Effects of nighttime low frequency noise on the cortisol response to awakening and subjective sleep quality

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Abstract

The effects of night-time exposure to traffic noise (TN) or low frequency noise (LFN) on the cortisol awakening response and subjective sleep quality were determined. Twelve male subjects slept for five consecutive nights in a noise-sleep laboratory. After one night of acclimatisation and one reference night, subjects were exposed to either TN (35dB L_{Aeq}, 50dB L_{Amax}) or LFN (40dB L_{Aeq}) on alternating nights (with an additional reference night in between). Salivary free cortisol concentration was determined in saliva samples taken immediately at awakening and at three 15-minute intervals after awakening. The subjects completed questionnaires on mood and sleep quality. The awakening cortisol response on the reference nights showed a normal cortisol pattern. A significant interaction between night time exposure and time was found for the cortisol response upon awakening. The awakening cortisol response following exposure to LFN was attenuated at 30 minutes after awakening. Subjects took longer to fall asleep during exposure to LFN. Exposure to TN induced greater irritation. Cortisol levels at 30 minutes after awakening were related to 'activity' and 'pleasantness' in the morning after exposure to LFN. Cortisol levels 30 minutes after awakening were related to sleep quality after exposure to TN. This study thus showed that night time exposure to LFN may affect the cortisol response upon wake up and that lower cortisol levels after awakening were associated with subjective reports of lower sleep quality and mood.
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Keywords: Cortisol awakening response; Sleep; Low frequency noise; Traffic noise

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Introduction

A multitude of functions in the human body are subject to circadian patterns, related to normal daily activities and rest during sleep. Disruption of this pattern, as caused by shift work or jet lag, may cause negative effects in terms of fatigue, somatic symptoms and subjective dissatisfaction.

A major intrinsic marker of the circadian rhythm is the level of circulating corticosteriods derived from activity within the hypothalamus-pituitary-adrenal (HPA) axis. Acute activation of this same HPA axis also represents a major physiological response to environmental stressors. Thus the degree to which environmental stressors can impact upon the circadian rhythm becomes of interest. The cortisol response to awakening is an index of adrenocortical activity. Previous studies have shown that the amount of cortisol in saliva (a true reflection of circulating physiologically active, or free, cortisol) increases after awakening with a peak at about 30 minutes, after which it declines [1,2]. The response is rather robust over weeks or months [3], but has also been found to be suppressed by the burnout syndrome [4] and increased in high levels of work overload [2] and in relation to unemployment stress [5].

Environmental noise is a potential disruptor of the normal circadian pattern due to its effects on sleep and recreation [6]. The effect of noise on the circadian rhythm of cortisol is not widely documented. Acute effects of noise on cortisol have however been studied by Brandenberger *et al.* [7]. Measurements of plasma cortisol levels were undertaken every ten minutes from 0800 to 1500 h among subjects exposed to noise at levels from 85 dBA to 105 dBA. The noise did not alter the normal decline of cortisol compared to that seen during a control day. The chronic effects of occupational industrial noise were studied by Melamed and Bruhis [8]. During normal conditions with noise exposure of 85 to 95 dB LAeq, urinary cortisol was almost as high in the afternoon (at 1300h) as in the early morning (0630h). However, when the workers were equipped with earmuffs, a normal diurnal decline in cortisol was observed. Low frequency noise was shown in one study to alter the circadian pattern of plasma cortisol [9]. Subjects were exposed to continuous low frequency noise at 90 dBA for eight hours or 84 dBA for 24 hours. During the first hour of exposure, the normal pattern of declining cortisol was reversed, and plasma cortisol remained significantly elevated for both noise levels 24 hours after the onset of the noise, despite the fact that the subjects exposed to 90 dBA had been in quiet conditions for 16 hours. No significant changes were observed in heart rate, blood pressure or catecholamine level, indicating that the stress response to noise was mediated primarily by HPA axis activity.

The auditory system is permanently alert, even during sleep. Excitations of the system are subcortically connected via the amygdala to the HPA axis [10]. Thus, noise can influence the release of different stress hormones, especially in sleeping persons during the early morning phase. Increased urinary cortisol levels collected in the morning have previously been found in humans when exposed to nocturnal aircraft or road traffic noise during sleep [11,12] and among children exposed to more than 60 dBA, day and night weighted [13].

While the effects on sleep and well being of transportation noises are rather well documented [14–17], much less is known of effects on sleep caused by low frequency noise (< 200 Hz). Low frequency noise is common in both occupational and domestic environments. Several case studies and some epidemiological studies indicate that low frequency noise affects sleep quality, particularly with reference to the time taken to fall asleep and tiredness in the morning [18,19].

As noise may affect the quality of sleep, it is reasonable to assume that the normal pattern of cortisol may be influenced by noise exposure during sleep. In view of the ubiquitous nature of low frequency noise exposure in many dwellings, the effects of this type of noise are particularly interesting. No studies

have previously been performed to elucidate the effect on the normal cortisol awakening response following noise exposure during sleep.

The aim of the present study was to assess the influence of nocturnal low frequency noise and road traffic noise on the cortisol awakening response and subjective sleep quality.

Materials and methods

General outline

The study group comprised 12 male subjects who slept for five consecutive nights in a bedroom in a noise laboratory. The first night was a quiet acclimatisation night, the 2nd and the 4th nights were quiet reference nights and the 3rd and 5th nights were either low frequency or traffic noise exposure nights. The order of the exposure noises was randomised among the four subject groups. The design of the exposure week is shown in Table 1. The effects of the noise exposure were determined by taking saliva samples for cortisol determination and by questionnaires evaluating sleep quality and mood. The study was approved by the ethics committee at the medical faculty of Göteborg University.

Participants and experimental design

The 12 test participants were recruited by advertising. They were all male students at Göteborg University and had an average age of 24.5 years, (SD = 4.17). All were non-smokers and reported themselves to have normal hearing. Their sensitivity to noise and to low frequency noise as well as their subjective judgement of the noise exposure during sleep in their homes was recorded by questionnaires but was not a part of a selection processes.

Noise exposures

During the acclimatisation night and reference nights, the only sound in the bedrooms was background noise from the ventilation, which was at a level of 24 dBA and below the normal hearing threshold [20] for the frequency range below 160 Hz.

During the nights with noise exposure, the noises were played back through hidden loud speakers in the room. The road traffic noise (TN) comprised recordings from a motorway to which was added a number of lorry passages. Between 23.00 h and 08.00 h the noise comprised a total of 75 small vehicle passages. There were approximately ten lorry passages per hour between 23.00 h and 00.00 h and in the early morning between 05.00 h and awakening (usually around 0800). Between 01.00 h and 05.00 h the

Table 1
Design for the exposure week

Group	Night				
	1	2	3	4	5
I n = 6	Acclimatisation	Reference 1	TN	Reference 2	LFN
II n = 6	Acclimatisation	Reference 1	LFN	Reference 2	TN

number of lorry passages was reduced to two to three per hour in order to resemble a realistic environmental exposure. The TN had an L_{Aeq} level of 35 dB and a maximum noise level of 50 dBA. The noise was filtered to mimic normal attenuation provided by a window.

The low frequency noise (LFN) was a recorded ventilation noise to which was added sound pressure levels in the frequency region of 31.5 to 125 Hz using a sound processing system (Aladdin Nyvalla DSP Stockholm). Furthermore, the third octave band at 50 Hz was amplitude-modulated with an amplitude frequency of 2 Hz. The L_{Aeq} level of the ventilation noise was 40 dBA. The LFN was played continuously with two interruptions at 00.30 h to 01.00 h and 04.30 h to 05.00 h, during which time it was reduced to the background level. After these 30-minute interruptions the level was gradually increased to 40 dBA. This was done in order to mimic the noise characteristics of common low frequency noise sources such as compressors and heat pumps that start and stop at certain intervals. The equivalent third octave band frequency spectra for the two noises in relation to the normal hearing threshold [20] are shown in Fig. 1.

Exposure facilities and experimental procedures

The noise laboratory is built like an apartment with three bedrooms, a combined kitchen and television room, a shower and toilet. The subjects had their own keys and could come and leave as they wanted. The size of each bedroom was $3 \times 2 \times 2.4$ meters.

On a separate day before the beginning of the exposure, subjects were given written and oral instructions for the procedures. They were also instructed as to how and when to take the saliva samples. During the experimental week, subjects came to the laboratory on Sunday evening and left on Friday morning. They were instructed to go to bed no later than 2300 h and were allowed to wake up according to their own routines. None of the subjects woke up later than nine in the morning. They were not permitted to drink alcohol or smoke cigarettes during the week of the experiment. The subjects were informed that they were
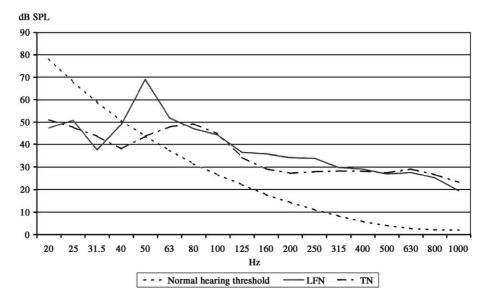

Fig. 1. Third octave band frequency spectra of the two noise exposures.

to take nil by mouth other than water and to not brush their teeth until the sampling procedure was completed. They were encouraged to sit in an armchair during the procedure, but were allowed to stand up or walk around in the facilities as long as they did not miss their next sampling time.

Cortisol determination

Four saliva samples were taken at 15-minute intervals every morning: directly after waking up and then 15, 30 and 45 minutes after awakening. The saliva was collected on a cotton salivette (Sarstedt Ltd, UK) that the subject chewed for three minutes, after which the subject placed the salivette in a sterile plastic tube. The samples were then frozen at -70 °C until analysed. Saliva was recovered from the cotton dental swab by centrifugation at 1000 g and cortisol was determined by commercial RIA, (Corticote ICN Biomedicals Ltd., Thane UK) inter and intra assay % CV = < 10%, as previously described [21]. Saliva samples for cortisol analysis were taken every morning; the samples from the first night were taken only to allow the subjects to practise and were not analysed.

Questionnaires

The subjects' sensitivity to noise and low frequency noise was measured using the question "are you sensitive to noise in general?" respectively "are you sensitive to low frequency noise". The answers were given on five response alternatives ranging from 1 "not at all" to 5 "extremely sensitive". Sensitivity to noise was also measured using the total number of points scored in a noise sensitivity evaluation questionnaire [22]. The questionnaire could give a total of 20 to 120 points, the higher the score the higher sensitivity to noise.

During the experimental week subjects were asked to fill out a questionnaire every morning after waking up and every night before they went to bed. In the morning, subjects were asked how they felt at that time and ratings were given on 0–10 scales with the endpoints "awake- tired", "relaxed-tense", and "friendly-irritated". Questions were also posed on how long (time in minutes) they estimated that it had taken to fall asleep the night before, whether they had woken up during the night and the reason for having woken up. Finally, the subjects were asked to rate their sleep quality with the endpoints "good-bad".

In the evening, the subjects were asked to rate how they felt at that time, i.e. just before they went to bed, with the endpoints "awake-tired", "relaxed-tense" and "friendly-irritated". The same questions were asked with reference to how they had felt during the afternoon. Subjects were also asked to compare their feelings of stress and tiredness during the day, as compared to a "normal" day, using one of the response alternatives: much less, somewhat less, about the same, somewhat more and much more. They were also encouraged to give their own comments on, for example, why the day had differed from a normal day.

A questionnaire evaluating mood [23] was completed in the evenings and the mornings. The questionnaire consisted of 71 adjectives describing feelings that formed the following six mood dimensions: social orientation, pleasantness, activity, extroversion, calmness and control. The subject could choose between four response alternatives: "I agree completely", "I somewhat agree ", "I do not agree" and "I do not agree at all".

In total, there were five morning and five evening questionnaires. The last evening questionnaire was given to the subjects after the last exposure night and they were asked to answer it in the evening and mail it to the department the next day. Due to an administrative mistake, three subjects did not fill in the last evening questionnaire.

Statistical analysis of the data

Cortisol concentrations were analysed as the square root of the measurements in order to normalise initially skewed distributions. The two noise exposure nights were analysed in relation to the reference night by a 4 (sampling periods) × 3 (exposure conditions) × 2 (groups) analysis of variance (ANOVA). The first two conditions were within-subject factors and group was a between-subject factor. Among polynomial contrasts the quadratic function was of more interest than the linear.

Pair-wise comparisons were made using Student's t-test. The subjective data were not normally distributed and were analysed using non-parametric tests. Pair-wise comparisons were made using Wilcoxson's rank test. Analyses of relationships were done using Spearman's correlation. The statistical analyses were carried out using SPSS (SPSS base 10.0 for Windows). All tests were two-tailed and a p-value below 0.05 was considered statistically significant; a p-value up to 0.10 is reported as a tendency toward significance.

Results

Saliva cortisol

No significant order effect was found for the two groups. The average values of the cortisol levels at each sampling point for reference nights 1 and 2 and after nights with traffic noise and low frequency noise are shown in Fig. 2.
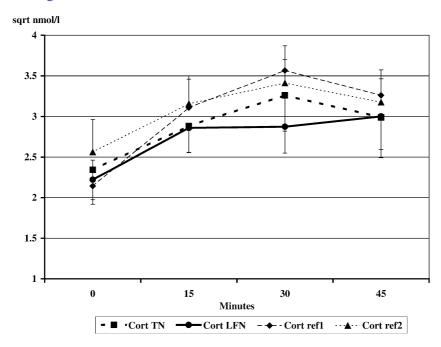

Fig. 2. Average values and standard deviations of saliva cortisol at different times after waking up and for the different conditions. For clarity only positive or negative values of standard deviations are shown.

We observed the expected cortisol response to awakening following the reference nights with cortisol levels reaching a peak at 30 minutes after which they declined. The levels of cortisol measured directly after awakening was somewhat higher for reference night 2 than reference night 1 although the difference did not reach significance (mean value 2.56 vs 2.14, $t = -2.052$, $p = 0.062$). No statistical difference between reference nights was found for the other sampling times. As reference night 1 was not proceeded by a night with noise exposure, and thus not confounded by noise, reference night 1 was used as the reference night for the cortisol analysis.

The main effect of time was highly significant $F(1,10) = 165,53$ $p < 0.0001$). The peak seen after the reference night at 30 minutes was not present after nighttime exposure to LFN and cortisol levels were similar at 15, 30 and 45 minutes post awakening.

A significant interaction between noise condition and time was found for the awakening cortisol response $F(1,10) = 8.150$, $p < 0.02$). The total mean for the three conditions were 3.019 for the reference condition, 2.870 for the TN and 2.739 for the LFN condition. However more importantly, the average level of cortisol after 30 minutes was significantly attenuated after nights with LFN as compared to the reference night ($t = 3.307$, $p < 0.01$).

Subjective responses

The median values of sensitivity to noise were 2.0 for noise in general, 74.5 for Weinstein noise sensitivity and 4.5 for sensitivity to low frequency noise. None of these parameters differed between groups and no significant interactions were found between sensitivity and cortisol for any of the noise conditions.

Fig. 3 shows the median value of subjective sleep quality for the initial acclimatisation night, reference night 1, reference night 2, and nights with noise conditions. The figure indicates that sleep quality improved from the first acclimatisation night to the second night (reference night 1) and that a further acclimatisation to the environment took place from the first reference night to the second. A similar pattern of acclimatisation was also found for most of the other subjective responses. The subjective responses after noise exposure nights were therefore analysed in relation to reference night 2. There was a tendency to a lower sleep quality ($z = 1.80$, $p = 0.07$) after nights with TN as compared to the reference night.

Fig. 4 shows the median value of tiredness in the morning, afternoon and evening for the three exposure conditions. The figure illustrates that the median value of tiredness in the morning tended to be highest after LFN while tiredness in the afternoon and in the evening were similar after the two noise exposure nights. No significant differences were however found in comparison with the reference night condition.

Table 2 gives the median values for the different sleep related parameters for the reference night and the two noise conditions. A higher value represents a more negative outcome, more tense, more tired etc. The participants generally took a longer time to fall asleep in the noise conditions. The difference was statistically significant for the LFN condition ($z = 2.408$, $p < 0.05$).

The subjects generally tended to feel more tense and irritated in the morning after the nights with noise exposure. A significant difference in comparison with the reference night was found for irritation in the morning after TN ($z = 2.045$, $p < 0.05$), while there was a tendency to a difference after nights with LFN, ($z = 1.725$, $p = 0.08$). No significant differences could be found for the other variables.
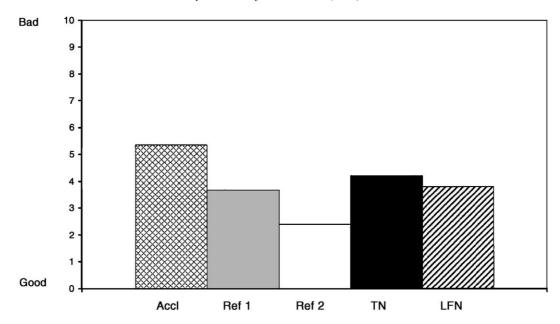
Fig. 3. Median values of sleep quality after the acclimatisation night (Accl), reference night 1 (ref 1), reference night 2 (ref 2) and the noise exposure nights (TN respective LFN).
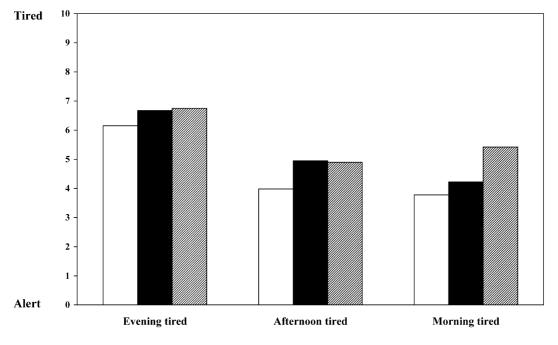

Fig. 4. Median values of tiredness reported in the evening, afternoon and morning after the reference night and the noise exposed nights. Unfilled bars = reference night, black bars = traffic noise and diagonally lined bars = low frequency noise.

Table 2
Median values of subjective sleep evaluations

Response variable	Ref night	Traffic noise (TN)	Low frequency noise (LFN)
Time to fall asleep (min)	20	35	39*
Morning:			
Tensed	3.0	4.3	4.0
Irritated	2.8	4.6*	4.2
Afternoon:			
Tensed	3.1	4.0	2.6
Irritated	2.4	2.5	2.4
Evening:			
Tensed	3.2	1.8	2.4
Irritated	1.9	1.6	2.4

* = p<0.05.

The median value of the number of wake ups during the night was 0.5 for the reference night, 1 for TN and 0 for LFN. Two of 12 subjects reported that they woke up because of noise from traffic.

Analysis of relationships between subjective reports generally showed that there were significant relationships between a low sleep quality, time to fall asleep, tiredness in the morning and tiredness in the evening, Although these relationships did not differ between exposure conditions during the night.

The subjective reports of sleep quality were analysed in relation to the cortisol levels measured 30 and 45 minutes after awakening (Table 3). For both noise conditions, a better subjective sleep quality (low scale value) was related to higher levels of cortisol at these sampling points. However, the correlation was significant only for TN and cortisol levels at 30 minutes post awakening. Table 3 also shows that there were tendencies to relationships between higher ratings of tiredness in the morning and lower cortisol levels for both noise exposure conditions.

A tendency to a relationship was also found for a higher rating of irritation in the morning after LFN exposure and lower cortisol levels after 45 minutes. After nights with LFN, higher cortisol levels were significantly related to higher degrees of activity and pleasantness.

Table 3
Correlation coefficients between subjective response and cortisol levels at 30 and 45 minutes

Response variable	Cortisol level at 30 minutes				Cortisol levels at 45 minutes			
	TN		LFN		TN		LFN	
	r	p-value	r	p-value	r	p-value	r	p-value
Sleep quality	− 0.66	<0.05	− 0.34	>0.10	− 0.55	0.062	− 0.31	>0.10
Morning:								
Tiredness	− 0.53	0.075	− 0.33	>0.10	− 0.40	>0.10	− 0.56	0.059
Irritation	− 0.21	>0.10	− 0.44	>0.10	− 0.05	>0.10	− 0.50	0.097
Activity	− 0.23	>0.10	0.60	<0.05	− 0.20	>0.10	0.56	0.06
Pleasantness	0.04	>0.10	0.59	<0.05	0.14	>0.10	0.51	0.094

Discussion

This pilot study was designed to be hypothesis generating in character and the results must be interpreted with caution. The exposure conditions were developed to resemble normal sleeping. The subjects slept in home-like conditions and no invasive devices, such as electrodes, interfered with the subject's normal sleep. Nevertheless, sleeping in a new environment will alter the normal sleep for at least the first two nights, after which sleep is comparable to the normal sleep at home [15]. Comparisons of results obtained in laboratory and field studies using similar methods show fairly good agreement for difficulties in falling asleep and perceived sleep quality, while awakenings are less frequently reported in the field [24]. The effects recorded in this study are the result of acute exposures and in order to study the relevance of these effects for long-term health, field studies are necessary. As this was the first study to evelute effects of noise on cortisol response upon awakening, it was however necessary to study the mechanisms under controlled condition. Little is known of the habituation due to noise on the cortisol response upon wake up. Previous studies on urine cortisol have found increased levels of urine cortisol after chronic exposure to transportation noise [11–13]. This would indicate that the secretion of cortisol as measured in urine show little habituation to noise. The study reported here indicated that the habituation to the new sleep environment affected the subjective sleep parameters, while the effect of the cortisol response was less clear. If the subject had not properly habituated to the new sleeping conditions, the difference in subjective ratings between the reference night and night with noise exposure could have been even greater. In new studies this should be investigated by extending the number of reference nights.

The main finding of this study was that the cortisol awakening response was altered following nocturnal exposure to low frequency noise. Most notably, levels of cortisol had not peaked by 30 minutes post awakening after exposure to low frequency noise, and these attenuated levels of cortisol were related to tiredness and negative mood. Other studies investigating exposure to transportation noises have reported higher levels of cortisol in urine collected in the morning. The findings here of an attenuated response soon after awakening are however not necessary contradictory as the cortisol levels, which had not peaked, may have continued to rise beyond the sampling time of this experiment to give a net increase in levels over a more prolonged time period. Alternatively, noise exposure might have led to cortisol secretion earlier during the night, which would be detectable in the urine but not in saliva samples, which more readily reflect the current circulating levels. A recent study among children living close to a road with 24 h lorry traffic supports the latter hypothesis as a significant correlation was found between the maximal levels of low frequencies in the noise, measured as max C-weighted levels, and urine cortisol levels sampled in the first half of the night, while no correlation was found between noise level and the excretion in the second half of the night [25].

Subjective reports of the time required to fall asleep and how the subjects felt in the morning showed that the noise exposures affected the subjects' sleep. Although the intention was not to compare the two noise exposures, the data indicate that the responses during exposure to traffic noise and low frequency noise differed. The cortisol response was attenuated only after nights with low frequency noise. For low frequency noise, a correlation was found between cortisol levels 30 and 45 minutes after awakening and tiredness, activity and pleasantness experienced in the morning while, for traffic noise, a correlation was found for sleep quality. Low frequency noise mainly affected the time to fall asleep, which was reported to be nearly twice as long as for the reference night, and the feeling of being irritated in the

morning. These effects were also found in a field study among subjects annoyed by low frequency noise [19]. Traffic noise mainly had an effect on sleep quality and the feeling of being irritated in the morning. Effects on sleep quality by traffic noise have been reported previously and significant decreases in sleep quality have been found in a noise exposure with 32 noise events at 45 dB LAmax [26].

There are some possible explanations for different response patterns after the two noises. One hypothesis could be that the subject reacted differently to the noises depending on their previous experience of sleeping in traffic noise as compared to low frequency ventilation noise. The reaction to the traffic noise exposure could therefore be in accordance with the stage of resistance, while the reaction to the ventilation noise would be in accordance to the alarm reaction, described by Seyle [27]. In this study there was a rather equal number of subjects who in their home slept in a bedroom facing a street (four subjects) and bedroom facing an inneryard (three subjects), hence there is not enough data to refute or support the hypothesis. The hypothesis deserves however further attention and should be look at in a study comprising a larger number of subjects. Another explanation to the different response may be the slight difference in the frequency spectra between the noises, where the difference is found in the frequency range of 40 to 80 Hz. It has previously been found in studies of annoyance that energy in the lower range of the low frequencies is more disturbing and annoying [28], although no information is available with regard to sleep disturbance. A further difference between the noises was that the low frequency noise, with the exception of the two periods during the night when it was turned off, was a continuous noise, while the traffic noise was of an intermittent character. In agreement with the effects seen in this study, Öhrström and Rylander [29] reported that continuous noise had a significantly smaller effect on sleep quality than intermittent noise. According to Eberhardt *et al.* [30], intermittent noise mainly has effects on deep sleep while continuous noise decreased REM sleep. Also, Ehrenstein and Muller Limroth [31] found that continuous noise had little effect on sleep pattern with the exception of the REM stage, which decreased. A reduction of REM sleep and increase of stage 2 sleep due to continuous noise was found by Tamura *et al.* [32] after exposure to ship noise at levels of 65 dBA. From these data, it may be hypothesised that continuous and intermittent noises affect different sleep stages and that this has consequences for subjective sleep disturbance and perhaps also for the cortisol response.

Previous studies have shown an association between normal sleep and nocturnal hormones in humans. The major portion of REM sleep occurs during the second half of the night, when the plasma cortisol levels are beginning to rise [33,34]. If sleep is disturbed, endogenous plasma cortisol levels vary depending on which sleep stages are affected. Decreased endogenous plasma cortisol levels during REM sleep have been reported, while shallow sleep and intermittent wakefulness have been associated with increased plasma cortisol levels [35,36]. This was furthered investigated by Born *et al.* [37] who found that a deprivation of REM as well as arousals in non-REM stages reduced rather than enhanced mean plasma cortisol levels as compared to baseline. Their findings suggest that not only disturbed REM stage but also the awakenings and subsequent light sleep have no stimulatory effect on the adrenocortical secretion. Recordings of sleep stages would be necessary to further evaluate the relationships between noise exposures, effects of sleep stages and cortisol response upon waking up.

In conclusion, this study has shown that night time exposure to low frequency noise at a level of 40 dBA disrupts the normal cortisol response to awakening and that this disruption is associated with self-reported measures of sleep quality and mood. However, the small sample size suggests that these

preliminary findings be treated with caution. A further study using a larger sample is currently under-way.

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