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Physiological effects of wind turbine noise on sleep

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Abstract

In accordance with the EU energy policy, wind turbines are becoming increasingly widespread throughout Europe, and this trend is expected to continue globally. More people will consequently live close to wind turbines in the future, and hence may be exposed to wind farm noise. Of particular concern is the potential for nocturnal noise to contribute towards sleep disturbance of nearby residents. To examine the issue, we are implementing a project titled Wind Turbine Noise Effects on Sleep (WiTNES). In a pilot study described in this paper, we performed an initial investigation into the particular acoustical characteristics of wind turbine noise that might have the potential to disturb sleep. Six young, healthy individuals spent 5 nights in our sound exposure laboratory. During the final 3 nights of the study, the participants were exposed to wind turbine noise, which was synthesised based on analysis of field measurements. Exposures involved periods of different amplitude modulation strengths, the presence or absence of beats, different blade rotational periods, and outdoor $L_{AEq,8h}$=45 or 50 dB with indoor levels based on the windows being fully closed or slightly open. Physiological measurements indicate that nights with low frequency band amplitude modulation and $L_{AEq,8h}$=45 dB, slightly open window ($L_{AEq,8h}$=33 dB indoors) impacted sleep the most. The presence of beats and strong amplitude modulation contributed to sleep disturbance, reflected by more electrophysiological awakenings, increased light sleep and wakefulness, and reduced REM and deep sleep. The impact on sleep by these acoustic characteristics is currently the focus of interest in ongoing studies.

Keywords: wind turbine, sleep, polysomnography

Physiological effects of wind turbine noise on sleep

1 Introduction

According to the European Wind Energy Association, there was almost 13 000 MW of wind power installed across the EU in 2015 [1]. This represents a 6.3% increase over the previous year. Annoyance from wind turbine noise has previously been evaluated, primarily in cross-sectional studies [2]. However, long term health consequences, including sleep disturbance, have not been studied and the physiological effects are not known. Present debate on one side argues that "sound from wind turbines does not pose a risk of... any adverse health effect in humans" [3]. On the other side, there have been claims for symptoms including impairment of mental health [4]. Studies such as [4] have been subject to criticism, both in terms of the conclusions drawn from the data and the experimental design itself. Whilst many claims of adverse effects are anecdotal, sleep disturbance is one of the issues most frequently reported and supported by previous cross-sectional studies [5].

There is ample evidence illustrating that adequate sleep is necessary for maintaining good health. Disturbed sleep can hence be of consequence for immediate and long-term health [6]. Night time noise has the potential to adversely affect sleep, which has been recognised by the World Health Organisation and reflected by their publication of night time noise limits [7]. The Environmental Noise Directive (2002/49/EC) recognises that community noise is potentially harmful and so requires that all EU member states map the noise exposure of their populations. Despite this, wind turbines are often erected in quiet rural areas, where sleep disturbance due to wind turbine noise is reported more frequently [8]. However, reported effects of this noise on sleep may be biased by perceived annoyance and so objective measures of sleep structure and other physiological response, for instance cardiovascular effects, are clearly needed.

Although objective measures have been made on the human effects of numerous environmental sources, particularly traffic noise [6,9,10], the majority of studies on the effects of wind farm noise have used only subjective means, and only using calculated equivalent sound levels in dBA at the façade based on simplified sound radiation and propagation models. A notable recent study has however examined wind turbine noise using wrist actigraphy [11]. Compared to traffic noise where much research has been performed, little is known regarding how noise from wind turbines objectively influences sleep. The aim of the current project is therefore to determine whether noise from wind turbines can impact on human sleep, and how any such impacts are manifested.

2 Methods

2.1 Overall study design

An experimental study using a within-subject design was implemented to investigate the effect of wind turbine noise on sleep. Prior to the study described in this paper, an initial explorative pilot study was performed. In this pre-pilot, which involved six young and healthy participants, it was found that EEG awakenings occurred more frequently during nights with indoor noise levels of 34

dBA (window closed) than nights with 30 or 34 dBA (window slightly open). Indications were also found that amplitude modulation, rotational speed of the turbine blades and/or the presence of strong “beats” contributed to sleep fragmentation, reflected by EEG arousals, awakenings and changes to a lighter sleep stage. These findings served as the basis for the development of the specific characteristics of wind turbine noise examined in the present work.

2.2 Study protocol

Participants slept for 5 consecutive nights in a sound environment laboratory, which is furnished to imitate a typical home environment. The first night served as a habituation to the environment and the sleep measurement apparatus. The second night was an exposure-free control night to measure baseline sleep. Nights 3-5 were exposure nights where wind turbine noise (WTN) was introduced (see 2.4). Participants arrived at the lab by 20:00 each evening and were instructed to begin trying to fall asleep at 23:00. They were woken by an automated alarm call at 07:00 each morning. Low level artificial background noise simulating ventilation noise was introduced into the bedrooms throughout the study.

Sleep was measured using polysomnography (PSG) using standard electrode placements and sampling and filter frequencies [12]. PSG data were manually scored by a single sleep technologist according to current guidelines [12]. Electroencephalogram (EEG) arousals were classed according to current criteria [13]. Arousals of longer than half an epoch (>15s) were classed as awakenings.

Participants completed questionnaires within 15 minutes of awakening each morning. The questions and their validation against objective sleep measurements are reported elsewhere, but summarily include items on sleep quality and disturbance, nocturnal restoration, and subjective sleep assessment [14]. The current paper focusses on objective measures of sleep only, and therefore the questionnaire data will not be reported.

2.3 Participants

Six young healthy persons took part in the study (mean age 24 years SD±2.3, mean BMI 20.7 SD±0.4, 5 women). All were students recruited via public advertising, provided informed written consent prior to the start of the study, and were free to stop taking part at any time. They were financially compensated for participating. Each individual underwent a hearing test to a screening level of 15 dB HL from 125 Hz to 8 kHz using pure tone audiometry.

2.4 Noise exposures

Noise exposure was synthesised based upon extensive analysis of recorded wind turbine sound signals. The development of these synthesised exposures is outside the scope of the current paper, but are presented here in summary and will be described in detail in a journal article that is currently under preparation. Three 8-hour periods of WTN were generated, corresponding to the three study exposure nights (Night A, Night B, Night C). Each individual hour of each night contained a 2*2*2 arrangement of high and low turbine rotational periods, weak and strong amplitude modulation, and the presence or absence of strong beats. Each hour therefore contained eight distinct noise scenarios, each of which was 400s in duration. The presentation

order of these eight scenarios varied in an 8×8 Latin square design across the eight study hours of each night (23:00 to 07:00), with each scenario only preceding and following any other scenario only once. Every hour concluded with 400s free of WTN. A detailed overview of the noise scenarios is given in Table 1. A description of the differences between Night A, Night B and Night C are also given in Table 1 (window closed or slightly open, outdoor noise level, and/or the frequency bands of the amplitude modulation).

Table 1 Noise scenarios implemented across the three exposure nights

Night	Noise level	Period	RPM	AM strength	AM frequency bands	Strong beats
Night A Window slightly open	$L_{AEq,1h,outdoor}$=45 dB $L_{AEq,1h,indoor}$=33 dB	1.	13	Weak	MF	No
		2.	17	Weak	MF	No
		3.	13	Strong	MF	No
		4.	17	Strong	MF	No
		5.	13	Weak	MF	Yes
		6.	17	Weak	MF	Yes
		7.	13	Strong	MF	Yes
		8.	17	Strong	MF	Yes
		9.	No turbine noise – background only			
Night B Window slightly open	$L_{AEq,1h,outdoor}$=45 dB $L_{AEq1h,,indoor}$=33 dB	1.	13	Weak	LF	No
		2.	17	Weak	LF	No
		3.	13	Strong	LF	No
		4.	17	Strong	LF	No
		5.	13	Weak	LF	Yes
		6.	17	Weak	LF	Yes
		7.	13	Strong	LF	Yes
		8.	17	Strong	LF	Yes
		9.	No turbine noise – background only			
Night C Window closed	$L_{AEq,1h,outdoor}$=50 dB $L_{AEq1h,,indoor}$=30 dB	1.	13	Weak	LF	No
		2.	17	Weak	LF	No
		3.	13	Strong	LF	No
		4.	17	Strong	LF	No
		5.	13	Weak	LF	Yes
		6.	17	Weak	LF	Yes
		7.	13	Strong	LF	Yes
		8.	17	Strong	LF	Yes
		9.	No turbine noise – background only			

RPM=Rotations per minute (of turbine blade); AM=Amplitude modulation; MF=Middle Frequencies (500–2500 Hz); LF=Low Frequencies (80–500 Hz)

2.5 Statistical analysis

All analyses were performed in IBM SPSS 22. Data were analysed in a repeated-measurement ANOVA. Given the exploratory nature of the work, all statistical tests with $p<0.1$ are reported. Post hoc analysis was performed even if no significant main effect was observed. Corrections for multiple comparisons were abdicated in order to minimise the likelihood of missing potentially important findings [15].

3 Results

3.1 Differences between nights

The mean values of sleep macrostructure variables which were found to differ between at least two nights ($p<0.1$) are given in Table 2. Compared to the control night,

- participants in Night A took 7.2 minutes longer to fall asleep, had a reduction in continual time in N2 (“intermediate”) sleep of 10.6 minutes, and had a reduction in continual time in slow wave (“deep”) sleep of 5.4 minutes.
- participants in Night B had an average increase of wakefulness of 5.8 minutes, corresponding to a reduction in sleep efficiency of 2.6%, and had a reduction in the time spent in slow wave sleep of 4.8%.
- participants in Night C had a reduction in continual time in N2 sleep of 11.4 minutes, had a reduction in continual time in slow wave sleep of 9.2 minutes and had a reduction in the time spent in slow wave sleep of 0.8%.

Participants in Night A and Night C had a reduction in continual time in N2 sleep of 8.4 and 9.2 minutes respectively than in Night B. Participants in Night B woke up for the first time 31 minutes earlier than in Night C.

No effects were seen for rapid eye movement (REM) sleep latency, SWS latency, sleep period time, maximum uninterrupted REM or N1 (“light”) sleep duration, %N1, %N2, %REM, number or frequency of EEG arousals, number or frequency of EEG awakenings, or number or frequency of sleep stage changes.

Generally, sleep was least fragmented during the night with no WTN (the control night), with a higher percentage of SWS, lower wakefulness, longer sustained SWS and N2, and shorter sleep latency compared to nights where a difference was found. However, differences between exposure nights are rather limited, with lower sustained N2 in Night B than Night A, and an earlier first awakening in Night B than Night C. Based on these PSG macrostructure variables, it appears that of all the exposure nights, the condition with the most deleterious effects on sleep was Night B, which had low frequency band amplitude modulation and LAEq,8h=45 dB, slightly open window (LAEq,8h=33 dB indoors). Taking all of the above together, the results suggest that there is some evidence that nights with WTN can contribute to sleep disturbance, but how exactly this contribution occurs is unclear.

Table 2 Differences in sleep macrostructure between nights. Mean values and standard deviations (±) are shown for sleep variables for which at least one significant (p<0.01) difference was found between nights.

Variable	Control	Night A	Night B	Night C	Statistics	
					Main effect	Post-hoc
SL (min)	10.3±8.4	17.5±10.6	17.0±11.4	21.3±25.5	-	Con:A (p=0.064)
WASO (min)	14.5±6.5	15.0±7.2	20.3±7.3	17.9±10.0	-	Con:B (p=0.031)
First awakening (min)	39.8±30.0	58.8±51.4	26.3±34.7	57.3±59.6	-	B:C (p=0.076)
Max N2 (min)	38.3±8.0	27.7±6.6	36.1±9.0	26.9±5.7	F(3,15)=8.792 p=0.001	Con:A (p=0.005) Con:C (p=0.005) B:A (p=0.029) B:C (p=0.011)
Max SWS (min)	40.2±10.3	34.8±10.2	32.9±16.9	31.0±8.9	-	Con:A (p=0.079) Con:C (p=0.035)
SE (%)	94.8±1.9	93.2±3.1	92.2±2.1	91.8±7.2	-	Con:B (p=0.073)
SWS (%)	22.8±4.9	21.7±5.3	18.0±3.7	22.0±4.0	F(3,15)=4.070 p=0.027	Con:B (p=0.055) Con:C (p=0.050)

SL=Sleep Latency following lights-out; WASO=Wakefulness After Sleep Onset; First awakening=Time of first EEG awakening following sleep onset; Max N2=Maximum uninterrupted period in sleep stage N2; Max SWS=Maximum uninterrupted period in Slow Wave Sleep; SE=Sleep Efficiency; SWS=Percentage of time asleep in Slow Wave Sleep

3.2 Differences between sound character periods

Sound character periods 1 to 8, plus the quiet period, were compared across the control and exposure nights. The following main effects were found:

- Time awake in period 7: F(3,15)= 3.325, p=0.048 (see Figure 1A)
- Percentage of time in N1 sleep in period 6: F(3,15)= 3.201, p=0.054 (see Figure 1B)
- Percentage of time in N1 sleep in period 7: F(3,15)= 2.568, p=0.093 (see Figure 1B)
- Percentage of time in SWS sleep in period 4: F(3,15)=11.454, p<0.001 (see Figure 1B)
- Percentage of time in REM sleep in period 8: F(3,15) =3.041, p=0.062 (see Figure 1B)

Post-hoc comparisons between nights for periods where main effects were found are given in Table 3.

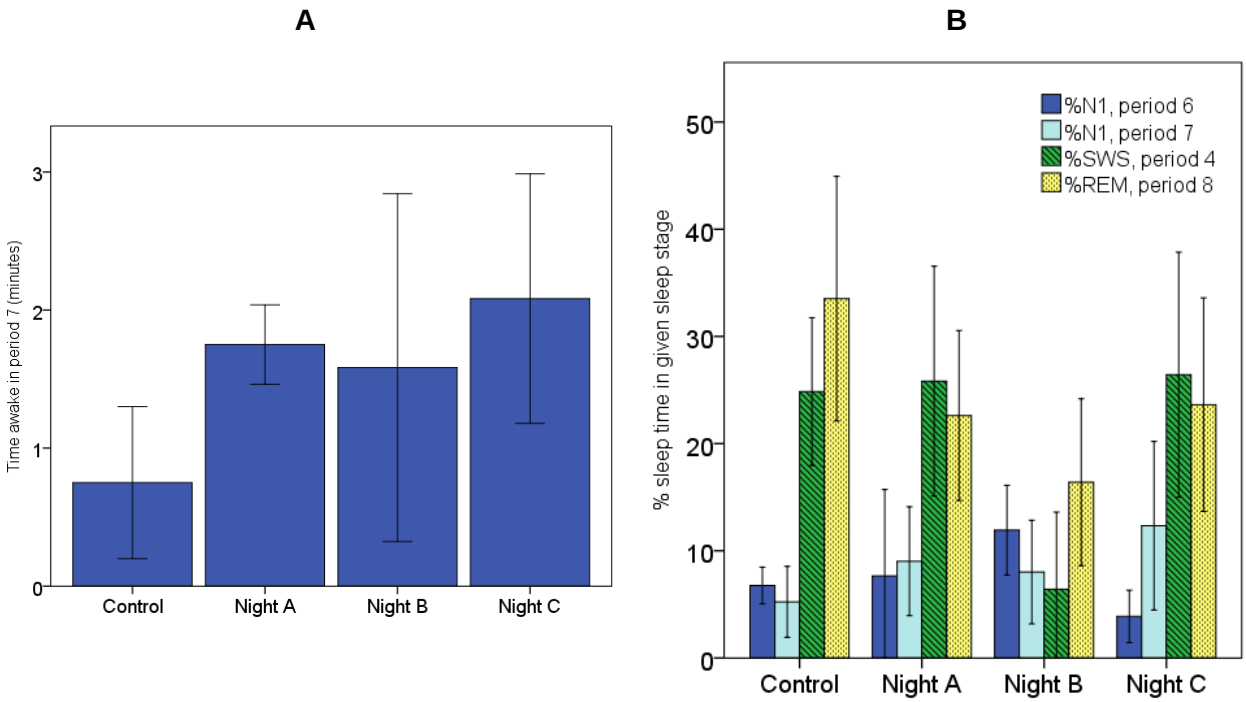

Figure 1A Time awake in sound character period 7 **1B** Distribution of time in difference sleep stages in different sound character periods between nights. Error bars indicate 95% confidence intervals.

Table 3 Significant post hoc effects between nights for sleep macrostructure variables in periods with the same sound character. No significant post hoc effects were found for TST or %N2. Only periods where a main effect was observed are shown.

Variable	Sound character period								
	1	2	3	4	5	6	7	8	9
Wake (min)							Con:A p=0.018 Con:C p=0.017		
N1 (%)						Con:B p=0.029 Con:C p=0.079 B:C p=0.006	Con:C p=0.036		
SWS (%)				Con:B p=0.009 A:B p=0.006 B:C p=0.001					
REM (%)								Con:B p=0.053	

Data for EEG arousals, awakenings and sleep stage changes (SSCs) for all three exposure nights were pooled for all sound periods sharing common sound character. Main effects were found for the frequency of SSCs ($F(5,25)=2.299$, $p=0.075$) and awakenings ($F(5,25)=2.146$, $p=0.093$). No

effects were found for arousals. From Figure 2 it appears that SSCs occur more frequently during periods with weak AM and no beats, and that awakenings occur more frequently during periods with strong AM and beats. These findings were not statistically significant however.

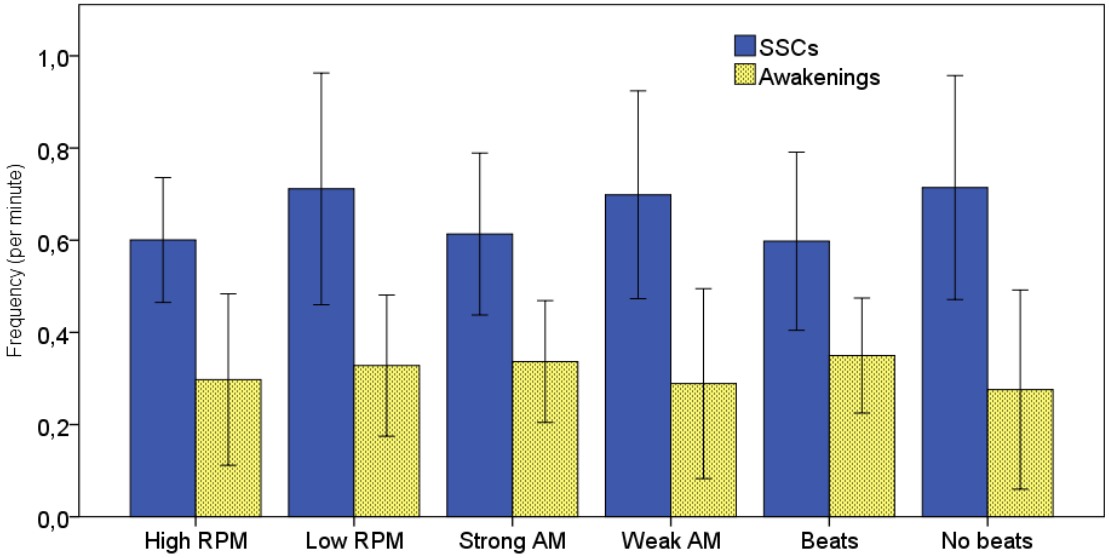

Figure 2 Frequency of SSCs and awakenings during periods with high and low RPM, strong and weak amplitude modulation, and beats or no beats. Data presented are summed from all three exposure nights. Error bars indicated 95% confidence intervals.

3.2.1 Summary of effects of sound character periods

In summary, the effects of individual sound character periods on different sleep parameters are:

- Effects on N1: Period 7 (low RPM, strong AM, beats), Period 6 (high RPM, weak AM, beats)
- Effects on W time: Period 7 (low RPM, strong AM, beats)
- Effects on SWS: Period 4 (high RPM, strong AM, no beats)
- Effects on REM: Period 8 (high RPM, strong AM, beats)
- Effects on SSCs and awakenings: Low/high AM, no beats/beats

4 Discussion

In this study, the effects of wind turbine noise on sleep were investigated using physiological measures. There is some evidence that compared to control nights with no noise, sleep during nights with WTN had a reduced amount of SWS, more time spent awake, increased sleep latency and a reduction in sustained SWS and N2 sleep. The amount of REM sleep, SWS and WASO were affected by sound characters with strong amplitude modulation. N1 (“light”) sleep was more prevalent during noise with beats. SWS in particular has been identified as important for declarative memory in humans [16]. Furthermore, SWS is considered to be important for physical restoration [17] and is accordingly prioritised after sleep deprivation [18]. REM sleep is believed to be important for cognition [19]. Despite the observed physiological disruptions, it is unclear at

present whether the size of these effects of WTN on SWS and REM would interfere with any associated biological processes.

During sleep, the body reacts more strongly to an abrupt change in acoustic environment than a gradual change. For instance, awakening probability is linked with noise rise time [20]. As such it was hypothesised if WTN had negative effects on sleep, such effects would manifest during high rotational speeds, strong amplitude modulation and the presence of beats. The indication, albeit non-significant, that the frequency of SSCs was lower during periods of high RPM, lower during periods of strong amplitude modulation and lower during periods with beats could therefore appear surprising. However, during periods of strong amplitude modulation and periods with beats there seems to be a higher frequency of awakenings, which do not include changes to a wake stage. In other words, rather than the participants simply changing sleep stage, full awakenings seemed to occur instead during these periods.

Although the frequency of awakenings and sleep stage changes were influenced by the strength of AM, the total number of awakenings and sleep stage changes did not differ between across any of the experimental nights, including the control. This means that in order for these reactions to occur at a higher rate during certain times, they are not occurring during other times when they may have spontaneously appeared as part of the natural rhythm of sleep. These awakenings and SSCs are being redistributed throughout the night, which may have implications for certain neuronal processes performed during sleep, such as the clearance of waste products that accumulate during wakefulness that has been demonstrated in animal studies [21].

This small-scale experiment served as a pilot study, and was therefore limited by a number of factors. The participants cannot be considered as representative of the population who are exposed to WTN at home. Together with the small sample size, the conclusions drawn should not be taken outside of the context of the work; to provide input for future work with a more appropriate study sample, size, and exposure design. The chosen noise levels were higher than those recommended in Sweden [22], but were not unrealistically high for other countries, in order to increase the likelihood of inducing a physiological response. The rationale for this decision was to increase the expected effect size so as to better detect what elements of the sound character contributed to response in this small pilot study. An ongoing study has the aim of examining sleep under the influence of noise from wind turbines at more commonly occurring levels.

5 Conclusions

Physiological measurements indicate that nights with low frequency band amplitude modulation and $L_{AEq,8h}$=45 dB, slightly open window ($L_{AEq,8h}$=33 dB indoors) impacted sleep the most. In particular, amplitude modulation and the presence of beating were important constituents of the wind turbine noise contributing to sleep disruption.

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