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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)