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Adverse effects of train noise and vibration on human heart rate during sleep –an experimental study

Authors: Dr. Ilona Croy[1]; Michael Smith[1],MSc; Prof. Kerstin Persson Waye[1]

[1]University of Gothenburg, Department of Occupational and Environmental Medicine, University of Gothenburg, Medicinaregatan 16, 405 30 Göteborg, Sweden

Corresponding author:
Dr. Ilona Croy
Occupational and Environmental Medicine
Medicinaregatan 16
University of Gothenburg
Box 414, 405 30 Göteborg, Sweden
+46 (0)31 786 6814
Ilona.croy@amm.gu.se

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Abstract

Objectives: Transportation of goods on railways is increasing and the majority of the increased numbers of freight trains run during the night. Transportation noise has adverse effects on sleep structure, affects heart rate during sleep and is linked to cardiovascular disease. Freight trains also generate vibration and little is known regarding the impact of vibration on human sleep. A randomized laboratory study was conducted to examine how a realistic nocturnal railway traffic scenario influences the heart rate (HR) during sleep.

Methods: Twenty four volunteers slept in the laboratory for six consecutive nights: one habituation night, one control and four experimental nights in which train noise and vibration was reproduced. In the experimental nights, 20 or 36 trains with low or high vibration characteristics were presented. Polysomnographical data and ECG were recorded.

Results: The train exposure lead to a significant change of HR within one minute of exposure onset ($p=0.002$), characterized by an initial and a delayed increase of HR. The high vibration condition provoked an average increase of at least 3bpm per train in 79% if the participants. Cardiac responses were in general higher in the high vibration compared to the low vibration condition ($p=0.006$). No significant effect of noise sensitivity and gender was revealed, although there was a tendency for men to exhibit stronger HR acceleration then women.

Conclusions: Freight trains provoke HR accelerations during sleep, and the vibration characteristics of the trains are of special importance. In long term, this may affect cardiovascular functioning of persons living close to railways.

Article summary

Article Focus:

The risk of cardiovascular diseases is higher increased in people living close to railways. This is because noise of bypassing trains affects sleep and induces heart rate accelerations in humans. Trains do not only emit noise, but also vibration and the influence of vibration is poorly understood.

Key message:

Our study shows, that freight train noise and vibration provokes heart rate accelerations in sleep. The HR response is characterized by two peaks, whereby the second one is even more pronounced and may dependent on the vibration amplitude.

Strengths and limitations:

The influence of nocturnal vibration on HR response has been studies for the first time under very well controlled laboratory conditions. However further studies with increased sample size should be carried out to analyze the influence of gender, ager and sensitivity more in detail.

Introduction

As the European market share of freight traffic is expected to increase from 8% in 2001 to 15% in 2020 [1], it is important to estimate the impact of this on the health of persons living close by to railway lines. Railway noise is related to disturbed sleep [2-6] and there are indications of increased cardiovascular disease in persons living close to railways [7]. There is some research about the impact of traffic noise on sleep and cardiovascular disease, however few have looked into rail traffic and the impact of rail traffic related vibration has been widely neglected.

The impact of other traffic sources like aircraft and road traffic on cardiovascular disease has been examined more closely. Babisch summarizes that traffic exposure enhances the risk for hypertension by between 1.5 to 3 times and enhances the percentage of persons using antihypertensive drugs [8]. Epidemiological studies found an enhanced risk for myocardial infarction in persons exposed to high traffic noise levels, however the effect sizes were rather small. Selander et al examined long term exposure to traffic noise exceeding 50dB $L_{Aeq,24h}$ and

found an increased probability of this occurrence [9]. A similar effect was found by Babisch et al [10]. They report that the exposure to traffic noise for more than 10 years increased the likelihood of myocardial infarction in men. However, this effect became only significant at levels of 70dB or above during daytime. In accordance Grazuleviciene et al report an increased probability of myocardial infarction in middle aged men in relation to traffic noise [11]. Soerensen et al [12] also found an increased incidence rate ratio in relation to traffic exposure. In this study too the effect was more pronounced for men. After adjusting their data for confounders the authors could show that traffic noise increases myocardial infarction risks independently of air pollution.

Disturbed sleep might be one factor leading to enhanced cardiovascular risk in persons exposed to traffic. It has been suggested that sleep disturbances contribute to cardiovascular disease through the pathway of enhanced inflammatory processes [13] or, related to this, through the pathway of enhanced stress reaction affecting the cardiovascular system [14]. Underpinning this, Portela et al found that patients with sleep-disordered breathing, like sleep apnea, exhibit a higher risk for cardiovascular diseases [15]. In a field study, Carter et al examined the influence of nocturnal traffic exposure on 7 older men with cardiac arrhythmia, who lived close to a busy road [16]. They found an increased likelihood of single ventricular premature contradictions (a form of cardiac arrhythmia) after noise peaks.

Traffic noise influences sleeping structure [17] and leads to increased awakenings [17 18] throughout the night. People living close to busy roads and railways or in close proximity to airports therefore often report poor sleep quality [3 4 6]. A Dutch study revealed that the number of persons reporting strong difficulties falling asleep due to traffic noise increased from 18% to 23% between 1998 and 2003 [19].

Two environmental exposures commonly arising from traffic are noise and vibration. As described, traffic noise disturbs sleep, but the effect of traffic vibration is not well understood. Studies investigating annoyance show that 1) traffic vibration causes annoyance, 2) annoyance increases with increasing vibration level and 3) vibration enhances the annoying effect of noise [20-22]. Furthermore, higher vibration amplitude is related to higher reported sleep disturbances in persons exposed to railway traffic [6]. A study on human fetuses suggests that short experimental external vibration exposure alters sleep stage [23]. Some indication that vibration exposure also influences cardiovascular function comes from working-life studies. Björ et al showed that persons who were exposed to high vibration during working and leisure time had a 1.6 times enhanced risk of acute myocardial infarction [24]. The coherence between vibration exposure and cardiovascular disease was supported by a study on mine workers exposed to whole body vibration [25].

The effect of nocturnal train vibration exposure on HR has - to our best knowledge - not been examined. However, some authors looked into the influence of train noise. Two laboratory studies on the effect of nocturnal traffic noise on cardiac arousals revealed that HR increases significantly due to train noise [26 27]. In the study conducted by Griefahn et al [26] 24 participants slept in the laboratory for a total of 12 nights and were exposed to aircraft, railway and road traffic noise. Resulting HR accelerations, indicating autonomic arousals, to each of those exposures were revealed. Rail vehicles lead to stronger reactions compared to road or aircraft noise. Accelerations were especially pronounced if the participants awoke. However, HR reaction was also very clearly detectable without awakenings. A similar effect has been found by Tassi et al. [27]. For this study 38 participants slept for three nights in the laboratory and were exposed to sounds of different types of trains. The same HR acceleration due to traffic noise was found and mean HR amplitude increased with increasing noise intensity of the trains.

As Di Nisi et al found out, traffic exposure induced HR changes seem not to habituate during sleep [28], therefore they might "bear a pathogenic potential for the genesis of cardiovascular disease"[26]. It is the aim of the present study to detect if nocturnal noise and vibration exposure from freight trains provoke HR accelerations.

Methods

Participants and procedure

Twenty four volunteers (11 men, 13 women, 19-28 years, mean age 22.9+/- 2.8years) slept in our sleep laboratory. They were instructed to begin attempting to fall asleep at 23:00 each night, were awoken by an alarm call at 07:00 each morning, and were prohibited from sleeping outside of this period. Before attending they were asked about their noise sensitivity on a 5-point Likert scale. Fourteen participants (8 men, 6 women) rated themselves as being noise insensitive (points 1-2) and 10 rated themselves being highly sensitive (points 3-5). For each participant, the study consisted of one habituation night, one control night and after this, four experimental nights in which simulated trains passed. The arrangement of the experimental nights was randomized across participants. Gender and noise sensitivity were approximately equally spread around the randomization table. In two of the four experimental nights, the participants were exposed to 20 trains per night with all trains having high vibration one night and low vibration in the other (High20 and Low20 respectively). In the remaining two nights they were exposed to 36 trains per night again with either high or low vibrations (High36 and Low36). The acoustic signal for the trains was not varied with vibration amplitude. There were more train passages between the periods of 23:00-01:00 and 05:00-07:00 to reflect typical real world scenarios.

The sleep laboratory consists of three individual rooms furnished to simulate a typical bedroom with a bed, chairs, desk and small chest of drawers. Eighty eight ceiling loudspeakers reproduced

the low frequency content of noise below 125 Hz, and higher frequencies were generated by two loudspeaker cabinets in the room corners. Because of the unrealistically low background noise levels in the rooms (<14 dBA), for the duration of the trial ventilation noise was introduced at a level of 25 dBA measured at the pillow position on the bed. In addition to the bedrooms the participants have private access to a shared a communal space outfitted with a kitchen, dining area and living area with sofa and television.

Vibration and noise exposure were similar to another study conducted in our laboratory [29] and are only summarized here. Five different train passages were synthesized based upon analysis of accelerometer and sound level measurements performed in the field. Noise signals were high pass filtered to correspond to a fully closed window. The vibration exposure was an amplitude modulated 10 Hz signal applied along the lengthwise horizontal axis of the bed by electrodynamic shakers with a frequency response of 5-40 Hz mounted to the underside of the bed frame. The vibration began when the train noise signal exceeded 35 dB L_{AEq}, ensuring masking of any audible mechanical operation noise. Further acoustic and vibration data for each individual train is presented in Table 1.

Ethics Statement

The study followed the Declaration of Helsinki on Biomedical Research Involving Human Subjects and was approved by the Ethics Committee from the University of Göteborg (920-11). All participants provided written informed consent.

Cardiac response and polysomnography

During all experimental nights, physiological data was recorded using ambulatory polysomnogram devices (SOMNOscreen plus PSG+, SOMNOmedics GmbH, Germany). Cardiac activity was recorded via a modified lead II torso placement electrocardiogram (ECG) at a sampling frequency of 256 Hz as per the American Academy of Sleep Medicine [30]. To

identify sleep stage, electroencephalogram (EEG), electrooculogram (EOG) and submental electromyogram (EMG) were also recorded using standardized surface electrode placements and sampling and filter frequencies. To ensure good signal quality, following electrode attachment it was checked that the electrical impedance of each contact was ≤ 5kΩ. Thirty second epoch sleep staging was performed manually by trained sleep technicians. Additionally, participants wore two effort belts to record breathing rates and a finger pulse oximeter to record blood oxygen saturation, plethysmogram and pulse information, although this data shall not be reported here. Due to a technical fault, ECG was not recorded for one participant (female, noise sensitive) in the High20 night and so could not be included in analysis.

Data reduction and event related analysis

In order to examine event related changes of HR, data from the four exposure nights was analyzed. The continuously recorded ECG (sampling frequency of 256Hz) was converted into HR (in bpm) in 1s intervals through the whole night. EEG and ECG recordings were synchronized with the train exposure events. This allows a direct temporal association between the occurrence of a train and the participant's HR reaction and sleep stages. Analysis revealed that 9.9% of the train onsets occurred during wake stage, 8.1% during stage N1, 42% during stage N2, 21.4% during stage N3 and 17.8% during REM.

The procedure of HR analysis is visualized in figure 1. HRs below 35 and above 130bpm were considered invalid [26 31] and time intervals where this happened were excluded from the analysis. This was the case in 4.1% of the events. For analysis of event related HR change (bpm), only train events where participants were asleep were considered. This approach was chosen because Griefahn and colleagues showed that HR response to traffic noise differs depending on whether the participants do or do not awake [26]. The relatively few trains per night in our study do not allow additional examination of HR response in the case of awakenings. According to the literature, an event related awakening is defined as a sleep stage change to Wake from any other

stage in at least one of the two epochs (30s) following the train onset [32]. The first epoch was thereby defined as the first where at least 15s were under influence of the event. Additionally, events for which the participants were awake in the epoch preceding train onset were excluded from the analysis. The whole procedure left 72.4% of the train events in the low vibration condition and 66.1% of the train events in the high vibration condition for analysis. In accordance with the literature, the screening interval for cardiac activations was set to 60s after train onset [26 31]. In order to analyze change of HR, the average cardiac response of the 10s preceding the train event was subtracted from the cardiac response in each of the sixty 1s time intervals following the train onset.

In order to examine if there was a train related change of HR at all, 'fake trains' were calculated. Twenty occurrences of fake trains were introduced in the Low20 and High20 nights and 36 onsets in the Low36 and High36 nights. These fake trains were distributed at time intervals approximately equally spaced between the actual exposure events. Analyzed HR data for these consisted of 70s, where 10s served as baseline for the following 60s. As with the real trains, data where polysomnographical analysis indicated that participants were awake before onset or awoke during the two epochs following onset was excluded. The 60 seconds after train onset were subsequently averaged over nights with low or high vibration respectively.

Griefahn and colleagues report a maximum increase of HR about 13.2s after event onset [26]. We took this time interval +/- 3s as a base for the searching area for the maximum increase between 10 and 16s after train onset.

Statistical analysis

Data was analyzed using SPSS 20 (SPSS Inc., Ill, USA). In order to look for an overall effect of trains on the HR, the integral of the HR response was taken to determine the area under the curve AuC for the 60s after train onset. An Analysis of Variance (ANOVA) for repeated measurements was

calculated comparing the AuC in three factors: event (fake vs. real-train), vibration level (low vs. high) and number of trains (20 vs. 36). The effects of noise sensitivity and gender were also analyzed using ANOVA for repeated measurements with the in-between subject factor noise sensitivity/gender and the within subject factor vibration level (low vs. high). Post-hoc-comparisons are reported Bonferroni-corrected. The level of significance is set at α=0.05.

Results

Influence of train noise and vibration on HR

The AuC analysis for the change of HR 60s after event onset revealed a significant main effect of train, indicating that ***train events lead to an enhanced change of HR compared with the fake events*** (F22,1=12.0, p=0.002). There was a ***significant main effect of vibration level.*** A high change of HR could be observed in the high compared to the low vibration level (F22,1=7.6; p=0.01). The number of trains had no significant influence. The results are displayed in table 2.

Averaged over all trains within one night, an ***increase of HR of at least 3bpm*** was observed in 54% of the participants in Low36, 52% in the Low20 condition, 74% in the High20 night and 79% of participants in the High36 night. For the fake train events an average increase of HR of at least 3 bpm was observed in only 17 to 38% of the participants, depending on the exposure night.

As no significant influence of the number of trains was revealed, the four exposure conditions were combined into two conditions: a high vibration and a low vibration condition. This approach is advantageous for the signal-noise ratio, because in this way the number of analyzable trains could be increased to 56. For calculating the low vibration condition, train events from the Low20 and Low36 nights that matched the inclusion criteria described in the methods section were averaged to obtain one low vibration response. The same was done for the high vibration

condition. This procedure was repeated for the fake events, so that resulting responses for the low and high vibration exposures for both the actual and phantom events could be compared.

Characteristics of the HR curve

The HR curve shows a biphasic characteristic (see figure 2A). After approximately 9s a short initial response characterized by increase of HR takes place, lasting for around 6s. This ***initial response*** is significantly above the baseline between 10-13s for the low vibration condition, and between 10-15s for the high vibration condition (t-test, $p<0.05$). This response is in accordance with the proposed search area of the maximum increase.

An additional ***delayed response*** can also be observed. This response is characterized by a second increase of HR beginning around 17 seconds after train onset and lasting for about 20 seconds for the low vibration condition and about 30 seconds for the high vibration condition. The delayed response is significantly above baseline between 21-22s for the low vibration condition, and between 20-48s for the high vibration condition (t-test, $p<0.05$). This same response could be seen for each of the five individual train types (see figure 2B)

This response pattern could be observed in the individual participants, but the time delay after event onset showed a great variability. The individual maximum for each participant within the range of the initial response (10-15s) and the delayed response (17-50s) was calculated and is reported in table 2.

For the **initial response and for the delayed response**, ANOVA for repeated measurements revealed a significant main effect of event, indicating that the ***train-events lead to an enhanced change of HR compared with the fake-events*** (initial response:$F_{23,1}=13$, $p=0.001$; delayed response: $F_{23,1}=15.5$, $p=0.001$).

The vibration level had no significant influence on the initial increase of HR (F23,1=2.1, n.s.), but did on the delayed response. A higher delayed increase of HR was observed in the high compared to the low vibration level (F23,1=9.4, p=0.006).

Effect of order

The participant's HR responses of the four nights were compared with respect to the order of experimental nights using ANOVA for repeated measurements. There was no significant influence of order of experimental nights on the AuC response (F22,3 =1.7, n.s.), nor for the initial (F22,3 =2.0, n.s.) or delayed response (F22,3 =0.8).

Influence of sleep stage

The AuC, the initial maxima and the delayed maxima were compared in the sleep stages N3, N2 and REM. For wake stage and for stage N1 there are too few events to allow for comparison. The results are presented in table 2.

ANOVA for repeated measurements using the within subject factors sleep stage (stages N3, N2 and REM) and vibration level (low vs. high) was calculated.

The overall **AuC** revealed no significant influence of sleep stage (F23,2 =1.1, n.s.). The **initial response** revealed a significant influence of sleep stage (F23,2=5.1 p=0.009). Bonferroni-corrected post-hoc comparison reveals that the initial increase of HR was significantly higher in REM then in stage N2 sleep (p=0.002). The same significant pattern was seen in the **delayed response** (F23,2=4.8 p=0.013). Here again post-hoc comparison showed that HR increase was higher in REM sleep than in stage N2 (p=0.017).

Influence of Noise sensitivity and gender

Effect of sensitivity. The AuC, initial maximum and delayed maximum of HR change are compared between participants with low (N=14) and high (N=10) noise sensitivity. The results

are displayed in table 3. No significant main effects of noise sensitivity were found in the AuC ($F_{22,1}=0.007$, n.s.) nor in the HR initial ($F_{22,1}=2.8$, n.s.) and delayed response ($F_{22,1}=0.004$, n.s.). There were no significant interactions between noise sensitivity and vibration level.

Effect of gender. The results of comparison between men (N=11) and women (N=13) are shown in table 3. No significant difference of gender were found in the AuC ($F_{22,1}=2.5$, n.s.) nor in the HR initial ($F_{22,1}=0.5$, n.s.). There was a tendency for men to exhibit a stronger increase of HR in the delayed response ($F_{22,1}=4.1$, $p=0.055$).

Discussion

Our data shows that train noise and vibration exposure leads to cardiac arousals and the vibration might be of particular importance for this. In response to train noise and vibration HR acceleration with two maxima was observed. One sharp and short initial HR increase is revealed about 9s after train onset and a longer delayed increase is observed starting about 17s after train onset. In a similar study dealing with traffic noise only, Griefahn et al [26] found an increase of HR matching our initial response but no delayed increase. A similar initial increase of HR has been found for pure tones during sleep [33]. We propose that the second plateau we found is due to the added vibration exposure, which seems to enhance the adverse effect of traffic noise on HR. Supporting this interpretation we see a significant difference between the low and the high vibration level for the delayed, but not for the initial increase. The combined exposure might be a more pronounced alarming signal than noise alone and the HR alterations endure even after cessation of the actual exposure. In accordance with previous literature on sleep disturbance due to noise exposure [26 27], the highest alterations of HR were found in REM sleep. This was observed for the initial and for the delayed response.

Cardiac arousals are thought to be induced by brain stem activation [34]. This subcortical activation may present a form of primary arousal in sleep and - depending on the strength of activation and on sleep stage - lead to cortical activation and arousal. Increased HR indicates subcortical arousal and enhances the probability of a cortical arousal with implications for sleep structure [34]. However, even HR acceleration without concomitant cortical arousal has been shown to have adverse effects on sleep structure. Guilleminault et al showed that forced subcortical arousals go along with disturbed sleep and the persons are not necessarily aware of their disturbed sleep the following morning [35].

In the short term, arousals have positive effects by ensuring a higher level of behavioral responsiveness in the presence of external stimuli, while the sleep is protected as far as possible [36]. However, in the long term increased autonomic reaction might be harmful. As Kohler and Stradling point out in a review paper, recurrent arousals are one of the mechanisms leading to cardiovascular disease in obstructive sleep apnea [37]. In that way the autonomic arousals provoked by passing trains may - if continuing over years - contribute to the slightly enhanced myocardial infarction risk that has been found in people living in areas with high traffic exposure [9-12]. The study of Tassi et al suggests that people living in areas with high railway noise for more than ten years can partly adjust their sleep to this adverse exposure. However, even after this long time of exposure, significant alterations of HR due to nocturnal railway have been found in those persons [5]. The effects of noise exposure alone found in epidemiological studies are rather small. Our data suggest that the effect of chronic traffic exposure on cardiovascular disease might be higher if vibration is also taken into account. As described in the methods of this paper, we excluded those events from the analysis where persons woke up. This approach allows a focused estimation of HR reaction on traffic noise during sleep. However, as HR reactions are usually much higher if accompanied by awakenings [26], our results very likely underestimate the effects.

Our results are less clear regarding the impact of individual differences. An epidemiological study has shown that persons who state being sensitive to noise have reported more sleeping problems due to traffic than non-sensitive persons [38]. However, we found no significant effect of self-reported noise sensitivity on HR response towards nocturnal train events. During the day it has been shown that self-declared high noise sensitive persons have higher cardiovascular responses towards different ecologically relevant sound, including traffic noise [28]. The sample size of sensitive and non-sensitive participants in our study is relatively small and therefore non-significant results in particular have to be interpreted with caution. The sample size also limits any interpretation of gender effects. In coherence with the study of Griefahn, we found no significant gender difference in the initial HR amplitude [26]. We found a tendency for enhanced delayed HR response in men with a rather strong effect size. This corresponds to results from several field studies, where enhanced traffic exposure related cardiovascular diseases were found in men[10-12].

In conclusion, the combined exposure of freight train noise and vibration influenced HR during sleep, whereby HR amplitudes increase with increasing vibration level. This provoked acceleration may affect cardiovascular functioning in long term [5 26] and could be a mechanism explaining the previous findings of cardiovascular disease in persons exposed to traffic in general [8-12] and rail traffic in particular [7]. To study the specific influence of vibration in contrast to noise, further studies are carried out.

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Tables

TABLE 1: *Vibration and noise parameters applied to individual trains.*

Train	Nr of passages per night		Noise exposure				Vibration exposure (same for all trains)	
	20 trains/ night	36 trains/ night	L_{AEq} (dB)	L_{AFmax} (dB)	t > 35dB (s)	$T_{10\%-90\%}$ (s)	Unweighted acceleration (m/s^2 rms)	W_d Weighted peak acceleration (m/s^2)
1	4	8	44.0	48.4	11.5	8.9		
2	5	8	42.7	47.2	46.2	9.8	High = 0.072	High = 0.0204
3	4	8	44.5	49.8	23.7	8.4	Low = 0.036	Low = 0.0102
4	5	8	45.6	49.8	29.2	7.9		
5	2	4	42.4	47.2	56.9	9.2		

The vibration acceleration is reported according to the ISO 2631-1 standard.

TABLE 2: *Analysis of event related HR response for each of the four exposure nights and combined for low and high vibration exposure.*

		Low vibration		High vibration	
		20 trains/night N=24	**36 trains/night N=23**	**20 trains/night N=23**	**36 trains/night N=24**
		Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
AuC	train	41.7 (114.5)	77.2 (103.0)	19.4 (77.6)	52.3 (86.8)
	fake	-4.6 (83.7)	0.2 (54.4)	-7.4 (51.5)	2.2 (60.9)
		number of participants (percentage of sample population)			
Event-related HR change of at least 3bpm	train	15 (62.5%)	17 (73.9%)	13 (54.2%)	19 (79.2%)
	fake	9 (37.5%)	8 (34.8%)	4 (16.7%)	4 (16.7%)

		low vibration	**high vibration**
		Mean (SD)	Mean (SD)
AuC	train	21.2 (81.8)	62.3 (81.4)
Initial response	train	1.6(1.8)	2.3 (2.0)
	fake	0.7 (0.9)	0.7 (1.0)
Delayed response	train	2.6 (2.4)	3.7 (2.8)
	fake	1.1 (1.0)	1.3(0.9)
		Sleep stage related (train events only)	
AuC	stage N3	25.1 (166.9)	79.7 (114.8)
	stage N2	30.2 (95.8)	53.2 (90.3)
	REM	42.6 (162.4)	113.0 (203.1)
Initial response	stage N3	2.3 (2.7)	3.7 (4.0)
	stage N2	2.1 (2.2)	2.2 (1.9)
	REM	2.8 (2.8)	4.7 (3.3)
Delayed response	stage N3	4.5 (4.9)	5.9 (4.1)
	stage N2	3.6 (3.6)	4.5 (3.4)
	REM	5.6 (4.1)	7.5 (5.8)

Area under the curve (AuC), initial and delayed maximal increase of heart rate (HR) and number of participants with an event related change of HR of at least 3bpm are presented for each of the four experimental nights. AuC, initial and delayed maximal increase of HR are presented for the combined low and high vibration condition. Cave: the initial and delayed response is calculated as maximal increase within the first 10 to15 or 20 to 48 seconds after train onset. This difference in search area explains partly the differences between the initial and delayed increase of HR.

TABLE 3: *Train related HR characteristics in relation to gender and noise sensitivity of the participants.*

			low vibration	high vibration
			Mean (SD)	Mean (SD)
gender		AuC	48.1 (73.0)	81.2 (96.8)
		initial response	1.7 (1.4)	2.0 (1.9)
	Men N=11	delayed response	3.5 (2.8)	4.8 (3.7)
		AuC	-1.6 (84.6)	46.2 (65.4)
	Women	initial response	1.5 (2.1)	2.6 (2.2)
	N=13	delayed response	1.8 (1.7)	2.8 (1.5)
noise sensitivity		AuC	24.0 (62.0)	63.8 (93.3)
	non sensitive	initial response	1.1 (1.3)	2.0 (2.0)
	N=14	delayed response	2.3 (2.0)	4.0 (3.4)
		AuC	17.2 (107.3)	60.1 (65.9)
	Sensitive	initial response	2.4 (2.2)	2.8 (2.1)
	N=10	delayed response	3.0 (2.9)	3.4 (2.0)

Figure Legends

FIGURE 1. Visualization of the analytical procedure. For each of the four experimental nights, HR data is taken out for each of the train-events (black lines of the events per night) for each participant. Data is checked for artifacts and wake stage and then sampled into one average HR response for each participant with corresponding initial maximum, delayed maximum and area under the curve parameters. The grand average is built over all of the participants. The very same procedure is applied to the fake events (grey lines of the events per night).

FIGURE 2. A) Averaged HR response following the train events and fake events, respectively, in the low and high vibration exposure. In figure 1A, a clear HR increase after train events can be seen, while HR stays at baseline for fake events. The HR response can be divided into two components. An initial response occurring around 10 to 15 seconds after train onset and a delayed response occurring about 17 to 48 sec after train onset. The delayed response is significantly enhanced in the high vs. low vibration exposure. In figure 1B the averaged HR reaction in the high vibration condition is visualized for each of the five different train types. Although the different number of trains does not allow direct comparison, it can be seen, that in principal the same characteristics of the HR reaction is apparent for each train.

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Contributorship

Ilona Croy contributed to conception and design, analysis, interpretation and wrote the article

MIchael Smith contributed to conception and design, interpretation and approved the article

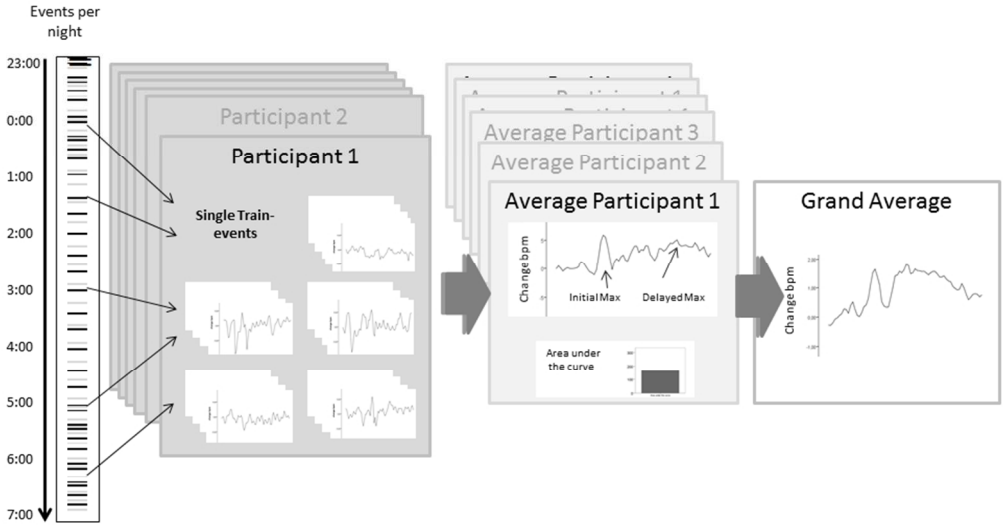
Kerstin Persson Waye contributed to conception and design, interpretation and approved the article

Data Sharing

Extra data is available by emailing Ilona Croy.

Competing Interests

None



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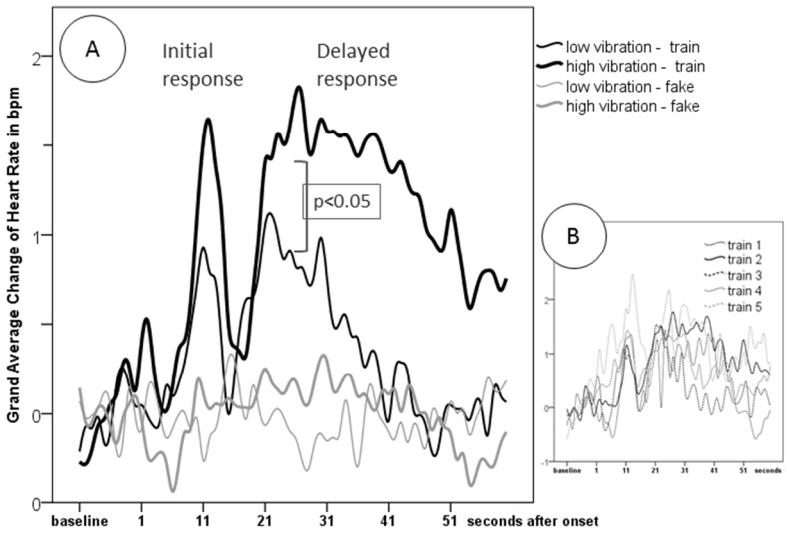

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Adverse effects of train noise and vibration on human heart rate during sleep –an experimental study

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Adverse effects of train noise and vibration on human heart rate during sleep –an experimental study

Authors: Dr. Ilona Croy[1]; Michael Smith[1],MSc; Prof. Kerstin Persson Waye[1]

[1]University of Gothenburg, Department of Occupational and Environmental Medicine, University of Gothenburg, Medicinaregatan 16, 405 30 Göteborg, Sweden

Corresponding author:
Dr. Ilona Croy
Occupational and Environmental Medicine
Medicinaregatan 16
University of Gothenburg
Box 414, 405 30 Göteborg, Sweden
+46 (0)31 786 6814
Ilona.croy@amm.gu.se

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Data sharing statement: Extra data is available by emailing Ilona Croy.

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Abstract

Objectives: Transportation of goods on railways is increasing and the majority of the increased numbers of freight trains run during the night. Transportation noise has adverse effects on sleep structure, affects heart rate during sleep and may be linked to cardiovascular disease. Freight trains also generate vibration and little is known regarding the impact of vibration on human sleep. A laboratory study was conducted to examine how a realistic nocturnal railway traffic scenario influences the heart rate (HR) during sleep.

Methods: Twenty four volunteers slept in the laboratory for six consecutive nights: one habituation night, one control and four experimental nights in which train noise and vibration was reproduced. In the experimental nights, 20 or 36 trains with low or high vibration characteristics were presented. Polysomnographical data and ECG were recorded.

Results: The train exposure lead to a significant change of HR within one minute of exposure onset ($p=0.002$), characterized by an initial and a delayed increase of HR. The high vibration condition provoked an average increase of at least 3bpm per train in 79% if the participants. Cardiac responses were in general higher in the high vibration compared to the low vibration condition ($p=0.006$). No significant effect of noise sensitivity and gender was revealed, although there was a tendency for men to exhibit stronger HR acceleration then women.

Conclusions: Freight trains provoke HR accelerations during sleep, and the vibration characteristics of the trains are of special importance. In long term, this may affect cardiovascular functioning of persons living close to railways.

Article summary

Article Focus:

Noise of passing trains affects sleep and induces heart rate accelerations in humans. This may be linked to cardiovascular disease. Trains do not only emit noise, but also vibration and the influence of vibration is poorly understood.

Key message:

Our study shows, that freight train noise and vibration provokes heart rate accelerations in sleep. The HR response is characterized by two peaks, whereby the second one is even more pronounced and may dependent on the vibration amplitude.

Strengths and limitations:

The influence of nocturnal vibration on HR response has been studied for the first time under very well controlled laboratory conditions. However further studies with increased sample size should be carried out to analyze the influence of gender, age and sensitivity in more detail.

Introduction

As the European market share of freight traffic is expected to increase from 8% in 2001 to 15% in 2020 [1], it is important to estimate the impact this may have on the health of persons living close by to railway lines. Railway noise is related to disturbed sleep [2-6] which in turn increases the risk of cardiovascular disease [7], supported by indications of increased cardiovascular disease in persons living close to railways [8]. Understanding how railway noise and vibration influence the cardiovascular system in sleep is therefore necessary.

The sympathetic tone is reduced in sleep which is reflected by a reduction of heart rate (HR) [9]. External noise events have the potential to disturb sleep and lead to event related increase of heart rate. This has been shown for pure tones [10] and more recently for traffic noise [11 12]. Noise induced HR changes do not seem to habituate during sleep [13], therefore they might “bear a pathogenic potential for the genesis of cardiovascular disease” [11]. Evidence that long term traffic exposure can increase the risk for cardiovascular disease comes from field studies.

Babisch summarizes that traffic exposure enhances the risk for hypertension by a factor of between 1.5 to 3 [14]. An increased risk for myocardial infarction in persons living close to railways has been found in several studies [15-18], although the effect sizes are rather small and the effects seem to be more pronounced in men [15 16 18].

Traffic noise influences sleeping structure [19] and leads to increased awakenings [19 20] throughout the night. People living close to busy roads and railways or in close proximity to airports therefore often report poor sleep quality [3 4 6]. A Dutch study revealed that the number of persons reporting strong difficulties falling asleep due to traffic noise increased from 18% to 23% between 1998 and 2003 [21]. Disturbed sleep might be one factor leading to enhanced cardiovascular risk in persons exposed to traffic. It has been suggested that sleep disturbances contribute to cardiovascular disease through the pathway of enhanced inflammatory processes [22] or, related to this, through the pathway of enhanced stress reaction affecting the cardiovascular system [23]. In a field study, Carter et al examined the influence of nocturnal traffic exposure on 7 older men with cardiac arrhythmia, who lived close to a busy road [24]. They found an increased likelihood of single ventricular premature contractions (a form of cardiac arrhythmia) after noise peaks.

Two environmental exposures commonly arising from traffic are noise and vibration. As described, traffic noise disturbs sleep and leads to event related HR changes, but the effect of traffic vibration is not well understood. Studies investigating annoyance show that 1) traffic vibration causes annoyance, 2) annoyance increases with increasing vibration level and 3) vibration enhances the annoying effect of noise [25-27]. Furthermore, higher vibration amplitude is related to higher reported sleep disturbances in persons exposed to railway traffic [6]. A study on human fetuses suggests that short experimental external vibration exposure alters sleep stage [28]. Indications that vibration exposure is disadvantageous for cardiovascular function have been found in people exposed to high levels of vibration at work [29 30].

The effect of combined traffic vibration and noise on the cardiovascular system in sleep has - to our best knowledge - not been examined. It is the aim of the present study to detect if nocturnal noise and vibration exposure from freight trains provoke HR accelerations.

Methods

Participants and procedure

Twenty four volunteers (11 men, 13 women, 19-28 years, mean age 22.9+/- 2.8years) slept in our sleep laboratory. Twenty three were students and one was in full-time employment, recruited by public advertisements placed around the campus of the University of Gothenburg. Eligible persons were required to be aged between 18 and 30 years, in healthy condition, maintain normal sleeping patterns and not use tobacco products. To decrease the probability of subclinical breathing difficulties or apnea, participants were required to have a BMI within the normal range of 18.5 - 24.99 [31]. Following initial screening, hearing was tested before volunteers were accepted. Manual audiometric testing was conducted using an Entomed SA201^{II} according to ISO 8253-1. Left and right ears were tested separately at frequencies of 250, 500, 1000, 2000, 3000, 4000, 6000 and 8000 Hz with hearing considered to be normal if measured thresholds were not more than a screening level of 20 dB HL.

The participants were instructed to begin attempting to fall asleep at 23:00 each night, were awoken by an alarm call at 07:00 each morning, and were prohibited from sleeping outside of this period. Before attending they were asked about their noise sensitivity on a 5-point Likert scale. Fourteen participants (8 men, 6 women) rated themselves as being noise insensitive (points 1-2) and 10 rated themselves being noise sensitive (points 3-5). For each participant, the study consisted of one habituation night and one control night proceeding four experimental nights in which simulated trains passed. The arrangement of the experimental nights was randomized

across participants. Gender and noise sensitivity were approximately equally spread around the randomization table. In two of the four experimental nights, the participants were exposed to 20 trains per night with all trains having high vibration one night and low vibration in the other (High20 and Low20 respectively). In the remaining two nights they were exposed to 36 trains per night again with either high or low vibrations (High36 and Low36). The acoustic signal for the trains was not varied with vibration amplitude. There were more train passages between the periods of 23:00-01:00 and 05:00-07:00 to reflect typical real world scenarios.

The sleep laboratory consists of three individual rooms isolated from external noise and vibration, furnished to simulate a typical bedroom with a bed, chairs, desk and small chest of drawers. Eighty eight ceiling loudspeakers reproduced the low frequency content of noise below 125 Hz, and higher frequencies were generated by two loudspeaker cabinets in the room corners. Because of the unrealistically low background noise levels in the rooms (<14 dBA), for the duration of the trial artificial ventilation noise was introduced at a level of 25 dBA measured at the pillow position on the bed. In addition to the bedrooms the participants had private access to a shared a communal space outfitted with a kitchen, dining area and living area with sofa and television. They were free to come and go as they desired during the daytime, being required to arrive by 20:00 each evening to ensure rest prior to bedtime and to allow sufficient time for electrode attachment.

Vibration and noise exposure were similar to another study conducted in our laboratory [32] and are only summarized here. Five different train passages were synthesized based upon analysis of accelerometer and sound level measurements performed in the field. Noise signals were low pass filtered to correspond to a fully closed window. The vibration exposure was an amplitude modulated 10 Hz signal applied along the lengthwise horizontal axis of the bed by electrodynamic shakers with a frequency response of 5-40 Hz mounted to the underside of the bed frame. The vibration began when the train noise signal exceeded an A-weighted equivalent

level (L_{Aeq}) of 35 dB, ensuring masking of any audible mechanical operation noise. Further acoustic and vibration data for each individual train is presented in Table 1.

Ethics Statement

The study followed the Declaration of Helsinki on Biomedical Research Involving Human Subjects and was approved by the Ethics Committee from the University of Göteborg (920-11). All participants provided written informed consent.

Cardiac response and polysomnography

During all experimental nights, physiological data was recorded using ambulatory polysomnogram devices (SOMNOscreen plus PSG+, SOMNOmedics GmbH, Germany). The time resolution of the onset was 4.7+/- 3.4 seconds.

Cardiac activity was recorded via a modified lead II torso placement electrocardiogram (ECG) at a sampling frequency of 256 Hz as per the American Academy of Sleep Medicine [33]. To identify sleep stage, electroencephalogram (EEG), electrooculogram (EOG) and submental electromyogram (EMG) were also recorded using standardized surface electrode placements and sampling and filter frequencies. To ensure good signal quality, following electrode attachment it was checked that the electrical impedance of each contact was ≤ 5kΩ. Thirty second epoch sleep staging was performed manually by a trained sleep technician. Additionally, participants wore two effort belts to record breathing rates and a finger pulse oximeter to record blood oxygen saturation, plethysmogram and pulse information, although this data will not be reported here. Due to a technical fault, ECG was not obtained for one participant (female, noise sensitive) in the High20 night and so could not be included in analysis.

Event related analysis and control condition

In order to examine event related changes of HR, data from the four exposure nights was analyzed. The continuously recorded ECG (sampling frequency of 256Hz) was converted into

HR (in bpm) in 1s intervals through the whole night. EEG and ECG recordings were synchronized with the train exposure events, thus allowing a direct temporal association between the occurrence of a train and the participant's HR reaction and sleep stages. Analysis revealed that 9.9% of the train onsets occurred during wake stage, 8.1% during stage N1, 42% during stage N2, 21.4% during stage N3 and 17.8% during REM.

The procedure of HR analysis is visualized in Figure 1. HRs below 35 and above 130bpm were excluded from the analysis [11 34]. This was the case for 4.1% of the events. For analysis of event related HR change (bpm), only train events where participants were asleep were considered. This approach was chosen because Griefahn and colleagues showed that HR response to traffic noise differs depending on whether the participants do or do not awake [11]. The relatively few trains per night in our study do not allow additional examination of HR response in the case of awakenings. An event related awakening has been defined as a sleep stage change to Wake from any other stage in at least one of the two epochs (30s) following the train onset [35]. The first of these was determined by that having at least 15s under influence of the event. Additionally, events for which the participants were awake in the epoch preceding train onset were excluded from the analysis. The whole procedure left 72.4% of the train events in the low vibration condition and 66.1% of the train events in the high vibration condition for analysis. In accordance with the literature, the screening interval for cardiac activations was set to 60s after train onset [11 34]. In order to analyze change of HR, the average cardiac response from the 10s preceding the train event was used as a baseline value for each given event and subsequently subtracted from the cardiac response in each of the sixty 1s time intervals following the train onset.

In order to examine if there was a train related change of HR at all, ***'fake trains'*** were calculated. Fake trains were defined as time intervals of 60s not accompanied by a real train event, distributed at time intervals approximately equally spaced between the actual exposure events.

Twenty of these were introduced in the Low20 and High20 nights and 36 in the Low36 and High36 nights. . Analyzed HR data for these consisted of 70s, where 10s served as baseline for the following 60s. As with the real exposures, data where polysomnographical analysis indicated that participants were awake before onset or awoke during the two epochs following onset was excluded. All event-related 60speriods were subsequently averaged over nights with low or high vibration respectively. The whole procedure was the same for each of the four nights to avoid any potential investigator bias.

Griefahn and colleagues report a maximum increase of HR about 13.2s after event onset [11]. We took this time interval +/- 3s as a base for the searching area for the maximum increase between 10 and 16s after train onset.

Statistical analysis

Data was analyzed using SPSS 20 (SPSS Inc., Ill, USA). In order to identify any overall effect of trains on the HR, the integral of the HR response was taken to determine the area under the curve (AuC) for the 60s after train onset. An Analysis of Variance (ANOVA) for repeated measurements was calculated comparing the AuC in three factors: event (fake vs. real-train), vibration level (low vs. high) and number of trains (20 vs. 36). The effects of noise sensitivity and gender were also analyzed using ANOVA for repeated measurements with the between subject factor noise sensitivity/gender and the within subject factor vibration level (low vs. high). Post-hoc-comparisons are reported Bonferroni-corrected. The level of significance is set at α=0.05.

Results

Influence of train noise and vibration on HR

The AuC analysis for the change of HR 60s after event onset revealed a significant main effect of train, indicating that ***train events lead to an enhanced change of HR compared with the fake events*** (F22,1=12.0, p=0.002). Furthermore there were significantly more awakenings in the train vs. fake events (F22,1 =40.3, p<0.001).

There was a ***significant main effect of vibration level.*** A higher change of HR and an increased number of awakenings could be observed in the high compared to the low vibration level (HR: F22,1=7.6; p=0.01; Awakenings: F22,1=6.5; p=0.014). The number of trains had no significant influence on the HR change. The results are displayed in table 2.

Averaged over all trains within one night, an ***increase of HR of at least 3bpm*** was observed in 54% of the participants in Low36, 52% in the Low20 condition, 74% in the High20 night and 79% of participants in the High36 night. For the fake train events an average increase of HR of at least 3 bpm was observed in only 17 to 38% of the participants, depending on the exposure night.

As no significant influence of the number of trains was revealed, the four exposure conditions were combined into two conditions: a high vibration and a low vibration condition. This approach is advantageous for the signal-noise ratio, because in this way the number of analyzable trains could be increased to 56. For calculating the low vibration condition, train events from the Low20 and Low36 nights that matched the inclusion criteria described in the methods section were averaged to obtain one low vibration response. The same method was performed for the high vibration condition. This procedure was repeated for the fake events, so that resulting responses for the low and high vibration exposures for both the actual and phantom events could be compared.

Characteristics of the HR curve

The HR curve shows a biphasic characteristic (see figure 2A). After approximately 9s a short initial response characterized by increase of HR takes place, lasting for around 6s. This ***initial response*** is significantly above the baseline between 10-13s for the low vibration condition, and between 10-15s for the high vibration condition (t-test, $p<0.05$). This response is in accordance with the proposed search area of the maximum increase.

An additional ***delayed response*** can also be observed. This response is characterized by a second increase of HR beginning around 17s following train onset and with a duration of around 20s for the low vibration condition and about 30s for the high vibration condition. The delayed response is significantly above baseline between 21-22s for the low vibration condition, and between 20-48s for the high vibration condition (t-test, $p<0.05$). This same response could be seen for each of the five individual train types (see figure 2B)

This response pattern could be observed in the individual participants, but the time delay after event onset showed a great variability. The individual maximum for each participant within the range of the initial response (10-15s) and the delayed response (17-50s) was calculated and is reported in table 2.

For the **initial response and for the delayed response**, ANOVA for repeated measurements revealed a significant main effect of event, indicating that the ***train-events led to an enhanced change of HR compared with the fake-events*** (initial response:$F_{23,1}=13$, $p=0.001$; delayed response: $F_{23,1}=15.5$, $p=0.001$).

The vibration level had no significant influence on the initial increase of HR ($F_{23,1}=2.1$, n.s.), but affected the delayed response. A higher delayed increase of HR was observed in the high compared to the low vibration level ($F_{23,1}=9.4$, $p=0.006$).

Effect of order

The participant's HR responses of the four nights were compared with respect to the order of experimental nights using ANOVA for repeated measurements. There was no significant influence of order of experimental nights on the AuC response ($F_{22,3} = 1.7$, n.s.), nor for the initial ($F_{22,3} = 2.0$, n.s.) or delayed response ($F_{22,3} = 0.8$).

Influence of sleep stage

The AuC, the initial maxima and the delayed maxima were compared in the sleep stages N3, N2 and REM. For wake stage and for stage N1 there were too few events to allow for comparison. The results are presented in table 2.

ANOVA for repeated measurements using the within subject factors sleep stage (stages N3, N2 and REM) and vibration level (low vs. high) was calculated.

The overall **AuC** revealed no significant influence of sleep stage ($F_{23,2} = 1.1$, n.s.). The **initial response** revealed a significant influence of sleep stage ($F_{23,2} = 5.1$ $p=0.009$). Bonferroni-corrected post-hoc comparison reveals that the initial increase of HR was significantly higher in REM then in stage N2 sleep ($p=0.002$). The same significant pattern was seen in the **delayed response** ($F_{23,2} = 4.8$ $p=0.013$). Here again post-hoc comparison showed that HR increase was higher in REM sleep than in stage N2 ($p=0.017$).

Influence of Noise sensitivity and gender

Effect of sensitivity. The AuC, initial maxima and delayed maxima of HR change are compared between participants with low (N=14) and high (N=10) noise sensitivity. The results are displayed in table 3. No significant main effects of noise sensitivity were found in the AuC ($F_{22,1} = 0.007$, n.s.) nor in the initial ($F_{22,1} = 2.8$, n.s.) or delayed response ($F_{22,1} = 0.004$, n.s.). There were no significant interactions between noise sensitivity and vibration level.

Effect of gender. The results of comparisons between men (N=11) and women (N=13) are shown in table 3. No significant differences were found in the AuC (F22,1=2.5, n.s.) or in the HR initial (F22,1=0.5, n.s.) between genders. There was a tendency for men to exhibit a stronger increase of HR in the delayed response (F22,1=4.1, p=0.055).

Discussion

Our data shows that train noise and vibration exposure leads to an increased number of awakenings and to cardiac arousals, and that vibration might be of particular importance. Heart rate acceleration with two maxima was observed following trains noise and vibration. One rapid and distinct initial HR increase is evident around 9s after train onset and a lengthier delayed increase is observed starting about 17s after train onset. In a similar study dealing with traffic noise alone, Griefahn et al [11] found an increase of HR matching our initial response but no delayed increase. A similar initial increase of HR has been found for pure tones during sleep [36]. We propose that the second plateau is due to the additional vibration exposure, which seems to either act in isolation to result in cardiac response, or alternatively enhance or interact with any delayed effects of rail noise on HR. Supporting this interpretation we see a significant difference between the low and the high vibration level for the delayed, but not for the initial increase. The combined exposure might be a more pronounced alarming signal than noise alone and the HR alterations endure even after cessation of the actual exposure. In accordance with previous literature on sleep disturbance due to noise exposure [10-12], the highest alterations of HR were found in REM sleep. This was observed for both the initial and delayed response.

Cardiac arousals are likely induced by brain stem activation [37]. This subcortical activation may present a form of primary arousal in sleep and - depending on the strength of activation and on sleep stage - lead to cortical activation and arousal. Increased HR indicates subcortical arousal

and enhances the probability of a cortical arousal with implications for sleep structure [37]. However, even HR acceleration without concomitant cortical arousal has been shown to have adverse effects on sleep structure. Guilleminault et al showed that forced subcortical arousals accompany disturbed sleep and the persons are not necessarily aware of their disturbed sleep the following morning [38].

In the short term, arousals have positive effects by ensuring a higher level of behavioral responsiveness in the presence of external stimuli, while the sleep is protected as far as possible [39]. However, in the long term increased autonomic reaction might be harmful. As Kohler and Stradling point out in a review paper, recurrent arousals are one of the mechanisms leading to cardiovascular disease in obstructive sleep apnea [40]. In this manner the autonomic arousals provoked by passing trains may - if continuing over years –contribute to the slightly enhanced myocardial infarction risk that has been found in people living in areas with high traffic exposure [15-18]. The study of Tassi et al suggests that people living in areas with high railway noise for more than ten years can partly adjust their sleep to this adverse exposure. However, even after such long-term exposure, significant alterations of HR due to nocturnal railway have been found [5]. In epidemiological studies, the effects of noise exposure alone are rather small [15-18]. Our data suggests that the effect of chronic traffic exposure on cardiovascular disease might be higher if vibration is additionally taken into account. As described in the methods of this paper, we excluded events from the analysis where persons awoke. This approach allows a focused estimation of HR reaction from traffic noise during sleep. However, as HR reactions are usually greater if accompanied by awakenings [11], our results very likely underestimate the effects. We found an average train-related increase of 3bpm in 79% of participants, supporting previously reported data [10]. It is not possible with the present study design to estimate the clinical relevance of these results. However, an increase of HR in sleep perhaps reflects a higher

sympathetic tone [9] and this may contribute to the enhanced the risk for hypertension in long term traffic exposure [14].

Our results are less clear regarding the impact of interindividual differences. Previous epidemiological research has shown that persons who state being sensitive to noise report greater sleeping problems due to traffic than non-sensitive persons [41]. However, we found no significant effect of self-reported noise sensitivity on HR response towards nocturnal train events. During the day it has been shown that self-declared highly noise sensitive persons have higher cardiovascular responses towards different ecologically relevant sound, including traffic noise [13]. The sample size of sensitive and non-sensitive participants in our study is relatively small and therefore non-significant results in particular have to be interpreted with caution. The sample size also limits any interpretation of gender effects. In coherence with the study of Griefahn, we found no significant gender differences in the initial HR amplitude [11]. We found a tendency for enhanced delayed HR response in men with a rather strong effect size. This corresponds to results from several field studies, where enhanced traffic exposure related cardiovascular diseases were found in men [15 16 18].

We are aware of the limitations of the study: Regarding internal validity, the time resolution of the measurement could potentially lead to a smoothing of the averaged data, underestimating our results. Nevertheless, a clear effect of enhanced HR reaction in high vibration conditions can be seen. We argue that vibration exposure is responsible for the second plateau. However, a vibration alone condition i.e. in the absence of noise, is needed to confirm this. With respect to external validity, the study is limited to the homogenous group of young and healthy participants. To generalize the results, a broader age range would be preferable and the study should be replicated in field conditions.

In conclusion, the combined exposure of freight train noise and vibration influenced HR during sleep, whereby HR amplitudes increase with increasing vibration level. This provoked acceleration may affect cardiovascular functioning in long term [5 11] and could be a mechanism explaining the previous findings of cardiovascular disease in persons exposed to traffic in general [14-18] and rail traffic in particular [8]. To study the specific influence of vibration in contrast to noise, further studies are required.

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Tables

TABLE 1: *Vibration and noise parameters applied to individual trains.*

Train	Nr of passages per night: 20 trains/ night	Nr of passages per night: 36 trains/ night	Noise exposure: L_{AEq} (dB)	Noise exposure: L_{AFmax} (dB)	Noise exposure: t > 35dB (s)	Noise exposure: $T_{10\%-90\%}$ (s)	Vibration exposure (same for all trains): Unweighted acceleration (m/s^2 rms)	Vibration exposure (same for all trains): W_d Weighted peak acceleration (m/s^2)
1	4	8	44.0	48.4	11.5	8.9		
2	5	8	42.7	47.2	46.2	9.8	High = 0.072	High = 0.0204
3	4	8	44.5	49.8	23.7	8.4	Low = 0.036	Low = 0.0102
4	5	8	45.6	49.8	29.2	7.9		
5	2	4	42.4	47.2	56.9	9.2		

The vibration acceleration is reported according to the ISO 2631-1 standard.

TABLE 2: *Analysis of event related HR response for each of the four exposure nights and combined for low and high vibration exposure.*

		Low vibration		High vibration	
		20 trains/night N=24	36 trains/night N=23	20 trains/night N=23	36 trains/night N=24
		Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
HR AuC	train	41.7 (114.5)	77.2 (103.0)	19.4 (77.6)	52.3 (86.8)
	fake	-4.6 (83.7)	0.2 (54.4)	-7.4 (51.5)	2.2 (60.9)
Number of event related awakenings per night	train	3.3 (2.3)	4.2 (2.6)	5.2 (3.2)	5.6 (3.4)
	fake	1.5 (0.7)	1.5 (0.6)	1.2 (0.4)	1.8 (0.8)
		number of participants (percentage of sample population)			
Event-related HR change of at least 3bpm	train	15 (62.5%)	17 (73.9%)	13 (54.2%)	19 (79.2%)
	fake	9 (37.5%)	8 (34.8%)	4 (16.7%)	4 (16.7%)
		low vibration		**high vibration**	
AuC	train	Mean (SD) 21.2 (81.8)		Mean (SD) 62.3 (81.4)	
Initial response	train	1.6(1.8)		2.3 (2.0)	
	fake	0.7 (0.9)		0.7 (1.0)	
Delayed response	train	2.6 (2.4)		3.7 (2.8)	
	fake	1.1 (1.0)		1.3(0.9)	
		Sleep stage related (train events only)			
AuC	stage N3	25.1 (166.9)		79.7 (114.8)	
	stage N2	30.2 (95.8)		53.2 (90.3)	
	REM	42.6 (162.4)		113.0 (203.1)	
Initial response	stage N3	2.3 (2.7)		3.7 (4.0)	
	stage N2	2.1 (2.2)		2.2 (1.9)	
	REM	2.8 (2.8)		4.7 (3.3)	
Delayed response	stage N3	4.5 (4.9)		5.9 (4.1)	
	stage N2	3.6 (3.6)		4.5 (3.4)	
	REM	5.6 (4.1)		7.5 (5.8)	

Area under the curve (AuC), number of event related awakenings, initial and delayed maximal increase of heart rate (HR) and number of participants with an event related change of HR of at least 3bpm are presented for each of the four experimental nights. AuC, initial and delayed maximal increase of HR are presented for the combined low and high vibration condition. Cave:

the initial and delayed response is calculated as maximal increase within the first 10 to15 or 20 to 48 seconds after train onset. This difference in search area explains partly the differences between the initial and delayed increase of HR.

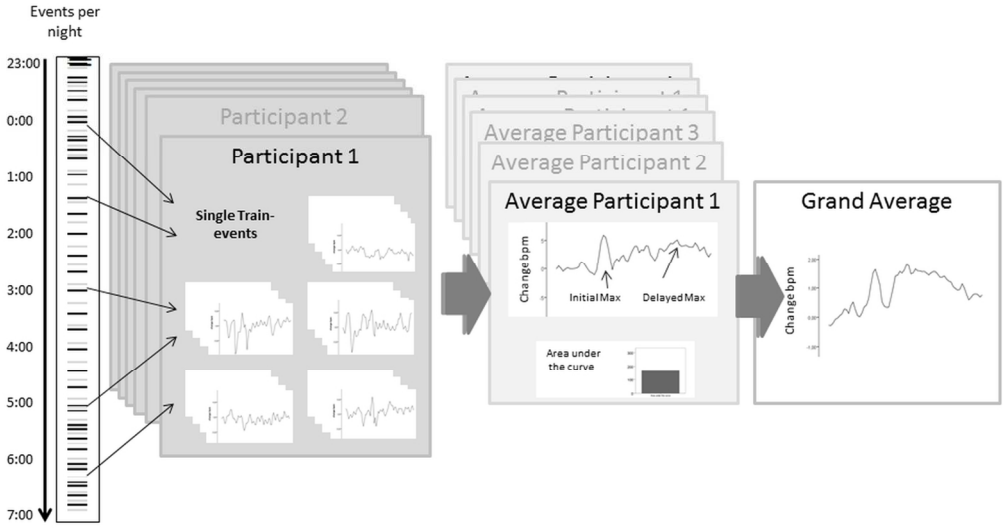
TABLE 3: *Train related HR characteristics in relation to gender and noise sensitivity of the participants.*

			Low vibration	High vibration
			Mean (SD)	Mean (SD)
Gender		AuC	48.1 (73.0)	81.2 (96.8)
		Initial response	1.7 (1.4)	2.0 (1.9)
	Men N=11	Delayed response	3.5 (2.8)	4.8 (3.7)
		AuC	-1.6 (84.6)	46.2 (65.4)
	Women	Initial response	1.5 (2.1)	2.6 (2.2)
	N=13	Delayed response	1.8 (1.7)	2.8 (1.5)
Noise sensitivity	Non sensitive	AuC	24.0 (62.0)	63.8 (93.3)
		Initial response	1.1 (1.3)	2.0 (2.0)
	N=14	Delayed response	2.3 (2.0)	4.0 (3.4)
		AuC	17.2 (107.3)	60.1 (65.9)
	Sensitive	Initial response	2.4 (2.2)	2.8 (2.1)
	N=10	Delayed response	3.0 (2.9)	3.4 (2.0)

Figure Legends

FIGURE 1. Visualization of the analytical procedure. For each of the four experimental nights, HR data is taken out for each of the train-events (black lines of the events per night) for each participant. Data is checked for artifacts and wake stage and then sampled into one average HR response for each participant with corresponding initial maximum, delayed maximum and area under the curve parameters. The grand average is built over all of the participants. The very same procedure is applied to the fake events (grey lines of the events per night).

FIGURE 2. A) Averaged HR response following the train events and fake events, respectively, in the low and high vibration exposure. In figure 1A, a clear HR increase after train events can be seen, while HR stays at baseline for fake events. The HR response can be divided into two components. An initial response occurring around 10 to 15 seconds after train onset and a delayed response occurring about 17 to 48 sec after train onset. The delayed response is significantly enhanced in the high vs. low vibration exposure. In figure 1B the averaged HR reaction in the high vibration condition is visualized for each of the five different train types. Although the different number of trains does not allow direct comparison, it can be seen, that in principal the same characteristics of the HR reaction is apparent for each train.



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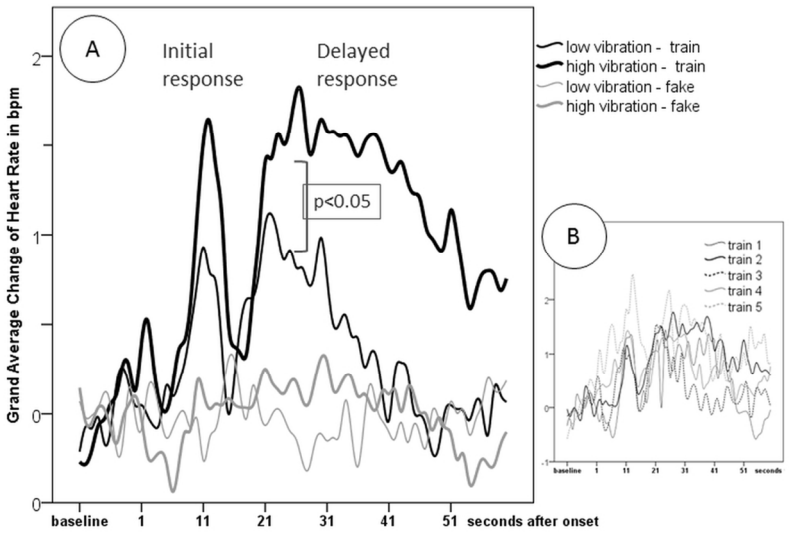
119x90mm (300 x 300 DPI)



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90x127mm (300 x 300 DPI)

Effects of train noise and vibration on human heart rate during sleep –an experimental study

Authors: Dr. Ilona Croy[1]; Michael Smith[1],MSc; Prof. Kerstin Persson Waye[1]

[1]University of Gothenburg, Department of Occupational and Environmental Medicine, University of Gothenburg, Medicinaregatan 16, 405 30 Göteborg, Sweden

Corresponding author:
Dr. Ilona Croy
Occupational and Environmental Medicine
Medicinaregatan 16
University of Gothenburg
Box 414, 405 30 Göteborg, Sweden
+46 (0)31 786 6814
Ilona.croy@amm.gu.se

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Abstract

Objectives: Transportation of goods on railways is increasing and the majority of the increased numbers of freight trains run during the night. Transportation noise has adverse effects on sleep structure, affects heart rate during sleep and may be linked to cardiovascular disease. Freight trains also generate vibration and little is known regarding the impact of vibration on human sleep. A laboratory study was conducted to examine how a realistic nocturnal railway traffic scenario influences the heart rate (HR) during sleep.

Methods: Twenty four volunteers slept in the laboratory for six consecutive nights: one habituation night, one control and four experimental nights in which train noise and vibration was reproduced. In the experimental nights, 20 or 36 trains with low or high vibration characteristics were presented. Polysomnographical data and ECG were recorded.

Results: The train exposure lead to a significant change of HR within one minute of exposure onset ($p=0.002$), characterized by an initial and a delayed increase of HR. The high vibration condition provoked an average increase of at least 3bpm per train in 79% if the participants. Cardiac responses were in general higher in the high vibration compared to the low vibration condition ($p=0.006$). No significant effect of noise sensitivity and gender was revealed, although there was a tendency for men to exhibit stronger HR acceleration then women.

Conclusions: Freight trains provoke HR accelerations during sleep, and the vibration characteristics of the trains are of special importance. In long term, this may affect cardiovascular functioning of persons living close to railways.

Article summary

Article Focus:

Noise of passing trains affects sleep and induces heart rate accelerations in humans. This may be linked to cardiovascular disease. Trains do not only emit noise, but also vibration and the influence of vibration is poorly understood.

Key message:

Our study shows, that freight train noise and vibration provokes heart rate accelerations in sleep. The HR response is characterized by two peaks, whereby the second one is even more pronounced and may dependent on the vibration amplitude.

Strengths and limitations:

The influence of nocturnal vibration on HR response has been studied for the first time under very well controlled laboratory conditions. However further studies with increased sample size should be carried out to analyze the influence of gender, age and sensitivity in more detail.

Introduction

As the European market share of freight traffic is expected to increase from 8% in 2001 to 15% in 2020 [1], it is important to estimate the impact this may have on the health of persons living close by to railway lines. Railway noise is related to disturbed sleep [2-6] which in turn increases the risk of cardiovascular disease [7], supported by indications of increased cardiovascular disease in persons living close to railways [8]. Understanding how railway noise and vibration influence the cardiovascular system in sleep is therefore necessary.

The sympathetic tone is reduced in sleep which is reflected by a reduction of heart rate (HR) [9]. External noise events have the potential to disturb sleep and lead to event related increase of heart rate. This has been shown for pure tones [10] and more recently for traffic noise [11 12]. Noise induced HR changes do not seem to habituate during sleep [13], therefore they might “bear a pathogenic potential for the genesis of cardiovascular disease” [11]. Evidence that long term traffic exposure can increase the risk for cardiovascular disease comes from field studies.

Babisch summarizes that traffic exposure enhances the risk for hypertension by a factor of between 1.5 to 3 [14]. An increased risk for myocardial infarction in persons living close to railways has been found in several studies [15-18], although the effect sizes are rather small and the effects seem to be more pronounced in men [15 16 18].

Traffic noise influences sleeping structure [19] and leads to increased awakenings [19 20] throughout the night. People living close to busy roads and railways or in close proximity to airports therefore often report poor sleep quality [3 4 6]. A Dutch study revealed that the number of persons reporting strong difficulties falling asleep due to traffic noise increased from 18% to 23% between 1998 and 2003 [21]. Disturbed sleep might be one factor leading to enhanced cardiovascular risk in persons exposed to traffic. It has been suggested that sleep disturbances contribute to cardiovascular disease through the pathway of enhanced inflammatory processes [22] or, related to this, through the pathway of enhanced stress reaction affecting the cardiovascular system [23]. In a field study, Carter et al examined the influence of nocturnal traffic exposure on 7 older men with cardiac arrhythmia, who lived close to a busy road [24]. They found an increased likelihood of single ventricular premature contractions (a form of cardiac arrhythmia) after noise peaks.

Two environmental exposures commonly arising from traffic are noise and vibration. As described, traffic noise disturbs sleep and leads to event related HR changes, but the effect of traffic vibration is not well understood. Studies investigating annoyance show that 1) traffic vibration causes annoyance, 2) annoyance increases with increasing vibration level and 3) vibration enhances the annoying effect of noise [25-27]. Furthermore, higher vibration amplitude is related to higher reported sleep disturbances in persons exposed to railway traffic [6]. A study on human fetuses suggests that short experimental external vibration exposure alters sleep stage [28]. Indications that vibration exposure is disadvantageous for cardiovascular function have been found in people exposed to high levels of vibration at work [29 30].

The effect of combined traffic vibration and noise on the cardiovascular system in sleep has - to our best knowledge - not been examined. It is the aim of the present study to detect if nocturnal noise and vibration exposure from freight trains provoke HR accelerations.

Methods

Participants and procedure

Twenty four volunteers (11 men, 13 women, 19-28 years, mean age 22.9+/- 2.8years) slept in our sleep laboratory. Twenty three were students and one was in full-time employment, recruited by public advertisements placed around the campus of the University of Gothenburg. Eligible persons were required to be aged between 18 and 30 years, in healthy condition, maintain normal sleeping patterns and not use tobacco products. To decrease the probability of subclinical breathing difficulties or apnea, participants were required to have a BMI within the normal range of 18.5 - 24.99 [31]. Following initial screening, hearing was tested before volunteers were accepted. Manual audiometric testing was conducted using an Entomed SA201^{II} according to ISO 8253-1. Left and right ears were tested separately at frequencies of 250, 500, 1000, 2000, 3000, 4000, 6000 and 8000 Hz with hearing considered to be normal if measured thresholds were not more than a screening level of 20 dB HL.

The participants were instructed to begin attempting to fall asleep at 23:00 each night, were awoken by an alarm call at 07:00 each morning, and were prohibited from sleeping outside of this period. Before attending they were asked about their noise sensitivity on a 5-point Likert scale. Fourteen participants (8 men, 6 women) rated themselves as being noise insensitive (points 1-2) and 10 rated themselves being noise sensitive (points 3-5). For each participant, the study consisted of one habituation night and one control night proceeding four experimental nights in which simulated trains passed. The arrangement of the experimental nights was randomized

across participants. Gender and noise sensitivity were approximately equally spread around the randomization table. In two of the four experimental nights, the participants were exposed to 20 trains per night with all trains having high vibration one night and low vibration in the other (High20 and Low20 respectively). In the remaining two nights they were exposed to 36 trains per night again with either high or low vibrations (High36 and Low36). The acoustic signal for the trains was not varied with vibration amplitude. There were more train passages between the periods of 23:00-01:00 and 05:00-07:00 to reflect typical real world scenarios.

The sleep laboratory consists of three individual rooms isolated from external noise and vibration, furnished to simulate a typical bedroom with a bed, chairs, desk and small chest of drawers. Eighty eight ceiling loudspeakers reproduced the low frequency content of noise below 125 Hz, and higher frequencies were generated by two loudspeaker cabinets in the room corners. Because of the unrealistically low background noise levels in the rooms (<14 dBA), for the duration of the trial artificial ventilation noise was introduced at a level of 25 dBA measured at the pillow position on the bed. In addition to the bedrooms the participants had private access to a shared a communal space outfitted with a kitchen, dining area and living area with sofa and television. They were free to come and go as they desired during the daytime, being required to arrive by 20:00 each evening to ensure rest prior to bedtime and to allow sufficient time for electrode attachment.

Vibration and noise exposure were similar to another study conducted in our laboratory [32] and are only summarized here. Five different train passages were synthesized based upon analysis of accelerometer and sound level measurements performed in the field. Noise signals were low pass filtered to correspond to a fully closed window. The vibration exposure was an amplitude modulated 10 Hz signal applied along the lengthwise horizontal axis of the bed by electrodynamic shakers with a frequency response of 5-40 Hz mounted to the underside of the bed frame. The vibration began when the train noise signal exceeded an A-weighted equivalent

level (L_{Aeq}) of 35 dB, ensuring masking of any audible mechanical operation noise. Further acoustic and vibration data for each individual train is presented in Table 1.

Ethics Statement

The study followed the Declaration of Helsinki on Biomedical Research Involving Human Subjects and was approved by the Ethics Committee from the University of Göteborg (920-11). All participants provided written informed consent.

Cardiac response and polysomnography

During all experimental nights, physiological data was recorded using ambulatory polysomnogram devices (SOMNOscreen plus PSG+, SOMNOmedics GmbH, Germany). The time resolution of the onset was 4.7+/- 3.4 seconds.

Cardiac activity was recorded via a modified lead II torso placement electrocardiogram (ECG) at a sampling frequency of 256 Hz as per the American Academy of Sleep Medicine [33]. To identify sleep stage, electroencephalogram (EEG), electrooculogram (EOG) and submental electromyogram (EMG) were also recorded using standardized surface electrode placements and sampling and filter frequencies. To ensure good signal quality, following electrode attachment it was checked that the electrical impedance of each contact was ≤ 5kΩ. Thirty second epoch sleep staging was performed manually by a trained sleep technician~~s~~. Additionally, participants wore two effort belts to record breathing rates and a finger pulse oximeter to record blood oxygen saturation, plethysmogram and pulse information, although this data ~~shal~~will~~l~~ not be reported here. Due to a technical fault, ECG was not ~~recorded~~ obtained for one participant (female, noise sensitive) in the High20 night and so could not be included in analysis.

Event related analysis and control condition

In order to examine event related changes of HR, data from the four exposure nights was analyzed. The continuously recorded ECG (sampling frequency of 256Hz) was converted into

HR (in bpm) in 1s intervals through the whole night. EEG and ECG recordings were synchronized with the train exposure events, thus allowing a direct temporal association between the occurrence of a train and the participant's HR reaction and sleep stages. Analysis revealed that 9.9% of the train onsets occurred during wake stage, 8.1% during stage N1, 42% during stage N2, 21.4% during stage N3 and 17.8% during REM.

The procedure of HR analysis is visualized in Figure 1. HRs below 35 and above 130bpm were excluded from the analysis [11 34]. This was the case for 4.1% of the events. For analysis of event related HR change (bpm), only train events where participants were asleep were considered. This approach was chosen because Griefahn and colleagues showed that HR response to traffic noise differs depending on whether the participants do or do not awake [11]. The relatively few trains per night in our study do not allow additional examination of HR response in the case of awakenings. An event related awakening has been defined as a sleep stage change to Wake from any other stage in at least one of the two epochs (30s) following the train onset [35]. The first of these was determined by that having at least 15s under influence of the event. Additionally, events for which the participants were awake in the epoch preceding train onset were excluded from the analysis. The whole procedure left 72.4% of the train events in the low vibration condition and 66.1% of the train events in the high vibration condition for analysis. In accordance with the literature, the screening interval for cardiac activations was set to 60s after train onset [11 34]. In order to analyze change of HR, the average cardiac response from the 10s preceding the train event was used as a baseline value for each given event and subsequently subtracted from the cardiac response in each of the sixty 1s time intervals following the train onset.

In order to examine if there was a train related change of HR at all, ***'fake trains'*** were calculated. Fake trains were defined as time intervals of 60s not accompanied by a real train event, distributed at time intervals approximately equally spaced between the actual exposure events.

Twenty of these were introduced in the Low20 and High20 nights and 36 in the Low36 and High36 nights. . Analyzed HR data for these consisted of 70s, where 10s served as baseline for the following 60s. As with the real exposures, data where polysomnographical analysis indicated that participants were awake before onset or awoke during the two epochs following onset was excluded. All event-related 60speriods were subsequently averaged over nights with low or high vibration respectively. The whole procedure was the same for each of the four nights to avoid any potential investigator bias.

Griefahn and colleagues report a maximum increase of HR about 13.2s after event onset [11]. We took this time interval +/- 3s as a base for the searching area for the maximum increase between 10 and 16s after train onset.

Statistical analysis

Data was analyzed using SPSS 20 (SPSS Inc., Ill, USA). In order to identify any overall effect of trains on the HR, the integral of the HR response was taken to determine the area under the curve (AuC) for the 60s after train onset. An Analysis of Variance (ANOVA) for repeated measurements was calculated comparing the AuC in three factors: event (fake vs. real-train), vibration level (low vs. high) and number of trains (20 vs. 36). The effects of noise sensitivity and gender were also analyzed using ANOVA for repeated measurements with the between subject factor noise sensitivity/gender and the within subject factor vibration level (low vs. high). Post-hoc-comparisons are reported Bonferroni-corrected. The level of significance is set at α=0.05.

Results

Influence of train noise and vibration on HR

The AuC analysis for the change of HR 60s after event onset revealed a significant main effect of train, indicating that ***train events lead to an enhanced change of HR compared with the fake events*** (F22,1=12.0, p=0.002). Furthermore there were significantly more awakenings in the train vs. fake events (F22,1 =40.3, p<0.001).

There was a ***significant main effect of vibration level.*** A higher change of HR and an increased number of awakenings could be observed in the high compared to the low vibration level (HR: F22,1=7.6; p=0.01; Awakenings: F22,1=6.5; p=0.014). The number of trains had no significant influence on the HR change. The results are displayed in table 2.

Averaged over all trains within one night, an ***increase of HR of at least 3bpm*** was observed in 54% of the participants in Low36, 52% in the Low20 condition, 74% in the High20 night and 79% of participants in the High36 night. For the fake train events an average increase of HR of at least 3 bpm was observed in only 17 to 38% of the participants, depending on the exposure night.

As no significant influence of the number of trains was revealed, the four exposure conditions were combined into two conditions: a high vibration and a low vibration condition. This approach is advantageous for the signal-noise ratio, because in this way the number of analyzable trains could be increased to 56. For calculating the low vibration condition, train events from the Low20 and Low36 nights that matched the inclusion criteria described in the methods section were averaged to obtain one low vibration response. The same method was performed for the high vibration condition. This procedure was repeated for the fake events, so that resulting responses for the low and high vibration exposures for both the actual and phantom events could be compared.

Characteristics of the HR curve

The HR curve shows a biphasic characteristic (see figure 2A). After approximately 9s a short initial response characterized by increase of HR takes place, lasting for around 6s. This ***initial response*** is significantly above the baseline between 10-13s for the low vibration condition, and between 10-15s for the high vibration condition (t-test, $p<0.05$). This response is in accordance with the proposed search area of the maximum increase.

An additional ***delayed response*** can also be observed. This response is characterized by a second increase of HR beginning around 17s following train onset and with a duration of around 20s for the low vibration condition and about 30s for the high vibration condition. The delayed response is significantly above baseline between 21-22s for the low vibration condition, and between 20-48s for the high vibration condition (t-test, $p<0.05$). This same response could be seen for each of the five individual train types (see figure 2B)

This response pattern could be observed in the individual participants, but the time delay after event onset showed a great variability. The individual maximum for each participant within the range of the initial response (10-15s) and the delayed response (17-50s) was calculated and is reported in table 2.

For the **initial response and for the delayed response**, ANOVA for repeated measurements revealed a significant main effect of event, indicating that the ***train-events led to an enhanced change of HR compared with the fake-events*** (initial response:$F_{23,1}=13$, $p=0.001$; delayed response: $F_{23,1}=15.5$, $p=0.001$).

The vibration level had no significant influence on the initial increase of HR ($F_{23,1}=2.1$, n.s.), but affected the delayed response. A higher delayed increase of HR was observed in the high compared to the low vibration level ($F_{23,1}=9.4$, $p=0.006$).

Effect of order

The participant's HR responses of the four nights were compared with respect to the order of experimental nights using ANOVA for repeated measurements. There was no significant influence of order of experimental nights on the AuC response ($F_{22,3} = 1.7$, n.s.), nor for the initial ($F_{22,3} = 2.0$, n.s.) or delayed response ($F_{22,3} = 0.8$).

Influence of sleep stage

The AuC, the initial maxima and the delayed maxima were compared in the sleep stages N3, N2 and REM. For wake stage and for stage N1 there were too few events to allow for comparison. The results are presented in table 2.

ANOVA for repeated measurements using the within subject factors sleep stage (stages N3, N2 and REM) and vibration level (low vs. high) was calculated.

The overall **AuC** revealed no significant influence of sleep stage ($F_{23,2} = 1.1$, n.s.). The **initial response** revealed a significant influence of sleep stage ($F_{23,2} = 5.1$ $p = 0.009$). Bonferroni-corrected post-hoc comparison reveals that the initial increase of HR was significantly higher in REM then in stage N2 sleep ($p = 0.002$). The same significant pattern was seen in the **delayed response** ($F_{23,2} = 4.8$ $p = 0.013$). Here again post-hoc comparison showed that HR increase was higher in REM sleep than in stage N2 ($p = 0.017$).

Influence of Noise sensitivity and gender

Effect of sensitivity. The AuC, initial maxima and delayed maxima of HR change are compared between participants with low (N=14) and high (N=10) noise sensitivity. The results are displayed in table 3. No significant main effects of noise sensitivity were found in the AuC ($F_{22,1} = 0.007$, n.s.) nor in the initial ($F_{22,1} = 2.8$, n.s.) or delayed response ($F_{22,1} = 0.004$, n.s.). There were no significant interactions between noise sensitivity and vibration level.

Effect of gender. The results of comparisons between men (N=11) and women (N=13) are shown in table 3. No significant differences were found in the AuC (F22,1=2.5, n.s.) or in the HR initial (F22,1=0.5, n.s.) between genders. There was a tendency for men to exhibit a stronger increase of HR in the delayed response (F22,1=4.1, p=0.055).

Discussion

Our data shows that train noise and vibration exposure leads to an increased number of awakenings and to cardiac arousals, and that vibration might be of particular importance. Heart rate acceleration with two maxima was observed following trains noise and vibration. One rapid and distinct initial HR increase is evident around 9s after train onset and a lengthier delayed increase is observed starting about 17s after train onset. In a similar study dealing with traffic noise alone, Griefahn et al [11] found an increase of HR matching our initial response but no delayed increase. A similar initial increase of HR has been found for pure tones during sleep [36]. We propose that the second plateau is due to the additional vibration exposure, which seems to either act in isolation to result in cardiac response, or alternatively enhance or interact with any delayed effects of rail noise on HR. Supporting this interpretation we see a significant difference between the low and the high vibration level for the delayed, but not for the initial increase. The combined exposure might be a more pronounced alarming signal than noise alone and the HR alterations endure even after cessation of the actual exposure. In accordance with previous literature on sleep disturbance due to noise exposure [10-12], the highest alterations of HR were found in REM sleep. This was observed for both the initial and delayed response.

Cardiac arousals are likely induced by brain stem activation [37]. This subcortical activation may present a form of primary arousal in sleep and - depending on the strength of activation and on sleep stage - lead to cortical activation and arousal. Increased HR indicates subcortical arousal

and enhances the probability of a cortical arousal with implications for sleep structure [37]. However, even HR acceleration without concomitant cortical arousal has been shown to have adverse effects on sleep structure. Guilleminault et al showed that forced subcortical arousals accompany disturbed sleep and the persons are not necessarily aware of their disturbed sleep the following morning [38].

In the short term, arousals have positive effects by ensuring a higher level of behavioral responsiveness in the presence of external stimuli, while the sleep is protected as far as possible [39]. However, in the long term increased autonomic reaction might be harmful. As Kohler and Stradling point out in a review paper, recurrent arousals are one of the mechanisms leading to cardiovascular disease in obstructive sleep apnea [40]. In this manner the autonomic arousals provoked by passing trains may - if continuing over years –contribute to the slightly enhanced myocardial infarction risk that has been found in people living in areas with high traffic exposure [15-18]. The study of Tassi et al suggests that people living in areas with high railway noise for more than ten years can partly adjust their sleep to this adverse exposure. However, even after such long-term exposure, significant alterations of HR due to nocturnal railway have been found [5]. In epidemiological studies, the effects of noise exposure alone are rather small [15-18]. Our data suggests that the effect of chronic traffic exposure on cardiovascular disease might be higher if vibration is additionally taken into account. As described in the methods of this paper, we excluded events from the analysis where persons awoke. This approach allows a focused estimation of HR reaction from traffic noise during sleep. However, as HR reactions are usually greater if accompanied by awakenings [11], our results very likely underestimate the effects. We found an average train-related increase of 3bpm in 79% of participants, supporting previously reported data [10]. It is not possible with the present study design to estimate the clinical relevance of these results. However, an increase of HR in sleep perhaps reflects a higher

sympathetic tone [9] and this may contribute to the enhanced the risk for hypertension in long term traffic exposure [14].

Our results are less clear regarding the impact of interindividual differences. Previous epidemiological research has shown that persons who state being sensitive to noise report greater sleeping problems due to traffic than non-sensitive persons [41]. However, we found no significant effect of self-reported noise sensitivity on HR response towards nocturnal train events. During the day it has been shown that self-declared highly noise sensitive persons have higher cardiovascular responses towards different ecologically relevant sound, including traffic noise [13]. The sample size of sensitive and non-sensitive participants in our study is relatively small and therefore non-significant results in particular have to be interpreted with caution. The sample size also limits any interpretation of gender effects. In coherence with the study of Griefahn, we found no significant gender differences in the initial HR amplitude [11]. We found a tendency for enhanced delayed HR response in men with a rather strong effect size. This corresponds to results from several field studies, where enhanced traffic exposure related cardiovascular diseases were found in men [15 16 18].

We are aware of the limitations of the study: Regarding internal validity, the time resolution of the measurement could potentially lead to a smoothing of the averaged data, underestimating our results. Nevertheless, a clear effect of enhanced HR reaction in high vibration conditions can be seen. We argue that vibration exposure is responsible for the second plateau. However, a vibration alone condition i.e. in the absence of noise, is needed to confirm this. With respect to external validity, the study is limited to the homogenous group of young and healthy participants. To generalize the results, a broader age range would be preferable and the study should be replicated in field conditions.

In conclusion, the combined exposure of freight train noise and vibration influenced HR during sleep, whereby HR amplitudes increase with increasing vibration level. This provoked acceleration may affect cardiovascular functioning in long term [5 11] and could be a mechanism explaining the previous findings of cardiovascular disease in persons exposed to traffic in general [14-18] and rail traffic in particular [8]. To study the specific influence of vibration in contrast to noise, further studies are required.

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Tables

TABLE 1: *Vibration and noise parameters applied to individual trains.*

Train	Nr of passages per night: 20 trains/ night	Nr of passages per night: 36 trains/ night	Noise exposure: L_{AEq} (dB)	Noise exposure: L_{AFmax} (dB)	Noise exposure: t > 35dB (s)	Noise exposure: $T_{10\%-90\%}$ (s)	Vibration exposure (same for all trains): Unweighted acceleration (m/s^2 rms)	Vibration exposure (same for all trains): W_d Weighted peak acceleration (m/s^2)
1	4	8	44.0	48.4	11.5	8.9		
2	5	8	42.7	47.2	46.2	9.8	High = 0.072	High = 0.0204
3	4	8	44.5	49.8	23.7	8.4	Low = 0.036	Low = 0.0102
4	5	8	45.6	49.8	29.2	7.9		
5	2	4	42.4	47.2	56.9	9.2		

The vibration acceleration is reported according to the ISO 2631-1 standard.

TABLE 2: *Analysis of event related HR response for each of the four exposure nights and combined for low and high vibration exposure.*

		Low vibration		High vibration	
		20 trains/night N=24	**36 trains/night N=23**	**20 trains/night N=23**	**36 trains/night N=24**
		Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)
HR AuC	train	41.7 (114.5)	77.2 (103.0)	19.4 (77.6)	52.3 (86.8)
	fake	-4.6 (83.7)	0.2 (54.4)	-7.4 (51.5)	2.2 (60.9)
Number of event related awakenings per night	train	3.3 (2.3)	4.2 (2.6)	5.2 (3.2)	5.6 (3.4)
	fake	1.5 (0.7)	1.5 (0.6)	1.2 (0.4)	1.8 (0.8)
		number of participants (percentage of sample population)			
Event-related HR change of at least 3bpm	train	15 (62.5%)	17 (73.9%)	13 (54.2%)	19 (79.2%)
	fake	9 (37.5%)	8 (34.8%)	4 (16.7%)	4 (16.7%)
		low vibration		**high vibration**	
AuC	train	Mean (SD) 21.2 (81.8)		Mean (SD) 62.3 (81.4)	
Initial response	train	1.6(1.8)		2.3 (2.0)	
	fake	0.7 (0.9)		0.7 (1.0)	
Delayed response	train	2.6 (2.4)		3.7 (2.8)	
	fake	1.1 (1.0)		1.3(0.9)	
		Sleep stage related (train events only)			
AuC	stage N3	25.1 (166.9)		79.7 (114.8)	
	stage N2	30.2 (95.8)		53.2 (90.3)	
	REM	42.6 (162.4)		113.0 (203.1)	
Initial response	stage N3	2.3 (2.7)		3.7 (4.0)	
	stage N2	2.1 (2.2)		2.2 (1.9)	
	REM	2.8 (2.8)		4.7 (3.3)	
Delayed response	stage N3	4.5 (4.9)		5.9 (4.1)	
	stage N2	3.6 (3.6)		4.5 (3.4)	
	REM	5.6 (4.1)		7.5 (5.8)	

Area under the curve (AuC), number of event related awakenings, initial and delayed maximal increase of heart rate (HR) and number of participants with an event related change of HR of at least 3bpm are presented for each of the four experimental nights. AuC, initial and delayed maximal increase of HR are presented for the combined low and high vibration condition. Cave:

the initial and delayed response is calculated as maximal increase within the first 10 to15 or 20 to 48 seconds after train onset. This difference in search area explains partly the differences between the initial and delayed increase of HR.

TABLE 3: *Train related HR characteristics in relation to gender and noise sensitivity of the participants.*

			Low vibration	High vibration
			Mean (SD)	Mean (SD)
Gender		AuC	48.1 (73.0)	81.2 (96.8)
		Initial response	1.7 (1.4)	2.0 (1.9)
	Men N=11	Delayed response	3.5 (2.8)	4.8 (3.7)
		AuC	-1.6 (84.6)	46.2 (65.4)
	Women	Initial response	1.5 (2.1)	2.6 (2.2)
	N=13	Delayed response	1.8 (1.7)	2.8 (1.5)
Noise sensitivity	Non sensitive	AuC	24.0 (62.0)	63.8 (93.3)
		Initial response	1.1 (1.3)	2.0 (2.0)
	N=14	Delayed response	2.3 (2.0)	4.0 (3.4)
		AuC	17.2 (107.3)	60.1 (65.9)
	Sensitive	Initial response	2.4 (2.2)	2.8 (2.1)
	N=10	Delayed response	3.0 (2.9)	3.4 (2.0)

Figure Legends

FIGURE 1. *Visualization of the analytical procedure. For each of the four experimental nights, HR data is taken out for each of the train-events (black lines of the events per night) for each participant. Data is checked for artifacts and wake stage and then sampled into one average HR response for each participant with corresponding initial maximum, delayed maximum and area under the curve parameters. The grand average is built over all of the participants. The very same procedure is applied to the fake events (grey lines of the events per night).*

FIGURE 2. *A) Averaged HR response following the train events and fake events, respectively, in the low and high vibration exposure. In figure 1A, a clear HR increase after train events can be seen, while HR stays at baseline for fake events. The HR response can be divided into two components. An initial response occurring around 10 to 15 seconds after train onset and a delayed response occurring about 17 to 48 sec after train onset. The delayed response is significantly enhanced in the high vs. low vibration exposure. In figure 1B the averaged HR reaction in the high vibration condition is visualized for each of the five different train types. Although the different number of trains does not allow direct comparison, it can be seen, that in principal the same characteristics of the HR reaction is apparent for each train.*