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Exposure to traffic pollution, acute inflammation and autonomic response in a panel of car commuters

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ABSTRACT

Background: Exposure to traffic pollution has been linked to numerous adverse health endpoints. Despite this, limited data examining traffic exposures during realistic commutes and acute response exists.
Objectives: We conducted the Atlanta Commuters Exposures (ACE-1) Study, an extensive panel-based exposure and health study, to measure chemically-resolved in-vehicle exposures and corresponding changes in acute oxidative stress, lipid peroxidation, pulmonary and systemic inflammation and autonomic response.
Methods: We recruited 42 adults (21 with and 21 without asthma) to conduct two 2-h scripted highway commutes during morning rush hour in the metropolitan Atlanta area. A suite of in-vehicle particulate components were measured in the subjects' private vehicles. Biomarker measurements were conducted before, during, and immediately after the commutes and in 3 hourly intervals after commutes.
Results: At measurement time points within 3 h after the commute, we observed mild to pronounced elevations relative to baseline in exhaled nitric oxide, C-reactive-protein, and exhaled malondialdehyde, indicative of pulmonary and systemic inflammation and oxidative stress initiation, as well as decreases relative to baseline levels in the time-domain heart-rate variability parameters, SDNN and rMSSD, indicative of autonomic dysfunction. We did not observe any detectable changes in lung function measurements (FEV1, FVC), the frequency-domain heart-rate variability parameter or other systemic biomarkers of vascular injury. Water soluble organic carbon was associated with changes in eNO at all post-commute time-points ($p < 0.0001$).
Conclusions: Our results point to measureable changes in pulmonary and autonomic biomarkers following a scripted 2-h highway commute.

1. Introduction

There is considerable evidence from observational and controlled studies linking traffic-related pollution and adverse health (HEI, 2010). Although the etiology of traffic pollution health effects is complex and may be mediated via numerous pathways (Brook et al., 2010), it is possible that biological response to traffic pollution components or mixtures is elicited following very short-term exposures (Ghio et al., 2003; Peters et al., 2004). Daily commuters may be especially vulnerable given their proximity and enhanced exposures to traffic-related pollution, as well as other non-chemical stressors including noise and psychosocial stress. While time spent daily in traffic may be limited, exposure assessments measuring in-vehicle pollutant concentrations indicate that even short durations inside vehicles (~30 min) can contribute substantially to total daily exposures to particulate matter (PM) (Adams et al., 2001; Boogaard et al., 2009; Rodes et al., 1998; Sioutas et al., 2005; Zuurbier et al., 2010). Despite this, there is still considerable uncertainty concerning in-vehicle exposures during typical commuting scenarios and corresponding cardiorespiratory responses for daily commuters.

Panel-based exposure studies afford unique opportunities to investigate the impacts of commuting on health, given their ability to accurately measure both real world exposures and health

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response on an individual level. An important panel study of highway patrolmen in North Carolina reported associations between in-vehicle PM exposures over 8 h shifts and acute changes in systemic inflammation biomarkers and cardiac autonomic function (Riediker et al., 2004). Among the notable findings from this study was that subclinical biological changes in cardiorespiratory response were observed in young, healthy, active adults following exposures to traffic PM at commonly experienced levels. Subsequent in-vehicle panel studies have provided additional indication that exposures experienced during scripted car or bus commutes may be associated with measures of heart rate variability (Adar et al., 2007; Laumbach et al., 2010; Shields et al., 2013; Wu et al., 2010) and pulmonary inflammation (Zuurbier et al., 2011).

Although suggestive, results from these initial commuter panel studies provide inconsistent evidence concerning the specific factors most associated with response or specific biological pathways most associated with exposures. Some of this inconsistency is likely due to the complexity of the in-vehicle microenvironment, comprising a combination of chemical, physical and psychosocial stressors. A more complete understanding of in-vehicle exposures and health for commuters is becoming increasingly necessary, as commuting durations as well as roadway congestion have steadily increased throughout the U.S. during the last 20 years. Over 10 million Americans spend greater than two hours each day commuting to and from their place of work, with 61% of those commuters driving alone (U.S. Census Bureau, 2011 American Community Survey Reports, 2011 Out-of-State and Long Commutes: 2011 Brian McKenzie).

To investigate in-vehicle exposures among daily car commuters and provide additional insight into the potential health effects of this activity, we conducted two large, panel-based exposure and health assessment studies in the metropolitan Atlanta area, including adults with and without asthma. The current analysis presents results from the initial Atlanta Commuters Exposure (ACE) study, ACE-1, which included measurements collected for over 80 morning rush hour commutes. We examined the hypothesis that exposures occurring during rush hour car commuting lead to acute changes in cardiorespiratory response, consistent with oxidative-stress mediated pathways of injury.

2. Methods

In-vehicle pollutant exposures and corresponding biomarker measurements were collected for 21 adults with self-reported asthma and 21 non-asthmatic adults between December 2009 and April 2011. Subjects used their personal vehicles to conduct a scripted commute lasting approximately 2 h during the morning rush hour period (7–9 AM) in the metropolitan Atlanta area. Commute routes began and ended at our environmental health laboratory at the Rollins School of Public Health of Emory University. Routes were similar among commutes and were designed to include heavily used commuting roadways with both gasoline and diesel engine vehicles. Trained field technicians accompanied subjects throughout the entire commute. Each subject conducted two scripted commutes as part of the protocol, with the exception of 3 subjects who withdrew from the study after conducting a single commute. The repeat commutes for a given subject were scheduled at varying time intervals from the initial commute, ranging from 2 weeks to 17 months, with a median between-commute interval of 4 months.

The driver's side window was alternately opened for 15 min and then closed for 15 min throughout the commute except during rain or uncomfortably cold temperatures. Subjects were allowed to use the vehicle's air condition or ventilation system but were asked to use the outside air setting throughout the commute.

2.1. Exclusion criteria

Subjects for this study were recruited largely by word of mouth and flyers posted on the Emory University and Centers for Disease Control and Prevention (CDC) campuses. To limit exposure to traffic pollution prior to the study commute, we restricted subjects to those living within close proximity (within 15 min drive) of our laboratory facility and commute start point. One subject, who lived approximately 20 miles from our facility, was met by field staff at their residence and began the commute from that location. Participants were considered 'Asthmatics" if they self -reported ever being diagnosed by a health provider of having asthma. All participants with asthma were instructed to continue normal medication regimens throughout their participation in the study.

We excluded individuals who were pregnant; had diabetes; a previous myocardial infarction; implantable cardioverter-defibrillators or pacemakers; used digoxin or beta blockers for treatment of hypertension or arrhythmias; or had non-asthma pulmonary disease such as COPD, emphysema, any type of lung cancer, or a forced expiratory volume in 1 s (FEV_1) less than 70% predicted at baseline. We excluded individuals who smoked. The study was approved by the Emory Institutional Review Board. Written informed consent was provided by all participants.

2.2. Biomarker measurements

Prior to sampling, each subject was administered a baseline questionnaire assessing factors related to both exposure and health, including proximity of subject residences to major roadways, potential exposures to indoor or outdoor pollution events, and recent health status. Approximately 30 min before each commute, a trained field technician and phlebotomist met with subjects at our laboratory facilities at Emory University to conduct initial baseline measurements (~6:30 AM). Biomarker measurements were also conducted during and immediately following the commute (0 h), as well as at hourly intervals for 3 hours after the commutes. In between measurements, participants were asked not to leave the surrounding area of the clinic.

The selected biomarker measurements were targeted primarily to assess acute response consistent with oxidative stress and inflammation pathways. The specific endpoints included those that have been shown in previous studies to be related to exposure to ambient particulate or gas-phase pollution (Brook et al., 2010; Ghio et al., 2003; Hertel et al., 2010; Mills et al., 2007; Park et al., 2010). For the current analysis, we examined lung function, exhaled nitric oxide (eNO), malondialdehyde (MDA) in exhaled breath condensate (EBC), C-reactive protein (CRP) and heart rate variability (HRV) parameters. Several additional circulating biomarkers of systemic inflammation including soluble intercellular adhesion molecule-1 (sICAM-1), soluble vascular adhesion molecule-1 (sVCAM1), interleukin 1 (IL-1β), interleukin 6 (IL-6), interleukin 8 (IL-8), and tumor necrosis factor alpha (TNF-α) were analyzed in plasma, which was collected at the pre-commute baseline and 3 h post-commute time points only.

The concentration of NO in exhaled breath, an indicator of acute bronchial inflammation and oxidative stress (Alving and Malinovschi, 2010), was measured first, using the portable NIOX MINO analyzer (Aerocrine, New Providence, NJ, USA). Participants were asked not to consume foods with high levels of nitrates (i.e. spinach, beets, radishes, celery, cabbage and cured meats) the night before the study and throughout the day of the study, in order to eliminate the effect of nutrition on eNO measurements. They were asked not to eat 30 min prior to each biomarker measurement session. FEV1 and forced vital capacity (FVC) measurements were performed with the use of an OHD KoKo spirometer (Occupational Health Dynamics, Birmingham, AL, USA). Metrics of lung function are presented as percent of age-, sex-, and race-specific predicted values (Hankinson et al., 1999). EBC was collected during a tidal breathing protocol with the use of a standardized breath-condensate collector which was stored at −80 °C prior to sampling (RTube, Austin, T, USA). Concentrations of MDA in the expired droplets of respiratory tract lining fluid, a marker of pulmonary lipid peroxidation in EBC were measured using a high-performance liquid chromatography (HPLC) technique to assess the progression of airway lipid peroxidation reactions (Lärstad et al., 2002). We measured CRP in blood obtained from finger prick samples collected at each of the measurement periods (Cholestech LDX system, Inverness Medical, Hayward, CA, USA). Blood was drawn by a trained phlebotomist at our clinical facility from an antecubital vein and immediately centrifuged to separate plasma. The suite of inflammation biomarkers in plasma were analyzed according to manufacturer's specifications at the National Health and Environmental Effects Research Laboratory of the US Environmental Protection Agency (Vascular Injury Panel II assay, Human Pro-inflammatory II 4-plex assay ultra-sensitive kit, MesoScale Discovery, Gaithersburg, MD). Blood pressure was measured using the Ambulo 2400 ABPM System (Tiba Medical, Portland, OR, USA).

Heart rate and heart rate variability (HRV) were recorded continuously throughout the commute and during the entire sampling day using a 5-lead Holter monitor (2010 Plus Philips Healthcare, Eindhoven, The Netherlands). For the current analyses, time and frequency domain HRV parameters were characterized, during a 10 min rest period at our clinical facility, performed in the sitting position, immediately prior to the collection of the other biomarker endpoints at each sampling time point. All normal-to-normal intervals from the 10-min recording windows were analyzed for time and frequency domain parameters in 10-min epochs using standard, validated algorithms on Zymed analysis software. The software automatically detected heart beats and labeled ectopic beats such as periventricular contractions or pretrial contractions. A trained technician working with an Emory cardiologist then visually viewed the ECG tracing, removing regions with noise, artifact and ectopy. Time domain parameters included the standard deviation of all normal to-normal intervals (SDNNs) and the square root of the mean squared difference between adjacent normal-to-normal intervals (rMSSDs);