Habitual traffic noise at home reduces cardiac parasympathetic tone during sleep

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A B S T R A C T

The relationships between road and rail traffic noise with pre-ejection period (PEP) and with respiratory sinus arrhythmia (RSA) during sleep, as indices of cardiac sympathetic and parasympathetic nervous system tone, were investigated in the field (36 subjects, with 188 and 192 valid subject nights for PEP and RSA, respectively). Two analyses were conducted. The first analysis investigated the overall relationships across the entire sleep period. A second analysis investigated differences in the relationships between the first and second halves of the sleep period. Separate multilevel linear regression models for PEP and RSA were employed. Potential covarates for each model were selected from the same pool of variables, which included: gender, age, body-mass index, education level, traffic noise source type, intake of medication, caffeine, alcohol and cigarette smoke, and hindrance during sleep due to the ambulatory recordings. RSA models were adjusted for respiration rate. Mean indoor traffic noise exposure was negatively related to mean RSA during the sleep period, specifically during the second half of the sleep period. Both respiration rate and age were negatively associated with RSA. No significant relationships were observed for PEP. The results indicate that higher indoor traffic noise exposure levels may lead to cardiac parasympathetic withdrawal during sleep, specifically during the second half of the sleep period. No effect of indoor traffic noise on cardiac sympathetic tone was observed.

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1. Introduction

Environmental noise from aircraft, road, or rail traffic during the night has been identified as a major cause of sleep disturbance (World Health Organization [WHO], 1999; Miedema and Vos, 2007; Muzet, 2007; WHO, 2007). In the European Union, 45% of citizens live in areas where environmental nighttime noise levels are considered uncomfortable for sleeping (WHO, 2007), and approximately 30% of citizens are exposed to night-time noise at levels known to cause sleep disturbance (WHO, 1999). Considering the extent to which urban populations are exposed to environmental night-time noise, a need for further knowledge of the impact of environmental noise on sleep exists. The focus of the present study was to investigate the influence of road and rail traffic noise on autonomic nervous system (ANS) activity during sleep at home.

During sleep overall parasympathetic nervous system (PNS) activity is increased compared to PNS activity during wakefulness, whereas sympathetic nervous system (SNS) activity is decreased during sleep depending on sleep stage (Gula et al., 2004; Burgess et al., 1997). Raised cardiac sympathetic tone leads to increased levels of beta-adrenergic receptor activity, thereby increasing myocardial contractility and heart rate (Sherwood et al., 1990; Brownley et al., 2000), as well as increasing blood pressure and vascular resistance. Increased cardiac parasympathetic tone leads to increased cardiac muscarinic receptor activity resulting in decreased heart rate (Dodd and Rolle, 1991; Brownley et al., 2000), and consequently a reduction in blood pressure. Alterations in autonomic nervous system activity during sleep could affect several aspects of sleep, which in turn may be related to well-being and long-term health (Muzet, 2007). A proposed function of sleep is that it protects the cardiovascular system, namely by lowering blood pressure levels and resetting baroreceptor sensitivity (Carter et al., 2002). Consequently, greater sympathetic dominance during sleep may counteract the protective function of sleep (Carter et al., 2002).

Night-time traffic noise has been found to induce alteration of cardiac ANS tone in humans during sleep without awaking them (e.g. Carter et al., 1994, 2002; Hofman et al., 1995). In order to assess the real-life impact of traffic noise exposure on ANS activity during sleep, field studies are most suitable, as laboratory sleep studies consistently find stronger effects (Pearsons et al., 1995; Basner et al., 2004). However, the limited number of field studies within this area of research has not given a clear pattern of outcomes. Whereas Hofman et al. (1995) found a decrease in mean heart rate, Wilkinson and Campbell (1984) found a statistically significant increase in mean heart rate associated with a reduction in indoor traffic noise exposure during sleep. Neither Wilkinson and Campbell, nor Vallet et al. 0167-8760/$ – see front matter © 2008 Elsevier B.V. All rights reserved.

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found a significant difference in mean heart rate variability (HRV) as a result of changed noise exposure levels. A limitation of these early field study designs was that room acoustics and subject-related conditions were changed to alter the noise condition and that the subjects were aware of the implications of these changes. A further limitation was the lack of specificity in describing cardiac autonomic tone (i.e. cardiac sympathetic and parasympathetic tone were not separately determined), which may in part account for the contrasting past results.

Heart rate as a metric is insufficient when analyzing cardiac autonomic control as it is regulated by both the cardiac sympathetic and parasympathetic branches of the ANS. Likewise, HRV is influenced by both the cardiac sympathetic and parasympathetic branches, although the (relative) contribution of each may be discerned by using appropriate measures (i.e. time domain methods or power spectral density analysis) (Taskforce, 1996). The most prominent component of HRV at rest occurs within the respiration frequency band (i.e. high frequency, HF), at approximately 0.15–0.4 Hz, (Bernston et al., 1997). Heart rate variability within the high frequency band is also known as respiratory sinus arrhythmia (RSA) and has been found to be sensitive to muscarinic receptor blockade (Martimkani et al., 2006). Thus, RSA is considered an index of cardiac parasympathetic tone (Taskforce, 1996; Bernston et al., 1997). In addition, the isovolumic contractile phase time-interval of ventricular systole, also known as pre-ejection period (PEP), is inversely related to beta-adrenergic receptor activity and myocardial contractility, and is widely accepted as a non-invasive index of cardiac sympathetic tone (Sherwood et al., 1990; Brownley et al., 2000).

The present study incorporated separate indices of both cardiac sympathetic tone and cardiac parasympathetic tone, namely PEP and RSA. During the analysis the effects of personal attributes (e.g. age, gender, body-mass index) and noise characteristics (e.g. type of noise source) were assessed, and if relevant, statistically controlled for. Also, during the analysis of RSA, respiration rate was statistically controlled for since respiration rate is a known confounder of the relationship between RSA and cardiac parasympathetic tone (Grossman et al., 1991; Grossman and Taylor, 2007). Slow-wave sleep (SWS) is most during the analysis of sleep period time, compared to during the first half of the sleep period, whereas largest during stage 2 and REM sleep (Griefahn et al., 2008). Thus, an increase in cardiac sympathetic tone (decrease in RSA) and a decrease in cardiac parasympathetic tone (decrease in RSA) also, the impact of road and rail traffic noise was expected to be larger during the second half of the sleep period, compared to during the first half of the sleep period.

2. Materials and methods

2.1. Subjects

The sample consisted of 36 subjects (17 men, 19 women; aged 18–62 years) from seven residential areas of cities and villages (i.e. ‘locations’) in the Netherlands (see Table 1). The participants formed a sub-sample of a larger study sample consisting of 262 subjects (Passchier-Vermeer et al., 2007a,b). Participants of this larger study were recruited by way of leaflet sent to approximately 2600 addresses within twelve residential locations inviting residents to take part in the study. Selection of residential areas was based on information gathered from on-site inspection, and from local traffic and environment databases. Locations where residents were exposed to a single source of either road or rail traffic noise were selected. Although the overall response rate was roughly 7%, a non-response analysis did not reveal selection bias.

From seven of the locations, participants who were not excluded on the basis of the following criteria were invited to take part in the sub-study: being pregnant, currently receiving medication for cardiovascular conditions or sedatives (such as beta-blockers or benzodiazepines), or having a known history of cardiovascular disease or having received treatment for cardiovascular-related conditions. Selection of these seven locations was based purely on logistical and budgetary restrictions within the study. Also, all individuals aged 65 years or above were excluded from participating in the sub-study as the chance of undiagnosed cardiovascular-related conditions, when taking into account the elevated cardiovascular risk associated with advancing age (e.g. Snidman et al., 2007) especially after the age of 65 (e.g. Lakatta, 2002), is considerable. Consequently, 38 individuals volunteered to take part in the sub-study. Of these, 36 participants took part and completed the sub-study (i.e. two individuals withdrew from the sub-study prior to completion; their data were not taken into further consideration). All participants of the sub-study gave written informed consent and were rewarded with €100 (roughly US $130) on completion. Permission to conduct the study was given by the Medical Ethics Committee of the University Medical Center of Utrecht.

2.2. Traffic noise exposure

Of the seven study locations used during the present study, three locations were situated in the vicinity of busy roads (i.e. ‘road traffic locations’); one location was near a motorway, one was near an urban access road, and the other was near a main provincial road. Traffic

| Table 1

| Traffic noise exposure | Indoor noise exposure | (calculated from the averaged values per subject) of the physiological parameters, indoor traffic noise exposure level metric across the entire sleep period time, age and body-mass index given for each combination of traffic location (i.e. source) and gender. |
|---|---|---|---|---|---|---|
| Age | BMI | PEP | RSA | logRSA | RR | L
| ns | ns | ns | ns | ns | ns | ns |
| 18.8 | 28.2 | 16.4 | 10.7 | 0.0 | 0.0 | 0.0 |
| 108.7 | 58.1 | 1.72 | 14.7 | 23.4 | 41.4 | 24.9 |
| 10.4 | 100.5 | 0.18 | 2.8 | 5.8 | 11.1 | 4.1 |
| 12.1 | 60.0 | 0.31 | 1.8 | 11.0 | 13.8 | 4.1 |
| 9.7 | 45.7 | 0.65 | 18.2 | 25.6 | 38.3 | 28.4 |
| 12.3 | 52.2 | 0.23 | 2.5 | 7.4 | 13.0 | 28.4 |
| 13.5 | 32.3 | 0.23 | 2.5 | 7.4 | 13.0 | 28.4 |

Note: PEP = pre-ejection period, (ms); RSA = respiratory sinus arrhythmia, (ms); logRSA = base-10 logarithm transformed RSA, (log_{10}(ms + 1)); RR = respiration rate, (breaths/minute); L = indoor traffic noise exposure during the sleep period time, (dB(A)); Age = years; BMI = body-mass index, (kg/m²). Significance of equality of means t-tests:
- \( p \leq .10; \) \( p \leq .01; \) \(^{**}\) = not significant.
- \( * \) comparing means between males vs. females; \( * \) comparing means between road vs. rail traffic locations; \( "p \leq .10; \) \( "p \leq .01; \) \(^{**}\) = not significant.
noise exposure along the motorway showed little fluctuation, with the individual passages generally not being clearly audible. In contrast, at the other road traffic locations individual passages were clearly audible (lasting between 10 and 20 s). At the remaining four study locations, night-time traffic noise was mainly caused by railway traffic (i.e. ‘rail traffic locations’), including both passenger and freight trains. The traffic noise exposure at these locations was characterized by passages varying from 10 s to more than 1 min (in 2.5% of cases) in length.

Night-time noise measurements were made at each location using one outdoor noise monitor (Larson Davis, model 870) and up to twelve indoor noise monitors (Larson Davis, model 820) depending on the number of participants per location. Per location, the outdoor noise monitor was positioned in the vicinity of the busy road or railway track, whereas a single indoor noise monitor was positioned inside the bedroom of each participating subject (i.e. bedroom site). Each noise monitor continuously recorded the A-weighted sound level per second between 22:00 and 09:00 h during each study night, whereby sounds at different frequencies were weighted according to the A-weighting method, which is a representation of the frequency sensitivity of human hearing (ISO, 2003, 2006).

The most widely used noise metric, i.e. the ‘average’ noise level (ISO, 2003, 2006), was used to characterize the subjects’ traffic noise exposures within the bedroom (i.e. indoor) during sleep. Here it is called the ‘indoor traffic noise exposure level during the sleep period time’ ($L_{SPT}$) to indicate that it is assessed for each individual sleep period time. Because the sound levels of traffic passages were most accurately determined at the outdoor meter, $L_{SPT}$ (in the bedroom) was assessed by subtracting the ‘outdoor–indoor difference’ ($D$) from the traffic noise level measured outdoors during the sleep period ($L_{	ext{outdoor,SPT}}$):

$$L_{SPT} = L_{	ext{outdoor,SPT}} - D.$$  

Per bedroom, the ‘outdoor–indoor difference’ ($D$) was determined as follows. The mean value of the difference between the maximum outdoor sound level ($L_{	ext{outdoor,max}}$) and the maximum indoor sound level ($L_{	ext{indoor,max}}$) emitted by the 100 loudest noise events during the otherwise quietest period of the night was taken as the bedroom site-specific ‘outdoor–indoor difference’ ($D$):

$$D = \frac{1}{100} \cdot \sum_{i=1}^{100} \left( L_{\text{outdoor,max}} - L_{\text{indoor,max}} \right)_i.$$  

In addition to indoor traffic noise exposure metrics ($L_{SPT}$ and $L_H$), which characterize the traffic noise exposure within the bedroom during (half) the sleep period, a variable (source) that coded the type of traffic noise location (i.e. road or rail) was used to investigate the possible influence of noise source type on cardiac autonomic nervous system tone.

### 2.3 Physiological recordings

Both thoracic impedance cardiography (ICG) and electrocardiography (ECG) were recorded using the Vrije Universiteit Ambulatory Monitoring System (VU-AMS, version 4.6); a system for which good cross-instrument comparison with standard laboratory set-ups has been found (De Geus et al., 1995). Implementation of the system and processing of the biosignals were in accordance with earlier described procedures (see: De Geus et al., 1995; De Geus and van Doornen, 1996; Willemsen et al., 1996). Briefly, this system uses a six electrode configuration to record both ECG (via three electrodes) and ICG (via four electrodes). Disposable, pre-gelled Ag/AgCl electrodes (ULTRA-TRACE, CONMED Corp., Utica, NY, USA) were used. The ECG signal is amplified and fed through a 17 Hz band-pass filter. The R-wave is recognized by a level detector with automatic level adjustment, and a millisecond counter read and reset at each R-wave peak to record the sequential R-wave to R-wave time intervals to extract the raw inter-beat-interval (IBI) data in milliseconds (ms). The rest of the ECG signal is discarded. Following amplification and precision rectification, the ICG signal is filtered at 72 Hz (low pass) to obtain the basic thoracic impedance ($Z_0$) signal, which is then filtered at 0.1 Hz (high pass) to obtain the dZ signal. To obtain the respiration signal, the dZ signal is sampled at 100 s and band-pass filtered between 0.1 and 0.4 Hz. To identify the variation in thoracic impedance associated with cardiac systole the dZ is filtered at 30 Hz to obtain the dZ/dr signal.

To calculate PEP, the dZ/dr signal was sampled at 250 Hz for a period of 512 ms around each R-wave, following which the corresponding segments were ensemble averaged across consecutive 30 second periods. All 30 s ensembles within single hour periods starting from sleep-onset were then further aggregated by large scale ensemble averaging to obtain single ICG complexes per hour of each sleep period (for details and validity concerning large scale ensemble averaging see Riese et al., 2003). Automatically detected B-points (onset of dZ/dr upstroke) of all large scale ICG complexes were visually inspected and, if necessary, manually corrected according to the guidelines proposed by Sherwood et al. (1990). Finally, PEP was calculated, in milliseconds, as the time interval between the R-wave...
and B-point, plus a constant 48 ms for the Q-wave to R-wave interval (De Geus and van Doornen, 1996; Willemsen et al., 1996).

From the respiration (dZ) signal, the VU-AMS software automatically detects both the inspiration period (IP) and expiration period (EP) of each breath (both in milliseconds, ms), and displays the respiration signal along with the continuous IBI signal for visual inspection. Respiration rate (RR, in breaths/minute) was calculated per cycle as: \( RR = \frac{60,000 \times (IP + EP)}{\text{cycle}} \). The respiration period (i.e. IP + EP) of each breath was compared to a running average respiration period of the preceding 20 breaths. All breaths that were 50% longer or shorter than the running average were automatically rejected, and visually verified. Similarly, the relative threshold to detect breaths was automatically calibrated, after which the amplitude of the dZ signal of each breath was visually inspected. All breaths with an amplitude <0.05 Ω with respect to the relative threshold were rejected (i.e. limited thorax movement during respiration). Sections of data displaying upper limb movement artefacts were removed manually. Using the “Peak-to-Trough” time-domain method, RSA (per breath) was calculated as the time difference, in milliseconds, between the longest IBI during heart rate deceleration during expiration minus the shortest IBI during heart rate acceleration during inspiration (De Geus et al., 1995; Bernstam et al., 1997). The Peak-to-Trough method provides RSA estimates that are highly correlated to RSA estimates obtained by the frequency-domain methods (Grossman et al., 1990). Their correlation is also known to remain stable across time, ambulatory conditions, and a wide range of resting heart rate and respiration rate ranges (Goedhart et al., 2007).

### 2.4. Procedure

The subjects took part in the study for six consecutive nights (i.e. Wednesday to Monday). In advance of participation they were informed of the purpose and set-up of the study and asked to fill in a questionnaire. The following data per subject were derived from the questionnaire: age (age, continuous: years), gender (gender, dichotomous: female(0), male(1)), highest attained level of education (education, number of (prescription) medications currently taken (medication, continuous), hearing impairment (hearing impairment, dichotomous: none(0), at least some(1)), self-reported noise sensitivity (sensitivity, combined scale: not(0)–extremely(110), Weinstein, 1978), and the number of years living in the current residence (resident years, continuous). Subjects indicated their height and weight, so that body-mass index (BMI, continuous: in kg/m²) could be calculated (because of missing data, the height and weight of three subjects were estimated by the fieldworkers). Fieldworkers visited each subject at home during the day-time prior to the first study night to set up all necessary equipment (i.e. noise monitors, VU-AMS). For each subject, the skin at the spot-electrode sites was first rubbed clean with alcohol and any excessive chest hair was shaven, where after the fieldworkers placed the electrodes and attached the VU-AMS measuring device. The signal quality was verified by online monitoring using a laptop PC to ascertain whether the basic thoracic impedance (Zth) remained within a range of 5–15 Ω, whether the respiratory impedance signal clearly responded to the subject’s breathing pattern and remained within a range of approximately dZ ± 0.5 Ω, and whether the ECG R-wave peaks were detected. Poor signal quality was improved by replacement and repositioning of one or more electrodes. The correct position of each electrode and proper operation of the measuring device was carefully demonstrated to the subjects by the fieldworkers. Each subject was also given a diagram indicating the correct electrode sites and the lead wire attachment scheme, in addition to sufficient replacement spot-electrodes in case any electrodes needed replacing. In addition each subject wore an actimeter (type AW4, Cambridge Neurotechnology Ltd, UK) on the wrist of their non-dominant arm so that upper limb movement could be registered (the analysis of actimetry data will be part of a separate report). The subjects were given instructions to a diary during the study, and were informed whom to contact in case of a problem with the equipment or in an emergency. The following data per sleep period were derived from the diary: the daily bedtimes (in-bed/out-of-bed times), the number of cigarettes smoked after 20:00 h (cigarettes, continuous), the number of alcohol or caffeine containing beverages consumed after 20:00 h (alcohol, continuous: 1 unit equal to 10 mg of alcohol per beverage; and caffeine, continuous: 1 unit equal to 85 mg per 125 ml), use of sedatives (sedatives, dichotomous: no(0), yes(1)), and earplugs (earplugs, dichotomous: no(0), yes(1)), self-reported hindrance due to the measuring device or its electrodes during sleep (hindrance, dichotomous: none(0), at least some(1)), and whether electrodes had come loose or had been replaced (loose/replaced, dichotomous: no(0), yes(1)).

Once familiarized with the VU-AMS equipment the subjects were asked to keep to their usual sleep behavior and bedtimes. The subjects slept with the VU-AMS measuring device either lying on their bedside table or under their pillow, and with the actimeter attached to their wrist. After getting up in the morning subjects detached lead wires and the measuring device, and were permitted to remove all electrodes if they so wished, provided that they noted this in their diary. Halfway through the study (during the day prior to night 4) each subject was checked upon, thereby providing the opportunity for fresh electrodes to be positioned by the fieldworkers and the signal quality to be verified once more, in addition to downloading the recorded data and replacing the measuring device’s battery. Finally, following completion of the study (night 6), the fieldworkers again visited the subjects’ homes to collect the recorded data, the equipment and diaries.

### 2.5. Data reduction

The actimeter data together with the diary time entries were used to determine both sleep-onset and awake-onset times per sleep period, from which the sleep period time (SPT) was calculated. Only data recorded during a sleep period time or for a maximum of the first 10 h per sleep period were selected for further analysis. The minimum amount of recorded ICG and ECG data within each hour per sleep period required for analysis was taken as 5 min. To avoid bias, PEP and RSA were derived without prior knowledge of the traffic noise exposure levels. The IBI, IP and EP (in ms) from each subject were individually inspected and selected so that only physiologically plausible data remained (i.e. IBI range: 250 s ≤ 1800; IP range: 300 s ≤ 9000; and EP range: 300 s ≤ 10,000). Also, to exclude potential outlying values, the highest 1% RSA (per breath) values were removed.

The RSA and RR per breath data were first aggregated per hour. Subsequently, the PEP, RSA, and RR data were aggregated per sleep period time, yielding the mean values for PEP, RSA, and RR per sleep period time (i.e. PEPspt, RSAspt, RRspt). Similarly, PEP, RSA and RR (per hour) data were aggregated across half of the sleep period time (H), thereby yielding mean values for PEP, RSA, and RR for each half of the sleep period time (i.e. PEPspt, RSAspt, RRspt). Across all subjects and all sleep periods there were 1417 mean PEP per hour values (i.e. hour means), and 1392 RSA hour means within the dataset. Of these hour means, 1.3% and 2.9% were based on between 5 and 10 min worth of valid PEP and RSA data, respectively. In total there were 188 PEPspt values (i.e. sleep period means) and 192 RSAspt values in the dataset, with a minimum of a one hour mean per sleep period mean. In all, only one PEPspt value and three RSAspt values were derived from a single hour mean. Approximately 5.9% of the PEPspt values and 8.3% of the RSAspt values were derived from less than five hour means.

Individual means (i.e. mean values per subject) of the day-to-day characteristics found to lack within-subjects variation (i.e. alcohol, caffeine, and cigarettes) were used during the analyses. Variables...
found to have a very low prevalence (<1%, i.e. earplug and sedatives) were rejected from all further analyses. The education variable was dichotomized so as to distinguish between levels below and above the first stage of tertiary education, i.e. low education: below college or university level (< ISCED-97 level 5A); high education: at least college or university level (≥ ISCED-97 level 5A; UNESCO, 1997).

2.6. Statistical analysis

The distributions of all of the PEP and RSA data were inspected visually, followed by calculation of the degree of skewness, kurtosis and the Shapiro–Wilk statistic. Non-normally distributed variables were numerically transformed and the distribution investigated again. Multilevel linear regression analysis with random effect factors was performed using SPSS 15.0 (SPSS Inc, Chicago, IL, USA). Covariates were pre-selected via separate regression analyses. Covariates of each final model were identified via backward elimination of the pre-selected covariates. Two analyses were conducted. The first analysis investigated mean cardiac autonomic nervous system tone during the sleep period (PEPSPT and RSASPT) in relation to the indoor traffic noise exposure during the sleep period (LSPR). The second analysis compared the relationship between cardiac autonomic nervous system tone (PEPSPT and RSASPT) and indoor traffic noise exposure (LSPR) during sleep between the first and second halves of the sleep period.

2.6.1. Across the entire sleep period time

Basic models were set-up with either PEPSPT or RSASPT as the dependent variable and logRSA as a fixed effect factor. A random effect factor for subject was entered into both basic models. RRH was also entered into the basic RSASPT model as a fixed effect factor. Covariates for each model were pre-selected from the following pool of variables: gender, age, BMI, education, resident years, hearing impairment, sensitivity, medication, source, caffeine, alcohol, cigarettes, hindrance, and loose/replaced. Each of these variables was individually entered into the basic PEPSPT and RSASPT models, and only variables that contributed with p < .10 were taken into further consideration. For PEPSPT only the variable source was pre-selected, whereas for RSASPT the variables age, hindrance and resident years were pre-selected. Subsequently, a backward elimination procedure was employed to derive the final model (elimination criterion per step of procedure: p_0 > .05; applicable to covariates only).

2.6.2. 1st vs. 2nd half of sleep period time

The analysis procedure used to compare the first and second halves of the sleep period time was similar to the analysis across the entire sleep period time. Basic models were set-up with PEPSPT or RSASPT as the dependent variable and logRSA as a fixed effect factor. Random effect factors for night and subject were entered into both models, with RRH entered into the RSASPT basic model as a fixed effect factor. A variable (H) distinguishing between the first (H = 0) and second (H = 1) halves of the sleep period time and its interaction term with LSPR (i.e. H * LSPR) were entered as fixed effect factors into both basic models. Covariates for each model were selected by the same procedure and from the same pool of variables as described above. Again, only source was entered into the backward elimination procedure for PEPSPT whereas age, hindrance and resident years were entered for RSASPT.

2.6.3. Post hoc analyses

A number of post hoc analyses were carried out once the final models were derived. The influence of the total indoor noise exposure were investigated by temporarily substituting the indoor traffic noise exposure metric within a final model (i.e. LSPR or LSPR) with the corresponding total indoor noise exposure metric (i.e. LoutdoorSPT or LoutdoorSPT respectively). This procedure was repeated to investigate the influence of the outdoor noise exposure (i.e. temporarily substituting LSPR and LSPR with LoutdoorSPT and LoutdoorSPT respectively). A further post hoc analysis, aimed at investigating the possibility of a first-night effect, was conducted by adding a variable coding the first study night (i.e. night1, coded as: night1 = 1, nights 2 through 6 = 0) and its interaction term with the indoor traffic noise exposure metric (either LSPR or LSPR) to a final model.

3. Results

3.1. General

Respiratory sinus arrhythmia data showed high positive skewness, and was therefore log-transformed (i.e. logRSA = log10(x + 1), with x = RSA in ms) to obtain a better estimation of a normal distribution. Pre-ejection period data showed no significant visual skewness or kurtosis, however the Shapiro–Wilks statistic was significant (p < .05), which could not be rectified by transformation. Pre-ejection period was analyzed according to the procedure described above. Table 1 displays a summary of the sample means and standard deviations (i.e. the non-weighted mean of the averaged values per subject) of the PEPSPT, RSAEP, logRSAEP, RRSPR and the indoor traffic noise exposure level during the sleep period (LSPR), along with the sample means of age and BMI for males and females at both the road and rail traffic locations. Table 2 provides a comparative overview of the sample means, standard deviations and range of the physiological (PEPH, RSAH, logRSAH, RRH) and noise exposure (LSPR, LoutdoorSPT, LoutdoorSPT) data between the first and second halves of the sleep period time.

3.2. Across entire sleep period time

Table 3 (left) provides an overview of the parameter estimates of the final PEPSPT and logRSAEP models.

The indoor traffic noise exposure level during the sleep period (LSPR) had no significant effect on PEPSPT (t_{106} = -0.99, p = .391). None of the investigated covariates was significantly related to PEPSPT.

Neither the total indoor noise level during the sleep period (LoutdoorSPT), nor the outdoor noise exposure level during the sleep period (LoutdoorSPT) was significantly related to PEPSPT.

Mean respiration rate during the sleep period (RRH) was negatively related to logRSAEP (t_{175} = -2.97, p = .003). In the final logRSAEP model adjusted for mean respiration rate, the indoor traffic noise exposure level during the sleep period (LSPR) had a significant negative effect on logRSAEP (t_{161} = -2.76, p = .006). Age was a significant covariate of logRSAEP (t_{148} = -3.69, p = .001). All other investigated covariates were not significantly related to logRSAEP.

Neither the total indoor noise exposure level during the sleep period time (LoutdoorSPT) nor the outdoor noise exposure level during the sleep period time (LoutdoorSPT) was significantly related to logRSAEP.

Table 2

Sample means, standard deviations, minimum and maximum values of the physiological parameters, (traffic) noise exposure metrics given for the first and second halves of the sleep period time.
Addition of both night1 and its interaction with LSPT to the final logRSA_{SPT} model indicated a significant effect for the main term of night1, \( t_{(142)} = -2.21, \ p = .029 \), and a non-significant interaction term between night1 and LSPT, \( t_{(141)} = 1.73, \ p = .086 \). The regression of logRSA_{SPT} on LSPT remained significant following the addition of night1 and its interaction with LSPT, \( t_{(167)} = -2.32, \ p = .022 \).

3.3. 1st vs. 2nd half of sleep period time

Table 3 (right) provides an overview of the parameter estimates of the final PEPH and logRSAH models.

The mean PEP during the second half of the sleep period did not significantly differ from the mean PEP during the first half of the sleep period, as indicated by the non-significant regression of PEPH on night1, \( t_{(199)} = 1.00, \ p = .317 \). Indoor traffic noise exposure (I_{in}) was not significantly related to PEPH during the first half of the sleep period, \( t_{(175)} = 1.20, \ p = .232 \). Similarly, I_{in} was not significantly related to PEPH during the second half of the sleep period, \( t_{(175)} = -2.73, \ p = .066 \). Neither the total indoor, nor outdoor, noise exposure levels (I_{indoor} and I_{outdoor}), respectively were significantly related to PEPH during the first or second halves of the sleep period time.

Respiratory sinus arrhythmia was significantly higher during the second half of the sleep period time compared to during the first half, as indicated by the significant regression of logRSAH on night1, \( t_{(180)} = 4.82, \ p < .001 \). Indoor traffic noise exposure (I_{in}) was not significantly related to logRSAH during the first half of the sleep period, as indicated by the regression of logRSAH on the main term for I_{in}, \( t_{(180)} = -1.74, \ p = .083 \). Indoor traffic noise exposure was however significantly related to logRSAH during the second half of the sleep period, as indicated by the regression of logRSAH on the interaction term H*LSPT, \( t_{(183)} = -3.06, \ p = .003 \).

There was no significant effect of the total indoor noise exposure level (I_{indoor}) on logRSAH, during either the first half, \( t_{(179)} = -2.83, \ p = .003 \), or second half, \( t_{(181)} = -0.79, \ p = .432 \), of the sleep period time. The outdoor noise exposure level (I_{outdoor}) was not significantly related to logRSAH during the first half of the sleep period, \( t_{(199)} = -0.4, \ p = .696 \). However, during the second half of the sleep period, \( t_{(199)} = 2.26, \ p = .028 \), although effect of the night1*LSPT interaction term was not significant, \( t_{(183)} = 1.42, \ p = .159 \). The regression of logRSAH on H*LSPT remained significant following the addition of night1 and its interaction with LSPT, \( t_{(183)} = -3.04, \ p = .003 \).

4. Discussion

Although no evidence of the impact of traffic noise on cardiac sympathetic tone during sleep was found, there is clear evidence of the hypothesized negative relationship between indoor traffic noise exposure and cardiac parasympathetic tone. Gender was not a covariate of PEP or RSA, while age was negatively related to RSA, which is consistent with earlier reported findings (e.g. Murata et al., 1992; Uchino et al., 1999). The present findings suggest that increased indoor traffic noise exposure levels averaged across the sleep period time are related to cardiac parasympathetic withdrawal during sleep, however the findings do not suggest any effect of traffic noise on cardiac sympathetic tone. The findings from a more detailed analysis suggest that the relationship between indoor traffic noise exposure with cardiac parasympathetic withdrawal during sleep exists only during the second half of the sleep period. To the authors’ knowledge, this is the first field study to indicate the potential influence of road and rail traffic noise on cardiac parasympathetic nervous system tone during sleep.

Sleep is characterized by an overall increase in parasympathetic activity and decrease in sympathetic activity in comparison to wakefulness (Gula et al., 2004; Burgess et al., 1997). The involvement of altered ANS activity in relation to low sleep quality was studied by Johns et al. (1976), who found that sleep quality was inversely related to sympathetic activity sleep. Also, increased sympathetic dominance of the sympatho-vagal balance during individual sleep periods has been suggested to play an important role in the pathway leading to low sleep quality (Hall et al., 2004) and to insomnia (Bonnet and Arand, 2003). Much of the focus of earlier studies has been on sympathetic activation as part of a mechanism of sleep disturbance and have interpreted their findings accordingly (e.g. Carter et al., 1994, 2002; Bonnet and Arand, 2003; Hall et al., 2004), although not without exception (e.g. Di Nisi et al., 1990).

A proposed function of sleep is the protection of the cardiovascular system (Di Nisi et al., 1990; Carter et al., 2002), the effects of which are presumably mediated by increased parasympathetic activity, considering that for instance increased cardiac parasympathetic tone reduces heart rate, and subsequently blood pressure (Dodd and Role, 1991; Brownley et al., 2000). Several studies (e.g. Babisch et al., 2003, 2005; De Kluizenaar et al., 2007) have provided evidence of an association between long-term 24-hour road traffic noise exposure and increased prevalence of cardiovascular-related disease, including the association between road traffic noise exposure and the increased risk of hypertension amongst middle-aged subjects (De Kluizenaar et al., 2007). Although these aforementioned studies did not exclusively investigate this relationship for night-time traffic noise exposure, their findings indicate the potential health consequences of long-term traffic noise exposure. Altered cardiac autonomic nervous system tone during sleep through exposure to traffic noise could potentially play a role in these earlier described relationships.

The relationship between the average levels of cardiac parasympathetic tone and indoor traffic noise exposure during the sleep period appears to exist only during the second half of the sleep period. During the first half of the sleep period a statistically non-significant negative trend between cardiac parasympathetic tone and indoor traffic noise exposure was observed. The mean levels of both cardiac parasympathetic tone and indoor traffic noise exposure were
higher during the second half of the sleep period compared to the first, suggesting that the reported negative relationship is unlikely to be a statistical artefact (i.e. that RSA happens to be low at a point in the sleep period during which the traffic noise levels are higher). It is more likely that at difference in sleep depth is the reason behind the different response to traffic noise during the first and second halves of the sleep period. As previously reported for autonomic responses (i.e. heart rate responses) to traffic noise events during sleep, in the absence of awakening the autonomic response during SWS is considerably smaller than during either stage 2 or REM sleep (Griefahn et al., 2008). Considering that SWS is most prominent during the early part of the sleep period (Griefahn et al., 2008), it is unsurprising that the relationship between cardiac parasympathetic tone and indoor traffic noise exposure differs between the first and second halves of the sleep period.

No influence of traffic noise on cardiac sympathetic tone activity during the sleep period was found which is surprising in light of earlier findings (e.g. Carter et al., 1994, 2002). There may be a number of explanations for this.

First, by allowing the subjects to reattach or replace loose electrodes, variation in how often new electrodes were applied and in the exact positioning of the electrodes occurred. Skin irritation experienced by a number of subjects may have contributed to this variation. Reduced electrode adhesion over time and incorrect positioning of the electrodes may have caused signal noise, which could have negatively affected the derivation of PEP. As measurement of change in thoracic impedance due to respiration is assumed to be less susceptible to minor inaccuracies in electrode positioning, the expected impact on the derivation of RSA is much smaller. However, self-reported occurrence of electrodes coming loose during sleep or being replaced was not related to either PEP or logRSA, nor was the relationship between traffic noise exposure level and logRSA affected. It appears therefore reasonable to assume that signal noise and signal losses associated with loss of electrode adhesion and replacement of electrodes was not significantly related to PEP or logRSA.

Second, the response of the sympathetic autonomic nervous system to traffic noise exposure during sleep may occur within a time frame much shorter than investigated here, as show for heart rate response by Griefahn et al. (2008). The fact that no evidence of a relationship between traffic noise level and logRSA was not statistically significant suggesting that the relationship between cardiac parasympathetic activity and indoor traffic noise exposure levels during sleep was not significantly different during the first study night compared to during the other study nights. The present findings do not indicate a significant effect of noise source (i.e. road or rail traffic) on cardiac sympathetic or parasympathetic tone. The pattern of rail traffic noise exposure, generally characterized by intermittent loud traffic passages followed by periods of relative quiet, may vary considerably from road traffic noise. Indeed, earlier findings suggest that aspects of traffic noise, other than the noise levels, may be relevant. For instance, the percentages of highly noise annoyed and highly sleep disturbed individuals within urban areas due to traffic noise are larger for road traffic noise sources than for rail traffic noise sources at equal noise levels (Miedema and Oudshoorn, 2001; Miedema and Vos, 2007). A similar distinction in the response of the autonomic nervous system to noise from different traffic sources may exist, although the present results, as well as earlier findings (e.g. Carter et al., 2002), do not suggest this.

4.1. Limitations

The present study design brings with it several limitations. First, inherent of most field study designs, not all sources of variation could be fully controlled or accounted for. For instance, the participants' activities during the daytime were not taken into account, and although extensive effort was taken to assess the times of sleep-onset and awakening in the morning, intermediate awakenings were not taken into account. Also, adherence to the study procedure and to the positioning of electrodes was largely unsupervised, although ICG and ECG signal quality was verified on two separate occasions per subject. A further limitation was that, as sleep stages were not distinguished, the physiological effects of traffic noise described here were not corrected for sleep stage. Although in interpreting RSA respiration rate was statistically controlled for, other confounding factors of RSA, such as tidal volume (Grossman et al., 1991), were not taken into account. Also, PEP and RSA refer to cardiac autonomic nervous system activity, and do not necessarily generalize to other aspects of sympathetic or parasympathetic activation. While the study sample was relatively large in comparison to those of the more frequent laboratory-based sleep studies, in epidemiological terms the sample was small.

Despite the lack of control over several study conditions and accountability of all variation, the present field study design and study conditions enhance the ecological validity of the results, as these reflect the actual response to real-life road and rail traffic noise exposure during the sleep period.

5. Conclusions

The present field study findings indicate the potential influence of road and rail traffic noise on cardiac parasympathetic tone during sleep, specifically during the second half of the sleep period. No evidence of an effect of traffic noise on cardiac sympathetic tone was found. Further research is required to verify these results, and to study
the long-term consequences of noise-induced cardiac parasympathetic withdrawal on health.

Acknowledgements

This study was financed by the Netherlands Ministry of Housing, Spatial Planning and the Environment (VROM) and commissioned by the National Institute for Public Health and the Environment (RIVM).

The authors kindly thank Dr. Winni Hofman of the Department of Psychology of the University of Amsterdam (UvA) for her helpful comments and advice during this study.

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