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Effects of environmental noise exposure on 24-h ambulatory vascular properties in adults ☆☆☆

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ABSTRACT

Exposure to environmental noise has been associated with hypertension, but the related mechanism of vascular structural changes is unclear. This repeated-measure study investigated the effects of noise exposure on the 24-h ambulatory vascular structural properties in 66 adults aged 18–32 years. Individual noise exposure and personal vascular parameters were measured simultaneously in all subjects. Linear mixed-effects regressions were used to estimate the effects. A 1-A-weighted decibel (dBA) increase was significantly associated with the transient effects of 1.39 (95% confidence interval: 1.07, 1.79) %mL/mmHg in arterial compliance at nighttime but −1.70 (−2.05, −1.10) kdynes · s/cm^5 in arterial resistance during the daytime and −2.38 (−3.44, −1.64) kdynes · s/cm^5 in arterial resistance at nighttime among all subjects. Such effects were observed in arterial distensibility only during the daytime after the 30-min (−1.84 [−2.61, −1.29] %/mmHg) and 60-min (−2.06 [−2.95, −1.44] %/mmHg) time-lagged noise exposures. For 24-h environmental noise, a 1-dBA increment was significantly associated with a sustained increase of 1.25 (1.10, 1.42) %mL/mmHg in arterial compliance but a decrease of 2.12 (−2.51, −1.80) kdynes · s/cm^5 in arterial resistance. Environmental noise exposure may have transient and sustained effects on adult vascular properties.

1. Introduction

Exposure to environmental noise has been demonstrated to be associated with an increased risk of myocardial infarction and stroke (Babisch et al., 2005; Belojevic and Saric-Tanaskovic, 2002; Huss et al., 2010; Selander et al., 2009; Sorensen et al., 2011; Willich et al., 2006). This association may exist because noise, a psychosocial stressor, activates the autonomic nervous and endocrine systems and affects the homeostasis of the human organism, producing persistent changes in endocrine regulation and metabolic function that promote the development of atherosclerosis, hypertension and cardiovascular diseases (Babisch, 2011; Ising et al., 1999).

Many epidemiological studies have reported that traffic noise exposure is associated with hypertension. Exposure to road traffic noise above 60 A-weighted decibels (dBA) may cause the transient elevation of blood pressure in preschool children (Belojevic et al., 2008; Regecova and Kellerova, 1995) and older children (Babisch et al., 2009). Residents exposed to a road-traffic noise level above 55 dBA may have a higher risk of hypertension (Barregard et al., 2009; Bodin et al., 2009; Chang et al., 2011; de Kluizenaar et al., 2007; Jarup et al., 2008; Leon Bluhm et al., 2007). In addition, transient and sustained increases of systolic blood pressure and diastolic blood pressure under environmental noise exposure ≥55 dBA have been found in adults aged 18–32 years (Chang et al., 2009).

However, the relative mechanism of environmental-noise-induced hypertension in vascular structural changes is not clear. Because the macro- and micro-vascular changes in arterial function and physical properties that are caused by hypertension occur prior to the development of clinical disease (Berenson et al., 1992), arterial compliance and distensibility have been used to measure vascular stiffness (Riley et al., 1986; Wada et al., 1994).

Abbreviations: dBA, A-weighted decibel; Leq, equivalent sound level; TWA, time-weighted average

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It is of interest to study the early changes in vascular properties because reduced arterial compliance and distensibility may relate to increased systolic blood pressure, left ventricular hypertrophy and acceleration of arteriosclerosis (Urbina et al., 2002, 2005).

One industry-based study has reported that automobile workers exposed to 85 ± 8 dBA had lower means of 24-h ambulatory arterial compliance and arterial distensibility compared with those exposed to 59 ± 4 dBA (Chang et al., 2007). These findings were limited to higher levels of occupational exposure in middle-aged males; however, the effects induced by lower levels of environmental noise have not been investigated. The purpose of this study was to determine whether environmental noise exposure has effects on the ambulatory vascular structural properties in adults.

2. Materials and methods

2.1. Subjects

The recruitment and selection of young adults as study subjects have been described previously (Chang et al., 2009). In short, participants were recruited through an on-campus advertisement at the China Medical University in 2007. Sixty-nine students responded to the advertisement, but only sixty-six volunteers were selected because three persons reported on a questionnaire to have been diagnosed with hypertension prior to joining the study. No study subjects had any pulmonary diseases. All of the study subjects lived at least 10 km from any airport; hence, their source of environmental noise was mainly the road traffic.

During the study period, a standardized questionnaire was also used to collect personal information related to additional risk factors for hypertension, such as age, height, weight, exercise habits, cigarette smoking, alcohol drinking, tea consumption, coffee consumption and family history of hypertension. Cigarette smokers were defined as those who had smoked cigarettes on more than 3 day per week for at least 6 months; alcohol, tea and coffee drinkers were defined using the same criterion. Regular exercisers were defined as those who had participated in a sporting activity at least 3 times per week for 6 months or more (Chang et al., 2009, 2011). In addition, each subject's height and weight were used to calculate his or her body mass index (BMI). To avoid interference between the measurements of noise exposure and ambulatory vascular parameters, each participant was required to refrain from certain activities during the study period, including cigarette smoking, alcohol drinking, tea or coffee consumption, drug use, regular exercise and the use of portable media players (Chang et al., 2009). The Institutional Review Board of the School of Public Health (China Medical University) had approved this study, and informed consent was obtained from each participating subject.

2.2. Ambulatory vascular property monitoring and recording

Each study subject carried a portable, non-invasive, automated monitoring and recording system (DynaPulse model 5000A, Pulse Metric, San Diego, California, USA) for 24 h to complete the continuous measurements of structural changes in their vascular properties. Because 3 sets of ambulatory monitoring devices were available in this study, the measurements included 22 weekdays (i.e., Tuesday to Thursday) during September–November 2007 to obtain a dataset of 66 person-days. The readings of each subject's vascular parameters related to structural properties were recorded repeatedly every 30 min during the daytime (0800–2300 h) and every 60 min during the nighttime (2300–0800 h) using the DynaPulse system. These vascular parameters included arterial compliance, arterial distensibility and arterial resistance. In addition, the individual's ambulatory systolic blood pressure and diastolic blood pressure during a 24-h period were measured simultaneously using the same device.

The DynaPulse system can measure a subject's arterial pulsation signals, known as the arterial waveform, through a non-invasive cuff device. This instrument derives arterial distensibility using the waveform analysis of arterial pressure signals obtained from a standard cuff sphygmomanometer (Brinton et al., 1997). The pressure waveform is calibrated and incorporated into a physical model of the cardiovascular system that has been validated against separate data collected in the cardiac catheterization laboratory (Brinton et al., 1997), assuming a straight-tube brachial artery and T-tube aortic system. Arterial compliance is derived from waveform parameters by the following equation: arterial compliance$=\pi^2 \times D_0^2 \times (D_0+\mathrm{Lc})/[(\mathrm{d}P/\mathrm{d}t_{pp}) \times t_{pp}]$, where $\mathrm{d}P/\mathrm{d}t_{pp}$ is the amplitude from the peak positive pressure derivative to the peak negative pressure derivative, and t_{pp} is the time interval between the peak positive pressure and peak negative pressure derivatives. The effective cuff width (Lc) is defined as the cuff width divided by the square root of 2. The brachial artery diameter (D_0) is estimated using an empirically derived model based on sex, height, weight and mean arterial blood pressure and is validated using B-mode ultrasound ($n=1250$, $r=0.63$). Arterial distensibility is then calculated using the following formula: arterial distensibility$=$arterial compliance$/[\pi \times (D_0^2/4) \times \mathrm{Lc}] \approx 4\pi/[(\mathrm{d}P/\mathrm{d}t_{pp}) \times t_{pp}]$ (Urbina et al., 2002, 2005).

Previous validation studies of the DynaPulse instrument have demonstrated the high correlation between compliance measurements obtained with cardiac catheterization and those derived by the noninvasive method ($r=0.83$) (Brinton et al., 1997, 1998). The intra-class correlation coefficient for blind duplicate recordings was 0.72, indicating that most of the variability in measurements were due to inter-individual variation (Urbina et al., 2002, 2005). The vascular parameter data of all subjects during the daytime (0800–2300 h) and nighttime (2300–0800 h) periods were used to investigate the transient effects of environmental noise exposure on adults' vascular properties. All of the participants' vascular parameter measurements during a 24-h period were used to study the sustained effects of environmental noise exposure.

2.3. Noise exposure measurements

Personal noise-exposure levels were measured continuously using a personal noise dosimeter (Logging Noise Dose Meter Type 4443, Brüel & Kjær, Nærum, Denmark.), which can report the 5-min continuous equivalent sound level (Leq) at an exchange rate of 3 dBA and the time-weighted averages (TWAs) of the noise doses. The range of 50–120 dBA was used to measure all of the subjects' noise levels with 5-min readings during a 24-h period. As the noise-exposure levels were measured below the limit of detection, a value of 35.4 dBA (the $\sqrt{2}/2$ detected limit of noise level with 50 dBA) was used to replace the non-detected level for this study. The use of 35.4 dBA was because the raw data of the noise distribution during a 24-h period had a geometric standard deviation of 1.16 dBA that provided a more accurate estimation instead of using 1/2 of the detected limit when the data were not highly skewed (i.e., geometric standard deviation <3.0) (Hornung and Reed, 1990). To investigate the time-lag effects of acute noise exposure, 5-min exposure measurements were summarized into 30-min and 60-min time-moving-average segments for further analysis.

2.4. Statistical analysis

The Shapiro–Wilk test was applied first to determine the normality of the continuous variables. Spearman's rank correlations were conducted to correlate the noise levels with the vascular properties during the daytime, nighttime and a 24-h period.

To address the auto-correlation problem between the repeated measurements of vascular parameters and to increase the statistical power by combining information across study subjects, the linear mixed-effects regression models were applied to associate environmental noise exposure with vascular parameters by controlling important confounding factors of the participants (Littell et al., 1996). This regression model has both fixed-effect and random-effect covariance parameters. The fixed effect in this mixed-effects model included variables of sex, age, BMI, smoking status, regular exercise, noise exposure and family history of hypertension. All of these variables were potential confounders for hypertension that had been reported in previous studies (Chang et al., 2009, 2011; Jarup et al., 2008; Leon Bluhm et al., 2007). Individual subjects were treated as a random effect in the model. Because the distributions of age, BMI, 24-h average noise exposure, arterial compliance, arterial distensibility and arterial resistance among all subjects were abnormal (all p values <0.001 for the Shapiro–Wilk test), these continuous variables were first translated into a 10-based logarithm form to fit the normal distribution and then implemented in the regression models. All assumptions of the mixed models, such as the normality in the residuals, were met using influence and residual diagnostics.

The mixed-effects models were also used to compare the within-group differences in the mean values of ambulatory vascular properties and noise exposure during the daytime and nighttime. Three types of covariance structures were used to assess the fit of the mixed-effects regressions, including compound symmetric, unstructured and the first-order autoregressive models. The first-order autoregressive model was chosen as the best-fitted one due to the minimizing value of Akaike's Information Criterion (Cnaan et al., 1997) in all vascular parameters. The MIXED Procedure containing fixed and random effects in SAS 9.1 version (SAS Institute Incorporation, Cary, North Carolina, USA) was used to perform the linear mixed-effects regression in this study.

In addition, smoothing splines (GAMM procedure, mgcv-package, R version 2.13.2, R Foundation for Statistical Computing, Vienna, Austria) were used to explore the linear associations between the vascular parameters and noise levels (Appendix Figs. A.1–A.3) (Wood, 2004, 2006). Because the adjusted association between the log-transformed arterial distensibility and log-transformed noise levels was non-linear, the outcomes with noise levels less than the limit of detection were excluded to produce the linear association (Appendix Fig. A.4). Therefore, only outcomes with noise levels ≥ 50 dBA were used to investigate the associations between arterial distensibility and noise levels in the present study.