The Short-Term Association between Exposure to Noise and Heart Rate Variability in Daily Locations and Mobility Contexts

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The short-term association between exposure to noise and heart rate variability in daily destinations and mobility contexts

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Abstract

Background: Personal exposure to noise has been shown to be associated with concomitant increases and lagged decreases of short-term heart rate variability (HRV). It is however unknown whether this association differs between contexts defined by visited places or mobility as both exposure sources and expectations may be different between these contexts.

Method: Between July 2014 and June 2015, the RECORD MultiSensor Study collected sound level and heart rate data for 75 participants, aged 34–74 years, in their living environments for 7 days using a personal dosimeter and electrocardiography sensor on the chest. Their whereabouts were collected using a GPS receiver and a mobility survey. Short-term concomitant and lagged associations between sound level and HRV parameters were assessed within types of visited places and transport modes using mixed effects models with a random intercept for participants.

Results: Increases in sound level were associated with a concomitant increase in all HR/HRV parameters, and delayed decreases in the overall HRV. Interactions between the sound level and the visited place/mobility context were documented. Compared with home, the concomitant association of sound level with HR and rMSSD was doubled within active and private motorized transport modes respectively.

Conclusion: The association of sound level with HR/HRV varies between visited places/mobility contexts. Future studies investigating these context-dependent associations in ambulatory settings will need to assess additional acoustical factors relating to the visited environments as well as non-acoustical factors impacting the perception of noise.

Keywords: Noise; Heart rate variability; Autonomic nervous system; Mobility; Sensors

Introduction

Noise is an environmental stressor ubiquitous to our urbanized modern lives, from traffic related noise to occupational noise, and noise in the household. Auditory health effects of noise are well known (1), with an average of 16% of the adult-onset hearing loss around the world being related to occupational noise (2). There is however increasing evidence on the non-auditory effects of noise on health (3), linking noise exposure with an increased risk of developing coronary heart disease as well as hypertension.

The noise reaction model introduced by Babisch (4) links noise exposure and the later development of cardiovascular diseases through the activation of the autonomic nervous system and endocrine systems as noise acts as a psychosocial stressor.

In a previous study (5), we assessed the short-term association between personal exposure to sound level and heart rate variability (HRV) parameters as a proxy for the state of the autonomic nervous system. We observed a concomitant increase of HRV parameters with the sound level. The use of wearable sensors allowed us to assess this association in a real life setting, therefore avoiding the limitations of modelled exposure or of exposure measured only at home or work, which ignores exposure in different visited places or during trips.

However, the reaction of the autonomic nervous system to an elevated sound level is likely to vary depending on the context. Indeed, first, the sources of elevated sound levels obviously vary depending on the context, and the perception of sounds as noise may depend on the source. Second, the expectations of people regarding the appropriate sound level may vary from one context to the other (e.g. home compared to the workplace or trips) as was underlined in the rationale for the development of a noise sensitivity questionnaire in different situations in daily life (6). Thus, an elevated sound level may be differentially interpreted as noise, and therefore may elicit different physiological reactions. Overall, the aim of this study is to assess how the context, as defined by

visited places or mobility (modes of transport), interacts with sound level in its short term concomitant or lagged association with heart rate and heart rate variability.

Method

Population

Participants came from the RECORD Cohort Study (7, 8), and more particularly from the RECORD MultiSensor sub-study, which aimed at investigating the relationships between transport and health using sensor-based measurement. Details regarding the study sample as well as the data collection protocol have been published elsewhere (5, 9). In summary, participants of the RECORD study were born between 1928 and 1978 and were residing in the Ile-de-France region. They were recruited between 2007 and 2008 without a priori sampling during preventive checkups performed by the IPC Medical Centre on behalf of the French social security. During the second wave of the RECORD Study in which new participants were also recruited, a fraction of the participants joined the RECORD MultiSensor Study between July 2014 and June 2015 in which they underwent a physiological and environmental sound monitoring. For a period of 7 days during their waking hours, participants were instructed to wear an electrocardiography (ECG) sensor, a dosimeter, a GPS receiver, and an accelerometer. Participants wearing a pacemaker or with hearing problems were not included. Written informed consent was obtained from all participants. The RECORD Multisensor Study was approved by the French Data Protection Authority (CNIL) regarding both the ethical and data security aspects (No: DR-2013-568).

Mobility survey

The data extracted from the BT-Q1000XT GPS receiver (Qstarz International, Taipei, Taiwan) were pre-processed after the 7-day data collection (10) in order to identify the visited places as well as the start and end times of each trip stage, defined as a segment of a trip using a unique transport mode. These data were in turn consolidated during a phone mobility survey with the participants, producing

in the end a detailed timetable covering the 7-day observation period. This timetable consisted of a time-stamped list of the visited places and trip stages between them.

For the present analysis, visited places were recoded to “Home” and “non-Home” while transport modes were divided into “Active transport modes” (i.e., walking and biking), “Private motorized vehicles” (i.e., cars and motorcycles as a driver or as a passenger) and “Public transport” (i.e., bus, tramway, metro and train).

Each visit at a place and trip stage was cut into contiguous time windows for which summaries of heart rate variability, sound level, and physical activity were computed. Two time scales were used in this study: (i) 5-minute windows as they correspond to the recommended duration for the measurement of short-term HRV (11) and (ii) 1-minute windows in order to assess HRV dynamics that may be masked within 5-min windows. These windows represented the statistical units of this study. The definition of windows uses as a starting time point the beginning of each visit at a place and each trip stage. Offcuts at the end of each segment were excluded from the analysis as they had a duration shorter than 5 minutes or 1 minute.

Sound level measurement/indicators

Individual exposure to sound level was assessed with a wearable Class II dosimeter Wed007 - 01dB (ACOEM Limonest, France) allowing for A-weighted measurements - a weighting that corresponds to the sensitivity of the human ear - between 40 and 120 dB(A) (tolerance ± 1.0 dB) every second (LAeq,1s). During the day, participants were instructed to place the microphone near the ear and over the clothing, to wear the dosimeter on the belt and to charge the device overnight. All of the dosimeters were calibrated at the beginning of the study following the manufacturer’s instructions using a standard acoustic calibrator (1 KHz sine wave at 94 dB).

Based on the A-weighted Leq,1s (LAeq,1s), the equivalent sound level (LAeq) was computed within each time window. The LAeq is a representation of the constant sound level that would have been

produced with the same energy than the varying sound level actually produced during the given period. It is one of the main sound level indicators used in environmental noise assessment (12).

Heart rate variability

The participants wore a BioPatch BHM 3 (Zephyr Technology, Annapolis, MD, USA) on the chest, an easy to set-up two electrodes ECG that collects data only if it is correctly worn. The use of a similar two electrodes ECG has been validated against a 12 lead ECG for the measurement of HRV (13). Participants were instructed to put it on when they woke up – using new electrodes every day – and to remove it when they went to bed as they had to charge it overnight. Files containing the inter-beat (RR) intervals were generated by the BioPatch and then processed in order to compute HRV parameters for which the specific signal processing steps are detailed elsewhere (5). Heart rate (HR) in bpm, the standard deviation of normal to normal RR intervals (SDNN) in ms as well as the root mean square of the successive differences (rMSSD) in ms were computed for each time window following the standard definitions (11). Both the high frequency (HF: 0.15 to 0.4 Hz) band power and the rMSSD represent vagal cardiac influence (i.e., the influence of the parasympathetic branch of the autonomic nervous system on heart rate) (14), but the latter was selected as it is less affected by breathing (15). The low frequency (LF: 0.04 to 0.15 Hz) band power as well as the LF/HF ratio were not considered in this study as their physiological interpretation is regarded to be unclear in the latest literature (16). All of the outcomes were log transformed in order to correct for heteroscedasticity. The entire signal processing was carried out under R version 3.4.3 (17) and the calculation of HRV parameters through the “RHRV” package version 4.2.3 (18).

Accelerometry

In addition to the aforementioned sensors, the participants wore an Actigraph wGT3X+ tri-axial accelerometer (ActiGraph LLC, Pensacola, FL, USA) on the right hip with a dedicated elastic belt. They were asked to remove the belt only when sleeping and when they were in contact with water. Accelerometry was collected in 5-second epochs. The count values of each of the three axes were

combined to produce the vector magnitude which in turn was summed up over the 5-minute and 1-minute windows. Additionally the coefficient of variation of the vector magnitude within each window was computed by dividing its standard deviation by its mean. Windows with a mean vector magnitude equal to zero were given a coefficient of variation of zero.

Socio-demographic variables

Age, sex, level of education, and employment status (only used for descriptive purposes) were collected from the IPC administrative database and medical questionnaire and the RECORD questionnaire filled in during the health checkup.

Statistical analysis

Linear mixed models applied separately to the 5-minute and 1-minute measurement windows were used to estimate associations between individual sound level exposure and HRV parameters. To take the clustering of the repeated measures into account as well as the imbalanced number of windows between participants, a mixed model with a random intercept at the individual level was used (19). Short-term trends of HRV parameters over the day were taken into account with smoothing splines estimated for each participant. The temporal autocorrelation between the repeated measurements of each participant was taken into account using an autoregressive model of order 1 AR(1) (20). This covariance structure assigns to each pair of measurements, within a participant, a correlation that decreases as the time interval separating the measurements increases. The correlation is expressed as ρ^k, where k is the time interval (in minutes) separating each pair of observations and ρ the correlation (ranging between 0 and 1) of a pair of observations separated by a one minute interval (19).

Separate models were built for each electrocardiographic outcome in two stages: (1) with the concomitant and lagged sound levels and (2) with an additional interaction term between the context and both the concomitant and lagged sound levels. All models were adjusted for the context (as defined by the mobility survey, with “Home” as the reference value), for heart rate (when the

outcome was HRV), and for the accelerometer vector magnitude and its coefficient of variation. Since the outcomes were log transformed, the regression coefficients were back transformed [using the formula: (exp(β) - 1) x 100], representing percent changes in the mean outcome for a one unit increase.

In order to correctly assess the interaction between sound level and context, lagged sound level was defined as the sound level in the preceding time window(s) within the same context. For the 5-minute windows a single lag (-5min) was considered in order to limit the exclusion of short trips. For the 1-minute windows four lags were considered (-1min, -2min, -3min and -4min) and the interaction with the lagged exposure was assessed using the furthest lag. Observations for which such lagged variables could not be defined (e.g., the first 5-minute window within a context episode) were discarded.

To assess the degree of multicollinearity within the models, particularly with the addition of lagged sound levels, the generalized variance inflation factor (GVIF) was computed using the vif.lme function implemented in the "car" package (21). The GVIF is a generalization of the variance inflation factor (VIF) that can be applied to categorical explanatory variables (22). Values of VIF < 4 are usually considered to be acceptable (23).

As the high number of observations in the 1-minute windows based dataset (more than 250 000 data points) rendered model fitting impossible with our current equipment (due to the autocorrelation structure), the dataset was under-sampled by selecting 20% of the 1-minute windows spent at activity places (“Home” and “non-Home”). Observations within trips were all included.

Statistical analysis was done in R version 3.4.3 (17) using the “nlme” package version 3.1-131 (24) and the “lmeSplines” package version 1.1–10 (25). The calculation of confidence intervals of the association within each context, based on linear combinations, was done using the “multcomp” package version 1.4-8 (26). Plots were created using the “ggplot2” package version 2.2.1 (27).

Results

Descriptive statistics

Of the initial 78 participants, three were excluded as either their accelerometer, noise dosimeter or ECG sensor did not work or was not worn. Consequently, the study sample consisted of 75 participants whose characteristics are presented in **Table 1** (additional information regarding the distribution of contexts relative to the socio-demographic characteristics are available in **Table S1** of the supplementary material). For the 75 participants the collected raw measurements consisted of 6 329 hours of RR intervals, 13 843 hours of sound level measurements and 11 825 hours of accelerometer data. Merging the three data sources with the timetable of the mobility survey, based on the timestamps, produced 5 221 hours of simultaneous measurements. After quality check (e.g., excluding periods of non-wear for the accelerometer, or missing data for sound level and RR intervals), 4 253 hours of measurement were selected. Finally, when considering only the 5-min windows with an available lagged exposure, 3 994 hours were available for analysis, representing 47 933 windows of 5-min. The final sample of 1-min windows had 251 110 observations which was reduced to 65 502 after under-sampling.

Table 2 shows descriptive statistics on sound level and HR/HRV parameters within the different contexts considered, while **Figure 1** shows the density plots of measured sound levels within each context. Visited places ("Home" and "non-Home") had the lowest mean values and the largest standard deviations, while "Private motorized vehicles" had the highest mean value. The overall sound level had a mean value and standard deviation of 65.7 dB(A) (SD: 11.0) with values ranging from 32.6 dB(A) to 112.0 dB(A). Mean sound level values were higher in transport modes [72.5 dB(A) (SD: 7.7)] than in visited places [65.3 dB(A) (SD: 11.1)] with a statistically significant difference ($p < 0.0001$).

Linear mixed effects models

Mixed effects models were fitted for each combination of sound level and HRV indicators, adjusted for the visited place or mobility context in addition to the accelerometer vector magnitude and its coefficient of variation, heart rate (when the outcome was HRV), and short-term trend. All models included the temporal autocorrelation structure as it improved the model fit, assessed using the likelihood ratio test ($p < 0.0001$) (see **Tables S2** and **S3** of the supplementary material).

Based on the 5-minute windows, **Table 3**, shows the change in percentage in the outcome for a one dB(A) increase for models including concomitant and lagged sound levels. Sound level was positively associated with a concomitant increase in HR, SDNN and rMSSD. Lagged associations (-5min) differed between the outcomes as it was positive for HR and negative for SDNN and rMSSD, with a 95% CI for the latter that overlapped zero.

Similar patterns were observed when using 1-minute windows (**Table 4**) i.e. all concomitant and lagged (-1min) associations were positive while lagged associations starting at -2min were negative for SDNN and rMSSD with a 95% CI for the latter that overlapped zero. No multicollinearity issues were detected in the models as the generalized variance inflation factors were below 1.71 and 2.62 in the models based on 5-minute and 1-minute windows respectively (**Table S6** and **S7** of the supplementary material).

The same models were then fitted with the addition of an interaction between the mobility context variable and both the concomitant and lagged sound level variables, using “Home” as the reference level. **Figure 2** shows the estimated associations within each context based on the 5-minute windows while the coefficients are available in the supplementary material **Table S8**.

For HR, the overall interactions between the context and the concomitant and lagged sound levels were statistically significant ($p < 0.0001$ and $p = 0.0016$ respectively). Compared with the concomitant positive association documented between sound level and HR at home, the concomitant associations with HR was slightly stronger in non-Home (difference: +0.024, 95% CI:

+0.011 to +0.038) and twice as high during active transport modes (difference: +0.146, 95% CI: +0.082 to +0.210) while the 95% CI of the association within Public transport modes overlapped zero (estimate: 0.066, 95% CI: -0.080 to 0.211). Regarding lagged associations, the only statistically significant difference compared with Home was in non-Home which was slightly lower. The 95% CI of the lagged associations within all three transport mode contexts overlapped zero, with a point estimate for private motorized transport modes close to the null and a point estimate for public transports close to its concomitant association estimate.

For SDNN, the overall interaction between the context and the concomitant and lagged sound levels was statistically significant only for the latter ($p = 0.1562$ and $p < 0.0001$ respectively). Concomitant associations were positive in all contexts with a 95% CI overlapping zero only in the case of public transports (estimate: 0.452, 95% CI: -0.511 to 1.423). Lagged associations were negative across contexts, with the exception of public transports for which the point estimate remained close to that of its concomitant association. The 95% CI of lagged associations with SDNN overlapped zero for the three transport mode contexts. Compared with home, the lagged associations in non-home was significantly more negative.

Regarding rMSSD, the overall interaction between the context and the concomitant and lagged sound levels were both statistically significant ($p = 0.0002$ and $p = 0.0245$ respectively). Concomitant associations were positive across contexts with the exception of public transports for which the association was negative, with a 95% CI overlapping zero (estimate: -0.137, 95% CI: -1.294 to 1.034). Compared to home, the concomitant association within private motorized transport modes was doubled (difference: +0.756, 95% CI: +0.290 to +1.224) while the lagged association was slightly lower – and therefore negative – in non-home. Lagged associations were close to the null across contexts with a 95% CI overlapping zero for the three transport modes.

Similar patterns were observed when using 1-min windows, both for the concomitant and lagged associations (Supplementary Material **Figure S1** and **Table S9**). The only notable exception was the

concomitant association between sound level and HR within active transport modes which was lower (but still positive with a 95% CI excluding zero) than the one observed at home (as opposed to higher when considering 5-min windows).

Discussion

Summary of results

In this study, we assessed the concomitant and lagged short-term associations between sound level and HR and HRV across different contexts of daily life exposure (visited places/transport modes). We documented positive concomitant associations with HR, SDNN and rMSSD as well as lagged associations of smaller magnitude, that were positive with HR and negative with both SDNN and rMSSD, (however the confidence interval of the latter association overlapping zero). These associations were documented for 5-min windows and similar patterns were observed when using smaller windows (1-min) with a positive concomitant and lagged (-1min) association with the three outcomes and starting at lag -2min a negative association with SDNN and rMSSD.

When considering the interaction between the sound level and the context in their concomitant effects on HR and HRV, some differences were documented within transport modes. While the concomitant associations were positive across contexts and outcomes (with the exception of public transport modes in the model for rMSSD), a stronger association of sound level with HR within active transport modes and a stronger association with rMSSD within private motorized transport modes were documented, both doubled compared to the effect observed at home. Regarding lagged associations, they had a smaller magnitude across contexts, with the exception of public transport modes for which the point estimates remained close to those of the concomitant associations.

The overall positive concomitant and lagged associations between sound level and HR may be explained by sounds acting as stressors triggering the fight-or-flight response with the activation of the sympathetic branch of the autonomic nervous system leading to an increased HR (28). This association have been documented in various studies, both in controlled and ambulatory settings as was underlined by Idrobo Avila et al. in a review on the relationship between sound and electrocardiographic signals (29). In a study by Holand et al. (30) participants were exposed to an auditory startle stimulus while HR and blood pressure (BP) were continuously monitored. Their

results showed an increase followed by a decrease towards the baseline in both HR and BP in the 30s following the stimulus. The results of our study additionally suggest that sound stimuli may have measurable delayed effects on HR lasting at least 5 minutes. In a study by Kraus et al. (31) similar positive concomitant and lagged (up to 15 min) short-term associations between sound level and HR were documented during daily life activities.

The SDNN represents the overall HRV. A reduced SDNN has been linked to an increased risk of cardiovascular morbidity and mortality in populations with (32) and without known cardiovascular diseases (33). The short-term association between sound stimuli and SDNN has been explored in previous studies, with inconsistent results. Sim et al. (34) and Oh et al. (35) found no significant changes in SDNN in participants exposed to different types of noise. Björ et al. (36) found an increased total power (another measurement of overall HRV) in participants during exposure to an 85 dB(A) white noise (i.e., a sound containing all audible frequencies at equal intensities). Conversely, Walker et al. (37) documented a reduced SDNN during exposure to low frequency noise (31.5 to 125 Hz), but not to high frequency noise (500 Hz to 2 kHz), while Huang et al. (38) reported a decreased SDNN with increasing sound levels only for the cumulative lagged exposure over 30 min (among 5 min, 15 min, 30 min and 1 h).

The rMSSD represents the vagally mediated HRV (parasympathetic branch of the autonomic nervous system) with some studies suggesting that a reduced vagal tone is an independent risk factor for all-cause mortality (39). Similarly to SDNN, previous studies assessing the associations between sound stimuli and vagal tone have documented discrepant results. In the studies by Björ et al. (36), Lee et al. (40) and Sim et al. (34), which used HF power as a proxy for vagal tone, no changes were associated with exposure to white noise (for the first two studies) and to traffic and speech noise (for the third study). The study by Oh et al. (35) found a significant increase in vagal tone when exposed to a car horn sound, but only for HF power and not for rMSSD. Cho et al. (41) documented a decrease in HF power during exposure to low frequency (100 Hz) and high frequency (10 kHz) white noise but

not during 1 kHz white noise, while Walker et al. (37) found a significant decrease in HF power during exposure to low frequency noise (31.5 to 125 Hz) but not during high frequency noise (500 Hz to 2 kHz) and no changes in rMSSD. Regarding the two studies with a non-simulated sound exposure, Kraus et al. (31) reported a concomitant decrease in HF power for sounds below 65 dB(A) (and an increase for sounds above), and Huang et al. (38) reported a decrease in HF power with both concomitant and cumulative lagged sound levels, up to 15 min.

The diverging results between the studies may be explained by differences in study design (type and duration of exposure, controlled or real life setting, measurement during or before and after exposure) as well as differences in the specific parameter used to assess the vagal tone, which limits the comparability of the results.

The concomitant increase in SDNN and rMSSD with increasing sound levels remains difficult to explain. However, one potential explanation could be that this increase is driven by peaks in the sound level which repeatedly trigger startle responses characterized by a peak in HR within few seconds (30), therefore momentarily increasing SDNN. As these quick responses are driven by the inhibition and reactivation of the parasympathetic branch (42), this would also explain the concomitant increase in rMSSD. As observed when assessing the concomitant and lagged associations using 1-min windows, this increase lasted 2 minutes before being counterbalanced. This is suggested by the lagged negative associations between sound level and both SDNN and rMSSD which were however not statistically significant for the latter. Kraus et al. (31) found patterns of association similar to those documented in this study, i.e. a positive concomitant and negative lagged associations between sound level and SDNN using 5-min windows. In both the study by Kraus et al. and in a previous study by our team on the same dataset (5), this delayed reduction in SDNN was still statistically significant after 15 minutes (maximum lag explored in both studies).

Regarding the interactions between the context of exposure and the sound level in their effects on HR and HRV parameters, few differences were documented in this study. Compared with home, the

concomitant association in other visited places was slightly stronger for HR while the lagged associations were slightly weaker for the three outcomes in this context. Within transport modes, a doubled concomitant association of sound level with HR within active transport modes and with rMSSD within private motorized transport modes were observed while associations within public transport modes were statistically non-significant. These differences may be related with differences in both the nature of the sounds as well as the perceived control over them associated with each of these contexts. Indeed, compared to home, active and private motorized transport modes are dominated by road traffic noises while offering less control over the sound environments. Lagged associations within all transport modes contexts were statistically non-significant with higher confidence intervals compared to the estimates within visited places. This stems from the relatively low number of observations within transport modes (6.0% of the sample) particularly for public transport modes (1.4% of the sample).

Strengths and limitations

Strengths:

The use of wearable sensors in combination with the mobility survey allowed us to assess precisely and objectively both the exposure (sound level) and the outcome (HR and HRV) as well as the transport modes/visited places in a “real life” setting. The analyzed time windows were defined according to the mobility survey, allowing us to slice the continuous sensor measurements into 5-min and 1-min windows within each defined context. This method allowed us in turn, to contextualize the association between sound level and HRV by looking at the interaction between sound level and mobility contexts. This approach allowed us also to assess the associations in “real life” situations which seems necessary since an experiment on stress effects of noise by Ising et al (43) comparing a field and a laboratory experiment found no correlation between both setups, underlining the necessity for field measurements. Finally, another strength of this study lies in the large number of

observations (i.e., n=47 933 windows at the 5-min window level), with however a limited number of participants (n=75).

Limitations and potential explanations for observed differences:

The observed differences between the mobility contexts are however difficult to interpret. First, the comparability with other studies is limited by differences in study design (29) (controlled or ambulatory setting, concomitant or lagged association), and in the signal processing steps for the calculation of HRV indicators (44). Second, the physiological correlates of HRV indicators have been criticized lately, in particular their ability to measure changes in the sympathetic branch activity and therefore, their ability to represent the balance of the autonomic nervous system (45). These limitations could be overcome by complementing the HRV assessment with that of galvanic skin response which is modulated by sympathetic activity and proved to be feasible in an ambulatory setting if confounders are taken into account (46). As for the comparability with other studies, this would require the standardization of the processing and reporting of HRV indicators across studies, which could be achieved by an update of the recommendations for HRV measurement and interpretation which date back to 1996 (11).

Another limitation of this study, which is related to the ambulatory “real life” assessment approach, lies in the unmeasured confounders that may impact HRV, namely air pollution (47), vibrations (48), body posture (49), and other psychological stressors (50). These confounders could be taken into account in future studies either with the use of additional sensors, or questionnaires on mobile phones. This approach would prove useful in order to disentangle the effects of various environmental stressors. Another confounder which was taken into account in this study is physical activity (51). Although we measured it objectively with accelerometry, the use of a single accelerometer placed at the hip may underestimate upper body movements and as well as total physical activity while cycling (52).

346 The observed differences between contexts could also be related to differences in the sound
347 environments within each context that a single indicator cannot encompass. Three distinct
348 components could be used to describe the acoustical characteristics of a sound environment: the
349 energetic, temporal, and spectral dimensions (53). The Leq, the traditionally used indicator in
350 environmental noise assessment, represents the energetic dimension, but it does not encompass
351 temporal variations (in this case within the 5-minute and 1-minute windows) and the spectral
352 components of the sound environments. Regarding the spectral dimension, in our study the
353 measurements were A-weighted, a weighting that accounts for the human ear sensibility as it is less
354 sensitive to low frequencies. As a consequence low frequency noises are underestimated even
355 though they have been shown to impact HRV (37) and are recognized as a special environmental
356 noise issue (54).

357 The three dimensions of sound environments (energetic, temporal, and spectral) can be accounted
358 for in future studies by using sound level dosimeters with octave band filters (or 1/3 octave band
359 filters) allowing for the concomitant measurement of sound level over split frequency bands. This
360 would allow to encompass the spectral dimension, while the temporal and energetic dimensions
361 could be taken into account by using different noise indicators, e.g., the range of noise levels (53).

362 Additionally, as the distinction between sound and noise lies in its subjective assessment, different
363 non-acoustical factors may modify the perception of a specific sound and the resulting physiological
364 reactions. These factors include the level of mental arousal, the meaning and predictability of the
365 sounds, and the perceived control over the sound source (55, 56). These non-acoustical factors
366 (perception) as well as the ongoing task during the real life assessment could be collected using two
367 complementary approaches, i.e., an enhanced version of our mobility survey and ecological
368 momentary assessment (57) with questionnaires on mobile phones. On one hand, this would allow
369 one to ask participants questions about their perception of current sound environments, on the

other hand this would also allow researchers to assess the participants' subjective stress in order to bridge the gap between subjective and objective stress (HRV).

This study attempted to evaluate the interactions between visited place/mobility contexts and sound levels in their association with HRV parameters. Interpretation of the results is however limited by the aforementioned factors which could be overcome by future studies aiming at assessing the effects of environmental stressors in an ambulatory unrestricted setting. Such will be the case of our ongoing Mobilisense project, for which sound level meters with 1/3 band octave filters will be used in combination with personal air pollution sensors as well as questionnaires on mobile phone.

Conclusion

In conclusion, in this study there was some evidence of differences in the association of sound level with HR and HRV parameters across visited places and mobility contexts. While the overall concomitant association between sound level and HR and HRV parameters was positive, some mobility contexts showed stronger positive associations. While our work represents an advance in the understanding of noise effects in real-life settings, the interpretation of those differences will have to be improved by the assessment of currently unmeasured factors. Overall, future studies assessing the effects of noise on the autonomic nervous system should supplement HRV monitoring with additional physiological measurements (e.g. Galvanic skin response) and environmental measurements (air pollutants) as well as complementary noise indicators while simultaneously assessing subjective factors on both the sound level exposure and related perceived stress.

Conflict of interest: The authors declare no conflict of interest

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Supplementary Information is available at the Journal of Exposure Science and Environmental Epidemiology website (https://www.nature.com/jes)

References

1. Basner M, Babisch W, Davis A, Brink M, Clark C, Janssen S, et al. Auditory and non-auditory effects of noise on health. The Lancet. 2014;383(9925):1325-32.

2. Nelson DI, Nelson RY, Concha-Barrientos M, Fingerhut M. The global burden of occupational noise-induced hearing loss. American journal of industrial medicine. 2005;48(6):446-58.

3. de Kluizenaar Y, Matsui T, editors. Recent progress in the field of non-auditory health effects of noise–trends and research needs. 12th ICBEN Congress on Noise as a Public Health Problem, June 18-22, 2017, Zurich, 1-12; 2017.

4. Babisch W. The noise/stress concept, risk assessment and research needs. Noise and health. 2002;4(16):1.

5. El Aarbaoui T, Méline J, Brondeel R, Chaix B. Short-term association between personal exposure to noise and heart rate variability: The RECORD MultiSensor Study. Environmental Pollution. 2017;231:703-11.

6. Schutte M, Marks A, Wenning E, Griefahn B. The development of the noise sensitivity questionnaire. Noise and Health. 2007;9(34):15.

7. Chaix B, Kestens Y, Bean K, Leal C, Karusisi N, Meghiref K, et al. Cohort Profile: Residential and non-residential environments, individual activity spaces and cardiovascular risk factors and diseases—The RECORD Cohort Study. International journal of epidemiology. 2011;41(5):1283-92.

8. Chaix B, Kestens Y, Duncan DT, Brondeel R, Méline J, El Aarbaoui T, et al. A GPS-based methodology to analyze environment-health associations at the trip level: case-crossover analyses of built environments and walking. American journal of epidemiology. 2016;184(8):579-89.

9. Chaix B, Kestens Y, Duncan S, Merrien C, Thierry B, Pannier B, et al. Active transportation and public transportation use to achieve physical activity recommendations? A combined GPS, accelerometer, and mobility survey study. International journal of behavioral nutrition and physical activity. 2014;11(1):124.

10. Wolf J, Schönfelder S, Samaga U, Oliveira M, Axhausen K. Eighty weeks of global positioning system traces: approaches to enriching trip information. Transportation Research Record: Journal of the Transportation Research Board. 2004(1870):46-54.

11. Camm AJ, Malik M, Bigger J, Breithardt G, Cerutti S, Cohen RJ, et al. Heart rate variability. Standards of measurement, physiological interpretation, and clinical use. European heart journal. 1996;17(3):354-81.

12. Ricciardi P, Delaitre P, Lavandier C, Torchia F, Aumond P, Charlotte. Sound quality indicators Night noise guidelines for urban places in Paris cross-validated by Milan dataEurope. The Journal of the Acoustical Society of America. 20152009;138(4):2337-48.

13. Nunan D, Donovan G, Jakovljevic DG, Hodges LD, Sandercock GR, Brodie DA. Validity and reliability of short-term heart-rate variability from the Polar S810. Medicine & Science in Sports & Exercise. 2009;41(1):243-50.

14. Thayer JF, Åhs F, Fredrikson M, Sollers III JJ, Wager TD. A meta-analysis of heart rate variability and neuroimaging studies: implications for heart rate variability as a marker of stress and health. Neuroscience & Biobehavioral Reviews. 2012;36(2):747-56.

15. Hill L, Siebenbrock A, Sollers J, Thayer JF. Are all measures created equal? Heart rate variability and respiration. Biomed Sci Instrum. 2009;45:71-6.

16. Laborde S, Mosley E, Thayer JF. Heart rate variability and cardiac vagal tone in psychophysiological research–recommendations for experiment planning, data analysis, and data reporting. Frontiers in psychology. 2017;8:213.

17. Team RC. R: A language and environment for statistical computing. 2013.

18. Rodríguez-Liñares L, Méndez AJ, Lado MJ, Olivieri DN, Vila X, Gómez-Conde I. An open source tool for heart rate variability spectral analysis. Computer methods and programs in biomedicine. 2011;103(1):39-50.

19. Fitzmaurice GM, Laird NM, Ware JH. Applied longitudinal analysis: John Wiley & Sons; 2012.

20. Kincaid C, editor Guidelines for selecting the covariance structure in mixed model analysis. Proceedings of the Thirtieth Annual SAS Users Group International Conference; 2005: SAS Institute Inc Cary NC.

21. Fox J, Weisberg S, Adler D, Bates D, Baud-Bovy G, Ellison S, et al. Package 'car'. Vienna: R Foundation for Statistical Computing. 2012.

22. Fox J, Monette G. Generalized collinearity diagnostics. Journal of the American Statistical Association. 1992;87(417):178-83.

23. O'brien RM. A caution regarding rules of thumb for variance inflation factors. Quality & quantity. 2007;41(5):673-90.

24. Pinheiro J, Bates D, DebRoy S, Sarkar D, Team RC. nlme: Linear and nonlinear mixed effects models (R package version 3.1-128, 2016). R software. 2017.

25. Ball R, Galecki MA. Package 'lmeSplines'. 2015.

26. Hothorn T, Bretz F, Westfall P, Heiberger RM, Schuetzenmeister A, Scheibe S, et al. Package 'multcomp'. 2017.

27. Wickham H, Chang W. ggplot2: An implementation of the Grammar of Graphics. 2018.

28. Münzel T, Gori T, Babisch W, Basner M. Cardiovascular effects of environmental noise exposure. European heart journal. 2014;35(13):829-36.

29. Idrobo Ávila EH, Loaiza Correa H, Van Noorden L, Muñoz Bolaños FG, Vargas Cañas R. Different types of sounds and their relationship with the electrocardiographic signals and the cardiovascular system-Review. Frontiers in physiology. 2018;9:525.

30. Holand S, Girard A, Laude D, Meyer-Bisch C, Elghozi J-L. Effects of an auditory startle stimulus on blood pressure and heart rate in humans. Journal of hypertension. 1999;17(12):1893-7.

31. Kraus U, Schneider A, Breitner S, Hampel R, Rückerl R, Pitz M, et al. Individual daytime noise exposure during routine activities and heart rate variability in adults: a repeated measures study. Environmental health perspectives. 2013;121(5):607.

32. Buccelletti E, Gilardi E, Scaini E, Galiuto L, Persiani R, Biondi A, et al. Heart rate variability and myocardial infarction: systematic literature review and metanalysis. Eur Rev Med Pharmacol Sci. 2009;13(4):299-307.

33. Hillebrand S, Gast KB, de Mutsert R, Swenne CA, Jukema JW, Middeldorp S, et al. Heart rate variability and first cardiovascular event in populations without known cardiovascular disease: meta-analysis and dose–response meta-regression. Europace. 2013;15(5):742-9.

34. Sim CS, Sung JH, Cheon SH, Lee JM, Lee JW, Lee J. The effects of different noise types on heart rate variability in men. Yonsei medical journal. 2015;56(1):235-43.

35. Oh B-S, Yeo YK, Wan FY, Wen Y, Yang Y, Lin Z, editors. Effects of noisy sounds on human stress using ECG signals: an empirical study. Information, Communications and Signal Processing (ICICS), 2015 10th International Conference on; 2015: IEEE.

36. Björ B, Burström L, Karlsson M, Nilsson T, Näslund U, Wiklund U. Acute effects on heart rate variability when exposed to hand transmitted vibration and noise. International archives of occupational and environmental health. 2007;81(2):193-9.

37. Walker ED, Brammer A, Cherniack MG, Laden F, Cavallari JM. Cardiovascular and stress responses to short-term noise exposures—A panel study in healthy males. Environmental research. 2016;150:391-7.

38. Huang J, Deng F, Wu S, Lu H, Hao Y, Guo X. The impacts of short-term exposure to noise and traffic-related air pollution on heart rate variability in young healthy adults. Journal of Exposure Science and Environmental Epidemiology. 2013;23(5):559.

39. Thayer JF, Yamamoto SS, Brosschot JF. The relationship of autonomic imbalance, heart rate variability and cardiovascular disease risk factors. International journal of cardiology. 2010;141(2):122-31.

40. Lee G-S, Chen M-L, Wang G-Y. Evoked response of heart rate variability using short-duration white noise. Autonomic Neuroscience. 2010;155(1-2):94-7.

41. Cho W, Hwang S-H, Choi H. The relationship between pure-tone noise and human bio-signal response. International Journal of Precision Engineering and Manufacturing. 2011;12(4):727-31.

42. Chen K-H, Aksan N, Anderson SW, Grafft A, Chapleau MW. Habituation of parasympathetic-mediated heart rate responses to recurring acoustic startle. Frontiers in psychology. 2014;5:1288.

43. Ising H, Michalak R. Stress effects of noise in a field experiment in comparison to reactions to short term noise exposure in the laboratory. Noise and Health. 2004;6(24):1.

44. Jarrin DC, McGrath JJ, Giovanniello S, Poirier P, Lambert M. Measurement fidelity of heart rate variability signal processing: The devil is in the details. International Journal of Psychophysiology. 2012;86(1):88-97.

45. Heathers J, Goodwin M. LF/HF HRV: The 'Life After Death'Of A Refuted Theory. 2017.

46. Doberenz S, Roth WT, Wollburg E, Maslowski NI, Kim S. Methodological considerations in ambulatory skin conductance monitoring. International Journal of Psychophysiology. 2011;80(2):87-95.

47. Buteau S, Goldberg MS. A structured review of panel studies used to investigate associations between ambient air pollution and heart rate variability. Environmental research. 2016;148:207-47.

48. Jiao K, Li Z, Chen M, Wang C, Qi S. Effect of different vibration frequencies on heart rate variability and driving fatigue in healthy drivers. International archives of occupational and environmental health. 2004;77(3):205-12.

49. Young FL, Leicht AS. Short-term stability of resting heart rate variability: influence of position and gender. Applied Physiology, Nutrition, and Metabolism. 2011;36(2):210-8.

50. Wallenius MA. The interaction of noise stress and personal project stress on subjective health. Journal of Environmental Psychology. 2004;24(2):167-77.

51. Hautala AJ, Karjalainen J, Kiviniemi AM, Kinnunen H, Mäkikallio TH, Huikuri HV, et al. Physical activity and heart rate variability measured simultaneously during waking hours. American Journal of Physiology-Heart and Circulatory Physiology. 2010;298(3):H874-H80.

52. Herman Hansen B, Børtnes I, Hildebrand M, Holme I, Kolle E, Anderssen SA. Validity of the ActiGraph GT1M during walking and cycling. Journal of sports sciences. 2014;32(6):510-6.

53. Can A, Michel S, De Coensel B, Ribeiro C, Botteldooren D, Lavandier C, editors. Comparison of noise indicators in an urban context. INTER-NOISE and NOISE-CON Congress and Conference Proceedings; 2016: Institute of Noise Control Engineering.

54. Leventhall H. Low frequency noise and annoyance. Noise and Health. 2004;6(23):59.

55. Rylander R. Physiological aspects of noise-induced stress and annoyance. Journal of Sound and Vibration. 2004;277(3):471-8.

56. Westman JC, Walters JR. Noise and stress: a comprehensive approach. Environmental Health Perspectives. 1981;41:291.

57. Moskowitz DS, Young SN. Ecological momentary assessment: what it is and why it is a method of the future in clinical psychopharmacology. Journal of Psychiatry and Neuroscience. 2006;31(1):13.

Figure legends

Figure 1: Density plots of LAeq sound levels within visited places and transport modes. Active: active transport modes, Priv.Mot: private motorized transport modes, Public: public transport modes.

Figure 2: Estimated associations (and their 95% CI) between concomitant and lagged sound levels and HR/HRV parameters in different mobility defined contexts using 5-min windows. Active: active transport modes, Priv.Mot: private motorized transport modes, Public: public transport modes. Asterisks denote a statistically significant difference with Home (reference level) *p<0.05, **p<0.01, ***p<0.001.

Table 1 Descriptive statistics of the participants characteristics (N = 75)

Variable	n (%)
Men	48 (64%)
Age	
[34-40]	16 (21.3%)
]40-50]	19 (25.3%)
]50-60]	23 (30.7%)
]60-74]	17 (22.7%)
Employment status	
Employed	49 (65.3%)
Unemployed	11 (14.7%)
Retired	13 (17.3%)
Other	2 (2.7%)
Annoyed by noise during work	17 (22.7%)
Educational level	
No education, primary, lower secondary	13 (17.3%)
Higher secondary, lower tertiary	23 (30.7%)
Intermediate tertiary	19 (25.3%)
Upper tertiary	20 (26.7%)

Table 2: Number of 5-min windows and summary statistics of sound level, heart rate and heart rate variability measurements per context (n = 47 933)

Context	n	Leq [db(A)]		HR (bpm)		SDNN (ms)		rMSSD (ms)	
		mean (sd)	median (range)	mean (sd)	median (range)	mean (sd)	median (range)	mean (sd)	median (range)
Home	25589	64.4 (11.3)	65.5 (32.6-107.0)	77.9 (13.0)	77.3 (44.8-143.4)	60.8 (30.1)	54.7 (6.2-270.9)	35.7 (24.9)	28.6 (1.8-261.2)
non-Home	19466	66.5 (10.6)	67.4 (34.3-102.4)	78.9 (13.5)	77.8 (46.6-171.1)	63.4 (29.7)	57.3 (7.8-258.5)	36.9 (26.2)	29.6 (2.1-238.1)
Active	791	70.4 (8.3)	71.4 (33.6- 91.9)	96.2 (19.0)	94.8 (52.3-164.2)	53.1 (33.4)	45.4 (8.8-242.3)	29.8 (29.0)	21.6 (2.2-201.8)
Priv.Mot	1434	73.7 (7.6)	72.8 (49.2-112.0)	78.8 (13.4)	77.1 (50.7-142.6)	48.1 (25.3)	43.0 (7.7-188.1)	31.1 (24.4)	23.7 (1.8-186.2)
Public	653	72.6 (6.6)	72.6 (40.8- 89.3)	78.6 (11.3)	77.6 (54.1-118.1)	51.3 (28.9)	43.7 (9.5-184.4)	27.9 (23.4)	22.0 (3.7-167.8)

Leq: Equivalent continuous sound level of A–weighted Leq,1s, HR: Heart rate in beats per minute, SDNN: Standard deviation of normal to normal RR intervals in milliseconds, rMSSD: Root mean square of the successive differences in milliseconds.

Table 3: Coefficients and 95% confidence intervals of the percentage change in the mean outcome associated with a one dB(A) increase in the sound level in the 5-minute windows.

	HR (bpm)		SDNN (ms)		rMSSD (ms)	
	β	95% CI	β	95% CI	β	95% CI
Concomitant	+0.141	[+0.135 to +0.148]	+0.894	[+0.849 to +0.939]	+0.600	[+0.546 to +0.655]
Lagged (−5min)	+0.033	[+0.026 to +0.039]	−0.315	[−0.358 to −0.272]	−0.050	[−0.102 to +0.002]

Models were adjusted for accelerometer vector magnitude and its coefficient of variation, heart rate (only for HRV outcomes), short-term trend and context. Complete models are available in the Table S3 of the supplementary material.

Abbreviations: HR: Heart rate, SDNN: Standard deviation of normal to normal RR intervals, rMSSD: Root mean square of the successive differences.

Table 4: Coefficients and 95% confidence intervals of the percentage change in the mean outcome associated with a one dB(A) increase in the sound level in the 1-minute windows.

	HR (bpm)		SDNN (ms)		rMSSD (ms)	
	β	95% CI	β	95% CI	β	95% CI
Concomitant	+0.112	[+0.105 to +0.119]	+0.755	[+0.698 to +0.813]	+0.526	[+0.460 to +0.592]
Lagged (−1min)	+0.076	[+0.069 to +0.083]	+0.180	[+0.119 to +0.241]	+0.141	[+0.072 to +0.210]
Lagged (−2min)	+0.016	[+0.009 to +0.023]	−0.129	[−0.190 to −0.068]	−0.067	[−0.136 to +0.002]
Lagged (−3min)	+0.016	[+0.009 to +0.023]	−0.060	[−0.122 to +0.001]	−0.025	[−0.094 to +0.045]
Lagged (−4min)	+0.016	[+0.008 to +0.022]	−0.160	[−0.217 to −0.104]	−0.059	[−0.124 to +0.006]

Models were adjusted for accelerometer vector magnitude and its coefficient of variation, heart rate (only for HRV outcomes), short-term trend and context. Complete models are available in the Table S3 of the supplementary material.

Abbreviations: HR: Heart rate, SDNN: Standard deviation of normal to normal RR intervals, rMSSD: Root mean square of the successive differences.

Figure 1

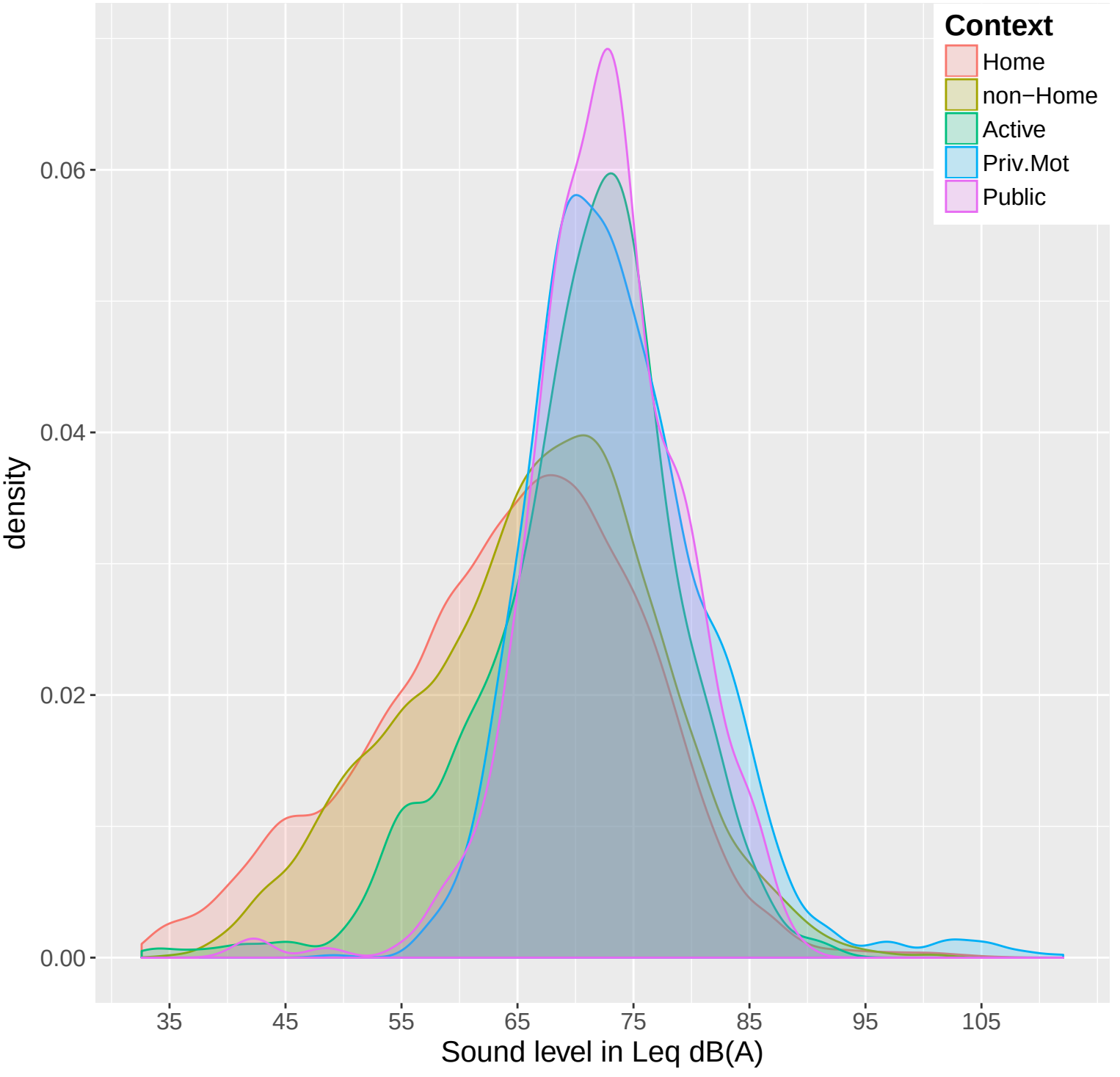
Context
Home
non−Home
Active
Priv.Mot
Public
density
0.06
0.04
0.02
0.00
35
45
55
65
75
85
95
105
Sound level in Leq dB(A)

Figure 2

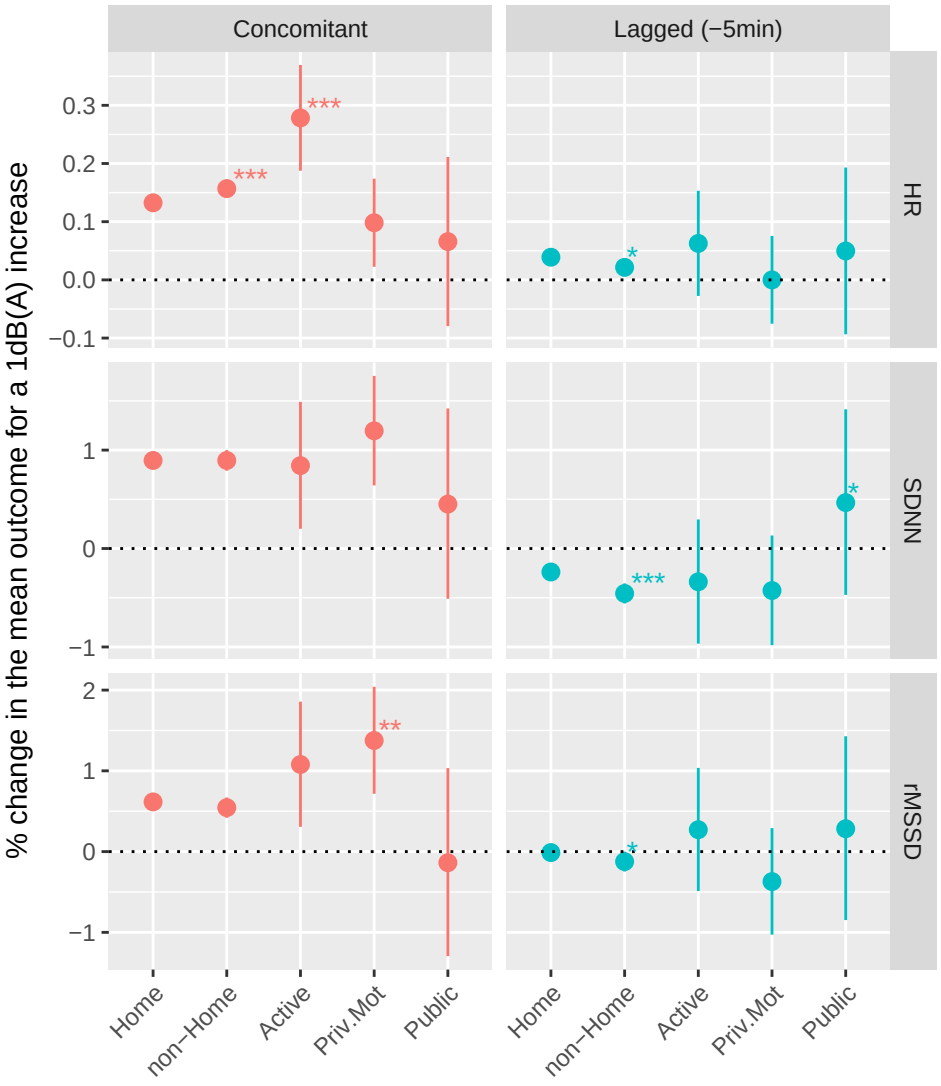
Concomitant
Lagged (−5min)
HR
SDNN
rMSSD
% change in the mean outcome for a 1dB(A) increase
0.3
0.2
0.1
0.0
−0.1
1
0
−1
2
1
0
−1
***
***
*
**
*
***
*
Home
non−Home
Active
Priv.Mot
Public
Home
non−Home
Active
Priv.Mot
Public

Table S1: Distribution of individual characteristics across contexts. Percentages are relative to each line.

		N (participants)	N (5-min windows)	Home	non-Home	Active	Priv.Mot	Public
Employment	Employed	49	31601	43.7%	50.4%	1.6%	2.5%	1.7%
	Unemployed	26	16332	72.2%	21.6%	1.7%	3.9%	0.7%
Sex	Women	27	17301	59.8%	34.8%	1.5%	2.8%	1.1%
	Men	48	30632	49.8%	43.9%	1.8%	3.1%	1.5%
Education	<3y tertiary	36	22672	54.0%	41.1%	1.2%	2.7%	1.1%
	≥3y tertiary	39	25261	52.9%	40.2%	2.1%	3.2%	1.6%
Age	<52y	36	24215	51.1%	44.0%	1.5%	2.1%	1.4%
	≥52y	39	23718	55.8%	37.2%	1.9%	3.9%	1.3%

Active: active transport modes, Priv.Mot: private motorized transport modes, Public: public transport modes.

Table S2: Model fit indices and likelihood ratio test between models with and without the autoregressive covariance structure of order 1 (for models based on 5-min windows)

Models	df	AIC	BIC	logLik	L.Ratio	p-value
SDNN: model without interaction, with AR	14	34083.59	34206.47	-17027.79	5735.237	<.0001
SDNN: model without interaction, without AR	13	39816.82	39930.93	-19895.41		
rMSSD: model without interaction, with AR	14	51862.88	51985.77	-25917.44	11467.62	<.0001
rMSSD: model without interaction, without AR	13	63328.50	63442.61	-31651.25		
HR: model without interaction, with AR	13	-147971.64	-147857.53	73998.82	60916.62	<.0001
HR: model without interaction, without AR	12	-87057.02	-86951.69	43540.51		
SDNN: model with interaction, with AR	22	34064.56	34257.67	-17010.28	5707.415	<.0001
SDNN: model with interaction, without AR	21	39769.98	39954.31	-19863.99		
rMSSD: model with interaction, with AR	22	51849.03	52042.13	-25902.51	11425.56	<.0001
rMSSD: model with interaction, without AR	21	63272.59	63456.92	-31615.30		
HR: model with interaction, with AR	21	-148007.85	-147823.52	74024.93	60709.56	<.0001
HR: model with interaction, without AR	20	-87300.29	-87124.74	43670.14		

HR: heart rate, SDNN: standard deviation of normal to normal RR intervals, rMSSD: root mean square of the successive differences, df: degrees of freedom

Table S3: Model fit indices and likelihood ratio test between models with and without the autoregressive covariance structure of order 1 (for models based on 1-min windows)

Models	df	AIC	BIC	logLik	L.Ratio	p-value
SDNN: model without interaction, with AR	17	84607.52	84762.05	-42286.76	5460.509	<.0001
SDNN: model without interaction, without AR	16	90066.03	90211.47	-45017.01		
rMSSD: model without interaction, with AR	17	105873.3	106027.9	-52919.67	9518.862	<.0001
rMSSD: model without interaction, without AR	16	115390.2	115535.6	-57679.10		
HR: model without interaction, with AR	16	-164894.7	-164749.3	82463.36	61748.6	<.0001
HR: model without interaction, without AR	15	-103148.1	-103011.8	51589.06		
SDNN: model with interaction, with AR	25	84583.56	84810.80	-42266.78	5441.775	<.0001
SDNN: model with interaction, without AR	24	90023.33	90241.49	-44987.67		
rMSSD: model with interaction, with AR	25	105852.7	106080.0	-52901.36	9477.673	<.0001
rMSSD: model with interaction, without AR	24	115328.4	115546.5	-57640.19		
HR: model with interaction, with AR	24	-164960.9	-164742.7	82504.44	61340.9	<.0001
HR: model with interaction, without AR	23	-103622.0	-103412.9	51833.99		

HR: heart rate, SDNN: standard deviation of normal to normal RR intervals, rMSSD: root mean square of the successive differences, df: degrees of freedom

Table S4: Coefficients and 95% CI of the percent change in the mean outcome associated with a one unit increase for the 5-min windows. The intercept represents the geometric mean of the outcome at home when all variables are set to zero.

	HR (bpm)		SDNN (ms)		rMSSD (ms)	
	β	95% CI	β	95% CI	β	95% CI
Intercept	+69.870	[+68.158 to +71.625]	+93.939	[+86.973 to +101.464]	+83.936	[+75.002 to +93.934]
LAeq	+0.141	[+0.135 to +0.148]	+0.894	[+0.849 to +0.939]	+0.600	[+0.546 to +0.655]
LAeq(-5min)	+0.033	[+0.026 to +0.039]	−0.315	[−0.358 to −0.272]	−0.050	[−0.102 to +0.002]
Context(non-Home)	−0.330	[−0.896 to +0.240]	−0.198	[−1.482 to +1.102]	+2.202	[+0.271 to +4.169]
Context(Active)	+3.389	[+2.592 to +4.191]	−35.825	[−38.028 to −33.544]	−25.895	[−29.303 to −22.323]
Context(Priv.Mot)	−3.633	[−4.336 to −2.925]	−21.689	[−23.816 to −19.504]	−13.034	[−16.317 to −9.623]
Context(Public)	−2.010	[−3.046 to −0.964]	−15.721	[−18.848 to −12.474]	−14.460	[−18.818 to −9.867]
VM	+4.616	[+4.551 to +4.682]	+14.438	[+13.879 to +15.001]	+11.031	[+10.357 to +11.710]
CV(VM)	−0.160	[−0.180 to −0.139]	−0.404	[−0.574 to −0.234]	−0.557	[−0.753 to −0.360]
HR	-	-	−1.150	[−1.200 to −1.099]	−1.783	[−1.851 to −1.715]

HR: heart rate, SDNN: standard deviation of normal to normal RR intervals, rMSSD: root mean square of the successive differences, LAeq: equivalent continuous sound level of A−weighted Leq,1s, Active: active transport modes, Priv.Mot: private motorized transport modes, Public: public transport modes, VM: Vector magnitude (standardized), CV: Coefficient of variation (range: 0-1).

Table S5: Coefficients and 95% CI of the percent change in the mean outcome associated with a one unit increase for the 1-min windows. The intercept represents the geometric mean of the outcome at home when all variables are set to zero.

	HR (bpm)		SDNN (ms		rMSSD (ms)	
	β	95% CI	β	95% CI	β	95% CI
Intercept	+67.297	[+65.588 to +69.051]	+77.473	[+71.590 to +83.841]	+94.925	[+85.436 to +105.468]
LAeq	+0.112	[+0.105 to +0.119]	+0.755	[+0.698 to +0.813]	+0.526	[+0.460 to +0.592]
LAeq(-1min)	+0.076	[+0.069 to +0.083]	+0.180	[+0.119 to +0.241]	+0.141	[+0.072 to +0.210]
LAeq(-2min)	+0.016	[+0.009 to +0.023]	−0.129	[−0.190 to −0.068]	−0.067	[−0.136 to +0.002]
LAeq(-3min)	+0.016	[+0.009 to +0.023]	−0.060	[−0.122 to +0.001]	−0.025	[−0.094 to +0.045]
LAeq(-4min)	+0.016	[+0.008 to +0.022]	−0.160	[−0.217 to −0.104]	−0.059	[−0.124 to +0.006]
Context(non-Home)	−0.360	[−0.902 to +0.185]	+1.646	[+0.338 to +2.971]	+4.532	[+2.773 to +6.320]
Context(Active)	+7.239	[+6.471 to +8.013]	−20.314	[−22.116 to −18.471]	−5.093	[−7.947 to −2.152]
Context(Priv.Mot)	−2.804	[−3.555 to −2.048]	−17.553	[−19.155 to −15.920]	−8.248	[−10.678 to −5.752]
Context(Public)	−0.979	[−2.001 to +0.052]	−17.343	[−19.447 to −15.184]	−12.427	[−15.465 to −9.279]
VM	+5.152	[+5.067 to +5.237]	+8.087	[+7.446 to +8.732]	+8.534	[+7.743 to +9.330]
CV(VM)	+0.198	[+0.160 to +0.236]	+4.914	[+4.573 to +5.256]	+1.682	[+1.300 to +2.065]
HR	-	-	−1.329	[−1.372 to −1.286]	−2.102	[−2.156 to −2.047]

HR: heart rate, SDNN: standard deviation of normal to normal RR intervals, rMSSD: root mean square of the successive differences, LAeq: equivalent continuous sound level of A–weighted $L_{eq,1s}$, Active: active transport modes, Priv.Mot: private motorized transport modes, Public: public transport modes, VM: Vector magnitude (standardized), CV: Coefficient of variation (range: 0-1).

Table S6: Generalized variance inflation factors associated with each independent variable for the models based on the 5-min windows. Columns represent separate models.

	HR	SDNN	rMSSD
LAeq	1.12	1.29	1.17
LAeq (-5min)	1.06	1.21	1.08
Context	1.09	1.23	1.20
VM	1.23	1.71	1.69
CV(VM)	1.10	1.16	1.13
HR	-	1.55	1.57

HR: heart rate, SDNN: standard deviation of normal to normal RR intervals, rMSSD: root mean square of the successive differences, VM: Vector Magnitude, CV: Coefficient of variation, LAeq: equivalent continuous sound level of A–weighted Leq,1s

Table S7: Generalized variance inflation factors associated with each independent variable for the models based on the 1-min windows. Columns represent separate models.

	HR	SDNN	rMSSD
LAeq	1.05	2.25	1.91
LAeq (-1min)	1.04	2.58	2.11
LAeq (-2min)	1.04	2.62	2.12
LAeq (-3min)	1.04	2.60	2.11
LAeq (-4min)	1.03	2.24	1.89
Context	1.10	1.49	1.42
VM	1.12	1.67	1.61
CV(VM)	1.04	1.07	1.06
HR	-	1.56	1.52

HR: heart rate, SDNN: standard deviation of normal to normal RR intervals, rMSSD: root mean square of the successive differences, VM: Vector Magnitude, CV: Coefficient of variation, LAeq: equivalent continuous sound level of A−weighted Leq,1s

Table S8: Coefficients and 95% confidence intervals of the percentage change in the mean outcome associated with a one dB(A) increase in the sound level in the 5−minute windows. The coefficients for the main effect represent the association in the reference level (Home) while the coefficients of the interaction terms represent differences relative to the reference level. The total effect within each context is obtained by summing up the coefficients for the main and interaction effects.

	HR		SDNN		rMSSD	
	β	95% CI	β	95% CI	β	95% CI
LAeq (ref : Home)	+0.132	[+0.124 to +0.141]	+0.895	[+0.838 to +0.951]	+0.616	[+0.548 to +0.684]
LAeq (-5min) (ref : Home)	+0.039	[+0.031 to +0.047]	−0.238	[−0.292 to −0.184]	−0.012	[−0.078 to +0.054]
LAeq*non−Home	+0.024	[+0.011 to +0.038]	−0.000	[−0.090 to +0.090]	−0.070	[−0.179 to +0.038]
LAeq*Active	+0.146	[+0.082 to +0.210]	−0.052	[−0.503 to +0.402]	+0.460	[−0.083 to +1.006]
LAeq*Priv.Mot	−0.034	[−0.088 to +0.019]	+0.298	[−0.093 to +0.691]	+0.756	[+0.290 to +1.224]
LAeq*Public	−0.067	[−0.169 to +0.036]	−0.439	[−1.114 to +0.240]	−0.748	[−1.560 to +0.070]
LAeq (-5min)*non−Home	−0.017	[−0.031 to −0.004]	−0.218	[−0.308 to −0.129]	−0.112	[−0.220 to −0.003]
LAeq (-5min)*Active	+0.024	[−0.040 to +0.087]	−0.100	[−0.547 to +0.350]	+0.283	[−0.254 to +0.823]
LAeq (-5min)*Priv.Mot	−0.039	[−0.092 to +0.014]	−0.189	[−0.585 to +0.209]	−0.359	[−0.826 to +0.110]
LAeq (-5min)*Public	+0.011	[−0.090 to +0.111]	+0.707	[+0.042 to +1.376]	+0.296	[−0.503 to +1.101]

HR: heart rate, SDNN: standard deviation of normal to normal RR intervals, rMSSD: root mean square of the successive differences, LAeq: equivalent continuous sound level of A−weighted Leq,1s, Active: active transport modes, Priv.Mot: private motorized transport modes, Public: public transport modes.

Table S9: Coefficients and 95% confidence intervals of the percentage change in the mean outcome associated with a one dB(A) increase in the sound level in the 1−minute windows. The coefficients for the main effect represent the association in the reference level (Home) while the coefficients of the interaction terms represent differences relative to the reference level. The total effect within each context is obtained by summing up the coefficients for the main and interaction effects.

	HR		SDNN		rMSSD	
	β	95% CI	β	95% CI	β	95% CI
LAeq (ref : Home)	+0.112	[+0.102 to +0.122]	+0.791	[+0.718 to +0.864]	+0.534	[+0.448 to +0.619]
LAeq (-4min) (ref : Home)	+0.017	[+0.007 to +0.027]	−0.103	[−0.174 to −0.031]	+0.000	[−0.084 to +0.085]
LAeq*non−Home	+0.040	[+0.024 to +0.056]	−0.033	[−0.137 to +0.071]	−0.067	[−0.191 to +0.058]
LAeq*Active	−0.049	[−0.074 to −0.025]	−0.157	[−0.354 to +0.041]	+0.113	[−0.118 to +0.344]
LAeq*Priv.Mot	−0.029	[−0.055 to −0.004]	+0.071	[−0.130 to +0.272]	+0.326	[+0.088 to +0.563]
LAeq*Public	−0.077	[−0.112 to −0.042]	−0.551	[−0.841 to −0.260]	−0.511	[−0.849 to −0.172]
LAeq (-4min)*non−Home	+0.000	[−0.016 to +0.017]	−0.191	[−0.295 to −0.088]	−0.171	[−0.296 to −0.046]
LAeq (-4min)*Active	−0.003	[−0.028 to +0.021]	+0.024	[−0.174 to +0.222]	+0.051	[−0.180 to +0.283]
LAeq (-4min)*Priv.Mot	−0.010	[−0.034 to +0.015]	−0.105	[−0.302 to +0.093]	−0.176	[−0.408 to +0.056]
LAeq (-4min)*Public	−0.002	[−0.036 to +0.032]	+0.198	[−0.088 to +0.485]	+0.085	[−0.246 to +0.418]

HR: heart rate, SDNN: standard deviation of normal to normal RR intervals, rMSSD: root mean square of the successive differences, LAeq: equivalent continuous sound level of A−weighted Leq,1s, Active: active transport modes, Priv.Mot: private motorized transport modes, Public: public transport modes.

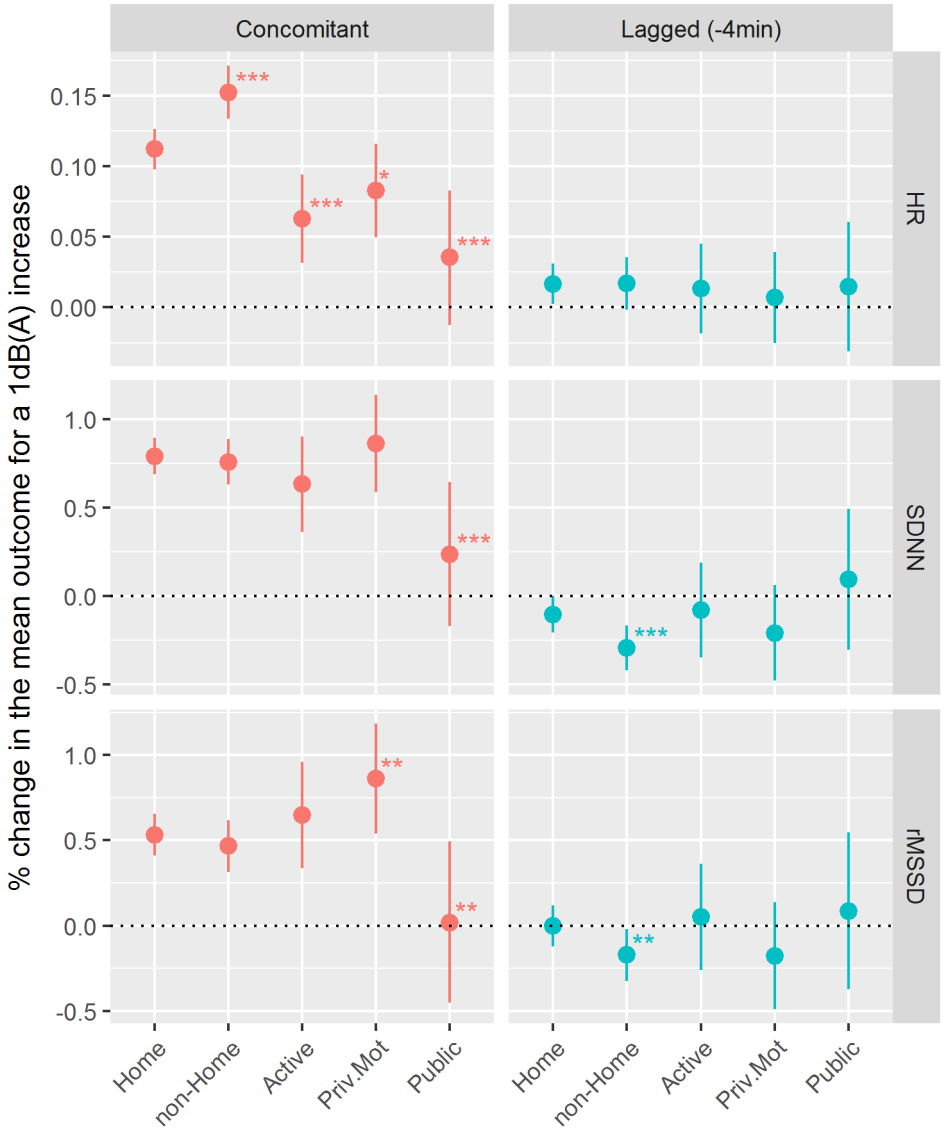

Figure S1: *Estimated associations (and their 95% CI) between concomitant and lagged sound levels and HR/HRV parameters in different mobility defined contexts using 1-min windows. Active: active transport modes, Priv.Mot: private motorized transport modes, Public: public transport modes. Asterisks denote a statistically significant difference with Home (reference level) *p<0.05, **p<0.01, ***p<0.001.*