Physiological reaction thresholds to vibration during sleep

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Summary
In the Night Noise Guidelines for Europe 2009, the World Health Organisation report that there is sufficient available evidence for biological effects of noise on sleep starting from $L_{AF,max,inside}$=32 dB. There is however no such evidence for vibration. Vibration is expected to affect an increasing number of people living close to rail or trafficked roads. It was therefore the objective to determine vibration amplitude thresholds for sleep disruption. These disruptions include cortical arousals and awakenings, alterations of sleep depth, and changes in cardiovascular activity. An experimental laboratory study was performed in a setting designed to closely resemble a home environment. Participants slept in the sleep laboratory for five consecutive nights, composed of a habituation night, a quiet control night, and three exposure nights. Sleep was measured using polysomnography, and heart rate measured using electrocardiogram (ECG). This pilot study involved five young, healthy participants, free of self-reported sleep problems. Exposure nights composed of 36 simulated freight train passages, with low level noise ($L_{AF,max}$=30dB) and vibration. Each series of 36 trains involved 12 trains per night with maximum vibration amplitudes of 0.1, 0.2 and 0.3 mm/s. The results indicate that alterations of sleep depth and cortical arousals may begin at 0.3 mm/s. A study involving a larger number of people is currently underway to further investigate thresholds for sleep disruption.

PACS no. 43.50.+y

1. Introduction

The WHO Night Noise Guidelines for Europe 2009 report that there is sufficient evidence for biological effects of noise on sleep, starting at $L_{AF,max,inside}$=32 dB [1]. Alterations in sleep structure and electroencephalogram (EEG) awakenings start to occur at $L_{AF,max,inside}$=35 dB. Changes in cardiovascular activity during sleep also occur as a result of noise exposure, but threshold levels have not been determined. Noise from freight trains in particular has been found to cause more frequent awakenings [2] and stronger cardiac response [3] than passenger trains. In the field, freight trains are often accompanied by low frequency vibration, with amplitudes commonly around 0.4 – 1.5 mm/s near railway lines [4]. As with noise, moderate to high level vibration from freight has been shown to contribute towards cortical EEG reactions and changes of heart rate during sleep [5-7]. However, it is not presently known at what threshold levels biological effects due to vibration begin to occur.

Vibration perception thresholds for vibration have been calculated for alert persons in the recumbent position, the same position adopted during sleep [8-10]. In the frequency range of freight vibration, approximately 5–10 Hz, this perception threshold corresponds to a maximum comfort weighted [11] amplitude of around 0.13–0.3 mm/s. ISO 2631-1 states that "*Fifty percent of alert, fit persons can just detect a W_k weighted vibration with a peak magnitude of 0.015 m/s^2*" [12]. At 10 Hz, this corresponds to a comfort weighted velocity of approximately 0.2 mm/s. Assuming that vibration amplitudes for perception and biological effect thresholds would be similar, it was therefore hypothesised that the reaction threshold for vibration during sleep would lie somewhere between 0.1 and 0.3 mm/s.

2. Methods

2.1. Study setting

The research group's sound environment laboratory was equipped to resemble an apartment, having a kitchenette, dining area and living space, showers, toilets and a private entrance. The

laboratory includes 3 private bedrooms, within each of which is a single bed with an electrodynamic transducer mounted to the underside of the bed frame. These transducers were within enclosures, and were used to introduce the desired vibration during the study nights. Loudspeakers mounted within the ceiling introduced the desired audio during the experiment.

2.2. Exposures

Artificial background ventilation noise L_{AEq}=25 dB was introduced into the bedrooms at all times. Vibrations of 0.1, 0.2 and 0.3 mm/s maximum velocity with 1s time weighting were used (Swedish comfort weighting [11]). The vibration signal was an amplitude modulated 10 Hz sinusoid, which is described in detail elsewhere [13]. The rise time of the vibration from 0 mm/s to the first maximum was 5.6s. The vibration signals of each train are presented in Figure 1. Vibration was accompanied by freight train noise exposure so that study participants could contextualise the vibration source. To avoid evoking a response to the noise rather than vibration, maximum noise levels of $L_{AF,max}$=30 dB, which falls below the biological reaction threshold, were used.
Two trains of different durations and at low noise levels were used each night (Table I). Each train occurred at 6 instances at 3 different vibration amplitudes. This yields a total of 2 trains × 3 amplitudes × 6 instances = 36 trains per night. The timing of the different vibration amplitudes was varied over the 3 exposure nights in a Latin square design. Trains started at 23:05:00, 23:15:00 and then subsequently every 13.5 minutes until 06:54:00.

Table I. Characteristics of trains in exposure nights.

Train	*Duration (s)*	$L_{AF,max}$ *(dB)*	$L_{A,Eq}$ *(dB)*
1	46.2	30	24.7
2	23.7	30	24.7

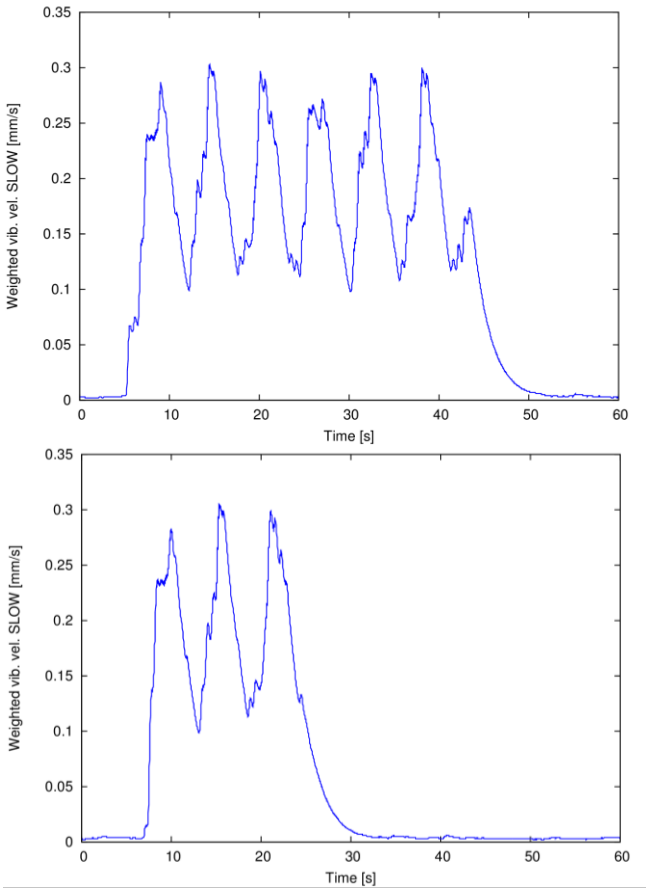

Figure 1. Vibration time history of train 1 (top) and train 2 (bottom) at maximum amplitude of 0.3 mm/s.

2.3. Sleep registration

Sleep was recorded via polysomnography (PSG) using surface EEG electrodes to register electrical brain activity, EOG to measure eye movements and submental EMG to record muscle tone. Electrode positions, impedances and sampling and filter frequencies were all in accordance with current guidelines [14]. Data were recorded offline onto an ambulatory PSG device (SOMNOscreen plus PSG+, SOMNOmedics, Germany), and analysed by a trained sleep technologist to identify sleep stage in 30s epochs and presence of EEG arousals [15]. Arousals of >15s were classified as awakenings.

Cardiac activity was recorded using a single modified electrocardiograph (ECG) Lead II. ECG electrode placement and sampling and filter frequencies were in accordance with guidelines [14].

2.4. Participants

Six healthy participants with good normal sleep were originally recruited, but one person dropped out after the habituation night. The remaining 5 participants (2 females and 3 males, mean age 23.2 SD±3.4 years) spent 5 consecutive nights in the laboratory. The first night was for adaptation to the environment and measurement apparatus and was not used in the analysis. The second night served as a control condition, to measure normal baseline sleep in the absence of train vibration or noise. Nights 3 to 5 served as experimental conditions, during which participants were exposed to railway freight vibration of different amplitudes during the night while they slept.

Participants were free to come and go as they wished during the daytime but were required to arrive at the laboratory by 20:00 each evening to allow sufficient time for relaxation and attachment of the sleep measurement apparatus. They were to begin attempting to fall asleep at lights-out at 23:00, and were woken by an alarm call at 07:00. During the study period they were prohibited from drinking caffeine after 15:00 and alcohol at any time. All participants were ranked as non-sensitive to noise according to a single item question reported previously [6]. They each provided informed written consent prior to commencement of the study, which was approved by the ethics committee of the University of Gothenburg.

2.5. Analysis

2.5.1. Event-related PSG

A computer routine was developed to determine whether exposure to any given train event was associated with a cortical response. In the routine, events where the participant was already awake were excluded in this routine. A 60s time window following the start of each train event was screened for the occurrence of an EEG arousal or awakening. The sleep stages in the three 30s epochs following train start were compared to the epoch immediately preceding train start to determine if a sleep stage change (SSC) to a less deep stage occurred. Changes to wake stage were not included, and rapid eye movement (REM) sleep was defined as the lightest stage for the purpose of analysis [16].

The prevalence of event-related arousals, awakenings and SSCs was used to calculate the probability of them being associated with train events of different vibration amplitudes. The control period was analysed at 36 time intervals corresponding to the times that trains would occur in the exposure nights. The resulting probabilities were the likelihoods of the reactions occurring spontaneously, in the absence of any stimulus.

2.5.2. Heart rate

A heart rate baseline was obtained by averaging 1s ECG samples in the 10s time window preceding the start of each train. This baseline was subtracted from the maximal heart rate occurring in the 60s time window following the start of each train to yield the maximum event-related heart rate change. Events where the participant was awake during the start of the train, or awoke during the train pass-by, were excluded from the analysis.

2.5.3. Statistical analysis

The event-related probabilities for each level of amplitude were compared to the spontaneous probability from the control condition in a generalised linear mixed model. Vibration amplitude was included as a fixed effect, and study participant was included as a random effect. Dunnet corrections were applied to the p-values, and the level of statistical significance was set at $\alpha=0.05$. Heart rate data were log-transformed before analysis to account for their non-normal distribution.

3. Results

Data were unavailable from a single exposure night in both weeks due to a technical issue. Data were therefore available from 10 participant exposure nights. The total number of observed event-related EEG arousals, awakenings and SSCs from these 10 nights for each vibration amplitude is presented in Table II.

Table II. Total number of observations of event-related cortical reactions used in probability calculations.

Amplitude (mm/s)	*Arousals (n)*	*Awakenings (n)*	*SSCs (n)*
Control	26	0	12
0.1	16	5	7
0.2	16	6	5
0.3	38	7	22

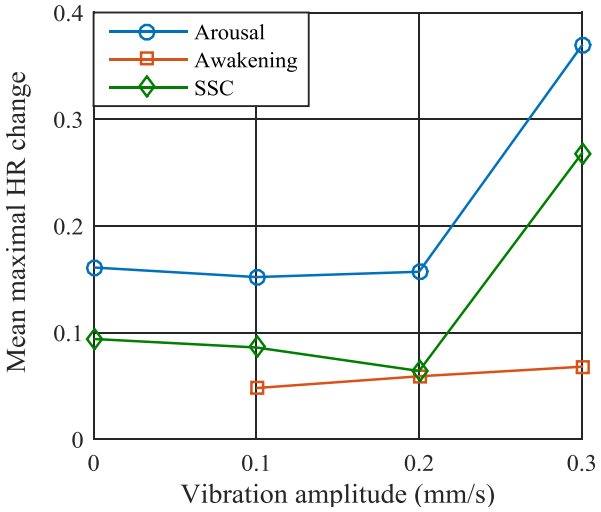

Figure 2. Mean probability of observing an EEG arousal, awakening, or sleep stage change (SSC) at different vibration amplitudes.

The mean probabilities of observing an arousal, awakening or SSC in the 60s following train start are presented in Figure 2. No awakenings were observed during the analysis periods in the control night, therefore it was not possible to determine the spontaneous awakening probability. Awakenings were therefore not investigated in the following analysis. Relative to the control condition, there are significantly higher probabilities of observing arousals ($p=0.007$) and SSCs ($p=0.02$) during 0.3 mm/s vibrations. No significant effects were seen at 0.1 or 0.2 mm/s.

The total number of events analysed in the heart rate analysis after excluding wake stages immediately prior to, or occurring during, trains is given in Table III. The maximum change in heart rate relative to baseline during the 60s following train start, averaged across all participants, is given in Figure 3. Relative to the control, no significant effects on heart rate were observed following 0.1, 0.2 or 0.3 mm/s vibrations.

Table III. Number of event-related samples used in heart rate change calculations. Events involving wake periods have been excluded.

Amplitude (mm/s)	*Total events (n)*	*Samples (n)*	*Excluded (n)*
Control	5*36=180	150	30
0.1	12*10=120	88	32
0.2	12*10=120	92	28
0.3	12*10=120	90	30

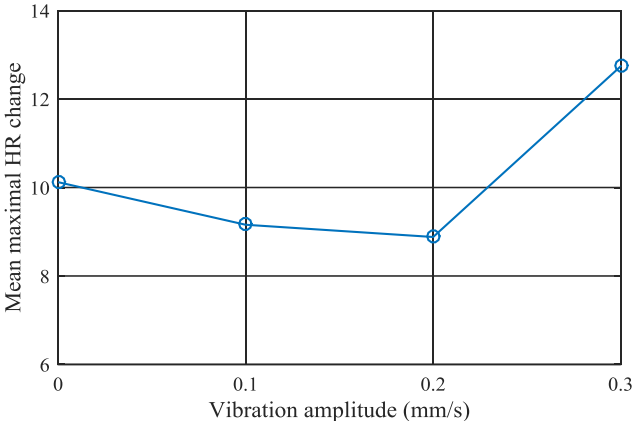

Figure 3. Mean of maximal heart rate change relative to 10s baseline for all participants.

4. Discussion

The results suggest that the physiological reaction threshold during sleep for vibration lies somewhere around 0.3 mm/s. The actual threshold might be slightly lower, but with the current data it cannot be said exactly where this would be.

The probabilities of EEG arousals and sleep stage changes were all statistically significantly higher during train vibration of 0.3 mm/s than would be expected of spontaneous reactions. No effect of the trains was observed at either 0.1 or 0.2 mm/s compared to the control for any of the physiological parameters examined. Therefore vibration at 0.1 or 0.2 mm/s did not have an effect on acute reactions, but as expected neither did the accompanying low-level noise contribute to response. The effects seen at 0.3 mm/s can therefore be attributed to the vibration exposure, and are not a result of any concurrent train noise.

Thirty six trains during the night, as used in the present work, is in line with realistic railway freight scheduling, and parts of Europe may even have up to 150 trains during the night [17]. Although EEG reaction probability to nocturnal noise has been shown to decrease as a function of the number of events [18], this has not been found for vibration [7]. The presented probabilities, and subsequent reaction thresholds, are therefore felt to be representative of what might occur in the field nearby to freight lines.

Current Swedish guidelines for vibration state that action must be taken by the local authorities if vibration from newly built, majorly refurbished, and existing railway lines in the bedroom during

night-time exceeds 0.7, 1.0 or 2.5 mm/s respectively [19]. These limits are rather much higher than a 0.3 mm/s reaction threshold. Taken together with the fact that the degree of heart rate change increases with even higher vibration amplitudes than in the present work [6], and EEG arousal and SSC probabilities increase further with stronger vibration [7], it is very possible that residents living close to freight lines are exposed to vibrations which may illicit physiological response. Indeed, recent work by the group suggests that vibration of 0.4 mm/s, only slightly above the threshold, may correspond to $L_{AF,max,indoor}$ = 50 dB [20]. In the short term, the impact of these acute reactions may be small, but sustained exposure may lead to chronic conditions in the long term, including cardiovascular diseases [21] and metabolic illness [22].

The study is limited by the low number of participants, meaning that the results should not be overstated. All participants were noise insensitive, and there are indications elsewhere that sleep differs between sensitivity groups [23]. Further work is necessary not only to improve the statistical power, but also to involve a greater diversity of participants. There is no guarantee that reaction thresholds for young, healthy people with good normal sleep would be the same as the thresholds for older persons for instance, whose sleep structures are already different. Nevertheless, the current findings provide a first indication of where the threshold for the biological effects of vibration may occur. Ongoing studies by the research group aim to further advance knowledge in this area.

Acknowledgement

This project was funded by Trafikverket, the Swedish Transport Authority. We thank Alf Ekblad and Katrin Nilsson for their input. We are grateful to Tania Kalafata, Hanna Herzberg, Karin Bergman and Margareta Nilsson for their assistance in conducting the study.

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