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Habitual traffic noise at home reduces cardiac parasympathetic tone during sleep

Jamie M.A. Graham *, Sabine A. Janssen, Henk Vos, Henk M.E. Miedema

Department of Environment and Health, TNO Netherlands Organisation for Applied Scientific Research, Delft, The Netherlands

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ABSTRACT

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 covariates 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.

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 night-time 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 Role, 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.

* Corresponding author. Environment and Health, TNO Netherlands Organisation for Applied Scientific Research, P.O. Box 49, 2600AA, Delft, The Netherlands. Tel.: +31 15 269 7305; fax: +31 15 276 3621.
E-mail address: jamie.graham@tno.nl (J.M.A. Graham).

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(1983) 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, (Berntson 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 blockage (Martinmaki et al., 2006). Thus, *RSA* is considered an index of cardiac parasympathetic tone (Taskforce, 1996; Berntson 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 prominent during primarily the first part of the night (Griefahn et al., 2008) and previous laboratory findings indicate that in the absence of awakenings the magnitude of autonomic responses (such as heart rate responses) to simulated traffic noise during sleep is smallest during SWS and largest during stage 2 and REM sleep (Griefahn et al., 2008). Therefore, a comparison of the relationship between cardiac sympathetic and parasympathetic tone with indoor traffic noise exposure during the second half of the sleep period compared to during the first half of the sleep period was made. Subjects were studied in their usual sleep conditions and were exposed to their habitual noise levels. The hypothesized impact of road and rail traffic noise during sleep is an increase in cardiac sympathetic tone (decrease in *PEP*) 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. Sniderman 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
Sample means and standard deviations (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*.

			Road traffic locations				Rail traffic locations				All locations	
			Male ($n=12$)		Female ($n=10$)		Male ($n=5$)		Female ($n=9$)		$N=36$	
	[1]	[2]	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*	*M*	*SD*
PEP_{SPT}	ns	†	108.7	16.4	107.0	10.5	104.1	12.1	97.9	12.3	104.9	13.5
RSA_{SPT}	ns	ns	58.1	29.5	39.0	16.7	50.5	35.5	60.0	45.7	52.2	32.3
$logRSA_{SPT}$	ns	ns	1.72	0.22	1.57	0.18	1.59	0.36	1.68	0.31	1.65	0.25
RR_{SPT}	ns	*	14.7	1.8	16.0	2.8	19.1	2.0	16.9	1.8	16.2	2.5
L_{SPT}	ns	ns	23.4	4.5	24.9	5.8	28.4	8.6	27.8	11.0	25.6	7.4
Age	ns	ns	41.4	13.1	34.7	11.1	42.0	15.8	36.0	13.8	38.3	13.0
BMI	ns	ns	24.9	2.1	24.4	4.1	26.8	3.2	24.0	4.1	24.8	3.4

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_{SPT} = indoor traffic noise exposure during the sleep period time, [dB(A)]; *Age* = years; *BMI* = body-mass index, (kg/m^2). Significance of equality of means *t*-tests: [1]comparing means between males vs. females; [2]comparing means between road vs. rail traffic locations; †$p \leq .10$; *$p \leq .01$; ^{ns} = 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 ($L_{\text{outdoor},SPT}$):

$$L_{SPT} = L_{\text{outdoor},SPT} - D. \quad (1)$$

Per bedroom, the 'outdoor–indoor difference' (D) was determined as follows. The mean value of the difference between the maximum outdoor sound level ($L_{\text{outdoor, max}}$) and the maximum indoor sound level ($L_{\text{indoor, max}}$) 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):

$$D = \frac{1}{100} \cdot \sum_{i=1}^{100} \left(L_{\text{outdoor,max}}\right)_i - \left(L_{\text{indoor,max}}\right)_i, \quad (2)$$

with $i = 1, 2, 3, \ldots, 100$, e.g. the 100 loudest traffic noise events detected on both the outdoor and indoor noise monitors at a bedroom site. The recorded indoor noise levels during these events were compared to an expected noise distribution of a typical situation indoors with traffic noise. If the observed distribution deviated from the expected distribution, the recorded data were not used to calculate D, in order to avoid contamination of the estimate of D with noise emitted indoors by sources other than the traffic noise source. The sample mean ($N = 36$) and standard deviation of D was 31.8 ± 7.9 dB (A). Cumulative distributions of the indoor traffic noise exposure level (L_{SPT}), the total (i.e. all noises) indoor noise exposure level ($L_{\text{indoor},SPT}$) as determined from the measurements made by the indoor noise monitors, and the outdoor noise exposure level ($L_{\text{outdoor},SPT}$), during the sleep period time are given in Fig. 1.

Noise metrics that express the noise exposure level during the first and second halves of each sleep period time were derived by the same method used to derive the sleep period time noise metrics (i.e. L_{SPT}, $L_{\text{indoor},SPT}$, $L_{\text{outdoor},SPT}$). These metrics are the indoor traffic noise exposure level (L_H), the total indoor noise exposure level ($L_{\text{indoor},H}$), and the outdoor noise exposure level ($L_{\text{outdoor},H}$) during half the sleep period time.

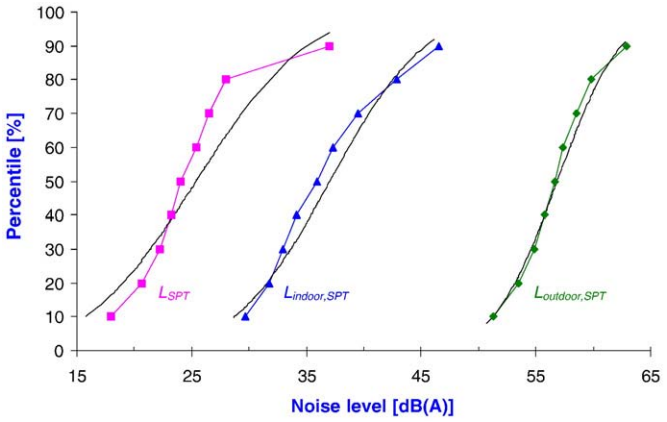

Fig. 1. Cumulative distributions of the indoor traffic noise exposure level (L_{SPT}), the total (i.e. all noises) indoor noise exposure level ($L_{\text{indoor},SPT}$), and the outdoor noise exposure level ($L_{\text{outdoor},SPT}$) expressed as percentiles within the 10–90% range of each distribution (data points with lines). Solid lines represent reference lines based on cumulative normal distributions. Displayed data are based on all valid data available per sleep period.

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/dt signal.

To calculate *PEP*, the dZ/dt 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/dt 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 (d*Z*) 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: $RR = 60{,}000 * (IP + EP)^{-1}$. 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 d*Z* 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; Berntson 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/m^2) 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 (Z_0) 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 d$Z \pm 0.5$ 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* data (in ms) from each subject were individually inspected and selected so that only physiologically plausible data remained (i.e. *IBI* range: $250 \leq x \leq 1800$; *IP* range: $300 \leq x \leq 9000$; and *EP* range: $300 \leq x \leq 10{,}000$). 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. PEP_{SPT}, RSA_{SPT}, RR_{SPT}). 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 the sleep period time (i.e. PEP_H, RSA_H, RR_H). 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 PEP_{SPT} values (i.e. sleep period means) and 192 RSA_{SPT} values in the dataset, with a minimum of a one hour mean per sleep period mean. In all, only one PEP_{SPT} value and three RSA_{SPT} values were derived from a single hour mean. Approximately 5.9% of the PEP_{SPT} values and 8.3% of the RSA_{SPT} 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 time (PEP_{SPT} and RSA_{SPT}) in relation to the indoor traffic noise exposure during the sleep period (L_{SPT}). The second analysis compared the relationship between cardiac autonomic nervous system tone (PEP_H and RSA_H) and indoor traffic noise exposure (L_H) during sleep between the first and second halves of the sleep period time.

2.6.1. Across the entire sleep period time

Basic models were set-up with either PEP_{SPT} or RSA_{SPT} as the dependent variable and L_{SPT} as a fixed effect factor. A random effect factor for *subject* was entered into both basic models. RR_{SPT} was also entered into the basic RSA_{SPT} 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 PEP_{SPT} and RSA_{SPT} models, and only variables that contributed with $p \leq .10$ were taken into further consideration. For PEP_{SPT} only the variable *source* was pre-selected, whereas for RSA_{SPT} 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: $p_{out} > .05$; 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 PEP_H or RSA_H as the dependent variable and L_H as a fixed effect factor. Random effect factors for *night* and *subject* were entered into both models, with RR_H entered into the RSA_H 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 L_H (i.e. $H * L_H$) were entered as fixed effect factors into both basic models. Covariates for each final 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 PEP_H, whereas *age*, *hindrance* and *resident years* were entered for RSA_H.

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. L_{SPT} or L_H) with the corresponding total indoor noise exposure metric (i.e. $L_{indoor,SPT}$ or $L_{indoor,H}$, respectively). This procedure was repeated to investigate the influence of the outdoor noise exposure (i.e. temporarily substituting L_{SPT} and L_H with $L_{outdoor,SPT}$ and $L_{outdoor,H}$, 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: night 1 = 1, nights 2 through 6 = 0) and its interaction term with the indoor traffic noise exposure metric (either L_{SPT} or L_H) to a final model.

3. Results

3.1. General

Respiratory sinus arrhythmia data showed high positive skewness, and was therefore log-transformed (i.e. $logRSA = \log_{10}(x+1)$, 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–Wilk 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 PEP_{SPT}, RSA_{SPT}, $logRSA_{SPT}$, RR_{SPT} and the indoor traffic noise exposure level during the sleep period (L_{SPT}), 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 (PEP_H, RSA_H, $logRSA_H$, RR_H) and noise exposure (L_H, $L_{indoor,H}$, $L_{outdoor,H}$) 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 PEP_{SPT} and $logRSA_{SPT}$ models.

The indoor traffic noise exposure level during the sleep period (L_{SPT}) had no significant effect on PEP_{SPT}, $t_{(99)} = -.09$, $p = .931$. None of the investigated covariates was significantly related to PEP_{SPT}. Neither the total indoor noise level during the sleep period ($L_{indoor,SPT}$), nor the outdoor noise exposure level during the sleep period ($L_{outdoor,SPT}$) was significantly related to PEP_{SPT}.

Mean respiration rate during the sleep period (RR_{SPT}) was negatively related to $logRSA_{SPT}$, $t_{(175)} = -2.97$, $p = .003$. In the final $logRSA_{SPT}$ model adjusted for mean respiration rate, the indoor traffic noise exposure level during the sleep period time (L_{SPT}) had a significant negative effect on $logRSA_{SPT}$, $t_{(168)} = -2.76$, $p = .006$. *Age* was a significant covariate of $logRSA_{SPT}$, $t_{(34)} = -3.69$, $p = .001$. All other investigated covariates were not significantly related to $logRSA_{SPT}$. Neither the total indoor noise exposure level during the sleep period time ($L_{indoor,SPT}$) nor the outdoor noise exposure level during the sleep period time ($L_{outdoor,SPT}$) was significantly related to $logRSA_{SPT}$, $t_{(171)} = -1.89$, $p = .061$; $t_{(161)} = -1.76$, $p = .081$, respectively.

Table 2
Sample means, standard deviations, minimum and maximum values of the physiological parameters, (traffic) noise exposure metrics given for the first half and second halves of the sleep period time.

		1st half of *SPT* ($H = 0$)				2nd half of *SPT* ($H = 1$)			
	[1]	*M*	*SD*	*Min*	*Max*	*M*	*SD*	*Min*	*Max*
PEP_H	*ns*	104.5	13.4	74.4	132.7	105.0	14.3	74.2	137.7
RSA_H	*ns*	49.1	30.5	9.5	151.8	55.1	35.9	10.7	166.1
$logRSA_H$	*ns*	1.62	0.25	1.02	2.18	1.67	0.26	1.07	2.21
RR_H	*ns*	16.3	2.6	12.3	21.9	16.1	2.4	12.9	21.6
L_H[a]	†	23.9	8.1	9.3	50.8	27.0	7.0	13.9	49.0
$L_{indoor,H}$[b]	*ns*	37.9	7.2	26.7	55.2	38.9	5.9	29.1	49.8
$L_{outdoor,H}$[c]	*	55.7	3.9	46.1	64.3	58.8	4.1	51.6	68.3

Note: $N = 36$. Significance of equality of means *t*-tests: [1]comparing means between *H1* vs. *H2*; †$p \leq .10$; *$p \leq .01$; *ns* = not significant.
[a] Indoor traffic noise exposure level during half of the sleep period time (L_H).
[b] Total (all noise) indoor noise exposure level during half of the *SPT* ($L_{indoor,H}$).
[c] Outdoor noise exposure level during half of the *SPT* ($L_{outdoor,H}$).

Table 3
Results of the final *PEP* and *logRSA* models (unstandardized regression coefficients and standard errors) from the analysis across the entire sleep period time (left), and the analysis comparing the 1st vs. 2nd half of the sleep period time (right), based on data from all 36 subjects.

	Entire *SPT*				1st vs. 2nd half of *SPT*			
	PEP[a]		*logRSA*[b]		*PEP*[a]		*logRSA*[b]	
	B	*SE*	*B*	*SE*	*B*	*SE*	*B*	*SE*
Intercept	105.0**	6.31	249.3×10^{-2}**	16.0×10^{-2}	100.8**	3.47	246.2×10^{-2}**	14.5×10^{-2}
L_{SPT}	−0.02	0.23	-0.67×10^{-2}*	0.24×10^{-2}	–	–	–	–
RR	–	–	-1.83×10^{-2}*	0.62×10^{-2}	–	–	-2.36×10^{-2}**	0.54×10^{-2}
Age	–	–	-1.00×10^{-2}**	0.27×10^{-2}	–	–	-1.05×10^{-2}**	0.27×10^{-2}
L_H	–	–	–	–	0.13	0.11	-0.27×10^{-2}†	0.16×10^{-2}
H	–	–	–	–	1.96	1.95	15.1×10^{-2}**	3.13×10^{-2}
H^*L_H	–	–	–	–	−0.05	0.07	-0.36×10^{-2}*	0.12×10^{-2}

Note: H = dummy variable indicating a half of the sleep period time, 1st half (0), 2nd half (1); number of subject-nights per model: [a]175, [b]179; †$p\leq.10$; *$p\leq.01$; **$p\leq.001$.

Addition of both *night1* and its interaction with L_{SPT} 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 L_{SPT}, $t_{(141)}=1.73$, $p=.086$. The regression of $logRSA_{SPT}$ on L_{SPT} remained significant following the addition of *night1* and its interaction with L_{SPT}, $t_{(167)}=-2.32$, $p=.022$.

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

Table 3 (right) provides an overview of the parameter estimates of the final PEP_H and $logRSA_H$ 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 PEP_H on H, $t_{(180)}=1.00$, $p>.317$. Indoor traffic noise exposure (L_H) was not significantly related to PEP_H during the first half of the sleep period, $t_{(175)}=1.20$, $p=.232$. Similarly, L_H was not significantly related to PEP_H during the second half of the sleep period, $t_{(175)}=-.73$, $p=.466$. Neither the total indoor, nor outdoor, noise exposure levels ($L_{\text{indoor},H}$ and $L_{\text{outdoor},H}$, respectively) were significantly related to PEP_H 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 $logRSA_H$ on H, $t_{(190)}=4.82$, $p<.001$. Indoor traffic noise exposure (L_H) was not significantly related to $logRSA_H$ during the first half of the sleep period, as indicated by the regression of $logRSA_H$ on the main term for L_H, $t_{(180)}=-1.74$, $p=.083$. Indoor traffic noise exposure was however significantly related to $logRSA_H$ during the second half of the sleep period, as indicated by the regression of $logRSA_H$ on the interaction term H^*L_H, $t_{(183)}=-3.06$, $p=.003$.

There was no significant effect of the total indoor noise exposure level ($L_{\text{indoor},H}$) on $logRSA_H$, during either the first half, $t_{(179)}=-.21$, $p=.834$, or second half, $t_{(181)}=-.79$, $p=.432$, of the sleep period time. The outdoor noise exposure level ($L_{\text{outdoor},H}$) was not significantly related to $logRSA_H$ during the first half of the sleep period, $t_{(199)}=-.04$, $p=.969$. However, during the second half of the sleep period, $L_{\text{outdoor},H}$ was significantly related to $logRSA_H$, $t_{(194)}=-2.85$, $p=.005$.

In the final $logRSA_H$ model there was a significant main effect of *night1*, $t_{(180)}=-2.22$, $p=.028$, although effect of the $night1^*L_H$ interaction term was not significant, $t_{(185)}=1.42$, $p=.159$. The regression of $logRSA_H$ on H^*L_H remained significant following the addition of *night1* and its interaction with L_H, $t_{(183)}=-3.04$, $p=.003$.

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 the *mean* traffic noise exposure level and the *mean* level of cardiac sympathetic tone during sleep was found does not imply that no relationship between cardiac sympathetic tone and traffic noise exposure during sleep exists. It may indicate that the present method of analysis was insufficient to detect such a relationship. Earlier studies have reported sympathetic response to traffic noise events during sleep (e.g. Carter et al., 2002; Griefahn et al., 2008). In the present situation, the response to individual traffic noise events may have been either a brief increase in cardiac sympathetic tone or a shift towards sympathetic dominance. Occasional sympathetic responses need not necessarily accumulate to a statistically significant increase in the mean level of cardiac sympathetic tone during sleep.

A third possibility is that there indeed is no effect of night-time traffic noise on sympathetic activity, indicating that the hypothesis tested during the present study, which was based on a straightforward linear reciprocal model of autonomic control, may be too simplistic. One of the alternative modes of autonomic control put forward by Berntson et al. (1991), such as 'uncoupled parasympathetic withdrawal', would be in line with the present findings.

No statistically significant evidence of a first-night effect for the relationship between cardiac parasympathetic tone and indoor traffic noise exposure level per sleep period was found. Addition of a variable coding for the first study night to the final *logRSA* model indicated that although cardiac parasympathetic tone during sleep was lower during the first study night compared to during the remaining study nights, this difference was not statistically significant. An explanation of the observed trend is that subjects may have slept less deeply during the first study night as they were unacquainted with the measuring device. Discomfort experienced while wearing the equipment was however not reflected in the level of cardiac parasympathetic tone during sleep, as self-reported hindrance due to the measuring device and its electrodes was not related to *logRSA*. Nevertheless, attenuated sleep depth during the first study night may have increased the subjects' responses to environmental stimuli, such as traffic noise, during sleep. To test this hypothesis of an interaction-effect, a variable coding for the indoor traffic noise exposure level times the first study night was added to the final *logRSA* model. This interaction variable 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.

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