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Cardiovascular and stress responses to short-term noise exposures—A panel study in healthy males

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ABSTRACT

Background: While previous epidemiological studies report adverse effects of long-term noise exposure on cardiovascular health, the mechanisms responsible for these effects are unclear. We sought to elucidate the cardiovascular and stress response to short-term, low (31.5–125 Hz) and high (500–2000 Hz) frequency noise exposures.

Methods: Healthy male (n = 10) participants were monitored on multiple visits during no noise, low- or high-frequency noise exposure scenarios lasting 40 min. Participants were fitted with an ambulatory electrocardiogram (ECG) and blood pressure measures and saliva samples were taken before, during and after noise exposures. ECGs were processed for measures of heart rate variability (HRV): high-frequency power (HF), low-frequency power (LF), the root of the mean squared difference between adjacent normal heart beats (N-N) intervals (RMSSD), and the standard deviation of N-N intervals (SDNN). Systolic blood pressure (SBP), diastolic blood pressure (DPB), and pulse were reported and saliva was analyzed for salivary cortisol and amylase. Multivariate mixed-effects linear regression models adjusted for age were used to identify statistically significant difference in outcomes by no noise, during noise or after noise exposure periods and whether this differed by noise frequency.

Results: A total of 658, 205, and 122, HRV, saliva, and blood pressure measurements were performed over 41 person days. Reductions in HRV (LF and RMSSD) were observed during noise exposure (a reduction of 19% (-35,-3.5) and 9.1% (-17,-1.1), respectively). After adjusting for noise frequency, during low frequency noise exposure, HF, LF, and SDNN were reduced (a reduction of 32% (-57,-6.2), 34% (-52,-15), and 16% (-26,-6.1), respectively) and during high frequency noise exposure, a 21% (-39,-2.3) reduction in LF, as compared to during no noise exposure, was found. No significant ($p < 0.05$) changes in blood pressure, salivary cortisol, or amylase were observed.

Conclusions: These results suggest that exposure to noise, and in particular, to low-frequency noise, negatively impacts HRV. The frequencies of noise should be considered when evaluating the cardiovascular health impacts of exposure.

1. Introduction

Noise, defined as unwanted sound, is a ubiquitous environmental and occupational stressor. While noise is quite common, it is a complex exposure due to its varying subjective (annoyance and sensitivity) and objective (loudness, frequency/pitch) characteristics. The effects of noise on hearing are well elucidated. However, in recent years particular interest has been in parsing out its effects on cardiovascular health. Associations of *long-term* noise exposures with actual disease manifestation such as hypertension, myocardial infarction, and ischemic heart disease have been observed (Basner et al., 2014; Babisch, 2011). What is less clear, is an understanding of the underlying mechanisms responsible for these cardiovascular effects as well as the role the subjective and objective components play in mediating these effects.

It is hypothesized that noise affects cardiovascular health through a stress mechanism via the autonomic nervous system and endocrine system. Over short time periods, noise exposed individuals experience increases in blood pressure, changes in heart rate variability (HRV), and the secretion of stress hormones including cortisol and amylase (Basner et al., 2014; Chang et al.,

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2009). Over longer time periods, continued exposure-response stress loops begin to affect the homeostasis of the human organism, giving rise to risk factors such as increased blood pressure, increased blood lipid concentrations, lower blood viscosity, and increased blood glucose concentrations that are well known for promoting the development of poor cardiovascular health (Basner et al., 2014).

In experimental studies, the stress mechanism hypothesis has been tested using short-term exposure to noise and biological stress response measures of blood pressure, HRV, salivary amylase, and cortisol. Results of such studies are mixed. For blood pressure, while Lee et al. (2010) found no changes across noise exposed groups, Lusk et al. (2004) and Zamanian et al. (2013) observed increased blood pressure with noise exposure. Likewise, Björ et al. (2007) found no changes in HRV between noise exposure groups; however, both Lee et al. (2010) and Kraus et al. (2013) found significant changes in HRV in groups exposed to higher noise intensities. Wagner et al. (2010) showed significant increases in both salivary cortisol and amylase after exposure to noise.

One of the major limitations of mechanistic studies of noise and stress is their implicit assumption that the sound pressure level is the most relevant characteristic. What is less known, however, is the influence of noise frequency on the stress response. The importance of considering noise frequency comes primarily from laboratory studies. These types of studies suggest that our body's organs respond to different frequencies differentially, with low-frequency noise being especially deleterious (Alves-Pereira and Branco, 2007). Studies of the effects of noise frequency on human health beyond the laboratory are quite limited. In an observational study of a population of oil mill workers in India, Kumar et al. (2008) found that with workers exposed to noise frequencies ranging from 350 to 700 Hz, roughly a third of them had cardiovascular problems (Kumar et al., 2008). Experimentally, a panel study found increased salivary cortisol levels in individuals exposed to low-frequency noise as compared to white noise (Waye et al., 2003).

We conducted a pilot study to investigate: (1) whether noise exposure produced acute changes in stress and cardiovascular responses; and (2) whether these responses differed based on noise frequency. Using a panel study design where participants were monitored on multiple visits during no noise, low- or high-frequency noise exposure scenarios, we evaluated changes of cardiac autonomic function as measured by HRV and blood pressure and on the endocrine system as measured by salivary cortisol and amylase.

2. Materials and methods

2.1. Study population and design

Between May and June of 2012, study participants were recruited to participate in this pilot study using a flyer placed in common areas of the UConn Health campus, a broadcast on a television screen within the UConn Health cafeteria, and through a broadcast email message sent to UConn Health employees. Upon scheduling of the study visit, the potential participants were screened to determine eligibility (male, 18–40 years old, no known hearing loss, and free from treated high blood pressure, known heart disease including irregular rhythm, heart failure, heart surgery, and history of heart attack). Participants were instructed to refrain from eating and drinking (water excluded) and from listening to loud music in the car or via headphones for 2.5 h prior to all subsequent study visits.

At the first visit, prior to beginning the study protocol, study participants gave informed consent and completed a standard audiometric screening to confirm normal hearing. Persons with pure-tone, air conduction hearing threshold levels determined by audiometry at frequencies from 125 to 8000 Hz of 20 dB hearing level or more were ineligible. The first visit lasted approximately 3 h due to the audiometric testing. The remaining visits lasted approximately 2 h each. All study methods were approved by UConn Health's and Harvard T.H. Chan School of Public Health's Institutional Review Boards.

Table 1
Study protocol and sampling scheme.

Time Period	Acclimation	Before	During			After		
	(10 min)	(10 min)	(40 min)			(30 min)		
Noise exposure scenarios								
Background 50 dB(A)	No noise	No noise	No noise			No noise		
High frequency 75 dB(A)	No noise	No noise	During noise			After noise		
Low frequency 75 dB(A)	No noise	No noise	During noise			After noise		
Biological sampling								
ECG monitor		X	X	X	X	X	X	X
Blood pressure		Y				Y		
Saliva		Y		Y		Y	Y	Y

Statistical analysis was performed by considering three exposure periods (no noise, during noise, and after noise). X indicates continuous ECG monitoring. Y indicates that a biological measurement occurred.

The study was performed in a reverberation room within the UConn Health Acoustics Laboratory. Study enrollees were asked to participate in up to five visits, during which they experienced different noise exposure scenarios including either: 1) no noise exposure (up to one visit); 2) low-frequency noise exposure (up to two visits); or 3) high-frequency noise exposure (up to two visits). The order of the scenarios was randomly administered with at least one day between scenarios. Individuals were scheduled during the same time of day, within an hour, for each session to control for natural circadian rhythm.

The study protocol for each visit is presented in Table 1. Each study visit was broken into four study periods. After a 10 min acclimation period with no noise exposure, participants spent an additional 10 min in the noise chamber with no noise exposure. Next, participants experienced one of the noise exposure scenarios (no noise, low-frequency noise, or high-frequency noise) for 20 min after which time the noise was stopped for 5 min for saliva monitoring followed by an additional 20 min of noise exposure. Finally, participants remained in the chamber for an additional 30 min “after noise” during which they were not exposed to any noise. Participants remained seated within the noise chamber for the duration of the study. Peaceful nature videos (without sound) were shown to the participant throughout their entire time in the noise chamber.

2.2. Noise exposures

Loudspeakers were positioned within the reverberation room at a standardized location for each participant and provided high- or low-frequency noise as needed. For the “low-frequency” scenario, the noise exposure was a low-frequency noise dominated by sound in the frequency range from 31.5 to 125 Hz with an overall sound level of 75 dB(A). For the “high-frequency” scenario, the noise exposure was dominated by sound in the frequency range from 500 Hz to 2 kHz at 75 dB(A). This sound level was chosen to avoid noise-induced changes in hearing threshold (Miller, 1974). For “no noise” exposure and periods of time before noise exposure, the average sound level in the reverberation room was 50 dB(A)

(i.e., the background noise). During each scenario, noise levels and frequencies were monitored with a calibrated sound level meter (Bruel and Kjaer, type 2260).

2.3. Stress and cardiovascular responses

To measure HRV, participants were outfitted with a standard 5-lead ambulatory GE SEER Light ECG Holter monitor that was worn for the duration of the experiment. Recordings were analyzed in the time and frequency domains for HRV by the Cardiovascular Epidemiology Group of Beth Israel Hospital, Boston, MA. Trained technicians, blinded to exposure, used standard criteria to identify and label all normal or abnormal beats. For the purposes of our analysis, HRV was summarized using the frequency domain measures of high-frequency power (0.15–0.40 Hz) (HF), primarily reflecting the activity the parasympathetic nervous system (PNS), and low-frequency power (0.04–0.15 Hz) (LF), reflecting the activity of both the sympathetic (SNS) and PNS. The time domain measures used included RMSSD, the root of the mean squared difference between adjacent normal heart beats (N-N) intervals, which represents a measure of overall degree of HRV and SDNN, the standard deviation of N-N intervals, which measures total heart rate variability (Kraus et al., 2013; Lee et al., 2010).

Systolic and diastolic blood pressure (SBP and DBP, respectively), as well as pulse measurements were taken using a standard blood pressure monitor at three times during the course of the experiment: at the end of the initial 20 min "no noise" period immediately before the noise exposure scenario commenced, immediately after the noise exposure scenario, and at the end of the "after noise" period (Table 1). Saliva was collected in sampling tubes using plastic straws at five times during the course of the experiment (Table 1). Participants were asked to use the straw to produce approximately 1 mL of saliva, directly into a 2 mL sample tube. Following collection, saliva samples were transferred for storage at −80 °C within 90 min of sample collection. Saliva was analyzed for salivary cortisol and alpha amylase using standard radio-immuno assay by the UConn Health Center Clinical Core Laboratory.

2.4. Statistical analysis

Heart rate variability data were averaged over five minutes to ensure a uniform time series. Three exposure periods were considered for our analysis: (1) 'No Noise', which included the baseline scenario as well as before any noise exposure commenced during the low- or high-frequency exposure scenario; (2) 'During Noise', which included the time during the low- or high-frequency noise exposure scenarios; and (3) 'After Noise', which included the time after the low- or high-frequency noise exposure study periods. Descriptive statistics (geometric mean, geometric standard deviation, minimum, maximum, range) were calculated for all health measures. We estimated mean levels and 95% confidence intervals (CIs) of salivary amylase and cortisol, blood pressure (SBP, DPB, and pulse) and HRV (HF, LF, RMSSD, SDNN) using mixed models, which accounted for the correlation of repeated measures within persons. An autoregressive covariance matrix was chosen as it was shown to minimize Akaike's information criterion.

Model 1 adjusted for age and exposure period, while Model 2 additionally adjusted for noise frequency and included an interaction of exposure and noise frequency.

The specification for both models is:

Model 1

$$Log\ Y_{ij}=\beta_0+\beta_1 Age_i+\beta_2 ExposurePeriod_{ij}+\varepsilon_{ij}$$

Where Y_{ij} = HRV, salivary amylase and cortisol, SBP, DBP, and pulse; i= individual (1–12) and j= visit (1–5), Exposure Period is a categorical variable with three categories: no noise (reference), during noise, or after noise; and age is linear.

We further evaluated the effect of noise frequency on the biological measures and included an interaction term (Model 2).

Model 2

$$Log\ Y_{ij}=\beta_0+\beta_1 Age_i+\beta_2 ExposurePeriod_{ij}+\beta_3 NoiseFrequency_{ij}+\beta_4 ExposurePeriod_{ij}*NoiseFrequency_{ij}+\varepsilon_{ij}$$

where Noise Frequency is a categorical variable with three categories: background (reference), low frequency, and high frequency where the noise is predominated by low-frequency sound or high-frequency sound. Effect estimates were multiplied by 100 and are presented as a percent.

Response variables were log-transformed to ensure normally-distributed residuals. Residual plots were assessed to check the normality of the residuals and fit of the model. All analyses were conducted using SAS (version 9.4; SAS Institute Inc., Cary, N.C.).

3. Results

Overall, we had a total of 13 study participants. However, three participants were excluded in our analysis—one for of violating study protocol and two because they only completed a "no noise" noise exposure. The mean age was 26 years, with a standard deviation of approximately 8 years. Of the 10 participants, 9 participated in the "no noise" exposure scenario, 7 in at least one of the low-frequency scenarios, and 9 in at least one of the high-frequency scenarios. Six participants completed all six sessions and 7 completed at least one of each of the exposure scenarios for a total of 41 person-days of monitoring. We obtained a total of 658, 205, and 122, HRV, saliva, and blood pressure measurements, respectively.

Descriptive statistics of these biological measurements throughout the course of the experiment are presented in Table 2. Mean HRV and salivary stress markers generally decreased during noise exposure, followed by an increase afterward. Pulse and systolic blood pressure increased after noise exposure, while diastolic blood pressure decreased.

The results from linear mixed model 1, which shows the association between the biological measures and exposure period, are shown in Table 3. While all HRV and salivary measures decreased during noise exposure, statistically significant declines were observed with LF and SDNN. All HRV parameters and salivary amylase had statistically significant increases after noise exposure, while cortisol continued to decrease after noise exposure. Systolic blood pressure decreased after noise exposure while pulse pressure increased. Both of these observed effects were statistically significant.

In Model 2 (Table 3), we considered the separate effects of low- and high-frequency noise exposures, using an interaction term. Similar to Model 1 results, all HRV parameters decreased during noise exposure. During low-frequency noise exposure, HF, LF, and SDNN all statistically significantly declined. During high-frequency noise exposure, only LF experienced statistically significant declines. While RMSSD decreased during both high- and low-frequency noise exposure scenarios, this effect was not statistically significant. Following noise exposure, HF, LF, RMSSD, and SDNN all increased. However, statistically significant increases were observed only during high-frequency noise exposure and only for HF.

For salivary amylase, declines were observed during low-frequency noise exposure while increases were observed during high-frequency noise exposure. However, neither of these effects were statistically significant. In contrast, there were small increases in cortisol during both low- and high-frequency noise

Table 2
Descriptive statistics of HRV, saliva, and blood pressure parameters by exposure period.

	No Noise					During Noise					After Noise				
	GM	GSD	Min	Max	Range	GM	GSD	Min	Max	Range	GM	GSD	Min	Max	Range
HRV Parameters			n=233					n=244					n=181		
LF (msec^2)	1999	59.1	199	12390	12191	1835	95.3	237	14489	14252	1839	82.9	426	13458	13033
HF (msec^2)	1070	49.3	33.1	9293	9260	920	77.3	54.4	11119	11064	989	69.7	90.3	10707	10617
RMSSD (msec)	45.9	0.96	10.2	138	128	42.7	1.64	13.5	122	109	44.7	1.44	17.1	136	119
SDNN (msec)	87.6	1.31	26.1	207	181	82.5	2.24	28.7	200	172	84.1	1.83	40.1	201	161
Saliva Parameters			n=81					n=31					n=93		
Amylase (U/mL)	96.1	7.8	9.5	342	332	86.1	9.6	15.7	190	174	105	7.7	20.0	344	324
Cortisol (U/dl)	0.13	0.008	0.03	0.50	0.47	0.13	0.01	0.04	0.34	0.30	0.12	0.01	0.04	2.71	2.67
Blood Pressure			n=61					n=0					n=61		
Diastolic (mm Hg)	73.5	0.91	53.5	103	49.0	*					75.5	0.93	60.0	99.0	39.0
Systolic (mm Hg)	114	1.21	99.5	143	43.0	*					113	1.07	99.0	135	36.0
Pulse (bpm)	64.0	1.29	47.5	86.5	39.0	*					60.6	1.31	48.5	85.5	37.0

* Blood pressure measurements were not taken during the noise exposure period.

Table 3
Percent change (95% CI) in stress and cardiovascular response by exposure period and noise frequency.

	Model 1		Model 2			
			Low frequency		High frequency	
HRV Parameters B(95% CI)	During noise	After noise	During noise	After noise	During noise	After noise
HF	−11 (−31, 8.6)	32 (0.12, 52)	−32 (−57,−6.2)*	18 (−8.1, 43)	−8.8 (−34, 16)	28 (2.9, 53)*
LF	−19 (−35,−3.5)*	16 (−0.2, 31)	−34 (−52,−15)*	2.4 (−17, 22)	−21 (−39,−2.3)*	12 (−6.4, 31)
RMSSD	−3.4 (−13, 5.7)	15 (5.3, 24)	−12 (−24, 0.7)	7.4 (−5.0, 20)	−4.1 (−16, 7.9)	13 (0.8, 25)*
SDNN	−9.1 (−17,−1.1)*	9.7 (1.6, 18)	−16 (−26,−6.1)*	4.7 (−5.3, 15)	−8.1 (−18, 1.6)	9.2 (−0.6, 19)
Saliva Parameters						
Amylase	−1.6 (−20, 16)	19 (4.4, 33)*	−3.7 (−38, 31)	19 (−12, 50)	5.3 (−29, 39)	23 (−7.3, 53)
Cortisol	−8.0 (−24, 8.1)	−14 (−27,−1.1)*	6.4 (−31, 44)	−16 (−51, 19)	12 (−25, 49)	21 (−14, 56)
Blood Pressure						
Diastolic Blood Pressure		−3.5 (−5.7,−1.2)*		−0.9 (−5.2, 3.4)		−3.2 (−7.4, 1.0)
Systolic Blood Pressure		−0.3 (−2.1, 1.5)		−0.9 (−4.8, 3.0)		−0.8 (−4.6, 3.0)
Pulse		7.7 (5.0, 10)*		5.4 (−0.7, 11)		2.4 (−3.6, 8.3)

All models are adjusted for age; Reference category is no noise.

* Denotes statistical significance (p-value $<$ 0.05).

exposures. After the noise exposure ended, there were further non statistically significant changes in levels of both amylase and cortisol.

Finally, for blood pressure, after both low- and high-frequency noise exposures, diastolic blood pressure increased while the systolic blood pressure appeared less affected and the pulse rate decreased. None of these effects were statistically significant.

4. Discussion

The main purpose for this study was to test whether noise exposure produced acute changes in stress and cardiovascular responses and whether these responses differed based on noise frequency operating under the hypothesis that noise acts on the body as physiological stressor. This stressor disrupts homeostasis by dysregulating the hypothalamic-pituitary-adrenocortical (HPA) and sympathetic-adrenal-medullar (SAM) axis by prolonging typical “fight or flight” responses such as increases in blood pressure or the secretion of cortisol (Recio et al., 2016). For cardiovascular activity, as measured by HRV, acute noise exposure may serve to dysregulate how the SNS and PNS work in tandem to generate a suitable stress response and where lowered HRV can be viewed as the inability to manage noise induced stress adequately (Recio et al., 2016) In model 1 where we considered associations between biological measures and exposure period, we observed statistically significant declines in HRV parameters LF and SDNN during noise exposure. Contrary to our biological hypothesis, we observed declines in both salivary amylase and cortisol during noise exposure. However, neither of these declines were statistically significant. A statistically significant decline was observed with diastolic blood pressure and statistically significant increases were observed with pulse. When we took into account noise frequency (model 2), we gained further statistically significant declines in the HRV parameter HF during low-frequency noise exposure and lost statistically significant declines in SDNN during high-frequency noise exposure. While we did see a suggestion of recuperation of HRV after noise exposure, gains were less during low-frequency noise exposure than with high-frequency noise exposure. Therefore, reductions in HRV—which we observed during both noise exposure overall and low frequency noise exposure, specifically—suggests that such noise may compromise the ability for SNS and PNS to generate a regulated response. In particular, these HRV results suggest three important findings: (1) Low-frequency noise

exposure has a more negative impact on the cardiovascular response than high-frequency noise exposure; (2) The HRV parameter LF is negatively impacted by noise exposure regardless of the frequency content of the noise exposure; (3) HRV effects resulting from low-frequency noise exposure tend to persist over time. No statistically significant changes were observed in saliva and blood pressure metrics with either low- or high-frequency noise exposure.

Our results showing a cardiovascular autonomic response to noise exposure are consistent with other studies—especially when a noise exposure frequency profile was directly or indirectly considered (Sim et al., 2015; Kraus et al., 2013; Roque et al., 2013; Björ et al., 2007; Yanagihashi et al., 1997). Sim et al. (2015) examined the effects of traffic, speech, or mixed (traffic and speech) noise (45 dB) on HRV and blood pressure in college aged men and found reductions in the HRV parameter LF, but only in the speech noise exposure group and only after noise exposure occurred. In both our pooled and separate analyses, we observed increases, albeit non-statistically significant, in LF after noise exposure.

Studies able to capture HRV activity during noise exposure tend to be consistent with our findings. For concurrent noise exposures, Kraus et al. (2013) examined the relationship between HRV in male and female adults and noise levels as study participants went about their daily routine activities. For sound levels >65 dB(A)– the range of noise exposure intensity used in our study– negative associations between SDNN and concurrent 5 dB(A) increases in noise exposure were observed. For sound levels <65 dB(A) LF and HF HRV were negatively associated with concurrent 5 dB increases in noise. SDNN was also negatively associated but with noise lagged by 5–15 min. Roque et al. (2013) considered the effects of baroque and heavy metal music on HRV in healthy females. Similar to our findings, statistically significant reductions were observed with LF during exposure to heavy metal music. Björ et al. (2007) exposed male and female subjects to four exposure conditions: only noise (broadband noise at 85 dB(A)), both noise and vibration, only vibration, and a control condition. While they did not observe a cardiovascular autonomic response with noise exposure alone, decreases in LF and HF were observed during exposure to the combination of noise and vibration groups and to vibration only. Yanagihashi et al. (1997) studied HRV in young females during three different noise exposure scenarios: (1) music from a synthesizer; (2) bird sounds; (3) mechanical sounds. They observed that HF was significantly lower during mechanical sounds exposures.

Contrary to our results, Lee et al. (2010), after exposing healthy adults to broadband white noise of intensities ranging from 50 to 80 dB(A), found statistically significant increases in LF during exposure to noise at sound levels of 50, 60, 70, and 80 dB(A). In a study similar to that of Roque et al. (2013), Ferreira et al. (2015) considered the effects of classical baroque (64 – 84 dB) and heavy metal (75–84 dB) music on HRV in healthy females. SDNN and LF were found to increase after exposure to classical baroque music. With heavy metal music, after noise exposure there were increases in SDNN, RMSSD, and LF.

Similar to our findings of non-statistically significant increases in blood pressure after exposure to low-frequency noise, Sim et al. (2015) found non-statistically significant increases in both systolic and diastolic blood pressure after noise exposure. Pauvonic et al. (2014) measured blood pressure during noise exposure and found increases during noise exposure for both men and women with returns to pre-exposure levels after the noise exposure ended. Both Chang et al. (2009) and Lusk et al. (2004) simultaneously measured systolic and diastolic blood pressure and noise levels as participants went about their daily activities during their work and non-work day. Chang et al. (2009), observed that a 5 dB (A) increase in the 24 h average environmental noise exposure significantly increased SBP and DPB in both young men and women. Lusk et al. (2004) found statistically significant associations between noise exposure and systolic and diastolic blood pressure.

For salivary amylase and cortisol, Wagner et al. (2010) saw increases in both measures after healthy male and female study participants were exposed to recorded road and rail traffic noise at a sound level of 75 dB(A). In our separate analysis we did see suggestions of increases in cortisol during noise exposure to both high- and low-frequency noise exposure, but the increases were not statistically significant.

Contrasting results between our study and others can most likely be attributed, at least in part, to differences in study design—in particular, differences in how the noise exposure was defined and differences in the time periods in which the cardiovascular and stress measures were analyzed. Noise exposures in all studies were different in terms of intensity, frequency profile, duration and context. Kraus et al. (2013), Lusk et al. (2004), and Chang et al. (2009), all looked at noise exposures as study participants went about their daily activities over a 24 h period. Paunovic et al. (2014), Roque et al. (2013), Ferreira et al. (2015), Yaganashi et al. (1997), all exposed their study participants to pre-recorded contextual noises in a carefully controlled experimental setting for less than an hour. Our study, along with Lee et al. (2010) and Björ et al. (2007) exposed study participants to non-contextual white noise of varying frequencies for just under an hour. All of the experimental studies exposed participants to each noise scenario in one setting whereas we allowed at least one day of wash-out between scenarios. It can be argued that each of these differences activate sympathetic and parasympathetic activity differentially, thus producing differential cardiovascular and stress responses.

Further, differences in when cardiovascular and stress measures were analyzed may also contribute to differences in observed responses to noise exposure. We had three distinct exposure time periods—no noise, during noise, and after noise. Our results were most consistent with those who examined responses during similar time frames.

Study population differences may also explain contrasting results. Our study only included healthy, young males. The cardiovascular response to noise may differ by gender. While no gender effect was observed by Björ et al. (2007), other studies have shown that females have a more pronounced cardiovascular response to noise (Paunovic et al. (2014), Chang et al. (2009), and Lusk et al. (2004)). Likewise, age appears to be an effect modifier of the cardiovascular response to noise with individuals ≥ 65 years old experiencing larger cardiovascular response to noise exposures with sound levels ≤ 65 dB(A) compared to younger individuals Kraus et al. (2013). Finally, Chang et al. (2009) found that hypertensive adults were more susceptible to noise exposure with a greater effect on systolic blood pressure.

Some limitations of our findings should be considered. First, the number of study participants was small and while we observed some statistically significant cardiovascular responses to noise, such a small sample size may have limited our ability to detect all cardiovascular responses to noise. While we considered healthy males, we did not collect information on existing stress and sensitivity to noise. Previous research suggests that sensitivity plays a significant role in how individuals biologically respond to noise exposure (Recio et al., 2016, Shepard et al., 2010). Waye et al. (2002), for example, found that with low-frequency noise exposure, subjects who were classified as highly noise sensitive maintained higher cortisol levels compared to low-sensitive subjects, suggesting differential stress responses based on an individual's noise sensitivity. Lusk et al. (2004) found that stress level positively influenced heart rate. Additionally, we used a comparatively narrow-band, non-contextual noise source, which may not have been perceived by our study participants as noise,

but rather sound. The difference in perception may lead to differences in response. Our noise exposure only varied by frequency profile, which does not allow us to consider differences in response due to different intensities of noise.

From our results, we know that there is a statistically significant decline in HRV with low-frequency noise exposure. How this relates to clinical outcomes is unclear. We did observe recuperation of negative dips in HRV after the noise exposure ended. Acutely, effects include typical fight or flight reactions such as increased blood pressure and cardiac output while indirect effects include hormone release (Kraus et al., 2013). Continued stimulation of these two systems leads to disease manifestation, which includes hypertension, myocardial infarction, arteriosclerosis, ischemic heart disease, and stroke (Babisch, 2014). Epidemiological studies do suggest that chronic exposure to noises with low-frequency profiles such as aircraft, rail, and road traffic noise is associated with negative cardiovascular and stress responses such as elevated cortisol (Selander et al., 2009); blood pressure (Haralabidis et al., 2008; Dratva et al., 2012); hypertension (Bluhm et al., 2007; Bodin et al., 2009; Babisch et al., 2005); myocardial infarction (Babisch et al., 2005; Selander et al., 2009); medication use (Floud et al., 2011); cardiovascular related hospital admissions (Hansell et al., 2013; Correia et al., 2013), and mortality (Hansell et al., 2013).

And while our sample size was quite small, our study design allowed each individual to serve as his own control, thereby enabling the examination of individual differences. We were also able to focus on the effect of noise frequency, which eliminates bias that may be introduced by known environmental noise sources. The study is also one of only a few that has looked at the effects of audible noise frequencies on such a wide variety of stress and cardiovascular outcomes.

5. Conclusions

In conclusion, the study adds to our understanding of the acoustic characteristic that drives the cardiovascular autonomic response to noise exposure. The results suggest that low-frequency noise in particular negatively impacts heart rate variability and these impacts may persist after noise exposure ends. In future studies, the dominant frequencies of noise should be considered when evaluating the cardiovascular health effects of noise.

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Approval for human subjects research

This study was approved by both the UConn Health and Harvard T.H. Chan School of Public Health Institutional Review Boards.

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