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Short communication

Impact of personally measured pollutants on cardiac function

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ABSTRACT

Epidemiological studies have shown associations between ambient air pollution and changes in heart rate variability (HRV). However, studies using personal air pollution measurements, especially with exposure averages <24 h, are still rare.

Between February and March 2008 HRV data as well as personal exposure to particulate matter <2.5 μm ($PM_{2.5}$), and particle number concentrations (PNC) were collected in five volunteers for up to 8.3 h on a 5 min resolution. Information about the participant's whereabouts was also collected. Mixed models were used to analyze concurrent and up to 30 min delayed effects of air pollutants as well as being in traffic on 5 min-averages of heart rate (HR), high and low frequency power (HF and LF), standard deviation of all normal-to-normal intervals (SDNN), and the root mean square of successive interval differences (RMSSD). Results are presented as %-change from the mean per increase in interquartile range of air pollutant.

In total, 474 5-min segments were available for analysis. We observed concurrent and delayed reductions in SDNN of about 0.8–1.0% in association with a 5.4 $\mu g/m^3$ increase in $PM_{2.5}$. However, being in traffic by car led to an increase of about 20% 10–14 min and 15–19 min later. An increase in $PM_{2.5}$ or PNC was associated with lagged decreases for RMSSD and HF. We detected concurrent reductions in RMSSD (−17.6% [95%-confidence interval: 29.1; −4.3]) when being in traffic by bike/foot. Being in traffic by car was associated with an immediate reduction in LF while more delayed increases in LF were observed when being in traffic by bike/foot. Air pollution and traffic effects on HR were less consistent.

These rapid changes in HRV within 30 min might be mediated by the autonomic nervous system in response to direct reflexes from receptors in the lungs.

Introduction

Epidemiological studies have found that exposure to air pollution is inducing changes in heart rate (HR) and heart rate variability (HRV), a parameter for the autonomous control of the heart (Brook et al., 2010; Rückerl et al., 2011; Pieters et al., 2012). There is strong evidence that a reduced HRV is an independent risk factor for cardiac mortality after myocardial infarction, ischemic sudden death and cardiac events such as arrhythmia (Reed et al., 2005; Lanza et al., 2006; Xhyheri et al., 2012). Especially a low standard deviation of all normal-to-normal intervals (SDNN) after myocardial infarction was associated with adverse outcomes such as mortality or cardiac complications (Buccelletti et al., 2009). While SDNN reflects sympathetic and parasympathetic modulation of the heart, the root mean square of successive interval differences (RMSSD) and high frequency power (HF) are measures of the parasympathetic activity (Xhyheri et al., 2012). A recent meta-analysis (Pieters et al., 2012) showed reductions in these HRV parameters due to increases in ambient $PM_{2.5}$ concentrations. Most of the studies published so far have used central measurement sites for exposure estimation rather than personal measurements. However, people spend most of the time indoors and have therefore variable

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exposures to air pollution from outdoor sources. Moreover, indoor air pollution can also rise to high levels (e.g. due to cooking or heating) even when the current-day outdoor levels are considered harmless. Measurements at central monitoring sites also miss short-term peaks in air pollution exposures of an individual e.g. in traffic. Only a few studies investigated the effects of personally measured particles with an aerodynamic diameter <2.5 μm ($PM_{2.5}$) on parameters of cardiac rhythm with inconsistent findings (Lanki et al., 2008; De Hartog et al., 2009; Folino et al., 2009; Wu et al., 2010a,b; He et al., 2011). So far there are no studies which explored the effects of personally measured ultrafine particles. The inhalation of air pollution might affect cardiac function within a few hours or even minutes. Accordingly, Peters et al. (2004) reported an association between exposure to traffic and the onset of a myocardial infarction (MI) within one hour afterward. It is hypothesized that particles impair the balance of the autonomic nervous system perceivable by an activation of the sympathetic tone and/or withdrawal of the parasympathetic tone (Brook et al., 2010).

The objective of this small study was to investigate immediate effects of personally measured $PM_{2.5}$, carbon monoxide (CO), and particle number concentration (PNC), a proxy for ultrafine particles, as well as of being in traffic on HR and HRV parameters on a 5 min-basis. Such a small number of participants were chosen as we aimed to evaluate our ability to collect personal measurements and to generate first hypotheses on personal air pollution effects especially of PNC on HR and HRV. In accordance with a recent meta-analysis (Pieters et al., 2012), we expected to observe a reduced HRV in association with increased personal measurements of air pollution exposure.

Methods

Five participants living in Augsburg were recruited from the KORA (Cooperative Health Research in the Region of Augsburg) survey 2000 (Holle et al., 2005). All participants gave written informed consent and the study protocol was approved by the Ethics Commission of the Bavarian Chamber of Physicians ("Bayerische Landesaerztekammer"). All participants were never smokers or stopped smoking at least twelve months before the study. None of the participants had ever experienced angina pectoris, heart attack or stroke. Between March 4 and 11, 2008 each participant was fitted once in the afternoon (around 3 pm) for up to 23 h with a long-term Holter 7-lead-ECG (Medilog AR4 recorder, Huntleigh Healthcare, Cardiff, United Kingdom). The participants left the study center and pursued their daily routines. Repeated 5 min-intervals of ECG parameters were available for each individual. ECG parameters of interest were HR, SDNN, RMSSD as well as low (LF, 0.04–1.5 Hz) and high frequency power (HF, 0.15–0.40 Hz) in normalized units.

In parallel, personal exposure was measured with a PEM sampler pDR-1200 (MIE Inc., MA, USA) for $PM_{2.5}$, (Lanki et al., 2002) a CO measurer (Langan Model T15n, Langan Products Inc., California, USA) (Langan, 2006), and an Ultrafine Particle Counter for PNC (P-TRAK, Model 8525, TSI, Minnesota, USA, size range: 0.2 to greater than 1 μm). Relative humidity and temperature were measured using Escort iLog EI-HS-D-32-L logger (Inc., Buchanan, VA). $PM_{2.5}$ concentrations were corrected for relative humidity and normalized using gravimetric results from the filter. CO concentrations were corrected for temperature. See Supplemental Material for a more detailed description of the air pollution measurements. Participants were instructed to carry the backpack containing the measurement devices (~4 kg in total) when moving around or doing activities that may affect the air quality. When staying in one place for a longer time, participants were allowed to place the backpack on a nearby table or a chair within a hand's reach, but not on the floor. None of the participants reported time periods when not carrying the backpack with him/her. Hourly means of ambient air temperature, relative humidity, and barometric pressure were measured at a central monitoring site in the urban background of Augsburg.

Additionally, participants filled in a diary (paper/pencil form) with an accuracy of 15 min, including questions on the whereabouts (e.g. being in traffic) and activities (e.g. cooking, cleaning) during the measurement period. These data were double-entered in electronic database by two different assistants. Double-entries were compared and corrected if necessary.

Mixed models with a random participant effect and a first order autoregressive covariance structure were used to analyze the association between ECG parameters and personally measured air pollutants as well as with the indicator variable "being in traffic" (by car vs. by bike/foot vs. not in traffic). HR, SDNN, RMSSD, and HF had to be log-transformed to fulfill the assumption of normally distributed residuals.

A confounder model was built for each outcome separately. Potential confounders were 1 h-averages of ambient air temperature, relative humidity, and barometric pressure measured within the same hour as the respective 5 min ECG-interval. The confounders were included linearly or smoothly as penalized spline depending on the model fit. The shape which minimized Akaike's Information Criterion was chosen. Barometric pressure was only included in case of model fit improvement.

Physical exertion leads to an increase in HR and a consecutive change in HRV. As participants are probably more likely to exercise (e.g. walking, running) outdoors physical activity might act as a confounder of the association between air pollution and HRV. Hence, we planned to adjust our models for physical activity. However, for only <4% of the 5 min-ECG intervals three participants stated to be physical active. Therefore, we adjusted our model for HR as a surrogate for physical exertion. Emotional stimuli might also influence HRV. Only one person reported to be mildly annoyed for about 25 min. Hence, we did not adjust our models for this variable.

After assessing the confounder model, lags of 5 min-averages of personally measured air pollutants concurrent and up to 30 min preceding the respective 5 min-ECG intervals were added to the model separately and effects were estimated linearly.

As a sensitivity analysis we performed polynomial distributed lag models (PDL, degree = 3) which included all 5 min-averages of an air pollution variable up to 1 h preceding the 5 min ECG-intervals simultaneously. In order to avoid multicollinearity, we used a distributed lag model which forces estimates to a polynomial shape.

Results are presented as percent change from the outcome mean per increase in interquartile range (IQR) of the respective air pollutant.

Results

Two men and three women with an average age of 46 years (range: 39–58 years) and body mass index of 29 kg/m^2 (25–33 kg/m^2) were part of our study. They spent on average 103 min (45–210 min) outdoors and thereby were mostly in traffic in these periods (average: 79 min, range: 15–210 min). A more detailed description of characteristics, whereabouts, and activities of each participant is given in Supplemental Table 1.

Personal PNC measurements lasted between 7.2 and 8.3 h because of limited amount of condensation liquid inside the device. Therefore, all further analyses were performed for this shorter period comprising 474 5 min-intervals. Table 1 shows the description of the air pollutants as well as the ECG parameters. On average personal $PM_{2.5}$ and PNC values were higher when the participants were not in traffic. This might be partly due to the use of air conditioning in cars. In accordance, $PM_{2.5}$ concentrations were