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**Short-term association between personal exposure to noise and heart rate variability:  
the RECORD MultiSensor Study**

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## **Abstract**

**Background:** Studies revealed long-term associations between noise exposure and cardiovascular health, but the underlying short-term mechanisms remain uncertain.

**Objectives:** To explore the concomitant and lagged short-term associations between personal exposure to noise and heart rate variability (HRV) in a real life setting in the Île-de-France region.

**Methods:** The RECORD MultiSensor Study collected between July 2014 and June 2015 noise and heart rate data for 75 participants, aged 34 to 74 years, in their living environments for 7 days using a personal dosimeter and electrocardiography (ECG) sensor on the chest. HRV parameters and noise levels were calculated for 5-minute windows. Short-term relationships between noise level and log-transformed HRV parameters were assessed using mixed effects models with a random intercept for participants and a temporal autocorrelation structure, adjusted for heart rate, physical activity (accelerometry), and short-term trends.

**Results:** An increase by one dB(A) of A-weighted equivalent sound pressure level ( $L_{eq}$ ) was associated with a 0.97% concomitant increase of the Standard deviation of normal to normal intervals (SDNN) (95% CI: 0.92, 1.02), of 2.08% of the Low frequency band power (LF) (95% CI: 1.97, 2.18), of 1.30% of the High frequency band power (HF) (95% CI: 1.17, 1.43), and of 1.16% of the LF/HF ratio (95% CI: 1.10, 1.23). The analysis of lagged exposures to noise adjusted for the concomitant exposure illustrates the dynamic of recovery of the autonomic nervous system. Non-linear associations were documented with all HRV parameters with the exception of HF. Piecewise regression revealed that the association was almost 6 times stronger below than above 65  $L_{eq}$  dB(A) for the SDNN and LF/HF ratio.

**Conclusion:** Personal noise exposure was found to be related to a concomitant increase of the overall HRV, with evidence of imbalance of the autonomic nervous system towards sympathetic activity, a pathway to increased cardiovascular morbidity and mortality.

**Keywords:** Noise, Heart Rate Variability, Sensors, Autonomic nervous system.

## 1 INTRODUCTION

2 Previous literature has established the effects of noise exposure on health [1-3]. In addition to  
3 hearing impairment after repeated exposure to high noise levels, there are effects of noise on  
4 sleep quality, hypertension, and the development of cardiovascular disease [4]. WHO  
5 estimates that the number of healthy life years lost due to noise exposure is between 1 and 1.6  
6 million years for the Western Europe population each year [5]. This quantification takes into  
7 account the impact of ischemic heart disease, cognitive disorders in children, sleep disorders,  
8 and of "noise annoyance" caused by long-term exposure to noise.

9 The general theory of stress [6] serves as a basis for the hypothesis linking noise exposure,  
10 the autonomic nervous system and the endocrine system, the homeostasis of the human body  
11 [7], and in the long run the development of cardiovascular diseases [8]. Babisch et al. [9]  
12 distinguished an indirect effect of noise mediated by the subjective perception and cognitive  
13 interpretation of sounds and a direct effect based on the interaction of the acoustic nerve with  
14 other parts of the central nervous system, the two of which are expected to affect the  
15 autonomic nervous system.

16 The autonomic nervous system regulates different functions of the body including heart  
17 rate. The study of heart rate variability (HRV) thus enables to explore whether and how the  
18 autonomic nervous system is disrupted by noise levels. In addition, HRV has also been  
19 identified as a risk factor with a reduced HRV being associated with the occurrence of  
20 cardiovascular events [10].

21 Several studies have documented associations between noise exposure and an increased  
22 heart rate [11-13]. However, few studies have examined the link between exposure to noise  
23 and HRV. In an experimental setting, an increase of sympathetic activity was observed in  
24 subjects exposed to a noise level of 95 dB(A) Leq during 135 minutes [14]. Another  
25 experiment showed that a noise level as low as 45 dB(A) affected HRV parameters [15].

26 To our knowledge, only three studies were carried out with a non-simulated exposure. One  
27 study asked 40 healthy participants to sit for 2 hours either in a traffic area or in a park [16].  
28 Associations between short-term exposure to noise and decreased HRV were found as well as  
29 associations with the sympathovagal balance with an increased sympathetic activity and  
30 decreased parasympathetic activity. A similar imbalance was found in the second study, in  
31 which 36 healthy participants were instructed to follow a pre-determined route covering  
32 various sites in the city of Tel Aviv for two sequential days [17]. In the third study [18], the  
33 only one in a non-experimental setting we are aware of, 110 individuals underwent personal  
34 noise monitoring and continuous electrocardiography (ECG) 4 times every 4 to 6 weeks  
35 during their daily activities. Associations were documented between noise exposure and HRV  
36 parameters in concomitant windows.

37 Overall, previous literature is scarce and primarily based on controlled settings or  
38 experimental designs, raising the question of the generalizability of the findings. Thus the  
39 objective of this study was to explore the concomitant and lagged short-term associations  
40 between personal exposure to noise and HRV in a real life setting in the Île-de-France region.

41

## 42 **METHODS**

### 43 **Data collection and processing**

#### 44 Population

45 Participants came from the RECORD Cohort Study [19], and more particularly from the  
46 RECORD MultiSensor sub-study, which aimed at investigating the relationships between  
47 transport and health using sensor-based measurement. Participants of the RECORD Cohort  
48 were born between 1928 and 1978, were residing at baseline in 10 districts of Paris and 111  
49 other municipalities of the Ile-de-France region, and were recruited without a priori sampling  
50 during preventive checkups performed by the IPC Medical Centre between 2007 and 2008.

51 During the second wave of the RECORD Study, a fraction of the participants were invited  
52 to enter the RECORD MultiSensor Study, between July 2014 and June 2015. After  
53 completing their health checkups, these participants were asked to wear an accelerometer and  
54 an ECG sensor during their waking hours, from the day of completion of the checkup D0 up  
55 to D8. Since the checkup could happen at different hours during D0, only measurements  
56 between D1 (3 am) and D8 (3 am) were taken into account, i.e., 7 days of data collection.  
57 Among the 129 participants of this group, 78 also carried a personal noise dosimeter. The  
58 inner clock of each ECG sensor, accelerometer, and noise dosimeter was synchronized with  
59 the Internet Time of the computer before giving it to the participants. Participants wearing a  
60 pacemaker or with hearing problems were not included. Written informed consent was  
61 obtained from all participants. The RECORD Multisensor Study was approved by the French  
62 Data Protection Authority (CNIL).

63

#### 64 HRV parameters

65 The participants wore a BioPatch BHM 3 (Zephyr Technology, Annapolis, MD) on the chest,  
66 an ECG with two electrodes, a technology which was validated against a 12-lead ECG for  
67 HRV measurement [20]. They were instructed to keep it on from the moment they woke up  
68 until they went to bed since they had to charge it overnight. The two electrodes were changed  
69 every day. From the ECG sampled at 1 kHz, inter-beat (RR) intervals were generated by the  
70 BioPatch at an 18Hz frequency. HRV parameters were calculated based on these RR  
71 intervals. The entire signal processing was carried out under R version 3.4.0 [21] and the  
72 calculation of HRV parameters through the RHRV package version 4.2.3 [22].

73

74 *Data selection.* The raw data of RR intervals extracted from the BioPatch were cut into  
75 continuous observation sequences of any length, corresponding to the sequences remaining

76 after excluding periods where the sensor was not worn or where the sensor had lost contact  
77 with the skin. The continuous sequences of less than 20 beats were excluded at this stage.

78

79 *Filtering.* Based on the RHRV package, two types of filters were applied to RR intervals: a  
80 fixed filter retaining the physiologically acceptable values [25-200 bpm] and a dynamic filter  
81 comparing the value of the RR interval with the value of the preceding interval, the value of  
82 the following interval, and with an average of the values of the 50 preceding intervals [23]. If  
83 the absolute difference in percentage was lower than a given threshold for at least one of the  
84 comparisons, the interval was retained. The threshold was computed every 50 intervals, taking  
85 into account the standard deviation of these intervals, but is bounded between 12% and 20%.

86

87 *Interpolation.* After filtering, the remaining RR intervals were interpolated at a frequency of  
88 4Hz [24] using a cubic spline function [25] in order to produce a uniformly sampled signal.  
89 Empty sequences with more than 30s of continuous filtered RR intervals were not  
90 interpolated. Interpolation had a dual function. On one hand, producing a uniformly sampled  
91 signal enabled us to meet the prerequisites of frequency analysis. On the other hand, it  
92 generated values for the filtered RR intervals.

93

94 *Windowing.* Each day (24h) was cut into successive and mutually exclusive windows of 5  
95 minutes for a theoretical maximum of 288 windows per day. Windows having less than 200  
96 beats or with more than 20% of RR interval removed during the filtering step (even if  
97 subsequently re-interpolated) were excluded [26].

98

99 *HRV parameters calculation.* The standard deviation of normal to normal RR intervals  
100 (SDNN) was computed for each window. It was expressed in milliseconds. The frequency



101 domain parameters were extracted through a Daubechie Least Asymmetric(8) wavelet  
102 transform [27]. This method was selected for its ability to decompose non-stationary signals,  
103 unlike the Fourier transform [28]. The frequency bands were defined according to the  
104 recommendations [29]:

- 105 • Low Frequencies (LF): 0,04 – 0,15 Hz
- 106 • High Frequencies (HF): 0,15 – 0,40 Hz

107 The power of each band (in  $\text{ms}^2$ ) was computed, as well as the LF/HF ratio. Total Power  
108 (TP), and normalized LF (LFnu) and HF (HFnu) bands powers (each divided by the sum of  
109 HF and LF bands powers) were also computed, but not considered in the models because of  
110 the mathematical redundancy of HFnu and LFnu with the LF/HF ratio [30, 31] and TP with  
111 SDNN, since the first one represents the variance of HRV, while the second represents its  
112 standard deviation [29].

113 The four parameters (SDNN, LF and HF bands, and LF/HF ratio) were expressed as  
114 continuous variables in descriptive statistics and were log-transformed in the models in order  
115 to correct for heteroscedasticity.

116

### 117 Individual noise exposure

118 The assessment of individual noise exposure was performed with a wearable Class II  
119 dosimeter Wed007 - 01dB (ACOEM Limonest, France) allowing noise level measurements  
120 between 40 and 120 dB(A) (tolerance  $\pm 1.0$  dB) every second. The measurement was A-  
121 weighted [dB(A)], a weighting that corresponds to the sensitivity of the human ear. During  
122 the day, participants were instructed to wear the dosimeter on the belt while placing the  
123 microphone near the ear and above the clothing and charge the device overnight.

124 Similarly to HRV parameters, noise data were aggregated in 5-minute windows. This  
125 aggregation used the notion of equivalent sound level ( $L_{eq}$ ). The  $L_{eq}$  is a representation of

126 the constant noise level that would have been produced with the same energy than the noise  
127 actually perceived during the given period. It is expressed in dB and is calculated as follows:

$$128 \quad L_{eq} = 10 \log \times \frac{1}{T} \int_0^T 10^{\frac{L(t)}{10}} dt$$

129  $L_{eq}$ : equivalent sound level

130  $L(t)$ : noise level at time  $t$

131  $T$ : period's length in seconds

132 The noise level was used in its continuous form and expressed in  $L_{eq}$  dBA. For each 5-minute  
133 window, the noise level of the three preceding windows was also computed in order to  
134 represent lagged noise at -5 minutes, -10 minutes, and -15 minutes.

135

136 Individual exposure to sound level was assessed with a wearable Class II dosimeter Wed007 -  
137 01dB (ACOEM Limonest, France) allowing for A-weighted measurements - a weighting that  
138 corresponds to the sensitivity of the human ear - between 40 and 120 dB(A) (tolerance  $\pm 1.0$   
139 dB) every second ( $L_{Aeq,1s}$ ). During the day, participants were instructed to place the  
140 microphone near the ear and over the clothing, to wear the dosimeter on the belt and to charge  
141 the device overnight. All of the dosimeters were calibrated at the beginning of the study  
142 following the manufacturer's instructions using a standard acoustic calibrator (1 KHz sine  
143 wave at 94 dB).

144

145 Based on the A-weighted  $L_{eq,1s}$  ( $L_{Aeq,1s}$ ), the equivalent sound level ( $L_{Aeq}$ ) was computed  
146 within each time window. The  $L_{Aeq}$  is a representation of the constant sound level that would  
147 have been produced with the same energy than the varying sound level actually produced  
148 during the given period. It is one of the main sound level indicators used in environmental  
149 noise assessment [32].

150

151 Accelerometer data

152 Participants wore an Actigraph wGT3X+ tri-axial accelerometer on the right hip with a  
153 dedicated elastic belt for the recruitment day and 7 additional days. They were asked to  
154 remove the belt only when sleeping and when they were in contact with water. Accelerometry  
155 was collected for 5 second epochs and aggregated over the 5-minute windows. Vector  
156 magnitude was used as an indicator of physical activity in the regression analysis and was  
157 computed as follows:

158 
$$VM = \sqrt{Axis\ 1^2 + Axis\ 2^2 + Axis\ 3^2}$$

159

160 Other covariates

161 Sociodemographic and health variables were collected from the IPC medical questionnaire  
162 and RECORD questionnaire filled in during the health checkup. Age, sex, medical histories  
163 (of hypertension, myocardial infarction, angina, and angioplasty), occupation, and educational  
164 level were considered. Age was coded as a continuous variable and the 4 medical history  
165 variables as separate binary variables.

166 Education was coded in 4 categories: low (no education, primary education, or lower  
167 secondary education); medium-low (higher secondary education and lower tertiary education,  
168 i.e., 1 or 2 years); medium-high (intermediate tertiary education: 3 or 4 years); and high  
169 (upper tertiary education: 5 years or more). Employment status was divided into employed,  
170 unemployed, retired, and other employment statuses.

171

172 **Statistical analysis**

173 Linear mixed models applied to the 5-minute measurement windows were used to estimate  
174 associations between individual exposure to noise and HRV parameters. To take the repeated  
175 measures into account, a mixed model with a random intercept at the individual level was

176 used. Short-term trends over the day were taken into account with smoothing splines  
177 estimated for each participant. Preliminary analyses showed no long term-trend at the scale of  
178 the week or the year.

179 We successively estimated models with linear associations including only the concomitant  
180 noise exposure variable and models including together the concomitant and progressively  
181 added lagged noise variables before adjusting for time-varying variables (heart rate and  
182 accelerometer vector magnitude). We conducted sensitivity analyses limiting the dataset to  
183 windows with no filtered RR intervals and to windows with stationary RR sequences.  
184 Stationarity within each 5-minute window was assessed with the augmented Dickey–Fuller  
185 test [33].

186 Non-linear associations were then considered including second to third degree polynomials  
187 and natural cubic splines separately for concomitant noise and lagged noise. The linear or  
188 non-linear associations minimizing the Bayesian information criterion (BIC) were selected for  
189 the final models. In addition to the non-linear associations, piecewise regressions were  
190 considered in order to produce interpretable coefficients. A common breakpoint value was  
191 chosen for all the HRV parameters for ease of interpretation by looking at the breakpoints  
192 minimizing the BIC for each HRV parameter.

193 In the nonlinear association models and the piecewise regression, temporal autocorrelation  
194 between the repeated measurements of each participant was taken into account by an  
195 autoregressive model of order 1 AR(1) [34, 35]. This covariance structure assigns to each pair  
196 of measures of a participant a correlation that decreases with the increase of the time interval  
197 separating the measures. The correlation is expressed as  $\rho^k$ , where  $k$  is the time interval  
198 separating each pair of observations (number of 5-minute windows) and  $\rho$  the correlation of a  
199 pair of successive observations (range between 0 and 1) [36]. All analyses were performed in

200 R version 3.4.0 [21]. Mixed models were estimated with the nlme package version 3.1-131  
201 [37] and smoothing splines with the lmeSplines package version 1.1-10 [38].

202

## 203 **RESULTS**

### 204 **Sample description**

205 From the initial sample of 78 individuals, 14 129 and 6381 hours of measurements were  
206 collected by the noise dosimeter and ECG sensor respectively (unlike the noise sensor, the  
207 ECG sensor did not collect data during sleep). Three participants were excluded because  
208 either the ECG sensor, the noise dosimeter, or the accelerometer did not work or was not  
209 worn. Only windows with concurrent measures of noise level, HRV, and accelerometry were  
210 retained. Afterwards, windows with any missing noise data were removed ( $n = 60$ ), as were  
211 those where more than 20% of the RR intervals stemmed from the interpolation and / or those  
212 with less than 200 beats ( $n = 592$ ). In the end, the study sample considered in this work  
213 comprised 53 969 windows of 5 minutes (4497.4 hours in total) of concomitant noise, HRV,  
214 and accelerometry measurements for 75 individuals over 7 days. *Table 1* summarizes the  
215 participants' main characteristics.

216 The sample included individuals aged 34 to 74 years with an average age of 51.5 years  
217 (SD: 10.4). It was mainly composed of men (64%), employed people (65.3%), and people  
218 with a high level of education (52% of the participants had 3 or more years of tertiary  
219 education). Of the participants, 21 (28%) had a history of hypertension defined as “self-  
220 reported blood pressure equal to or greater than 140 mmHg repeatedly”, while 12 participants  
221 (16%) were taking blood pressure lowering medications. None of the participants had a  
222 history of myocardial infarction or angina pectoris.

223

### 224 **Measurement windows and noise levels**

225 As shown in *Table 2*, the distribution of the number of measurement windows was equivalent  
226 across the days (ANOVA test,  $p = 0.25$ ), with a decline at the end of the week. *Figure 1*  
227 shows the distribution of measurement windows by time across the day (average of all days).  
228 Most of the measures were taken between 8am and 10pm, an interval covering 87% of the  
229 observations.

230 *Figure 2* shows the histogram of measured noise levels, ranging from 32.6 to 113.7 Leq  
231 dB(A) with an average of 66.1 and a standard deviation of 10.9 Leq dB(A).

232

### 233 **Correlations between the cardiovascular parameters**

234 *Figure 3* represents the correlation between the different cardiovascular parameters. TP and  
235 SDNN are highly correlated ( $r = 0.94$ ) as the first one represents the variance of HRV and the  
236 second one its standard deviation. HFnu and LFnu have a perfect correlation of -1, while they  
237 both share with LF/HF a correlation of 0.79, positive for LFnu and negative for HFnu. LF and  
238 HF are correlated between them ( $r = 0.80$ ) as well as with both TP and SDNN, with  
239 correlations ranging from 0.59 to 0.79. Heart rate is mildly correlated with most of the HRV  
240 parameters with the exception of HF.

241

### 242 **Concomitant measures of noise and HRV**

243 *Table 3* provides the average values of heart rate and HRV parameters over four increasing  
244 noise level categories. The mean values of all parameters showed an increasing trend  
245 confirmed by the Jonckheere-Terpstra trend test.

246

### 247 **Mixed-effects models: linear associations**

248 *Table 4* reports the linear relationships of HRV parameters with (A) concomitant and  
249 progressively added lagged noise, (B) adjusted for heart rate and accelerometer vector

250 magnitude. Since the HRV parameters were log-transformed, the associations in the Table  
251 represent changes in percentage of the mean outcome for an increase of one Leq dB(A). In the  
252 models including only concomitant noise, positive associations were documented with all four  
253 HRV parameters. When adding lagged noise, these associations were pulled towards higher  
254 values, while lagged noise was systematically negatively associated with all HRV parameters,  
255 with however smaller magnitudes than the concomitant noise. When adjusted for heart rate  
256 and accelerometer vector magnitude, SDNN and LF and HF powers associations with  
257 concomitant and lagged noise were pulled towards the positive while the association of  
258 LF/HF with noise was pulled towards the negative. The variance inflation factor of the  
259 independent variables for every HRV parameter remained below 3 (Supplementary material  
260 I).

261 Models C and D in Table 4 report also the associations for (C) windows including no  
262 filtered RR intervals and (D) for windows with stationary RR sequences as a sensitivity  
263 analysis. There are no noticeable changes in term of direction of the associations. The  
264 coefficients were also quite stable between the different models, with two exceptions: (i) the  
265 reduction of the association of HF power with concomitant noise between models (B) and (C),  
266 moving from 1.30% to 0.86%, the effects of which are also observable on LF/HF; (ii) the  
267 reduction in the association between concomitant noise and SDNN between models (B) and  
268 (D), moving from 0.97% to 0.72%.

269 History of hypertension, blood pressure lowering medication intake, educational level, and  
270 employment status were not associated with any of the HRV parameters and were therefore  
271 not included in the models.

272

273 **Mixed-effects models: non-linear associations and piecewise regression**

274 *Figure 4* represents both the non-linear associations between concomitant noise and the HRV  
275 parameters, as well as the piecewise regression with concomitant and lagged noise for models  
276 adjusted for heart rate, accelerometer vector magnitude, and short-term trend with a temporal  
277 autocorrelation structure. The specifications of each model are available in the supplementary  
278 material II.

279 The best association, based on the BIC, between noise and the different HRV parameters  
280 was the natural cubic spline with the exception of HF power, for which it was the linear  
281 association. Regarding piecewise regression (numerical values shown in supplementary  
282 material III), a breakpoint at 65 Leq dB(A) was chosen based on the examination of which  
283 breakpoint ranging from 61 to 66 Leq dB(A) gave the best BIC for each HRV parameter.

284 For concomitant noise levels below 65 Leq dB(A), a quasi-linear increase was documented  
285 for all HRV parameters, starting at the lowest measured noise levels. Piecewise regression  
286 showed that that the slope of the association was much weaker above 65 Leq dB(A) than  
287 below this noise level, at the most 6 times weaker for the SDNN and LF/HF ratio.

288 As shown in Supplementary material III, the piecewise regression associations between  
289 lagged noise exposure variables and the HRV parameters were either negative or null (after  
290 mutual adjustment and adjustment for concomitant noise). Below 65 Leq dB(A), the different  
291 lagged noises were all negatively associated, with the exception of the 5 min lagged noise  
292 with LF and HF powers which did not show any association. Above 65 Leq dB(A), the lagged  
293 noise variables did not show any association with the HRV parameters, with the exception of  
294 the 5 min lagged noise associations with SDNN and LF/HF. In this piecewise regression  
295 analysis, there was no identifiable pattern of associations with increasing lag.

296

## 297 **DISCUSSION**

### 298 **Summary of results**



299 This study aimed to explore the relationship between individual acute exposure to noise and 4  
300 HRV parameters (SDNN, LF, HF, LF/HF ratio). Concomitant noise was positively associated  
301 with all 4 HRV parameters, after adjustment for heart rate, accelerometry, short-term trend,  
302 and even after controlling for the lagged noise variables. After mutual adjustment for the  
303 concomitant and lagged exposures, the lagged noise exposures were negatively associated  
304 with all 4 HRV parameters. Analyses restricted to sequences without removed RR intervals or  
305 to stationary RR sequences supported the same conclusions. Piecewise regression with a  
306 breakpoint at 65 Leq dB(A) demonstrated that the association was stronger below this  
307 threshold.

308

### 309 **Strengths and limitations**

310 First, the use of wearable sensors enabled to accurately measure continuously over time the  
311 personal noise exposure, HRV parameters, and physical activity as a confounder. We could  
312 reduce misclassification biases that would have resulted from the use of proxy indicators of  
313 noise exposure derived from interpolated measurements or modeling work. Similarly, this  
314 approach allowed us to escape from controlled laboratory environments and permitted  
315 observation in a “real life” context. This non-constrained observation over a week in various  
316 living environments yielded a wide range of situations of exposure to noise and related  
317 reactions of the autonomic nervous system.

318 A related strength of this study lies in the large number of observations that were collected.  
319 Indeed, a total of 4497.4 hours of concomitant noise, HRV, and accelerometry measurements  
320 from 75 individuals were analyzed in this study. This is considerably more than in the three  
321 other studies that have addressed the problem, that relied on 156 hours (n = 40),  
322 approximately 100 hours (n = 36), and 1785 hours (n = 110) of observation, respectively [16-  
323 18], although the third one had a larger number of participants.

324 However, as a weakness resulting from this strength, this considerable amount of data  
325 could only be handled by automated processes, made of filtering and calculation rules. Thus,  
326 each of the signal processing steps leading to the calculation of the HRV parameters may be a  
327 source of measurement bias [39]. First, the identification of beats and RR intervals from the  
328 ECG was performed by the internal algorithm of the BioPatch. Although its ability to  
329 correctly measure heart rate at different levels of physical activity has been verified by the  
330 manufacturer, the specificity and sensitivity of the algorithm are unknown, as well as their  
331 variation according to the wearer's activity. The filtering of ectopic and abnormal beats can  
332 also be a source of bias. Filtering was performed on the sequences of intervals, which provide  
333 limited information for the identification and selection of valid heartbeats. The filtering step is  
334 crucial for calculating the HRV parameters, since even a small proportion of ectopic beats can  
335 seriously affect the measurements [40]. While the approach to retain only sequences without  
336 removed RR intervals was explored in this study, it could however introduce problematic  
337 selection biases [29]. The same statement applies to restricting data to stationary sequences.

338 The subsequent signal processing steps can also be a source of heterogeneity between  
339 studies, affecting their comparability. The latest recommendations related to the measurement  
340 of HRV were drafted in 1996 [29]; they do not cover all processing stages and do not include  
341 methods introduced since then (e.g., wavelet transformation for calculating HRV frequency  
342 domain parameters). Various software are available for the calculation of HRV parameters,  
343 but it seems that no clear consensus has been reached as to the exact sequence of signal  
344 processing steps needed from the recording to the calculation of HRV parameters [39].

345 Another limitation of the study pertains to the lack of information about confounders like  
346 tobacco and alcohol consumption during the observation week, or personal air pollution  
347 measurements which effects on HRV have been previously described [16]

348 Finally, the study design allowed us to analyze a large number of 5-min windows, but the  
349 small number of participants in this study ( $n = 75$ ) limits the use of individual-level variables  
350 as stratifiers (the association estimated in each stratum would likely not represent the true  
351 association in this stratum).

352

## 353 **Interpretation of findings**

### 354 General framework

355 Through the complementary HRV parameters, it is the state of the autonomic nervous system  
356 that is being assessed. The SDNN is a global measure of HRV, reflecting the combined state  
357 of the two branches of the autonomic nervous system. It thus masks the modulations of HRV  
358 caused by each branch of the autonomic nervous system. This decomposition of HRV is  
359 however possible through frequency domain parameters breaking down HRV according to the  
360 frequencies of heart rate modulations. Two of these frequency bands were considered in this  
361 study: the low frequency band LF (0.04 to 0.15 Hz) and the high frequency band HF (0.15 to  
362 0.40 Hz). The HF band reflects parasympathetic activity [41] while the LF band, which was  
363 initially described as a reflection of the sympathetic system [42], is currently considered as the  
364 result of the combined effects of the two branches of the autonomic nervous system [43]. The  
365 LF/HF ratio is in turn an index of the sympathetic / parasympathetic balance.

366 Several studies have shown that a reduction of the SDNN is associated with the occurrence  
367 of cardiovascular events and with cardiovascular mortality [10, 44-46]. The Framingham  
368 Heart study [47] has found, of numerous HRV parameters, the SDNN to be the best predictor  
369 of new cardiac events with a reduction of one standard deviation being linked with a 50%  
370 increase in the risk of cardiovascular events over 3.5 years.

371

### 372 Concomitant associations

373 In this study, an increase in noise level was associated with a concomitant increase in SDNN,  
374 in the power of the LF band and the HF band, and in the LF/HF ratio. The concomitant  
375 increase in SDNN with noise stems from the association of noise with both the LF band  
376 power and HF band power, suggesting that noise exposure increased both the low frequency  
377 and high frequency modulations of heart rate. Thus in this study, a higher exposure to noise  
378 was not associated with an overall concomitant reduction in HRV, which was also observed in  
379 other studies [18, 48]. An increase in personal noise exposure was also associated with a  
380 concomitant increase in the LF/HF ratio. Together with the observed increases with noise of  
381 LF and HF band powers, this higher LF/HF ratio reflects a comparatively larger increase in  
382 sympathetic activity than parasympathetic activity, implying an imbalance of the autonomic  
383 nervous system associated with noise exposure. These results are in line with the reaction  
384 scheme formalized by Babisch and colleagues [9], as noise acts as a stressor triggering the  
385 fight or flight response with an activation of the sympathetic branch of the autonomic nervous  
386 system. This was documented by several studies with either simulated [49, 50] or non-  
387 simulated [17, 18] noise exposure. It is this imbalance that is conceptualized as a bridge  
388 between noise exposure and the subsequent development of cardiovascular diseases [9]. In  
389 terms of heart rate, this sympathetic dominance over the parasympathetic system leads to high  
390 heart rate values, as the first increases heart rate and the role of the second is to decrease it.  
391 This is consistent with our finding that higher noise levels were also associated with an  
392 increased heart rate in adjusted models (data not shown).

393

#### 394 Mutually adjusted lagged and concomitant associations

395 When associations with concomitant and lagged noise were mutually adjusted, the  
396 associations of concomitant noise with HRV parameters were slightly pulled towards the  
397 positive while lagged noise showed negative associations with HRV parameters. The negative

398 association between lagged noise and SDNN might express a recovery of the autonomic  
399 nervous system, after the increase in SDNN with a concomitant exposure to noise, which was  
400 of much larger magnitude. Kraus and colleagues reported somewhat comparable patterns of  
401 associations with, e.g., a positive association of concomitant noise with SDNN but negative  
402 associations of lagged noise variables with SDNN when such exposure variables were  
403 mutually adjusted for [18]. As speculated by Kraus and colleagues, this positive and negative  
404 associations of, respectively, concomitant and lagged noise with HRV parameters may be  
405 attributable to an overreaction and self-regulation of the autonomic nervous system but further  
406 research is needed in order to better understand those short-term dynamics.

#### 407 408 Non-linear and piecewise regression

409 With the exception of the HF band power, non-linear associations were documented between  
410 concomitant noise level and the HRV parameters. These associations shared a similar shape,  
411 with a decreasing slope as the noise level increased. This was explicitly reflected with the  
412 piecewise regression showing that the slope was much steeper below than above 65 Leq  
413 dB(A), mainly for the SDNN and the LF/HF ratio.

414 In our study, the associations of concomitant and lagged noise levels with HRV started at  
415 the lowest observed noise levels (around 40 Leq dB(A)) and reached a maximum after 65 Leq  
416 dB(A). Kraus and colleagues reported similar results in the case of the SDNN and the LF/HF  
417 ratio with a similarly chosen breakpoint at 65 Leq dB(A).

418 This threshold is however of limited clinical significance as it hides individual variations.  
419 It was slightly different depending on the HRV parameter, may have been influenced by the  
420 distribution of noise values, and does not strictly identify the beginning of the plateau but is  
421 close to where the biggest shift in slope occurs.

422

#### 423 **Conclusion**

424 In this study, a higher exposure to noise in real life settings was associated with increases in  
425 the LF and HF band powers, thus with an increase in the overall HRV (as expressed with the  
426 SDNN). Our analysis of the lagged noise exposures adjusted for the concomitant exposure  
427 showed evidence of a recovery starting after a 5-minute lag. Non-linear and piecewise  
428 regressions allowed us to identify a breakpoint at 65 dB(A) Leq, below which the reported  
429 association was much stronger. Future research will be needed to better understand the  
430 dynamics through which and timescales over which noise exposure influences the autonomic  
431 nervous system. Perspectives for this work include a better characterization of the daily  
432 activity of the participants during the observation windows in order to contextualize the  
433 reported association, the use of different summary measures for noise, and the consideration  
434 of the effects of air pollution in conjunction with those of noise to assess both their potential  
435 for reciprocal confounding and their synergistic effects on cardiovascular health.

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**Table 1** Descriptive statistics for the sample (N = 75)

<b>Variable</b>	<b>n (%)</b>
Men	48 (64%)
History of hypertension	21 (28%)
Intake of antihypertensive medications	12 (16%)
Employment status	
Employed	49 (65.3%)
Unemployed	11 (14.7%)
Retired	13 (17.3%)
Other	2 (2.7%)
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** Distribution of the 5-minute windows per participant over the week\* (n = 53969)

<b>Day</b>	<b>Mean <math>\pm</math> <math>\sigma</math></b>
Monday	103.1 $\pm$ 70.1
Tuesday	105.3 $\pm$ 64.0
Wednesday	111.9 $\pm$ 61.8
Thursday	111.4 $\pm$ 60.2
Friday	103.3 $\pm$ 61.4
Saturday	95.9 $\pm$ 57.7
Sunday	88.7 $\pm$ 60.7
ANOVA	p = 0.25

\*Number of follow-up days = 6.0  $\pm$  1.5;  
number of windows per participant =  
719.6  $\pm$  268.3

**Table 3** Mean and standard deviation of HRV parameters and heart rate according to four increasing categories of noise level

Noise Leq [dB(A)]	n	HR (bpm)		SDNN (ms)		LF/HF (w.u)		LF power (ms <sup>2</sup> )		HF power (ms <sup>2</sup> )	
		Mean	$\sigma$	Mean	$\sigma$	Mean	$\sigma$	Mean	$\sigma$	Mean	$\sigma$
[30,45]	2395	71.0	±11.7	50.5	±26.6	12.8	±9.4	1119.9	±1516.0	447.3	±753.1
(45,65]	19767	75.6	±13.0	61.0	±29.0	15.7	±9.9	1453.5	±1666.5	477.1	±721.4
(65,80]	27560	82.4	±14.3	62.7	±28.3	17.4	±10.2	1603.9	±1816.5	545.0	±850.5
(80,110]	4246	86.3	±15.1	59.5	±27.7	16.9	±10.0	1650.3	±1887.8	627.4	±941.9
Trend		p < 0.0001		p < 0.0001		p < 0.0001		p < 0.0001		p < 0.0001	

Abbreviations: HR: Heart rate; Leq [dB(A)]: A-weighted equivalent sound pressure level in dB; bpm: beats per minute; ms: milliseconds; w.u: without unit

Trend tested using Jonckheere-Terpstra test with the alternative hypothesis being “increasing”

**Table 4** Linear associations between concomitant and lagged noise exposure variables and log-transformed HRV parameters

	SDNN				LF/HF				LF				HF			
	0 min	5 min	10 min	15 min	0 min	5 min	10 min	15 min	0 min	5 min	10 min	15 min	0 min	5 min	10 min	15 min
A	+0.52				+1.23				+1.01				+0.37			
	+0.83	-0.45			+1.33	-0.15			+1.41	-0.59			+0.74	-0.53		
	+0.88	-0.23	-0.37		+1.35	-0.08	-0.12		+1.51	-0.21	-0.65		+0.83	-0.17	-0.62	
	+0.90	-0.21	-0.22	-0.25	+1.35	-0.08	-0.08	-0.07	+1.54	-0.17	-0.41	-0.41	+0.86	-0.13 <sup>ns</sup>	-0.39	-0.39
B	+0.97	-0.16	-0.17	-0.19	+1.16	-0.14	-0.12	-0.12	+2.08	+0.01 <sup>ns</sup>	-0.31	-0.30	+1.30	+0.04 <sup>ns</sup>	-0.28	-0.25
C	+0.95	-0.12	-0.21	-0.12	+1.41	-0.06 <sup>ns</sup>	-0.14	-0.15	+2.08	+0.03 <sup>ns</sup>	-0.33	-0.25	+0.86	-0.03 <sup>ns</sup>	-0.25	-0.15
D	+0.72	+0.03 <sup>ns</sup>	-0.19	-0.17	+1.11	-0.03 <sup>ns</sup>	-0.10 <sup>ns</sup>	-0.10 <sup>ns</sup>	+1.92	+0.01 <sup>ns</sup>	-0.25	-0.34	+1.23	-0.10 <sup>ns</sup>	-0.23 <sup>ns</sup>	-0.35

For each HRV parameter, each line represents a different model. Associations represent changes in percentage of the mean outcome for an increase of one Leq [dB(A)]. They were estimated from models with a random effect at the individual level and adjusted for short-term trends. Models A include only concomitant and progressively added lagged noise, while models B are additionally adjusted for heart rate and accelerometer vector magnitude. Models C include only windows without filtered RR (n = 17 321). Models D include only stationary RR sequences (n = 14 350).

The associations are statistically significant ( $p < 0.05$ ) unless stated otherwise.

Abbreviations: Leq [dB(A)]: A-weighted equivalent sound pressure level in dB; SDNN: Standard deviation of RR intervals; LF/HF: Low frequency to high frequency ratio; LF: Low frequency band power in  $\text{ms}^2$ ; HF: High frequency band power in  $\text{ms}^2$ ; ns: not significant.

**Fig. 1** Distribution of the analyzed 5-minute windows over the day

**Fig. 2** Histogram of measured noise levels in Leq dB(A)

**Fig. 3** Correlations between the cardiovascular parameters

**Fig. 4** Plot of the nonlinear and piecewise associations between concomitant or lagged noise and the 4 HRV parameters, estimated from models with a random effect at the individual level and a temporal autocorrelation structure, adjusted for heart rate, accelerometer vector magnitude, and short term trend

Figure 1

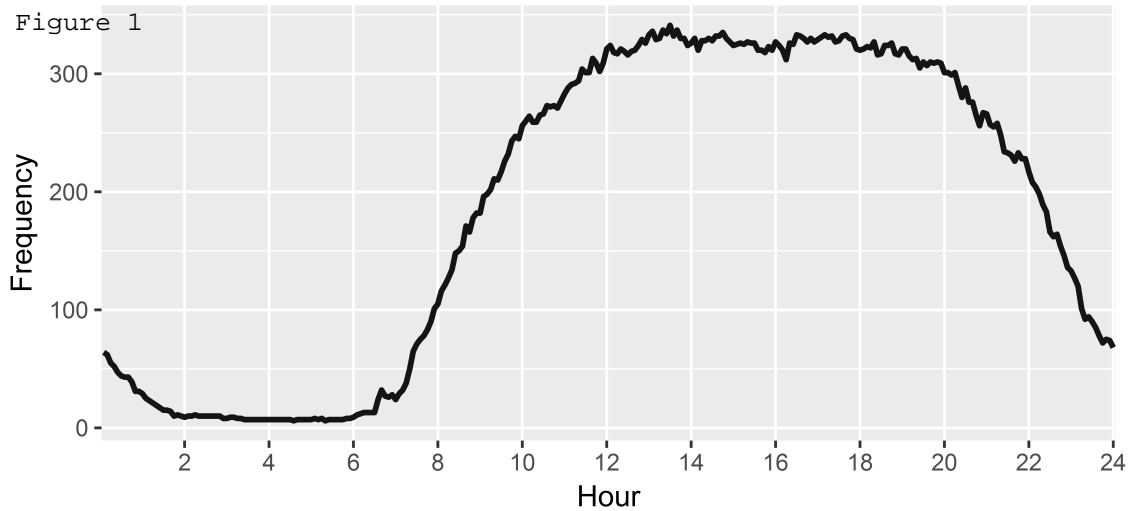
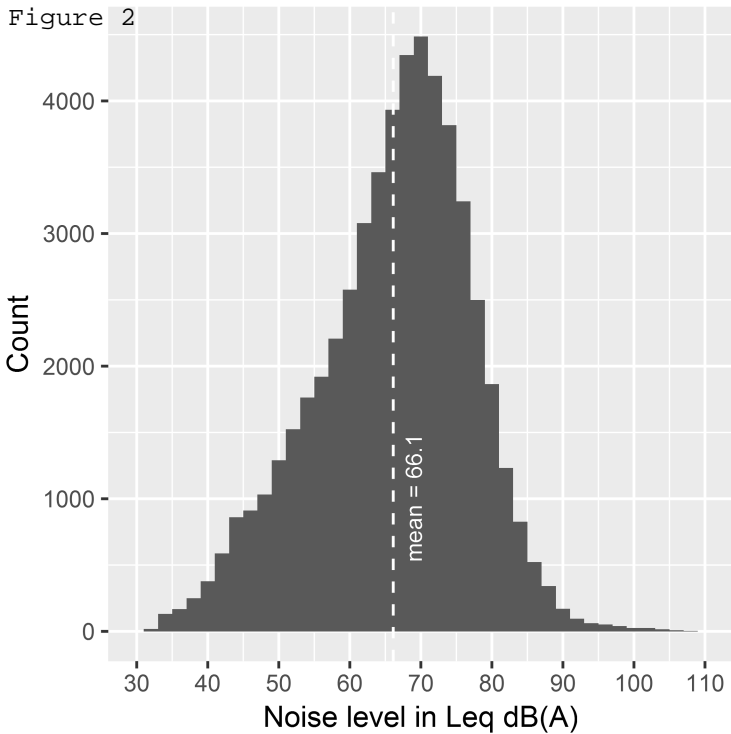


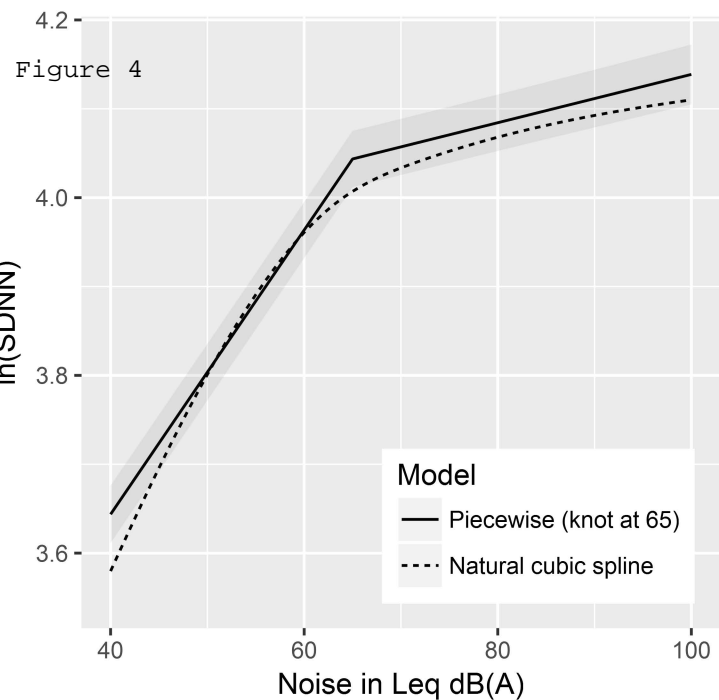
Figure 2





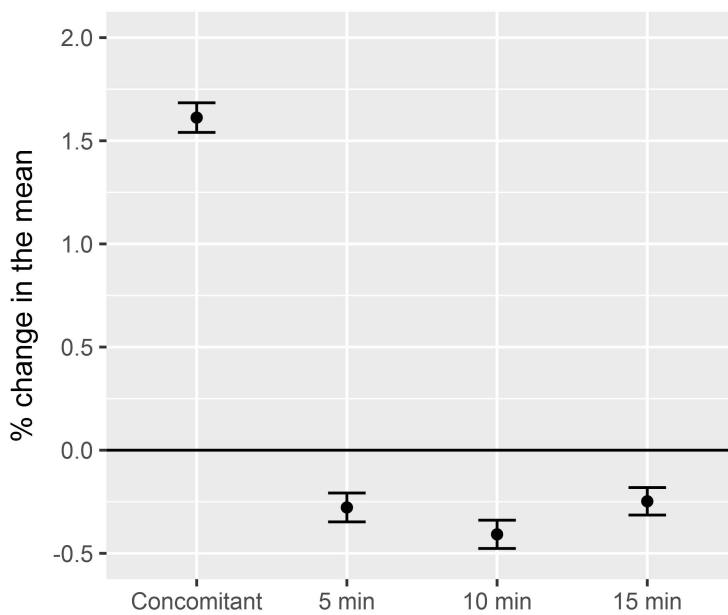


### Concomitant noise

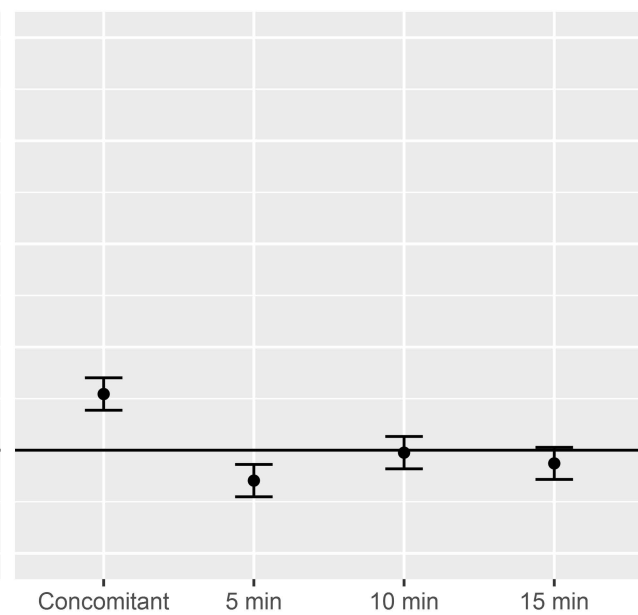


### SDNN

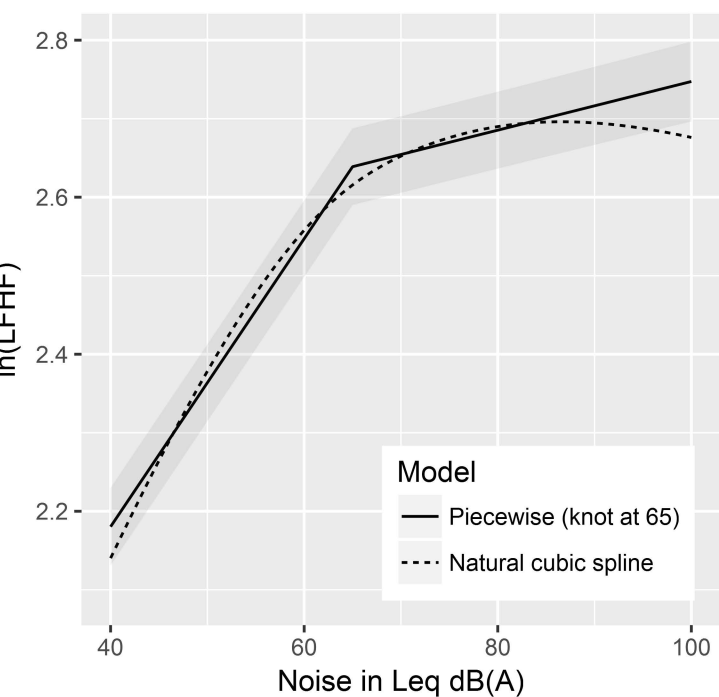
#### Noise < 65dB(A)



#### Noise > 65dB(A)

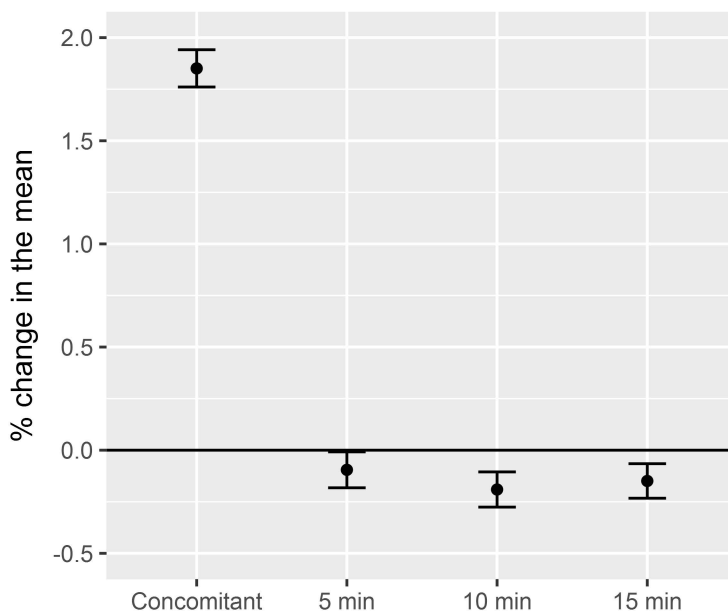


### Concomitant noise

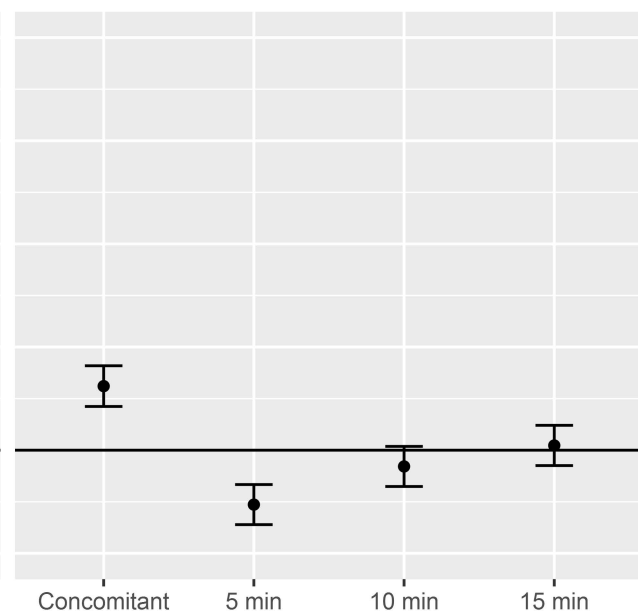


### LFHF

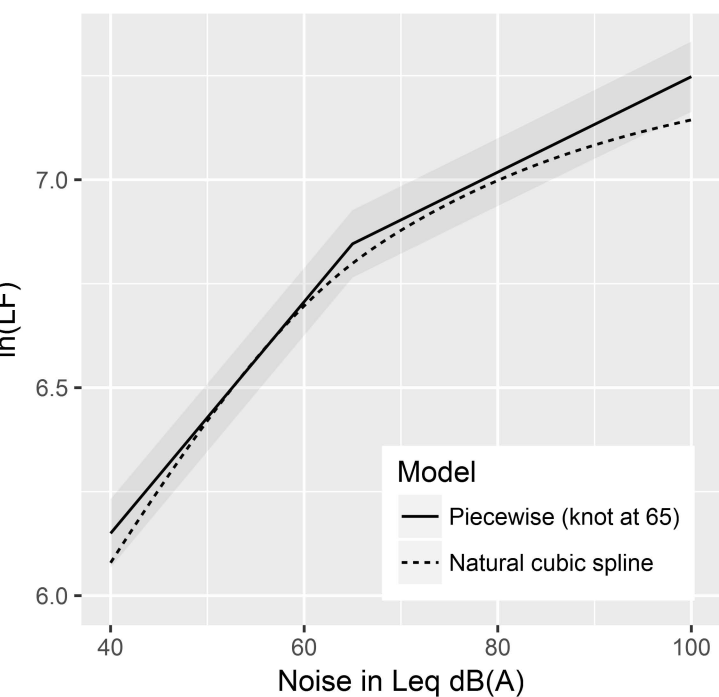
#### Noise < 65dB(A)



#### Noise > 65dB(A)

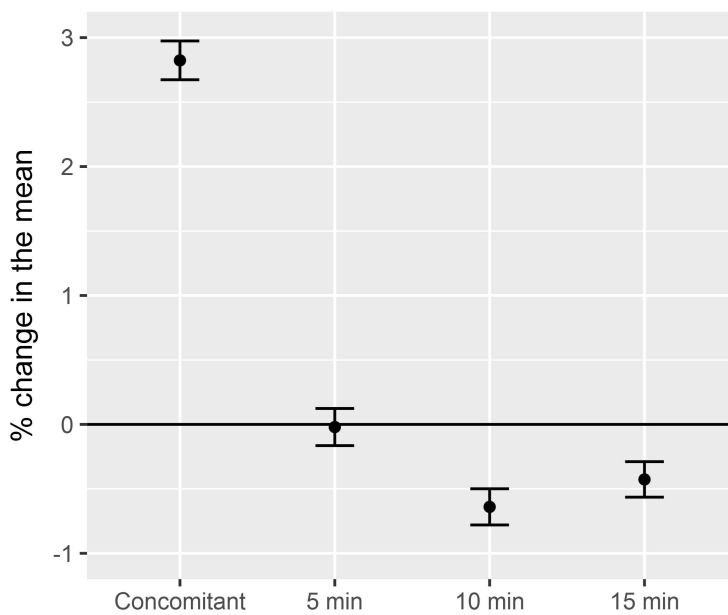


### Concomitant noise

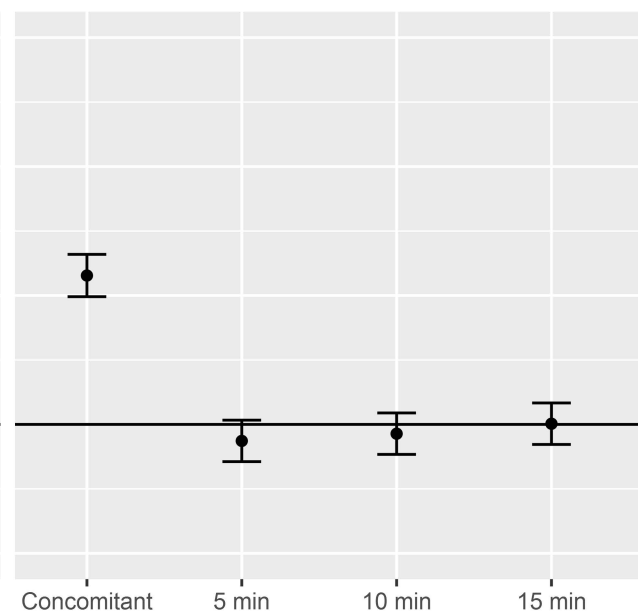


### LF

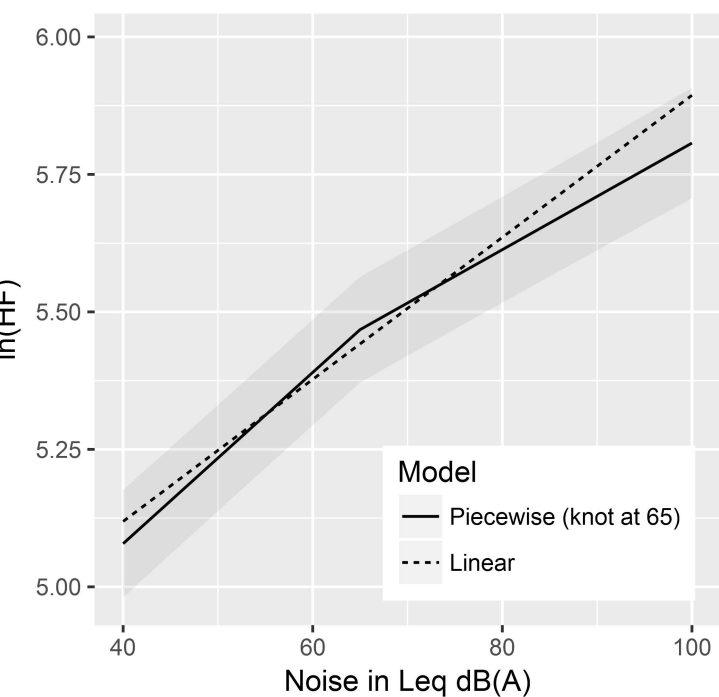
#### Noise < 65dB(A)



#### Noise > 65dB(A)

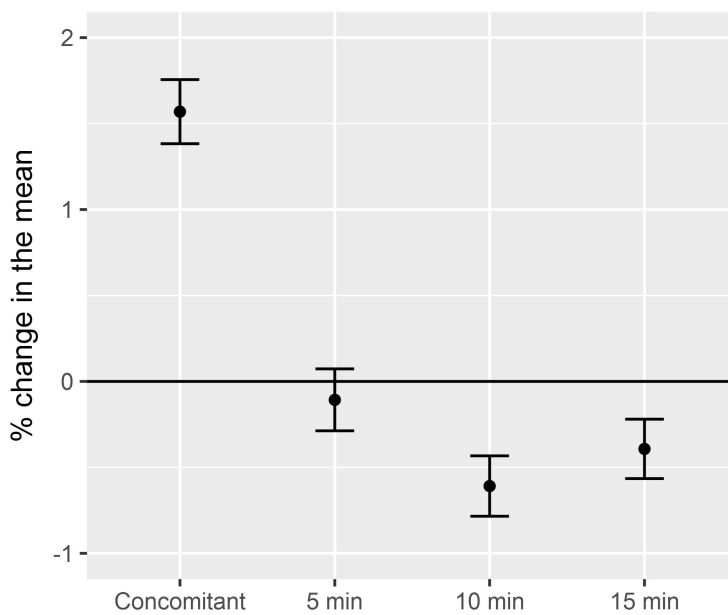


### Concomitant noise

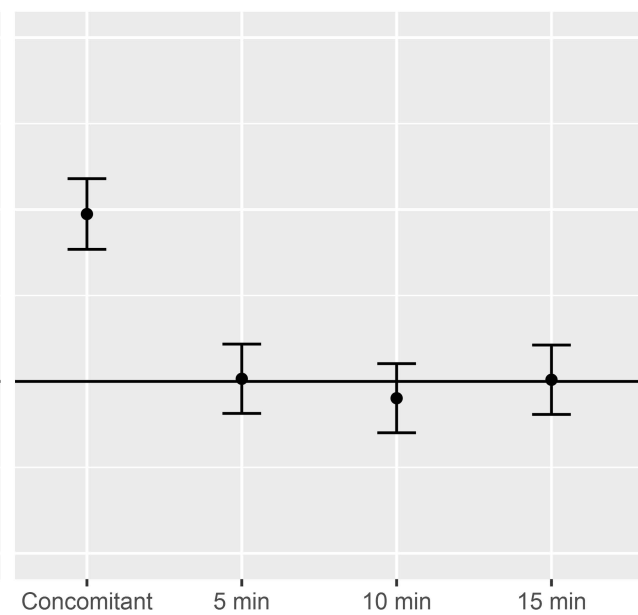


### HF

#### Noise < 65dB(A)



#### Noise > 65dB(A)



## Supplementary material

### I. Variance inflation factor of the linear association models

	<b>SDNN</b>	<b>LF/HF</b>	<b>LF</b>	<b>HF</b>
<b>Noise - 0 min</b>	2.07	2.03	2.05	2.05
<b>Noise - 5 min</b>	2.65	2.59	2.63	2.62
<b>Noise - 10 min</b>	2.65	2.59	2.62	2.62
<b>Noise - 15 min</b>	1.99	1.95	1.97	1.97
<b>Heart rate</b>	1.74	1.74	1.74	1.74
<b>Vector magnitude</b>	1.67	1.67	1.67	1.67

### II. Models' specification for the non-linear association models

$\ln(\text{SDNN}) = \text{ncs}(\text{Concomitant noise}) + \text{poly2}(\text{Lagged noise (5 min)}) + \text{poly2}(\text{Lagged noise (10 min)}) + \text{poly2}(\text{Lagged noise (15 min)}) + \text{Heart Rate} + \text{Vector magnitude}$

$\ln(\text{LFHF}) = \text{ncs}(\text{Concomitant noise}) + \text{poly1}(\text{Lagged noise (5 min)}) + \text{poly1}(\text{Lagged noise (10 min)}) + \text{poly1}(\text{Lagged noise (15 min)}) + \text{Heart Rate} + \text{Vector magnitude}$

$\ln(\text{LF}) = \text{ncs}(\text{Concomitant noise}) + \text{poly2}(\text{Lagged noise (5 min)}) + \text{poly2}(\text{Lagged noise (10 min)}) + \text{poly2}(\text{Lagged noise (15 min)}) + \text{Heart Rate} + \text{Vector magnitude}$

$\ln(\text{HF}) = \text{poly1}(\text{Concomitant noise}) + \text{poly1}(\text{Lagged noise (5 min)}) + \text{poly1}(\text{Lagged noise (10 min)}) + \text{poly1}(\text{Lagged noise (15 min)}) + \text{Heart Rate} + \text{Vector magnitude}$

All the models included a temporal autocorrelation structure and were adjusted for short term trends.

*Abbreviations:*

ncs = natural cubic spline with two boundary knots and two internal knots at the 33<sup>rd</sup> and 66<sup>th</sup> quantiles.

Polyx = x<sup>th</sup> degree polynomial

### III. Piece-wise regression: Numerical values

	<b>SDNN</b>	
	<65 dB(A)	>65 dB(A)
<b>Noise - 0 min</b>	+1.61 [+1.54 to +1.68]	+0.27 [+0.19 to +0.35]
<b>Noise - 5 min</b>	-0.28 [-0.35 to -0.21]	-0.15 [-0.23 to -0.07]
<b>Noise - 10 min</b>	-0.41 [-0.48 to -0.34]	-0.01 [-0.09 to +0.07]
<b>Noise - 15 min</b>	-0.25 [-0.31 to -0.18]	-0.06 [-0.14 to +0.01]
	<b>LF/HF</b>	
	<65 dB(A)	>65 dB(A)
<b>Noise - 0 min</b>	+1.85 [+1.76 to +1.94]	+0.31 [+0.21 to +0.41]
<b>Noise - 5 min</b>	-0.10 [-0.18 to -0.01]	-0.26 [-0.36 to -0.17]
<b>Noise - 10 min</b>	-0.19 [-0.28 to -0.11]	-0.08 [-0.18 to +0.02]
<b>Noise - 15 min</b>	-0.15 [-0.23 to -0.07]	+0.02 [-0.07 to +0.12]
	<b>LF</b>	
	<65 dB(A)	>65 dB(A)
<b>Noise - 0 min</b>	+2.82 [+2.67 to +2.97]	+1.15 [+0.99 to +1.32]
<b>Noise - 5 min</b>	-0.02 [-0.16 to +0.12]	-0.13 [-0.29 to +0.03]
<b>Noise - 10 min</b>	-0.64 [-0.78 to -0.50]	-0.07 [-0.23 to +0.09]
<b>Noise - 15 min</b>	-0.43 [-0.56 to -0.29]	+0.01 [-0.16 to +0.17]
	<b>HF</b>	
	<65 dB(A)	>65 dB(A)
<b>Noise - 0 min</b>	+1.57 [+1.38 to +1.76]	+0.97 [+0.77 to +1.18]
<b>Noise - 5 min</b>	-0.11 [-0.29 to +0.07]	+0.02 [-0.19 to +0.22]
<b>Noise - 10 min</b>	-0.61 [-0.78 to -0.43]	-0.10 [-0.30 to +0.10]
<b>Noise - 15 min</b>	-0.39 [-0.57 to -0.22]	+0.01 [-0.19 to +0.21]

The coefficients represent changes in percentage of the mean outcome for an increase of one Leq dB(A). 95% confidence interval are reported.