Acoustic, individual, and situational determinants of vegetative and cortical arousals induced by traffic noise during sleep

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INTRODUCTION

A recent publication by the World Health Organization (WHO) on the burden of disease from environmental noise concludes that an estimated 1.6 Million healthy life years are lost in the European Union annually (Fritschi et al. 2011). Notably, 903,000 healthy life years lost (or 55.8 %) were attributed to noise induced sleep disturbance, thus acknowledging the importance of undisturbed sleep of sufficient length for health and well-being.

Sleep recuperation is a very active process, and noise-induced disturbance of this process impairs restoration (Muzet 2007; Basner et al. 2010). At the same time, we are not aware of ourselves and our surroundings during sleep (i.e., unconscious). We have thus limited insight into our own sleep process, and self-reports may both underestimate (e.g., in insomnia) or overestimate (e.g., in obstructive sleep apnea) sleep duration and quality. Therefore, physiologic measurements (e.g., polysomnography or actigraphy) have been used to gather objective information on the effects of noise on sleep (Muzet 2007; Basner et al. 2010; Griefahn & Basner 2011). Due to the high methodological expense of physiologic measurements, the field has mostly seen small studies in healthy, young populations with limited representativeness for the exposed population (Basner et al. 2010). These studies consistently show that whether or not sleep is disturbed by environmental noise depends on individual, situational, and acoustical factors (see Figure 1) (Marks et al. 2008; Basner et al. 2011). However, this is not reflected in noise policy and regulation, where average noise levels (like L_{night}), integrating noise exposure during a specific period (e.g., 23:00 to 7:00) over several months, are often used as the sole criterion. Thus, important acoustical, individual, and situational variables that could be used to better predict and mitigate the effects of environmental noise on sleep are often not taken into account.

Basner argues that a variable should only be included in a prediction model on the effects of noise on sleep if (a) the variable has a relevant impact on sleep, (b) information on the distribution of the variable in the target population is available, and (c) the distribution of the variable in the target population differs relevantly from the distribution in the population that was used to generate the prediction model (Basner 2009). Furthermore, even variables that do not fulfill all of the above criteria may often be used to inform political decision making processes.

Here, we report on the results of a polysomnographic laboratory study on the effects of air, road, and rail traffic noise on awakenings, EEG arousals, and cardiac arousals where several acoustical, individual, and situational variables were taken into account (Basner et al. 2011).

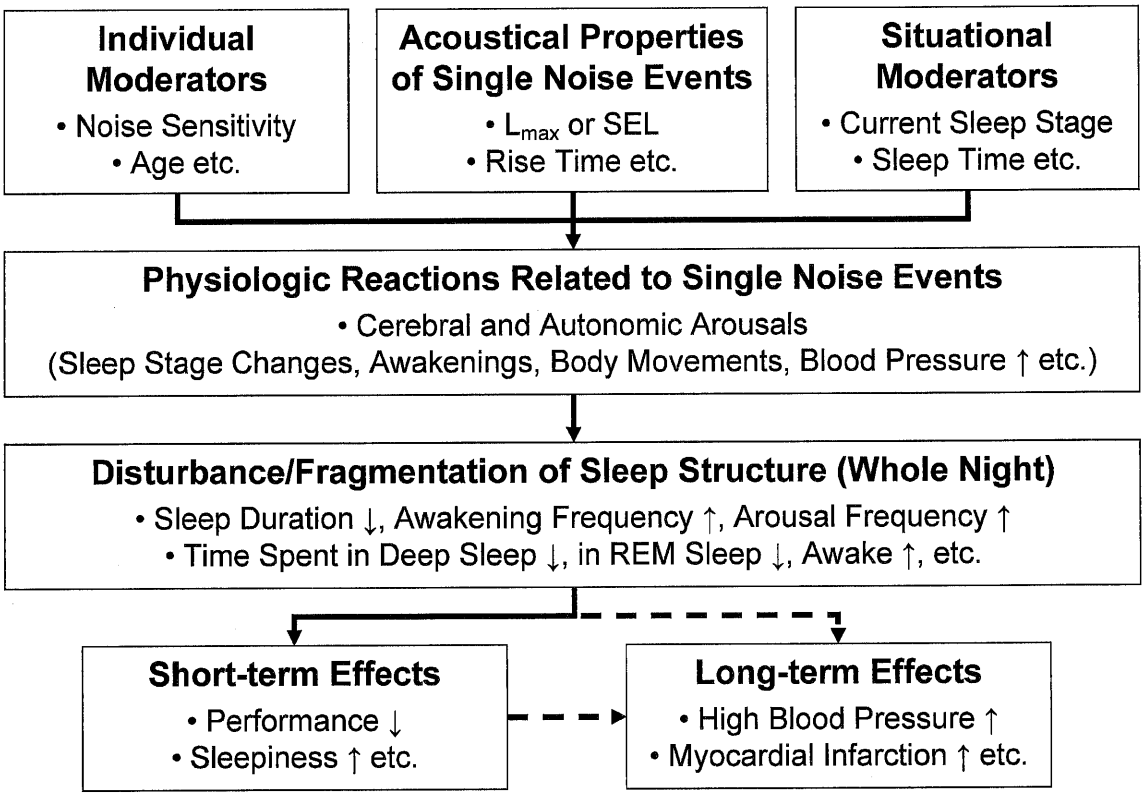

Figure 1: Flow chart on the effects of traffic noise on sleep. L_{max} = maximum sound pressure level, SEL = single event level.

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METHODS

We polysomnographically investigated 72 healthy subjects (40 ± 13 years, range 18-71 years, 32 male) for 11 consecutive nights each in the sleep laboratory of the German Aerospace Center (DLR) in Cologne. Physiological variables included the electroencephalogram (EEG: C3-A2, C4-A1), the electrooculogram (EOG), the electromyogram (EMG), the electrocardiogram (ECG), respiratory movements of rib cage and abdomen, and finger pulse amplitude. Additionally, subjects wore actigraphs 24 hours each day. The study included 8 nights with 8 hours time in bed and with exposure to 40, 80, or 120 pre-recorded air, road, and/or rail traffic noise events with $L_{AS,max}$ of 45, 50, 55, 60, or 65 dB, and one noise-free control night. The first night served as adaptation, was noise-free and excluded from the analyses (for a detailed description of the protocol see Basner et al. 2011). The study was approved by the local ethics committee. Subjects gave written informed consent prior to study participation and were free to discontinue the study any time.

This report concentrates on an event-related analysis on the effects of acoustical, individual, and situational variables on EEG awakenings, shorter cortical activations in form of EEG arousals (Bonnet et al. 1992), and changes in heart rate (representing vegetative arousals). Awakenings were defined according to Rechtschaffen & Kales. (1968) as sleep stage changes from any sleep stage other than wake to stage wake.

By design, noise events started exactly at the beginning of a sleep epoch, which was then defined as the first epoch under the influence of noise. A noise event was excluded from the analysis if the subject was already awake in the epoch preceding the first noise epoch. Therefore, noise events outside of the sleep period time, i.e. before sleep onset or after final awakening, were also excluded from the analysis. The first noise epoch and the epoch following it were screened for an EEG awakening, as this maximized the difference in awakening probability with and without noise exposure (i.e. signal to noise ratio).

For EEG arousals, a 60 s window following the beginning of a noise event was screened for arousal onset. This way, noise events from the three traffic modes were compared on an equal footage, and comparisons with the analysis based on EEG awakenings were facilitated. The noise event was only included if it fell within sleep period time (SPT) and if the 10-s interval preceding noise onset was free of EEG arousals.

For heart rate analysis, heart beats with inter-beat intervals (IBI) > 2 s or < 500 ms (corresponding to heart rates of < 30 bpm and > 120 bpm) were considered invalid (less than 0.2 % of all beats). Nights where valid heart beats covered less than 95 % of SPT were excluded from the analysis (N=28). For each noise event, maximum heart rate was identified in a 60 s time window following noise onset. Then, average heart rate was calculated for an interval ±10 s relative to the moment when maximum heart rate occurred. The same procedure was repeated for a 30 s time window preceding noise onset. The difference between average heart rate after and before noise onset was calculated and constituted the outcome variable for the event-related analysis. This difference increases both with amplitude and duration of a noise-induced heart rate response. Noise events were excluded from analysis if the screening window contained > 10 % invalid heart beats (see above) or a single heart beat with an IBI > 6 s.

Altogether, 31,266 noise events contributed to the analysis of awakening probability, 29,151 noise events contributed to the analysis of arousal probability, and 30,224 noise events contributed to the event-related heart rate analysis. Proc NLMIXED (SAS 9.2) was used to perform random subject effect regressions. For the awakening and arousal data, random subject effect logistic regression was performed. The dichotomous dependent variable was classified as 1 for an awakening or arousal and 0 for no awakening or no arousal. For the heart rate data, random subject effect linear regression was performed.

Several independent variables were considered as predictors or mediators: two indicator variables for traffic mode, contrasting air and rail traffic with road traffic (reference), maximum sound pressure level, age, gender, sleep stage in the epoch preceding the noise event with stage 2 as reference, elapsed sleep time since sleep onset, study night (2-11), the number of noise events per night with single exposure nights (40 noise events) serving as reference, noise-free interval between the end of the last and the beginning of the current noise event, noise event duration, sound pressure level rise time, and octave band energy for mid frequencies from 31.5 Hz to 8 kHz. Here, we report on the effects of individual, situational, and acoustical variables for the fully adjusted model. All continuous variables were mean-centered and inspected for linearity in the logit of awakening and arousal probability, and in heart

rate changes, respectively. Based on the results of this analysis, maximum sound pressure level (SPL) was the only variable to enter the models non-linearly.

RESULTS AND DISCUSSION

Table 1 summarizes the findings of the multivariable random subject effect regression models. Variables that promote EEG awakenings, EEG arousals, or cardiac arousals are shaded red, those that are associated with a decrease in EEG awakening or EEG arousal probability or heart rate are shaded green. The level of statistical significance is indicated by stars (★p<0.05, ★★p<0.01, ★★★p<0.001, ★★★★p<0.0001, statistically non-significant effects have a white background).

Table 1: Findings of the event-related analyses

	EEG Awakenings	**EEG Arousals**	**Cardiac Arousals**
Acoustical moderators			
$L_{AS,max}$ [dB]			
SPL rise time [dB/s]			
Octave energy 31.5 Hz [dB]			
Octave energy 63 Hz [dB]			★
Octave energy 125 Hz [dB]			
Octave energy 500 Hz [dB]			
Octave energy 4 kHz [dB]			
Octave energy 8 kHz [dB]			
Individual moderators			
Age [years]			★
Male gender			
Situational moderators			
Sleep stage S1 vs. S2			
Sleep stage SWS vs. S2	★★★★	★★★★	★★★★
Sleep stage REM vs. S2	★★★★	★★★★	★★★★
Elapsed sleep time [h]			
Study night [d]	★★	★	
80 vs. 40 noise events	★★★	★★★★	★★★★
120 vs. 40 noise events	★★★★	★★★★	★★★★

SPL = sound pressure level; $L_{AS,max}$ = maximum SPL; SWS = slow wave sleep; REM = rapid eye movement sleep; mid frequencies are given for octave bands; adopted from Basner et al. (2011)

Acoustical moderators

Rise time: Beside the maximum sound pressure level $L_{AS,max}$, central nervous system arousal probability increased with sound pressure level rise time. Nocturnal speed limits both decrease sound pressure levels and sound pressure level rise times, and could therefore effectively be used to decrease noise induced sleep disturbance.

Spectral composition: Sound energy especially in the high frequency domains > 3 kHz were associated with higher arousal probabilities. The spectral composition of the noise events was able to explain the differences between the effects of air, road, and rail traffic noise on cortical and cardiac arousal probability that were seen in descriptive statistics and unadjusted models. Because of atmospheric absorption of high frequency components of aircraft noise but not of road and rail traffic noise, aircraft noise was associated with significantly lower arousal probabilities relative to road and rail traffic noise (Marks et al. 2008; Basner et al. 2011). These findings may have implications for the design of active and passive noise control measures, and for the optimization of the sound design of the noise source. The variables "noise event duration" and octave bands with mid frequencies of 250 Hz, 1 kHz, and 2 kHz were statistically non-significant (P>0.05) and thus removed from the final models.

Individual moderators

Age: Our models indicate a non-significant increase in awakening probability, a significant increase in EEG arousal probability, and a significant decrease in heart rate response with increasing age. A similar study by Marks et al. (2008) found significant increases in awakening probability only after the data of air, road, and rail traffic noise were combined into a single model. However, one has to keep in mind that the effect of age was adjusted for several other situational and individual moderators. Thus, the age variable will only show effects in excess of what is already explained by the other variables (e.g., sleep stage distribution varies with age, and the momentary sleep stage is a potent predictor of arousal probability, see below). In this light, our findings corroborate that older age is an important risk factor for noise induced sleep disturbance.

Gender: Arousal probabilities were consistently higher for male relative to female study participants. Marks et al. (2008) saw no statistically reliable gender effect on awakening probability, and an earlier study on the effects of aircraft noise on sleep found slightly higher awakening probabilities for female subjects (Basner et al. 2004). These discrepancies may be explained by the relatively low sample sizes. Larger studies are needed to clarify whether one sex is more at risk for noise induced sleep disturbance than the other. Some epidemiological studies suggest that there are differences in the effects of noise-induced cardiovascular disease between men and women (Babisch et al. 2005).

Noise sensitivity: Although we did not test for the significance of subjectively perceived noise sensitivity as an individual moderator, both Marks et al. (2008) and Basner et al. (2004) found no statistically reliable effect of noise sensitivity on awakening probability.

Situational moderators

Sleep stage: The momentary sleep stage is a potent moderator for the effects of noise on arousal probability. Arousal thresholds were highest for slow wave sleep (SWS, also called "deep sleep"), followed by REM sleep, S2 sleep, and S1 sleep. As SWS is predominantly seen during the first half of the night, this part of the sleep period is often considered less vulnerable for the effects of noise on sleep (although it is unclear whether or not the disturbance of SWS is associated with a higher "physiological cost" compared to, e.g., the disturbance of S2 sleep). As SWS amounts de-

crease with age, older subjects are usually more easily aroused from sleep, while it is harder to arouse children. The latter are nevertheless regarded a group at risk, as they are in a vulnerable developmental life period. At the same time, subjects with a genetically determined shallow sleep or insomnia (who have trouble initiating and maintaining sleep) may be at greater risk for noise induced sleep disturbance.

Elapsed sleep time: Additional to these sleep stage effects, awakening probability and heart rate increased with elapsed sleep time (EEG arousal probability increased statistically non-significantly), again pointing to a higher vulnerability of sleep towards the end of the night. Due to the decreased sleep pressure in the early morning, subjects are not only more easily aroused from sleep, but it is also harder to re-initiate sleep after a spontaneous or noise-induced awakenings (Basner & Siebert 2010).

Repeated exposure: We also observed significant habituation effects for EEG awakenings, EEG arousals, and cardiac arousals within a single night (i.e., arousal probabilities per noise event were lower in nights with a higher number of noise events). This effect was also seen for EEG awakenings and EEG arousals across study nights (i.e., arousal probability decreased from study night 2 to study night 9). These habituation effects are most likely caused by a decrease in the importance of noise events due to repeated stimulation, and it seems biologically plausible in terms of sleep homeostasis and energy conservation. It is unclear whether this effect represents true habituation or whether it can be, at least in part, explained by increased arousal thresholds due to noise-induced sleep fragmentation in previous exposure nights or in preceding parts of the same night. According to Bonnet (1985), both is probably true. Interestingly, cardiac arousals did not habituate across nights, corroborating earlier findings (Griefahn et al. 2008) and stressing the potential relevance of nocturnal cardiac arousals for the genesis of long-term cardiovascular health effects. The variable "noise-free interval" was statistically non-significant ($P>0.05$) and thus removed from the final models.

CONCLUSIONS

These results stress that the degree of noise-induced sleep disturbance depends on several acoustical, situational, and individual variables. This knowledge can be used to improve predictions of aircraft noise effects on sleep and improve mitigation measures. Relying on average noise levels alone for regulation purposes fails to properly address the complex interactions between acoustical, situational, and individual factors. Noise prediction models need to be further improved in order to reliably predict the time course and spectral composition of single noise events. Field studies on more representative populations are needed for validation of our results in a setting with higher ecologic validity.

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