



Effects of continuous and intermittent transportation noise on sleep fragmentation

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ABSTRACT

Nocturnal transportation noise disturbs sleep by causing awakenings, sleep-stage changes or EEG arousals. However, it is unclear which acoustical characteristics cause these effects. Twenty-one healthy participants (age range 19-69 y) were exposed to four different nighttime noise scenarios along with two noise-free nights. Scenarios varied with respect to short-term exposure variation over time [Intermittency Ratio (IR) low, medium or high] and source (road or train), but had a constant hourly L_{Aeq} of 45 dB at the sleeper's ear. Sleep and EEG arousals were scored according to standard criteria. There was no significant difference in any of the global sleep variables (e.g. TST) between nights. However, we found an increase in additional arousals (calculated as a percentage difference between the number of arousals during noise nights and spontaneous arousal during noise-free nights) during REM sleep for all noise-disturbed nights irrespective of the noise source. Arousal probability was significantly related to the maximum sound pressure level and the slope of rise (dB/s). In sum, REM sleep seems particularly susceptible to fragmenting effects of nocturnal transportation noise.

Keywords: Transportation Noise, Sleep, Arousal Probability,
I-INCE Classification of Subjects Number: 62.5

1. INTRODUCTION

Nocturnal transportation noise disturbs sleep by causing awakenings, sleep-stage changes or EEG arousals [1, 2]. However, awakenings and arousals are also an integral component of normal non-disturbed sleep with spontaneous hourly arousal frequencies between 9.8 (18-20 years) and 21.2 (61-70 years) [3]. In order to attribute an observed arousal or awakening to a specific noise event, one has to disentangle spontaneous arousals from noise-induced arousals, which may partly only substitute spontaneous arousals. Moreover, bodily arousal reactions to external disturbing events comprise varying degrees and are best described by an arousal hierarchy that ranges from minimal vegetative arousals, brief EEG arousals with or without an associated body movement to full awakening [4]. Maximum sound pressure levels and sound exposure levels are widely used acoustical predictors to estimate sleep disturbances and fragmentation [5-7]. However, other acoustical parameters such as the slope of rise of the sound pressure level or an exposure variation over time might also influence

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arousal and awakening probabilities. The slope of rise for example was used to estimate awakening probability from rail and aircraft noise events [8], actimetry derived motility reactions to aircraft noise events [9] and cardiac acceleration to traffic noise [10].

2. METHODS

2.1 Participants and Procedure

Twenty-one participants (age range 19-69 y, 9 women) underwent a 6-day study protocol (six scheduled night-time sleep episodes of 8 hours and five scheduled wake episodes on consecutive days) in a sleep laboratory. Participants were healthy by medical history, physical examination and one night of polysomnographic screening. Exclusion criteria comprised: sleep disorders (e.g. periodic limb movements, sleep apnea or insomnia), hearing impairment (hearing devices or abnormal hearing thresholds according to age and sex), diseases of somatic origin (particularly cardiovascular diseases such as high blood pressure or cardiac arrhythmia) or drug and alcohol abuse. EEG (12 derivations), EMG, EOG and ECG were recorded continuously during the four noise nights and the two noise-free nights (the first night served as baseline night, and the last served as recovery night). The four noise scenarios were applied in a randomized incompletely balanced order.

2.2 Noise Scenarios

Five different noise scenarios were applied. One scenario was used during both the baseline and recovery night and comprised ambient sound (cricket chirps, distant traffic) with a constant hourly L_{Aeq} of 30 dB at the ear of the sleeper. The noise scenarios differed with respect to noise source (railway noise and different road traffic situations) and short-term exposure variation over time [Intermittency Ratio (IR) low, medium or high] [11]. All had a constant hourly L_{Aeq} of 45 dB at the sleeper's ear, which corresponds to an outdoor level of approx. 60 dB for a tilted window (see Table 1 for the acoustic properties and Figure 1 for the cumulative distribution of the sound pressure level for the used noise scenarios).

Table 1 – Acoustical characteristics of the noise scenarios

Scenario	L_{Aeq} [dB]	L_{AFmax} [dB]	L_{A5} [dB]	L_{A10} [dB]	IR [-]
Baseline	30	39	35	34	0.3
A	45	53	49	48	0.3
B	45	60	52	48	0.7
C	45	62	52	48	0.8
D	45	62	53	46	0.9

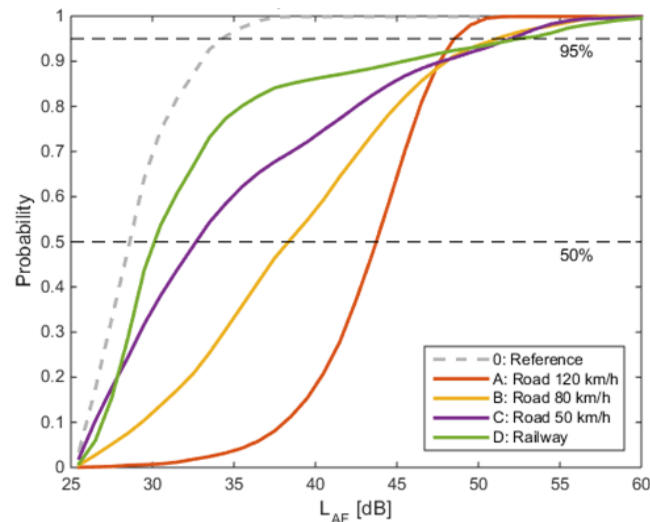


Figure 1 – Cumulative distribution function of the sound pressure level for the five applied noise scenarios

The auralisations were based on real world field recordings of single noise events that were filtered (e.g. simulation of a tilted window) and in the case of the road scenarios combined to simulate traffic based on measured real-world distributions. *Scenario A* (road, low intermittence) represents a 4-lane highway (speed limit of 120 km/h) with approximately 1000 vehicles per hour in a distance of 400 m. *Scenario B* (road, medium intermittence) represents a distance of 50 m from a 2-lane country road (speed limit of 80 km/h) with approximately 250 vehicles per hour. *Scenario C* (road, high intermittence) represents a 1-lane urban road (50 km/h) at a 15 m distance with approximately 100 vehicles per hour. For *scenario D* (train, high intermittence), recordings of eight freight and two commuting trains were combined so that the number of trains per hour was 10 (pseudorandom equidistant spacing at ≈ 300 s). The 80 distinct noise events of noise *scenario D* differed with respect to event duration, maximum sound pressure level (L_{AFmax}), sound exposure level (SEL) and positive slope of the sound pressure level (calculated according to [9] with a 10 dB difference). The event duration for the 10 events ranged between 15.7 and 114.7 s, L_{AFmax} ranged between 50 and 61.7 dB, SEL between 62.4 and 75 dB and the maximum slope of the sound pressure level between 0.5 and 3.4 dB/s. Sound files were played back from portable audio devices (Sound Devices type 702T) through one active monitor (Focal CMS 50) at a distance 2 m to the sleepers head. The sleep rooms were acoustically measured and calibrated using a sound level meter and by adjusting the playback volume.

2.3 Data Analysis

Polysomnograms and EEG arousals were scored manually by four experienced raters (accordance between raters was assured $> 85\%$) according to standard criteria ([12] for sleep stages and [13] for EEG arousals). A sleep stage change from any sleep stage to wake was defined as awakening. To determine noise effects on total number of arousals, the total number of arousals during the noise-free baseline night was subtracted from the respective noise nights to account for spontaneous, non-noise related arousals. For analysis of the sleep structure and sleep continuity variables we used repeated-measures analyses of variance. Sound pressure and polysomnography were recorded simultaneously to facilitate event-related analysis for noise *scenario D*. If an EEG arousal occurred within a certain time window after exceeding a threshold of 35 dB(A) of a single train noise event (TNE), it was considered noise associated. The analysis window size was set to 60 s (see for example [5, 6]). To estimate the probability of noise associated arousals, random subject effect logistic regression models were used which included acoustical (event duration, maximum sound pressure level, SEL, maximum slope of the sound pressure level), sleep related (epoch since sleep onset, dichotomous variables for sleep state in the epoch of the threshold exceedance for SWS and REM [e.g. 1 = REM, 0 = every other stage]) and subject variables (age [binary dummy variable with 1 = age > 55 years, 0 = age < 55 years], sex [female, male]).

3. RESULTS

3.1 Sleep Structure

Results for the standard PSG derived sleep parameters are displayed in Table 2. There were no significant differences between nights, i.e. the different noise scenarios for none of the sleep variables.

Table 2 – PSG derived sleep parameters for the different noise scenarios

Noise	TST [min]	SE [%]	WASO [min]	SL2 [min]	RL [min]	Stage 1 [%]	Stage 2 [%]	Stage 3 [%]	Stage 4 [%]	SWS [%]	NREM [%]	REM [%]
BL	442	92	31	14	92	14	53	7	7	14	81	19
	25	5	24	6	31	5	7	3	7	8	5	5
A	448	93	24	16	63	13	51	7	7	14	78	22
	36	8	34	13	22	5	8	3	6	8	5	5
B	441	92	29	18	62	13	50	8	8	15	78	22
	28	6	25	7	15	5	7	3	7	8	5	5
C	443	92	29	16	61	15	50	7	6	13	78	22
	28	6	27	7	12	5	7	3	6	7	5	5
D	449	93	23	17	68	14	50	7	7	14	78	22
	19	4	19	9	30	5	8	3	7	8	6	6
REC	442	92	25	21	54	15	48	7	7	14	77	23
	26	5	24	8	16	4	7	3	6	7	4	4

Mean values and standard deviations underneath. BL = baseline night (always first night), REC = recovery night (always last night), TST = total sleep time [stages 1–4 + REM], SE = sleep efficiency $[(TST/480)*100]$, WASO = wake after sleep onset, SL2 = latency to stage 2, RL = SL2 to first epoch of stage REM, for all sleep stages: percent of TST.

3.2 Sleep Continuity

Awakening reactions per hour, sleep stage changes per hour, ASDA EEG Arousals per hour of total sleep time and the total difference between the noise-free control night and the respective noise night during NREM and REM sleep (calculated for each participant individually) were used to describe sleep continuity and are shown in Table 3. There were no significant differences observed between nights for these sleep continuity measures.

Table 3 – Analysis of sleep continuity for the different noise scenarios

Noise	AWR	Stage	ASDA Arousals	ASDA Arousals n-c	ASDA Arousals n-c
	[N/h]	changes [N/h]	[N/h]	NREM [N]	REM [N]
BL	2.11 ± 1.14	21.08 ± 5.84	11.75 ± 4.75	-	-
A	1.73 ± 1.07	19.57 ± 5.72	12.09 ± 6.87	-1.25 ± 23.13	2.15 ± 8.81
B	1.93 ± 1.14	21.18 ± 7.59	11.78 ± 6.23	-3.40 ± 22.43	3.55 ± 7.99
C	2.19 ± 1.14	21.68 ± 6.47	12.46 ± 5.17	2.70 ± 21.66	2.15 ± 6.33
D	2.12 ± 1.25	21.41 ± 5.74	12.54 ± 5.26	3.60 ± 17.92	3.65 ± 8.34
REC	1.65 ± 0.78	21.18 ± 6.91	13.59 ± 6.30	-	-

Mean values ± standard deviations. BL = baseline night (always first night), REC = recovery night (always last night), AWR = Awakening reactions per hour of total sleep time, ASDA Arousals n-c is the difference between the first noise-free control night and the respective noise night calculated for each participant individually.

The percentage change for arousals in NREM sleep was significantly higher during nights with high intermittency as compared to low, medium intermittency and quiet nights, whereas percentage change for arousals in REM sleep was significantly higher for all noise scenarios as compared to the noise-free nights (repeated measures ANOVA revealed a significant interaction between noise and NREM/REM state: $F(4, 72) = 2.62, p = .030$). Moreover, differences between the individual noise-free control night and the respective noise night show fairly substantial inter-individual differences as indicated by the rather wide span between the upper and the lower quartile of the boxplots (see Figure 3).

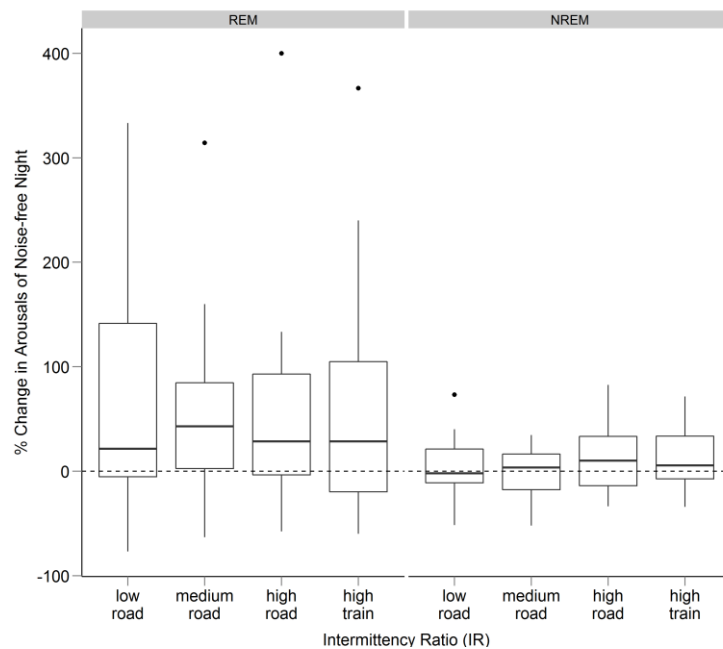


Figure 3 – Percentage change to noise-free conditions for the number of arousals during REM and NREM sleep for the different Intermimency Ratio classes. The dashed line indicates the average number of arousals during the noise-free night

3.3 Event-Related Analysis

1.680 single TNE's were applied over the course of the 21 nights with *scenario D*. We excluded 115 events (6.85 %) as participants were awake during these events, either during sleep onset at the beginning of the night, during the night or prior to lights on in the morning. Finally, 1.565 TNE's were considered for the analysis. Arousal probability decreased significantly during slow wave sleep as compared to all other sleep stages. REM sleep however did not significantly contribute to the prediction, while the factor age significantly influenced arousal probability: the older participants were more likely to display noise induced arousals than the young sleepers. Probability of a noise induced arousal increased significantly with two acoustical descriptors: the maximum sound pressure level (L_{AFmax}) and the maximum slope of the sound pressure level (dB/s). If both were included in a combined model, only the maximum slope of the sound pressure level significantly influenced arousal probability (see Table 4 for the coefficients of the logistic regression model).

Table 4 – Coefficients of a logistic regression model for arousal probability during a 60-s time window during and after TNE

Effect	Estimate	SE	z	p
Intercept	-2.85	0.89	-3.22	0.002
Slope of rise (dB/s)	0.24	0.06	4.33	< 0.001
L_{AFmax}	0.02	0.02	1.09	0.277
SWS during event (1 = yes)	-1.40	0.28	-4.96	< 0.001
REM during event (1 = yes)	0.10	0.14	0.67	0.504
Age (1 = older)	0.66	0.18	3.66	< 0.001

4. CONCLUSION

Sleep macrostructure and continuity were not significantly altered by nocturnal transportation noise in our preliminary analysis with the first sample of healthy sleepers. However, we found an increase of additional arousals (calculated as a percentage difference between the number of arousals during noise nights and spontaneous arousal during noise-free nights) during REM sleep for all noise-disturbed nights irrespective of noise source and short-term variation over time. Besides evaluating the all-night number of arousals, we will also apply the same methodology used for the event-related analysis to screen for spontaneous arousal reactions to “virtual” noise events during the noise-free nights. Arousal probability significantly increased with the maximum sound pressure level and the slope of rise—the latter being the better predictor for arousal reactions. Arousal probability was also positively related to age and negatively to slow wave sleep. However, there are still other predictors such as individual spindle density [14], which may explain inter-individual variability of noise-related responses during sleep. In sum, we have evidence that REM sleep is particularly susceptible to fragmenting effects of nocturnal transportation noise, and the slope of rise of the decibel level plays an important role in noise-related arousal reactions.

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