	31.5 [24.4 to 54.8]	100.5 [84.5 to 160.6]	46.8 [25.9 to 60.3]
47.3 [42.9 to 49.9]	21.8 [18.1 to 24.5]	24.4 [21.9 to 25.5]	20.5 [14.4 to 26.3]	30.8 [25.6 to 60.1]	111.5 [81.1 to 136.3]	65.3 [35.8 to 101.8]
46.4 [41.0 to 49.5]	20.9 [16.1 to 24.9]	24.8 [22.9 to 26.3]	23.8 [14.4 to 28.1]	35.3 [21.3 to 52.9]	104.5 [82.1 to 152.1]	52.8 [34.5 to 74.3]
43.7 [35.8 to 47.4]	25.6 [20.6 to 28.7]	25.7 [20.2 to 27.8]	28.0 [12.5 to 55.0]	39.5 [23.5 to 67.0]	120.5 [88.0 to 144.0]	70.0 [28.5 to 86.0]

most likely to exhibit sleep difficulties due to noise. This group showed some signs of more chronically disturbed sleep compared to the other groups, but at relatively modest levels below those typically used to classify chronic insomnia. However, it also remains unclear how representative the recruited sample might be of highly disturbed individuals. Several factors made this group particularly challenging to recruit including travel-distance to the sleep laboratory, COVID-19 travel restrictions, the time commitment necessary to accommodate the multi-night study protocol, and reluctance of some individuals to engage in research. In addition, the inclusion criterion of WTN-sleep disturbed and non-sleep disturbed participants residing <10 km from the nearest wind turbine meant that participants could live some distance away from wind turbines where habitual WTN exposure are likely to be more variable and lower than closer distances. Study inclusion inevitably relied on somewhat arbitrary cut-offs from self-reports where more direct assessments of habitual noise exposure would clearly be preferable. Thus, several potential recruitment biases may have influenced study participation and between-group comparisons. However, consistent WTN exposure effects would still be expected to be apparent from within-subjects comparisons between nights, for which this study also had substantially greater statistical power. Nevertheless, further research using higher noise exposure levels in noise-sensitive individuals remains warranted to establish WTN levels that objectively impact on the ability of nearby residents to sleep.

A further limitation was that the morning sleep diary did not capture the participant's perception of overnight WTN exposures compared to their usual experiences at home. The WTN-sleep disturbed group reported moderate WTN-related sleep disruption at their residence but showed no significant differences in self-reported sleep disruption between control and WTN exposure conditions, potentially reflective of lower-level WTN exposure in the laboratory compared to home environment. Alternatively, hyperawareness or hypervigilance towards the presence versus absence of WTN during the sleep period could have impacted responses on all study nights. Two-week at-home sleep diary measures were largely not different from in-laboratory sleep diary outcomes, apart from greater self-reported wake after sleep onset in the laboratory compared to home. This could reflect factors beyond WTN effects, such as participant discomfort in the laboratory due to sleep equipment and/or sleeping in a foreign environment [53], in addition to noise impacts. However, we attempted to control these effects via an acclimatization night, randomization of study nights, participant blinding of noise exposure conditions, and comparisons between noise exposure versus a quiet control night.

Further research

Although effects of higher WTN levels remain unclear, the current study found no evidence to support that average WTN levels experienced around 3 km from a wind farm has measurable impacts on objective or self-reported sleep outcomes in a carefully controlled laboratory setting. Although more representative of real-world WTN exposures, field studies lack sufficient control over extraneous variables such as weather, wind speed, study blinding, nocebo effects among many other variables likely to confound underlying cause-and-effect relationships. Thus, a key next step for further research is to identify specific WTN features and SPLs that are more likely to be problematic for sleep and how these relate to real-world WTN exposure in the field. This will require appropriately controlled daytime listening tests and overnight exposure studies to understand dose-response relationships with annoyance and sleep disturbance compared to other noise types. Ultimately, noise policies and guidelines require appropriate evidence to protect public amenity around industries that generate noise, particularly at night.

In addition to further studies using higher WTN SPLs, more subtle microstructural effects of WTN on sleep warrant further examination is given they are more sensitive to sleep disturbance than traditional measures of sleep macrostructure [54–56]. For example, using power spectral analysis, subtle yet significant SPL and sleep stage effects of WTN compared to RTN have been demonstrated in healthy sleepers [52]. Odds ratio product,

a sensitive objective marker of sleep depth has also been shown to identify subtle sleep changes to sleep with nocturnal traffic noise [57]. Other spectral features [58] and K-complex responses to WTN versus RTN [50] may also be more sensitive and useful markers of sleep disruption than traditional metrics and warrant further examination in WTN exposed residents who do and do not self-report WTN related sleep disruption at home and in the laboratory when exposed to much higher WTN SPLs.

The potential for WTN exposure to affect daytime outcomes such as mood, anxiety, and daytime performance also remains to be determined. Anecdotal data suggest that some residents living near wind farms report daytime impacts that they attribute to nocturnal WTN exposure [7, 59]. Given the potential for both psychological factors and/or microstructural effects on sleep quality [60–61], mood and daytime performance could clearly be impacted without necessarily objective changes in markers of sleep time or quality.

Conclusions

WTN impacts on PSG and sleep diary determined sleep macrostructure parameters were assessed in a carefully controlled sleep laboratory setting in a sample including four sub-groups: WTN exposed residents with and without self-reported prior WTN related sleep disruption, rural participants with no prior WTN exposure and RTN residents reporting RTN related sleep disruption. Despite an overall group main effect on PSG wake after sleep onset, there were no further significant noise condition or group main effects or group-by-noise interaction effects on other conventional objective and subjective markers of sleep time or quality. Overall, these results do not support that acute WTN exposures approximating median WTN exposure levels around 3 km from a windfarm, measurably impact sleep assessed using conventional sleep scoring metrics, including in individuals with self-reported sleep difficulties attributed to WTN living at a similar distance. However, further studies remain warranted to test for effects of higher WTN exposure levels on traditional sleep macrostructure outcomes, subtle microstructural sleep parameters, and impacts on next-day mood, anxiety, and performance.

Supplementary Material

Supplementary material is available at *SLEEP* online.

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Data Availability

The data underlying this article will be shared on request to the corresponding author.

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