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Exposure Error Masks The Relationship Between Traffic-Related Air Pollution and Heart Rate Variability (HRV)

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Abstract

Objective—We examined whether more precise exposure measures would better detect associations between traffic-related pollution, elemental carbon (EC) and nitrogen dioxide (NO₂), and HRV.

Methods—Repeated 24-h personal and ambient PM_{2.5}, EC, and NO₂ were measured for 30 people living in Atlanta, GA. The association between HRV and either ambient concentrations or personal exposures was examined using linear mixed effects models.

Results—Ambient PM_{2.5}, EC, and NO₂ and personal PM_{2.5} were not associated with HRV. Personal EC and NO₂ measured 24-h prior to HRV was associated with decreased rMSSD, PNN50, and HF and with increased LF/HF. RMSSD decreased by 10.97% (95% CI: -18.00,-3.34) for an IQR change in personal EC (0.81 ug/m³).

Conclusions—Results indicate decreased vagal tone in response to traffic pollutants, which can best be detected with precise personal exposure measures.

INTRODUCTION

Previously, we reported results examining the association between ambient pollution and HRV for 18 individuals with chronic obstructive pulmonary disease (COPD) and 12 individuals with recent myocardial infarctions (MIs) living throughout metropolitan Atlanta, GA.[1] In this examination, we found ambient PM_{2.5} concentrations to have opposing effects on HRV in our COPD and MI participants, resulting in significant pollution-associated increases in SDNN for individuals with COPD. Individuals with MIs consistently showed pollution-associated decreases in SDNN, although these decreases were not statistically significant. Although consistent across moving averages and HRV measures, observed associations were strongest for the 4-h as compared to 1-h and 24-h moving averages and for SDNN. For ambient concentrations of EC and NO₂, locally generated pollutants, similar trends were observed for individuals with MI and COPD; however, associations between ambient EC and NO₂ and all HRV measures were not statistically significant.

The observed insignificant findings for ambient EC and NO₂ and HRV are contrary to findings from other panel studies, such as that conducted in Boston, MA, where the SAM

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monitoring site was located in close proximity to the study participant residences. The divergent findings may be due to the fact that unlike in Boston, study participants in Atlanta lived in homes located across a wide area, in areas characterized by a range of population densities, land uses, and distances to major roads. As a result of this spatial spread and site heterogeneity, ambient EC and NO₂ concentrations were likely poor surrogates of exposure, since spatial variability in outdoor concentrations of both of these traffic-related pollutants has been shown to be substantial within large metropolitan cities, such as Atlanta.[2] Consequently, we hypothesized that our ability to detect associations with HRV using ambient EC and NO₂ concentrations may have been poor for our Atlanta cohort. To examine the impact of exposure error on our findings, we analyzed data from our Atlanta study to assess spatial variability in outdoor PM_{2.5}, EC, and NO₂ and to evaluate associations between HRV and ambient pollutant concentrations and personal pollutant exposures.

METHODS

In Fall 1999 and Spring 2000, repeated health and exposure measurements were made under a Harvard School of Public Health Human Subjects Committee approved protocol. Measurements were made for individuals living in metropolitan Atlanta who had a myocardial infarction (MI) 3-12 months prior to the start of the study or had physician-diagnosed moderate-to-severe COPD. Each of these individuals provided informed consent to participate in the study. Health and exposure measurements were made for each participant over seven consecutive days in one or both seasons, with exposure measurements beginning 24-h prior to the HRV measurement. Five participants were monitored simultaneously each seven-day period. Twenty-one and 22 individuals participated in the fall and spring, respectively, with 13 individuals participating in both seasons. A total of 30 individuals – 12 individuals with a recent MI and 18 individuals with COPD – participated in the study.

Participant recruitment, baseline health assessments and health monitoring have been described previously.[1] Briefly, technician-completed questionnaires on medical history, medication use, housing characteristics, and vitamin use were administered prior to enrollment in the study. Baseline spirometry and a resting 12-lead ECG were also performed (MAC6, Marquette Medical Systems Inc, Milwaukee, WI). Exclusion criteria were similar to the Boston HRV study conducted by Gold et al.[3] and included unstable angina, atrial flutter, atrial fibrillation, paced rhythm, left bundle branch block, on constant oxygen, smokers, or living with smokers and inability to walk on level ground.

Health Monitoring

Field technicians visited participants in their home between 7am and 11am on each monitoring day. At the beginning of each visit, field technicians administered a questionnaire on health status, medication use, vitamin intake, housing characteristics and activity patterns. Field technicians subsequently placed a Holter monitor (SEER MC ambulatory digital analysis recorder, GE Medical Systems, Milwaukee, WI) on each participant and led the participant through a standardized HRV protocol, as described previously.[3]

Briefly, the HRV protocol included continuous Holter monitoring using electrodes in a modified V5 and AVF position as participants rested, stood, walked outside, and performed paced breathing. The Holter tapes were analyzed using a Marquette MARS Workstation, with a standard 125 samples/second sampling rate.[4] All analyses were reviewed by a trained technician for noise and artifact. Ectopic beats were excluded in these analyses, with a linearly interpolated QRS based on the prior beat's RR interval and the subsequent beat's RR interval inserted, as is standard. Holter tapes were analyzed for several measures of

HRV, including the standard deviation of normal RR intervals (SDNN), the square root of the mean of the sum of squares of differences between adjacent NN intervals (RMSSD), the percent of the absolute differences between successive normal R-R intervals that exceed 50ms (PNN50), high frequency power of 0.15 – 0.40Hz (HF), low frequency power of 0.04 – 0.15Hz (LF), and the HF/LF ratio. A total of 300 Holter monitoring sessions were completed for 31 subjects, 28 of whom had 5 or more measurements. Of the 300 observations, 25 observations were excluded due to onset of atrial fibrillation, 3 due to second-degree block, and 3 were unreadable. One participant was excluded from all analyses due to atrial fibrillation. Of the 300 valid Holter sessions, 3 sessions were missing portions of the overall protocol. These sessions were not excluded from subsequent data analyses, as results did not differ based on their inclusion in the model.

Exposure Measurements

Personal exposures and indoor and outdoor concentrations to PM_{2.5}, sulfate (SO₄²⁻), elemental carbon (EC), O₃, and NO₂ were measured for each participant for seven consecutive 24-h periods, beginning 24h prior to the participants' first HRV measurement. All measurements were made using our multi-pollutant sampler.[5,6] In the fall, the sampler consisted of two SKC Personal Exposure Monitors (PEMs) to measure PM_{2.5} and EC,[7] one mini-PEM sampler to measure SO₄²⁻, and passive badges to measure O₃ and NO₂. The PM_{2.5} and EC SKC PEMs were modified to operate at flow rates of 2.0 LPM, with the number of nozzle holes reduced from ten to five for both PEMs. In the spring, the sampler consisted of a Harvard Personal Exposure Monitors (PEMs) for PM_{2.5},[6] two mini-PEM samplers to measure SO₄²⁻ and EC, and passive badges to measure O₃ and NO₂. [8,9] The performance of the Harvard and SKC PEMs were comparable (data not shown). All PM_{2.5} and PM₁₀ samples were collected on Teflon filters and included drain disk rings to minimize contamination.

The mini-PEM samplers, which are essentially miniaturized versions of the PEM, consisted of an inlet-impactor section to remove coarse particles followed by a 15mm Fluoropore or quartz filter to collect SO₄²⁻ or EC, respectively. SO₄²⁻ mini-PEM samplers were operated at flow rates of 0.5 LPM, except in the first sampling session when samplers were operated at 0.8 LPM. EC mini-PEM samplers were operated throughout the study at flow rates of 0.8 LPM. Impactor plates of all PEM and mini-PEM samplers were greased to minimize particle bounce.[6] The PEM and mini-PEM samplers were attached to a 10 cm long elutriator. Air was drawn through each of the samplers using a single personal sampling pump, whose flow was split into multiple air streams, such that one air stream flowed to each sampler. Passive O₃ and NO₂ badges were placed in the side of the elutriator, with their face exposed to the sample air stream to allow for constant sampler collection rates. Each of the passive badges contained a single cellulose filter, which was coated in either nitrite to collect O₃, or triethanolamine to collect NO₂.

Personal multi-pollutant monitors were worn on the shoulder strap of a backpack, containing a personal pump and a motion detector. The inlets of the samplers were placed at breathing height. Monitored individuals were asked to wear the sampler throughout each 24-h monitoring period, but were allowed to remove the samplers during stationary activities, such as eating or sleeping, and during activities where the monitor may be damaged, such as showering. If the participant was mobility-restricted or otherwise hampered, the samplers were attached to fixed objects near the participant's body, with the inlet protruding into the breathing zone. Indoor samplers were placed on a stand in the main activity room of the home, away from vents, doors, and windows, and approximately at sitting height. Outdoor samplers were placed approximately five feet high in the backyard, as far as possible from large stationary objects such as the home and trees.

Teflon filters for PM_{2.5} were weighed before and after sample collection on an electronic microbalance (Cahn Model C-31), with filters passed over Po²¹⁰ sources (alpha rays) prior to weighing. Mass concentrations were determined from the mass change (corrected for barometric pressure) and the volume of air sampled. SO₄²⁻, O₃, and NO₂ filters were extracted and analyzed using ion chromatography. Quartz fiber filters were analyzed for EC using Thermal Optical Reflectance (TOR).

Ambient Measurements

Ambient pollution and meteorological data were obtained from sites located within the metropolitan Atlanta area. Hourly ambient fine particle (PM_{2.5}) concentrations were measured at the Yorkville (YRK), Fort McPherson (FT) and Tucker (TU) stationary ambient monitoring (SAM) sites which were operated as part of the Assessment of Spatial Aerosol Composition in Atlanta (ASACA) and Southeastern Aerosol Research and Characterization (SEARCH) studies (National Research Council, 1999). Hourly ozone (O₃) concentrations were measured at SAM sites located at TU, YRK, and South Dekalb (SD), while hourly NO₂ concentrations were measured at the Tucker and SD sites. Black carbon concentrations and temperature and relative humidity were measured at the Jefferson Street (JST). All measurement methods and SAM sites were described previously.[1]

Analyses with ambient pollution data were conducted using 24-hour mean prior to the HRV protocol to correspond to personal exposure data. Calculated 24-h mean exposures were considered valid when more than 18 hours (75% of the hours) were available. Since participant homes and SAM sites were located throughout metropolitan Atlanta, the average concentration for the available SAM sites was used as the ambient PM_{2.5} and gaseous measure. Exposure data were void if data were only available from one SAM site.

Quality Assurance and Control

Standard QA/QC procedures were followed for this study. Blank filters were used to correct concentrations of all measured species. For PM_{2.5}, PM₁₀, SO₄²⁻, and EC, concentrations were corrected using the mean filter blank level for the respective pollutant, when mean blank levels differed significantly from zero. Corrections were generally made by season and by sample type. Corrections by sample batch or sample date were unnecessary, as blank values did not differ significantly by either of these parameters. Data points were voided due to sampling (e.g., pump or battery failures, tube disconnection) or laboratory (e.g., contamination) problems. Where possible, data for samples with negative levels or with concentrations below the method limit of detection (LOD) were flagged, but were left in the data set and subsequent data analyses.

Data Analysis

Pollution exposures were assessed using 24-h averaged ambient concentrations, personal exposures, outdoor concentrations, and indoor concentrations. Exposure periods from 0 to 24 hours prior to the HRV measurements (lag 0), from 24 to 48 hours prior to the HRV measurement (lag 1), and for the 48 hours prior to the HRV measurement (lag 0/1) were examined. Averaging periods for ambient concentrations were matched exactly to the time of the HRV measurement. While not matched explicitly to the time of the HRV measurement, personal exposures and indoor and outdoor concentrations were matched approximately to the HRV measurements, with the end time of the exposure measurement within two hours before or after the start of the HRV measurement. The association between personal pollutant exposures and corresponding ambient concentrations were compared using mixed models with compound symmetry covariance structure to account for the repeated measurements structure of the data. For comparison purposes, HRV associations with personal and ambient levels were calculated using matched data sets.

The association between pollution and HRV (for the entire 35-minute period) was examined separately for each exposure measure using linear mixed effect models (LME). The heart rate variability outcomes measured in the models were log transformed. Models controlled for time, meteorological, and subject-related variables, including random effects for subjects, and adjusting for BMI, temperature and relative humidity as continuous variables and sex, age, medication use, season, hour and day of week as categorical variables. The relationship between BMI and other continuous variables and HRV outcomes was tested using penalized splines and the Generalized Cross Validation method (GCV). Using the GCV, we found the relationship between BMI and HRV to be linear. We used natural splines with three degrees of freedom for temperature and relative humidity, with the degrees of freedom selected based on previous studies. Medication use was included as a categorical variable for four medication types: beta-blockers, calcium channel blockers, angiotensin converting enzyme (ACE) inhibitors, and bronchodilators (non-steroidal long and short-acting), with values based on whether the participant was or was not prescribed the medication. As the relationship between weather and the outcome variables were non-linear, both temperature and relative humidity were modeled using natural spline functions with 3 degrees of freedom. Models did not control for heart rate, which is inversely associated with heart rate variability. When heart rate was included in the model, model results were similar, although with slightly lower coefficients.

To identify factors that modify the association between HRV and pollutant exposures, disease status were included in the models. Medication use was controlled for in these models. Since only individuals with COPD took bronchodilator medications, models that were stratified by disease status did not control for bronchodilator medications. Effect modification by age and gender were also examined (data not shown), but all interaction terms were insignificant. Sensitivity analyses were performed examining indoor and outdoor pollution-associated effects on HRV. To allow comparisons with those for ambient concentrations and personal exposures, sensitivity analyses were performed using a matched dataset that included only those person-days for which all data, including HRV, personal, ambient, indoor, and outdoor measurements, were available. Analyses of pollution and HRV were conducted with data combined across seasons. All statistical analyses of exposure data were conducted using SAS, while health effects analyses were performed using R 2.7.1 (The Comprehensive R Archive Network: <http://cran.r-project.org/>). Unless otherwise indicated, statistical significance was determined based on a p-value of 0.05.

RESULTS

Participant Profiles

Participant characteristics have been described previously.[1] Briefly, exposure and health measurements were made for 30 individuals, 12 with recent MIs and 18 with self-reported moderate-to-severe COPD. In total, 265 observations were obtained over all participants, 159 for those with COPD and 106 for those with recent MIs. Participants lived throughout metropolitan Atlanta, in areas characterized by varying population densities and distances from road. All participants had air conditioning, with all but 3 having central air conditioning. Participants spent the majority of their time indoors at home (mean=76±15%) each day, with an average of 12% (±13%) spent in other indoor environments, 6% (±5%) spent in transit, and 4% (±6%) spent outside.

Pollution Exposures

Personal particulate and gaseous exposures varied substantially across the monitoring period (Table 1), with similar distributions for the fall and spring seasons. Personal pollutant exposures did not differ significantly for individuals with COPD as compared to MI. Mean

personal pollutant exposures were generally lower than corresponding ambient concentrations, with differences greatest for O₃, for which personal exposures were extremely low.

In the ambient environment, 24-h pollution concentrations were strongly correlated with one another, with the exception of O₃ for which correlations with other pollutants were weak (Table 2). For ambient PM_{2.5}, EC, and NO₂, Spearman correlation coefficients for pair-wise comparisons ranged between 0.47 (NO₂ vs. PM_{2.5}, $p < 0.0001$) and 0.59 (EC vs. PM_{2.5}, $p < 0.0001$). Those with ambient O₃ fell between 0.12 (NO₂, $p = 0.08$) and 0.34 (PM_{2.5}, $p < 0.0001$). Correlations among personal exposures were lower than those among ambient concentrations for all pollutant measures.

Personal PM_{2.5} exposures were strongly associated with corresponding ambient concentrations ($r = 0.63$, $p < 0.0001$) in both seasons and for both cohorts, with a slope of 0.65 (0.06) for personal-ambient comparisons (Table 2). For EC, the personal-ambient association was weaker and had a lower slope (0.26 ± 0.04) than that for PM_{2.5}, but remained highly significant ($r = 0.48$, $p < 0.0001$). The personal-ambient EC association did not differ by cohort, but varied by season, with a higher and significant slope in the fall (0.44 ± 0.05) as compared to spring (0.10 ± 0.06). Ambient NO₂ concentrations were poor proxies of corresponding personal exposures ($r = 0.12$, $p = 0.09$), with lower and insignificant slopes. Seasonal trends in the personal-ambient NO₂ association were similar to that observed for EC, as associations were significant in the fall (slope = 0.18 ± 0.05) but not spring (slope = -0.02 ± 0.13). For O₃, personal-ambient associations were insignificant, with extremely low slopes in both fall and spring.

Association between 24-h Ambient Pollution and HRV

Table 3 shows the association between ambient and personal pollution and HRV measure for all participants; while Figure 2 shows results for PM_{2.5}, EC, and NO₂ by disease group. The association between the 24-h moving average of ambient PM_{2.5} (lag 0, lag 1 and lag 0/1) and the standard deviation of normal RR intervals (SDNN) was not significant when data were analyzed for all participants or when stratified by disease group (Figure 2). Although not significant, HRV for individuals with an MI generally decreased with IQR changes in ambient PM_{2.5} concentrations, especially for the parasympathetic measures RMSSD, PNN50, and HF (Figure 2). For the 24-hr moving average, RMSSD rose by 2.4% (95% CI: -9.3, 15.6%) per inter-quartile range (IQR) increase in ambient PM_{2.5} (7.8 ug/m^3) for individuals with COPD, whereas RMSSD decreased by 13.2% (95% CI: -25.2, 0.75%) per IQR increase in ambient PM_{2.5} (8.1 ug/m^3) in individuals who were post-MI (Figure 2). Associations of ambient EC and NO₂ with HRV generally followed similar trends, with associations negative, although insignificant, especially for parasympathetic measures and for individuals with MI. Associations of HRV with O₃ were insignificant, when analyses were performed for all individuals and when stratified by disease group. With some exceptions, similar results were found when ambient concentrations averaged between 24-h to 48-h or averaged between 0-h to 48-h prior to the HRV measurements were used as the exposure measure.

Association between 24-h Personal Pollution and HRV

Personal PM_{2.5} exposures were not significantly associated with changes in any HRV measure (Table 3), irrespective of the exposure averaging period (lag 0, lag 1, lag 0/1) and disease group (Figure 2). In contrast, personal exposures to the traffic-related pollutants EC and NO₂ were significantly related to decreased HRV, particularly for measures of parasympathetic function rMSSD, PNN50, HF, and LF/HF ratio (Table 3). Associations were strongest for personal EC exposures measured the 24-h before the health measurement

(lag 0). Among all participants, RMSSD decreased by 10.97% (95% CI: -18.00, -3.34) for an IQR change in personal EC exposures (0.81 ug/m^3). The magnitude of these associations was substantially stronger than those observed for corresponding 24-h ambient concentrations and was strongest in MI participants. For example, an IQR change of 0.75 ug/m^3 in personal EC exposures was associated with a 13.47% (95% CI: -23.81, -1.73) decrease in RMSSD for MI participants as compared to a 7.53% (95% CI: -16.14, 1.96) decrease in RMSSD for COPD participants. Results for other measures of parasympathetic function were similar, with the impact again stronger in individuals with an MI as compared to those with COPD.

For NO_2 , personal exposures (lag 0) were also associated with decreased HRV. Effects were again generally most pronounced for the parasympathetic HRV measures. Associations between personal NO_2 exposures and these parasympathetic measures, however, were weaker and less consistent than those observed for personal EC. A -14.93% (95% CI: -25.66, -2.65) change in PNN50, for example, was associated with a 9.45 ppb increase in personal NO_2 exposures for all individuals, with the effect greatest and significant in individuals with COPD. In contrast, personal NO_2 -associated decrements in HF were significant and most pronounced in individuals with a recent MI, but were not significant when all individuals or individuals with COPD were included in the analysis.

As was the case with ambient concentrations, personal ozone exposures were not significantly associated with any HRV measure. When stratified by disease group, personal-HRV associations remained largely insignificant, although for HF, personal ozone exposures were significantly and positively associated for individuals with MI. Corresponding measures of parasympathetic function, such as rMSSD and PNN50, however, were insignificant for these individuals.

Relationship between HRV and Outdoor and Indoor Pollutant Concentrations

Indoor and outdoor home $\text{PM}_{2.5}$, EC, and NO_2 concentrations were not associated with any measure of HRV (Figure 3). Insignificant associations for indoor and outdoor concentrations were not due to lower sample numbers, as when matched to include only those data for which personal, indoor, outdoor, and ambient concentrations were all available, pollutant associations with HRV remained significant and of similar magnitude for personal EC and NO_2 exposures, but remained insignificant for ambient, indoor, and outdoor exposure measures.

DISCUSSION

Findings from our study provide direct evidence of the effect of exposure error on our ability to examine the autonomic response to traffic pollution, which is known to vary spatially. Changes in HRV, particularly those associated with parasympathetic control, were significantly and negatively associated with EC and to a lesser extent NO_2 , when measurements of personal exposure -- but not ambient, indoor and outdoor concentrations -- were used to assess their exposures. Notably, the importance of personal exposure measurements to our ability to detect associations with HRV was evident only for these traffic-related pollutants. Non-significant associations with HRV were found for 24-h ambient concentrations and personal exposures to $\text{PM}_{2.5}$ and ozone, regional pollutants that are more spatially uniform. Consistent with these findings, ambient EC and NO_2 concentrations were poorly associated with their corresponding personal exposures, providing evidence for why associations with HRV could not be observed using ambient EC and NO_2 concentrations as a proxy for personal exposures. In contrast, ambient concentrations were strong proxies of personal $\text{PM}_{2.5}$ exposures, thus resulting in similar findings when either personal or ambient $\text{PM}_{2.5}$ was used to assess exposures. Since ambient

NO₂ and EC originate primarily from motor vehicles, our findings suggest that motor vehicle-related pollution may be partly responsible for the observed effects of ambient particles on parasympathetic measures of HRV and that personal exposure measurements are needed to detect their effects on health.

These results are consistent with previous studies showing significant impacts of EC and NO₂ on HRV. Mobile source pollution (EC in particular), averaged over the hour to four hours before measurement, has been linked in several studies to decreased HRV. In a study of elderly individuals taken on field trips aboard a diesel-powered bus, for example, trip-level exposures to black carbon and ultra-fine particles (as well as to PM_{2.5}) were consistently and significantly associated with decreased HRV.[10] Similarly significant associations were found in a panel study of elderly individuals living near a major inner city roadway in Boston, where the effect of traffic-related black carbon (BC) on reduced HRV predominated.[11] Lower HRV was also linked with exposure to 1- to 4-hr moving averages of ultra-fine particle counts, also a marker of traffic, especially in elderly individuals with COPD.[12] In each of these studies, exposures were assessed using measurements of BC or traffic-related pollutants that were made in close proximity to the participants being studied.

Our observed associations are also consistent with plausible biological mechanisms, where traffic-particles directly affect the cardiovascular system by triggering pro-inflammatory cytokines and reactive oxygen species (ROS).[13,14] Once generated, the pro-inflammatory cytokines and ROS impact various cardiac-related processes, including autonomic cardiac control and particularly heart rate variability.[15] Since decreased HRV is an indicator of weakened autonomic control of the heart and is a known risk factor for sudden death,[16,17] our study findings together with those from earlier studies suggest that exposures to particles from mobile sources impair heart function and provide a possible explanation for the Peters et al.[18] finding of an increased risk of heart attack in the hour immediately after periods spent in traffic.

Interestingly, the effects of mobile source pollution on HRV could not be observed when indoor concentrations were used as the exposure measure, which was somewhat surprising given that individuals spent the majority of their time indoors at home and given the moderate but statistically significant correlations between personal exposures and indoor concentrations for both EC and NO₂. Although significant, these moderate correlations were not strong enough to detect associations with HRV, suggesting that indoor and outdoor home concentrations are unable to capture variation in important sources of exposure, such as occupational exposures, or mobile source exposures when in transit.

Important limitations of this investigation are the small number of individuals studied, with only 30 total study participants, and the relatively long averaging time of our indoor, outdoor, and personal exposure measurements, which allowed us only to examine the effects of 24-h and 48-h averaged exposures. As discussed above, several previous studies have shown that the effects of traffic-related pollution on HRV are strongest with exposures averaged one to four hour prior to HRV measurement. This combined with our ability to find significant findings with 24-h integrated personal exposures suggests that the relation between personal EC exposures and HRV may be even stronger for personal exposures averaged over one hour to four hour time frames. Despite this, our findings of that show associations between exposures to mobile source pollutants and HRV are consistent with our understanding of the spatial and temporal heterogeneity in mobile source exposures and with findings from previous epidemiological and toxicological studies. In addition, it is important to note that traffic can be a source not only of particle pollution, but also of stress, a known risk factor for adverse cardiac outcomes, and psychologic stress has been known to influence HRV and autonomic function [19]. A limitation of our study is that we did not

measure stress, and therefore we cannot disentangle the combination of pollution and stress exposures that “traffic” exposures may represent. Stress not associated with traffic, however, probably did not confound our results, as it is unlikely that non-traffic related stress is associated with traffic pollutant concentrations. Further study is needed to examine this possibility directly and to assess the impacts of personal exposures over shorter averaging periods.

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Figure 1.
Locations of participant's homes and the stationary ambient monitoring sites, Atlanta, GA.

Figure 2.

Percent Change in rMSSD per IQR in 24h Ambient and Personal Exposures

*% changes calculated using IQR for ambient concentrations to allow comparison. For PM_{2.5}, IQRs are 7.81 ug/m³ for all, 7.40 ug/m³ for MI, and 9.24 ug/m³ for COPD participants. For EC, IQRs are ug/m³ for all and MI participants and 1.81 ug/m³ for COPD participants. For NO₂, IQRs are 9.45 ug/m³, 9.13 ug/m³, 9.52 ppb for all, MI and COPD participants, respectively.

Figure 3.
Association between 24-h BC, 24-h NO₂ and HRV for Ambient, Outdoor, Indoor, and Personal Exposure Measures

Table 1

Summary of 24-h Pollution Exposures

Pollutant/ Disease Group ^I	Personal Exposures				Ambient Concentrations			
	N	Mean (SD)	50%	IQR	N	Mean (SD)	50%	IQR
PM _{2.5} (ug/m ³)	282	15.78 (8.75)	14.25	9.49	264	17.17 (7.24)	16.29	8.09
MI	113	14.65 (7.70)	13.89	9.51	106	17.28 (7.51)	16.36	8.22
COPD	169	16.54 (9.33)	14.44	9.25	158	17.09 (7.07)	16.29	7.96
EC (ug/m ³)	283	1.61 (0.71)	1.49	0.85	253	2.04 (1.25)	1.70	1.20
MI	115	1.72 (0.72)	1.61	0.78	99	1.99 (1.33)	1.68	1.22
COPD	168	1.53 (0.70)	1.39	0.88	154	2.08 (1.20)	1.80	1.19
SO ₄ ²⁻ (ug/m ³)	265	2.73 (1.58)	2.50	1.81				
MI	111	2.68 (1.47)	2.43	1.98	--	--	--	--
COPD	154	2.76 (1.66)	2.55	1.70				
NO ₂ (ppb)	277	11.60 (11.7)	8.08	7.17	224	17.13 (6.97)	17.96	9.48
MI	114	11.87 (8.82)	8.90	8.35	89	17.84 (6.88)	18.65	9.13
COPD	163	11.42 (13.33)	7.82	6.59	135	16.67 (7.01)	17.54	9.63
O ₃ (ppb)	282	2.54 (3.06)	1.51	3.49	250	29.38 (10.38)	31.18	16.02
MI	113	2.83 (2.96)	2.07	4.03	96	28.29 (9.62)	31.22	14.55
COPD	169	2.34 (3.12)	1.31	3.09	154	30.06 (10.80)	31.11	16.92

^I Values correspond to the 24-hour periods prior to the HRV measurements. Ambient measurements matched in time to personal exposure measurements.

Table 2

Correlation between Ambient and Personal PM_{2.5}

	Ambient			Personal		
	PM _{2.5}	EC	NO ₂	PM _{2.5}	EC	NO ₂
Ambient						
PM _{2.5}	--					
EC	0.59*	--				
NO ₂	0.47*	0.58*	--			
O ₃	0.34*	0.26*	0.12***			
Personal						
PM _{2.5}	0.63*	0.43*	0.25*	--		
EC	0.46*	0.48*	0.33*	0.45*	--	
NO ₂	0.20*	0.22*	0.12***	0.29*	0.49*	--
O ₃	0.08	-0.01	-0.09	0.14**	0.00	-0.03

^a Spearman correlation coefficients for comparisons of 24-h (9am-9am) ambient and personal levels.

* p<0.01

** 0.01< p<0.05

*** 0.05< p<0.10

Table 3

Association between 24-h Ambient and Personal Pollution and Overall HRV

HRV Measure – Pollutant	Ambient		Personal	
	% Change	95% CI	% Change	95% CI
SDNN				
PM _{2.5}	-1.0	-4.7, 2.8	0.32	-3.35, 4.12
EC	-1.0	-3.7, 1.7	-4.66	-7.99, -1.22
NO ₂	-0.3	-5.2, 4.9	-1.19	-3.65, 1.33
SO ₄ ²⁻	--	--	0.01	-3.48, 3.63
Ozone	-0.03	-8.40, 9.10	-0.03	-8.40, 9.10
RMSSD				
PM _{2.5}	-4.4	-12.4, 4.3	-3.50	-11.49, 5.22
EC	-3.6	-9.5, 2.8	-10.97	-18.00, -3.34
NO ₂	-3.1	-14.4, 9.8	-4.96	-10.20, 0.58
SO ₄ ²⁻	--	--	1.06	-6.97, 9.78
Ozone	-2.14	-30.18, 37.15	10.83	-12.63, 40.58
PNN50				
PM _{2.5}	-6.71	-19.69, 8.37	-6.70	-19.64, 8.33
EC	-0.34	-10.55, 11.04	-15.16	-26.33, -2.29
NO ₂	0.14	-17.99, 22.27	-11.03	-19.29, -1.93
SO ₄ ²⁻	--	--	-0.03	-13.39, 15.38
Ozone	-5.24	-17.72, 72.42	14.35	-20.34, 64.14
HF				
PM _{2.5}	-6.29	-18.27, 7.44	-5.49	-17.31, 8.02
EC	-2.36	-11.67, 7.92	-13.41	-23.95, -1.41
NO ₂	-0.70	-17.43, 19.42	-7.29	-15.34, 1.53
SO ₄ ²⁻	--	--	1.45	-10.95, 15.59
Ozone	-6.32	-42.37, 52.29	20.84	-13.47, 68.76
LF/HF				
PM _{2.5}	4.70	-1.46, 11.25	5.48	-0.80, 12.15
EC	2.60	-1.89, 7.29	6.22	0.15, 12.64
NO ₂	6.46	-1.93, 15.57	3.31	-0.80, 7.58
SO ₄ ²⁻	--	--	1.41	-4.34, 7.50
Ozone	2.16	-18.48, 28.01	-2.85	-16.87, 13.55

Percent change in overall SDNN (with 95% confidence intervals) expressed per IQR change in PM_{2.5}, EC, NO₂, ozone averaged over the 24-h immediately preceding HRV measurement. IQRs for ambient concentrations were 8.0 ug/m³, 1.2 ug/m³, 9.4 ppb, 15.99 ppb for PM_{2.5}, EC, NO₂, ozone respectively and were 8.68 ug/m³, 0.81 ug/m³, 6.83 ppb, and 9.10 ppb for personal exposures, respectively. Model does not include heart rate. Statistically significant estimates in bold.