Published in final edited form as:
J Expo Sci Environ Epidemiol. 2009 July ; 19(5): 525–533. doi:10.1038/jes.2008.46.

Acute exposure to elevated $PM_{2.5}$ generated by traffic and cardiopulmonary health effects in healthy older adults

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Abstract

There are evidences for exposure to vehicular emissions and adverse cardiopulmonary health effects. This study attempted to further explore these effects on elderly. This study monitored personal $PM_{2.5}$ concentrations and ambulatory electrocardiograms continuously for 24 h on 1 working day in 3 separate weeks for 11 school crossing guards. Spirometry was also performed before and after the morning shift. The traffic at each work location was video recorded during one of the three morning shifts. The increases in the average personal $PM_{2.5}$ concentrations (baseline $PM_{2.5}$ was subtracted) of 1.2–87 and 1.1–98 $\mu g/m^3$ were observed during the 1-h morning ($\Delta PM_{2.5\text{-ave-m}}$) and afternoon shift ($\Delta PM_{2.5\text{-ave-a}}$), respectively. Traffic count was not a significant predictor of the $\Delta PM_{2.5\text{-ave-m}}$ (P=0.78). Mean heart rate variability (HRV), measured as 5-min standard deviation of normal-to-normal (SDNN) beats during the 10-min rest periods, decreased 18–26% (P<0.02) 15 min, 2 and 4 h after the morning shift, but changes in SDNN (ΔSDNN) were insignificant post-afternoon exposure (−0.3 to −7% with P>0.53). ΔSDNN were negatively associated with $\Delta PM_{2.5\text{-ave-m}}$, with the strongest association at 2 h after the morning shift (P<0.01) but insignificant 4 h after the morning exposure. The peak $PM_{2.5}$ concentration ($\Delta PM_{2.5\text{-peak}}$, baseline $PM_{2.5}$ was subtracted) was not a significant predictor for ΔSDNN, and no clear effect of $PM_{2.5}$ exposure on heart rate was observed. There was no effect of PM exposure on lung function (P>0.16), either. In conclusion, acute exposure to the $PM_{2.5}$ resulting from mobile sources can cause acute decline in HRV in healthy older adults, suggesting one of the biological mechanisms for the adverse cardiovascular health effects associated with traffic-related air

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pollution. Traffic count may not be an appropriate surrogate measure of acute personal exposure to vehicular emission in traffic congested areas.

Keywords

$PM_{2.5}$; vehicular emission; heart rate variability; heart rate; lung function; traffic count

Introduction

Chronic exposure to vehicular emissions has been associated with cardiopulmonary mortality and morbidity (Brauer et al., 2002; Buckeridge et al., 2002; Hoek et al., 2002; Lin et al., 2002; Finkelstein et al., 2004; Kim et al., 2004; Slaughter et al., 2005; Wellenius et al., 2005). Pollutants that are related to vehicular emissions, especially fine particulate matter ($PM_{2.5}$), have been studied for the mechanisms underlying the observed associations between chronic exposure to vehicular emissions and cardiopulmonary health effects. As summarized by Rowan et al.(2007), numerous studies have reported reduced heart rate variability (HRV) in association with exposure to $PM_{2.5}$, which may represent autonomic nervous system changes that could mediate some of the cardiovascular effects of $PM_{2.5}$. Changes in heart rate (HR) in association with exposure to $PM_{2.5}$ air pollution were also observed, although inconsistent findings were reported (Peters et al., 1999; Pope et al., 1999; Ibald-Mulli et al., 2004; Adar et al., 2007). As opposed to other sources, some studies showed a stronger association between particles originating from traffic and cardiovascular endpoints (Laden et al., 2000; Gold et al., 2005; Park et al., 2005; Adar et al., 2007). Furthermore, the elderly appear to be more susceptible to the cardiopulmonary effects of PM exposure (Tsuji et al., 1994; Laden et al., 2000; Gold et al., 2005; Park et al., 2005; Schwartz et al., 2005; Adar et al., 2007).

Exposure to vehicular emissions, however, often includes short-term (minutes to hours) exposure to relatively high concentrations of air pollutants, such as exposure during commuting (Sabin et al., 2005; Adar et al., 2007). Many health effect studies that examined exposure to traffic-related PM used the 24- or 48-h average concentrations of $PM_{2.5}$ measured at central monitoring sites distant from traffic to represent personal $PM_{2.5}$ exposure levels (Tsuji et al., 1994; Liao et al., 1999; Pope et al., 1999, 2004; Gold et al., 2000; Laden et al., 2000). Given the dramatic decreases in concentrations and rapid changes in physical and chemical properties of $PM_{2.5}$ with time and distance from vehicular emissions (Zhu et al., 2002; Marr et al., 2004), integrated measurements of $PM_{2.5}$ obtained at distant central monitoring sites may underestimate the acute exposure as well as the potential health effects due to being near traffic sources.

The present study investigated the effects resulting from acute exposure to $PM_{2.5}$ freshly generated by vehicles on the cardiopulmonary health of older adults — school crossing guards. School crossing guards are likely to experience elevated $PM_{2.5}$ concentrations generated by both school buses and other vehicles given their very close proximity to traffic during their morning and afternoon work shifts. Moreover, many crossing guards are

elderly, and may therefore be more susceptible to the cardiopulmonary effects of air pollutants than the general population.

Materials and methods

Subject Information

This study was conducted on school crossing guards from Paterson, the third largest city in New Jersey (NJ). Research protocols and consent forms were approved by the Institutional Review Board of the University of Medicine and Dentistry of New Jersey (UMDNJ) and all the subjects signed the consent form before their participation in this study. Subject recruitment was conducted with the assistance of the Paterson Police Department. Traffic information about the locations where the crossing guards worked was initially provided by the police department. Site visits were conducted and the locations with a high volume of traffic were selected for inclusion in the study. Subsequently, study information was distributed by the police department to the crossing guards who worked at the selected locations. Eleven crossing guards volunteered to participate in the study. The demographic information of the subjects is presented in Table 1. The mean age of the crossing guards was 61 years. All subjects were currently non-smokers and generally healthy by self-reports. One subject reported a previous diagnosis of asthma by a physician but was asymptomatic during participation in the study. No subjects took β-blocker medications or other medications that are likely to affect cardiac function measurement. No subjects had diabetic or hypertensive diseases. Except one subject, all participants completed the 3 days of measurements and complied fully with the study protocols.

Study Protocols

All of the school crossing guards had the same working schedule: 0730–0830 hours for the morning shift and 1430–1530 hours for the afternoon shift. Thus, personal $PM_{2.5}$ concentration and electrocardiogram (ECG) monitoring started at ~0700 hours, a half-hour before crossing guards went on-duty in the morning, to ~0700 hours the following day, for a total of 24 h. Each subject was monitored on 1 working day in 3 separate weeks. $PM_{2.5}$ concentration was expected to differ among the 3 sampling days at each location and between locations given the day-to-day variation in meteorological conditions and different traffic volume between locations. Each subject was monitored on the same day of the week and at the same time of day to reduce the possible confounding effects of temporal changes in physiological and activity-related variables. The study was conducted from February to May 2005. All tests were conducted on days with no precipitation to avoid suppression of $PM_{2.5}$ concentrations. Temperature, humidity, and other meteorological data were obtained from Teterboro airport located 10 miles southwest of Paterson. The 24-h average temperature ranged from −11°C to 28°C and relative humidity ranged from 32% to 78%. During each monitoring period, subjects were instructed not to participate in vigorous physical activities, such as playing sports, bicycling, running, and so on. The subjects were asked to complete baseline (including demographic information, general information about living conditions, health status, medications, etc.), activity, and health symptom questionnaires.

Personal $PM_{2.5}$ Concentrations

The 24-h personal $PM_{2.5}$ mass concentrations ($\mu g/m^3$) for each subject were continuously monitored using a real-time personal $PM_{2.5}$ monitor (Side-Pak, Model AM510-1D11; TSI Inc., Shoreview, MN, USA). The monitor was placed either in a pouch or in a back pack so that it was easy for the subject to wear. The inlet of the sampling tube was clipped to the shirt within the participant's breathing zone. The $PM_{2.5}$ monitor has a dynamic measurement range of 1 $\mu g/m^3$ to 20 mg/m^3 and was fitted with an impactor to collect $PM_{2.5}$. The monitor was programmed to record sequential 1-min $PM_{2.5}$ measurements.

Traffic Count

The intersection was video recorded with a Sony Digital Video recorder (model GV-D200) for ~15 min during one morning shift for each subject but two locations were recorded twice. The 12 videos were subsequently transcribed using Virtual Timing Device software (Sama Sama Consulting). The standard template was modified to enable investigators to record the type of vehicles (bus, car, large truck, small truck or other) observed in the intersection. The traffic count by vehicle type and observation time was recorded for each video.

Electrocardiogram (ECG)

The ECG of each subject was recorded continuously for 24 h using a five-lead Holter monitor (Model Dynacord 3 Channel Model 423; Raytel Cardiac Services, Windsor, CT, USA). A trained field technician placed the electrodes on the subjects in the subjects' homes before his/her morning shift began. The protocol for the ECG monitoring included 10 min rest periods, during which the subjects were instructed to sit quietly in a chair, beginning at ~15 min before, and at 15 min, 2 and 4 h after the morning and afternoon shifts. A timer was given to the subjects to remind them to sit still for the designated time intervals, and the technician or subjects recorded the actual time interval on a sampling sheet. The tapes were sent to Raytel Cardiac Services for analysis of 5-min standard deviation of normal-to-normal (SDNN) intervals, a measure of overall HRV, by trained professionals.

Lung Function Measurement

The lung function of each subject was measured with a portable spirometer (Micro Inc., CT, USA) according to the quality criteria of the American Thoracic Society (American Thoracic Society, 1995), and the measurement was only performed before and right after the morning shift. Lung function was measured after the 10-min rest period for the ECG recording. The lung function parameters included forced vital capacity (FVC), forced expired volume in the first second ($FEV_{1.0}$), and peak expired flow (PEF). The expiratory maneuver was repeated until the three $FEV_{1.0}$ measurements within 10% of the maximum value were obtained. Due to the age of the subjects (mean of 61 years), a 10% variation quality criterion among repetitive measurements was used to consider the lung function measurement valid. Parameter values from these three trials were used for analysis.

Data Analysis

Descriptive statistics and mixed effect models (SAS 9.1) were used to examine (1) whether there were changes in HR, SDNN, and lung function measurements after the morning and afternoon work shifts; and (2) whether those changes were due to exposure to the corresponding $PM_{2.5}$ exposure. Both 1-h average and peak concentrations of $PM_{2.5}$ during each work shift were used for examining the effect of $PM_{2.5}$ exposure on the changes of the health endpoint measurements. To control for the potential effect from $PM_{2.5}$ exposure before each work shift, the average $PM_{2.5}$ background concentrations measured 10 min before each work shift were subtracted from the corresponding 1-h average and peak concentrations of $PM_{2.5}$, that is increases in $PM_{2.5}$ concentrations were used for analysis. $\Delta PM_{2.5\text{-ave-m}}$ and $\Delta PM_{2.5\text{-peak-m}}$ represent for the morning increases in the average and peak $PM_{2.5}$ concentrations, and $\Delta PM_{2.5\text{-ave-a}}$ and $\Delta PM_{2.5\text{-peak-a}}$ represent for the afternoon increases in the average and peak $PM_{2.5}$ concentrations, respectively. As most crossing guards worked in areas of high traffic, the increase in $PM_{2.5}$ concentration was primarily due to mobile sources, which allowed for examining the effect from exposure to traffic-related $PM_{2.5}$ on the cardiac and pulmonary parameters. To control the variability of an individual, the changes in health measures after work shift were used for analysis, that is changes in health outcomes (ΔHR, ΔSDNN, ΔFEV_1, ΔFVC, and ΔPEF) were obtained by subtracting baseline health measures before each work shift from those obtained after each work shift. Also, the potential effects from the morning $PM_{2.5}$ exposure on the afternoon health measures could be controlled. Finally, to avoid potential confounding effects of activities on the recorded signals, the ΔSDNN and ΔHR obtained at the designated resting periods (i.e. 15 min before, 15 min, 2, and 4 h after the morning and afternoon shifts) were used for the analysis of the associations with the $\Delta PM_{2.5}$ exposure. Temperature, relative humidity, and age were included in the model to control potential effects from these parameters. For all analyses, the natural log transformed $\Delta PM_{2.5}$ concentrations were used to normalize the distribution and reduce the influence of a small subset of extremely large values.

A multiple linear regression model was used to examine the associations between the traffic counts (predictor) and the morning increase in $PM_{2.5}$ concentrations ($\Delta PM_{2.5\text{-ave-m}}$) measured during the same time period (dependent variable), controlled by wind speed (another predictor). Both total traffic counts, as well as, the counts of diesel and non-diesel-powered vehicles were examined.

Results

Personal $PM_{2.5}$ Concentration

A total of thirty-one 24-h continuous $PM_{2.5}$ measurements were collected, but three measurements (two from subject 10 and one from subject 11) were excluded due to a malfunction of the $PM_{2.5}$ monitor. As expected, elevated personal $PM_{2.5}$ concentrations were observed for most monitoring sessions. The $\Delta PM_{2.5\text{-ave-m}}$ measured for each subject ranged from 1.1 to 87 $\mu g/m^3$ and the $\Delta PM_{2.5\text{-ave-a}}$ ranged from 1.2 to 98 $\mu g/m^3$ (Table 2). The average increase in $PM_{2.5}$ concentration for all the subjects in the morning was 35.2±25.9 $\mu g/m^3$, higher than that in the afternoon (24.1±22.1 $\mu g/m^3$). Many spikes of $PM_{2.5}$ concentrations were observed during the work shifts (Figure 1), and the 1-min peak $PM_{2.5}$

concentrations ($\Delta PM_{2.5\text{-peak}}$) were often 10 times higher than the background level (~15 $\mu g/m^3$), with the maximum $\Delta PM_{2.5\text{-peak}}$ of 278 $\mu g/m^3$ during a morning shift.

A wide range of the $\Delta PM_{2.5\text{-ave}}$ and $\Delta PM_{2.5\text{-peak}}$ was observed among the 3 different working days sampled for most of the subjects (Table 2). Different wind speeds on the sampling day is the primary factor affecting the $PM_{2.5}$ concentration, as discussed in the following section. The large variations in $PM_{2.5}$ concentrations among the 3 sampling days allowed the use of each subject as his or her own control in a mixed model to examine the effects of different $PM_{2.5}$ concentrations on the measured health endpoints.

Traffic Count Information

Different vehicle types and traffic counts were observed at each school location (Table 3). The traffic counts recorded from the 10 locations ranged from 10 to 25 vehicles/min during the morning shift. Although positive relationship were observed, the associations between $\Delta PM_{2.5\text{-ave-m}}$ and traffic count were not statistically significant (P=0.78, 0.83, and 0.79 for all vehicles, diesel-powered vehicles, or non-diesel-powered vehicles, respectively, Table 4). Only wind speed was inversely associated with $\Delta PM_{2.5\text{-ave-m}}$ (P=~0.05, Table 4) for all three vehicle categories examined.

Changes in SDNN

The ECG obtained from each subject was reviewed for qualifying beats before analysis. For example, the measurements from subject 2 were excluded from the analysis due to a low proportion of qualified heart beats (<60%) resulting from frequent ectopic heart beats. Overall, 25 measurements were used for analysis.

The mean 5-min SDNNs measured at each time point during the morning and afternoon sessions are presented in Figure 2 to examine the change of SDNN with time. The error bar represents the standard deviation of the measurements at each time point. After the morning shift, significant decreases in SDNN were observed, with an average decrease of 18% (P=0.02), 21% (P=0.006), and 26% (P=0.001) at ~15 min, 2 and 4 h, respectively, after the morning shift (Figure 2). To examine whether decrease in SDNN was due to exposure to PM during the morning shift, regression analysis was conducted for the change of SDNN (ΔSDNN) on the increase in PM (ΔPM). The ΔSDNN measured at all three time points were negatively associated with $\Delta PM_{2.5\text{-ave-m}}$ (Table 5), and the association between ΔSDNN and $\Delta PM_{2.5\text{-ave-m}}$ was the strongest and most significant (P=0.0002) 2 h after the morning exposure, with 40 ms decrease in HRV for every 10 $\mu g/m^3$ increase in $PM_{2.5}$. The association between ΔSDNN and $\Delta PM_{2.5\text{-ave-m}}$ was only marginal (P=0.06) 15 min after the morning exposure, and insignificant (P=0.78) 4 h after the morning work shift. Relative humidity was found to be a significant covariate for all ΔSDNNs examined (P-values of 0.03–0.05). However, temperature, age, subjects', and former smoking status were not found to be the significant predictors for the ΔSDNN.

Similar to the morning shift, decreases in SDNN were observed post-afternoon exposure, but the change in SDNN was small and insignificant, with an average decrease of 0.3% (P=0.94), 7% (P=0.55), and 7% (P=0.53) for a lag time of 15 min, 2 and 4 h, respectively

(Figure 2). ΔSDNN measured at all three time points were negatively associated with the $\Delta PM_{2.5\text{-ave-a}}$ (Table 5), but the association between ΔSDNN and $\Delta PM_{2.5\text{-ave-a}}$ were not significant for the lag time of 15 min (P=0.77) or 4 h (P=0.95) in the afternoon, and only marginal effects were observed (P=0.1) 2 h post-afternoon exposure.

The same analysis was performed to examine the effect of peak $PM_{2.5}$ exposure ($\Delta PM_{2.5\text{-peak}}$) on the change in SDNN (ΔSDNN). Both positive and negative associations between ΔSDNN and $\Delta PM_{2.5\text{-peak}}$ were observed 15 min or 4 h after the morning and afternoon exposures (Table 5), and none of the associations were found to be significant (P ranged from 0.2 to 0.86). A negative association was observed between the ΔSDNN measured at 2 h after both work shifts, and the corresponding $\Delta PM_{2.5\text{-peak}}$, however, was not significant, with P-values of 0.72 and 0.23 for the morning and afternoon exposures, respectively.

Changes in HR

Both increases and decreases in HR were observed after exposure to $PM_{2.5}$ during the morning and afternoon shifts (Table 5, Figure 3). The association between ΔHR and $\Delta PM_{2.5\text{-ave-m}}$ was only marginal for the lag time of 2 h in the morning (P=0.08). It was insignificant for the remaining time points (P-values ranged from 0.14 to 0.87) when we examined the association between either the $\Delta PM_{2.5\text{-ave}}$ or $\Delta PM_{2.5\text{-peak}}$ and ΔHR measured during both work shifts (Table 5).

Changes in Lung Function

The mean values of lung function parameters (FEV_1, FVC, and PEF) measured before and 15 min after the morning shift are presented in Figure 4. Small decreases in FEV_1, FVC, and PEF were observed after the morning shift, but the differences in the mean of lung function measures before and after the morning shift were not found to be significant (P>0.23). ΔFEV_1, ΔFVC, and ΔPEF were found negatively associated with the $\Delta PM_{2.5\text{-ave-m}}$ and $\Delta PM_{2.5\text{-peak-m}}$ (except ΔFEV_1 and ln $PM_{2.5\text{-ave-m}}$), however, as shown in Table 6, the association was not significant (P>0.16).

Discussion

$PM_{2.5}$ Exposure

The study showed acute exposure to $PM_{2.5}$ by being near or in traffic, and the increases in $PM_{2.5}$ concentrations during the morning shift were found to be higher than during the afternoon shift. This is because the morning shift (7:30–8:30 a.m.) coincides with the rush hour which has an increase in vehicular emissions generated by a variety of vehicles, including diesel-powered school and city buses. In addition, as the morning shift also often coincides with time periods having a low atmospheric mixing height, the concentrations of air pollutants in the intersection during the morning shift are expected to be higher than the afternoon shift.

The average personal concentrations of $PM_{2.5}$ measured for crossing guards were found to be higher (about two times) than those reported for patrol officers (Riediker et al., 2004) and

other commuters (Sabin et al., 2005; Adar et al., 2007). This is probably because school crossing guards often stand in the middle of traffic or next to school bus exhausts and other vehicles while crossing school children. Moreover, frequent idling of school buses and other vehicles during the morning shift generates higher emissions than moving vehicles, resulting in higher concentrations of $PM_{2.5}$ at the locations where school crossing guards work and therefore higher personal $PM_{2.5}$ concentrations. This may also partially explain a lack of association between $\Delta PM_{2.5\text{-ave}}$ and traffic volume measured in the study. Vehicles continue to emit air pollutants while idling, thus, personal $PM_{2.5}$ concentrations of crossing guards can be increased without increases in the number of vehicles passing through their work locations, that is the concentrations of air pollutants in the congested areas may not be proportional to the number of vehicles that pass through. These observations suggest that the use of traffic count as a surrogate for acute exposure to vehicular emissions may lead to underestimation of personal exposures to air pollutants in high trafficked areas. For those locations, besides considering the traffic count, the time vehicles are present in the monitoring locations should also be considered when estimating personal exposure to vehicular emissions.

Effects of $PM_{2.5}$ Exposure on HRV, HR, and Lung Function

For health outcomes, we did not find significant effect of peak $PM_{2.5}$ exposure ($\Delta PM_{2.5\text{-peak}}$) on the heath measures in the study, thus, the following discussion primarily focuses on the impact of the increases in the average $PM_{2.5}$ ($\Delta PM_{2.5\text{-ave}}$) during the 1-h work shifts.

$PM_{2.5}$ Exposure and SDNN

The present study found a consistent negative association between changes in SDNN and exposure to increases in $PM_{2.5}$ from vehicular exhaust, suggesting that exposure to elevated PM can result in decrease in SDNN. These observations are in agreement with the findings of previous studies (de Paula Santos et al., 2005; Schwartz et al., 2005; Adar et al., 2007). For example, Adar et al. (2007) reported significant associations between decreases in HRV and particle exposure in the elderly. Also, they reported a stronger association between the decreases in HRV and exposure to $PM_{2.5}$ experienced during the 2-h bus trip than that from non-bus periods. The findings from this study again suggest that exposure to $PM_{2.5}$ with traffic origin may alter autonomic nervous system, which may be one of the potential physiological mechanisms linking particle air pollution due to traffic and cardiovascular diseases.

An association between changes in SDNN and increases in $PM_{2.5}$ ($\Delta PM_{2.5\text{-ave-m}}$) was found highest 2 h after morning shift. In addition, the effect of $PM_{2.5}$ on HRV became insignificant 4 h after the morning exposure. These results suggested that acute exposure to $PM_{2.5}$ may alter HRV for a short period of time. Our observations are consistent with those reported by Vallejo et al. (2006), who reported the strongest effects on HRV for $PM_{2.5}$ concentrations that were 1.5 and 2 h before the HRV measurements. Nonetheless, it is worth noting that various lag times for the HRV change associated with exposure to elevated $PM_{2.5}$ have been reported previously. Gold et al.(2000) reported decreases in HRV in the 21 older adults after exposure to increases in ambient $PM_{2.5}$ over the previous 4 h; other researchers (Liao et al.,

1999; Pope et al., 1999) reported changes in HRV with a lag time of 24 h after exposure to elevated $PM_{2.5}$; and Park et al.(2005) reported strongest association of HRV with the 48-h moving averages of $PM_{2.5}$. The mechanisms underlying the effect of $PM_{2.5}$ on the autonomic system are not well understood and require further investigation.

The decreases in SDNN post-afternoon exposure were very small and insignificantly. The lack of significance in the afternoon sessions may be partially due to the lower afternoon $PM_{2.5}$ concentrations than in the morning (see Table 2). In addition, we noticed that the SDNN before the afternoon work shift (the afternoon base in Figure 2) was lower than the morning baseline (Figure 2), leading to a small difference in SDNN between post-afternoon and before afternoon exposure. It is not clear whether the lower afternoon SDNN baseline was due to the carry-over effect from the morning exposure on HRV or diurnal pattern of the HRV.

$PM_{2.5}$ Exposure and HR

Surprisingly, we did not observe any consistent or significant associations between changes in HR and increases in $PM_{2.5}$ exposure. Lack of such associations should not be attributed to the light activities of the subjects during their work shifts. As described in “Materials and methods” section, the HRs measured at 15 min, 2, and 4 h after work shift were used for analysis. Further, the subjects sat quietly for 10 min, and the ECG from a 5-min period towards the end of the 10 min period was examined. Thus, there should have been sufficient time for the HR to return to the subjects’ resting baselines before the 5-min ECG data period.

Different $PM_{2.5}$ effects on HR were reported previously. Ibald-Mulli et al. (2004) found a significant decrease in HR associated with $PM_{2.5}$ exposure in only one of four centers studied, but no significant associations were found for most subjects examined. In contrast, other investigators (Peters et al., 1999; Pope et al., 1999; Adar et al., 2007) reported increases in HR resulted from exposure to $PM_{2.5}$. The discrepancy observed in different studies are not known. This may be partially due to different exposures and subjects conditions examined in each study.

$PM_{2.5}$ Exposure and Lung Function

No significant changes in lung function measures were observed for the crossing guards after their acute exposures to elevated $PM_{2.5}$ concentrations during their work shifts. The findings are consistent with previous studies of effects on lung function with controlled PM exposure (Salvi et al., 1999; Devlin et al., 2003). For example, Salvi et al. (1999) observed increases in systemic and pulmonary inflammatory markers in healthy human volunteers after their 1-h exposure to diesel particles (300 $\mu g/m^3$ of PM_{10}), but found no changes in standard lung function measurements.

It is recognized that some limitations exist in the study design. First, the small size of the study may have resulted in insufficient power to detect some associations between health outcomes and elevated personal $PM_{2.5}$ levels. Second, personal exposures to other traffic-related air pollutants, such as CO and NO_2 that may affect the cardiopulmonary endpoints,

were not measured. Third, we did not control other exposure-related factors that might have confounded the association between $PM_{2.5}$ exposure and HRV, such as stress and noise during the work shift. Thus, we cannot determine whether the co-pollutants and/or other factors may have contributed to the effects observed. Nonetheless, the findings from the study suggest that common acute exposure to mobile source emissions may have important cardiovascular health consequences. Further studies are needed to investigate how the acute decline in HRV due to exposure to vehicular emission may be related to the increased risks of adverse cardiovascular events. Moreover, this study suggests that research is needed to examine the correlation between personal exposure levels of traffic generated air pollutants and traffic count in traffic congested areas to define the proper surrogates for future exposure and health studies associated with traffic air pollution.

Acknowledgments

This research was supported in part by the NIEHS sponsored UMDNJ Center for Environmental Exposures and Disease (NIEHS P30ES005022), Q. Meng is a US EPA/NCEA-DOE/ORISE research fellow. We thank Dr. Linda Bonanno from the New Jersey Department of Environmental Protection for providing contact information for the Paterson Police Department in New Jersey. We also thank the subjects for their participation and the support from the Paterson Police Department. The views expressed in this article are those of the authors and do not necessarily reflect the views or policies of the funding agencies and US EPA.

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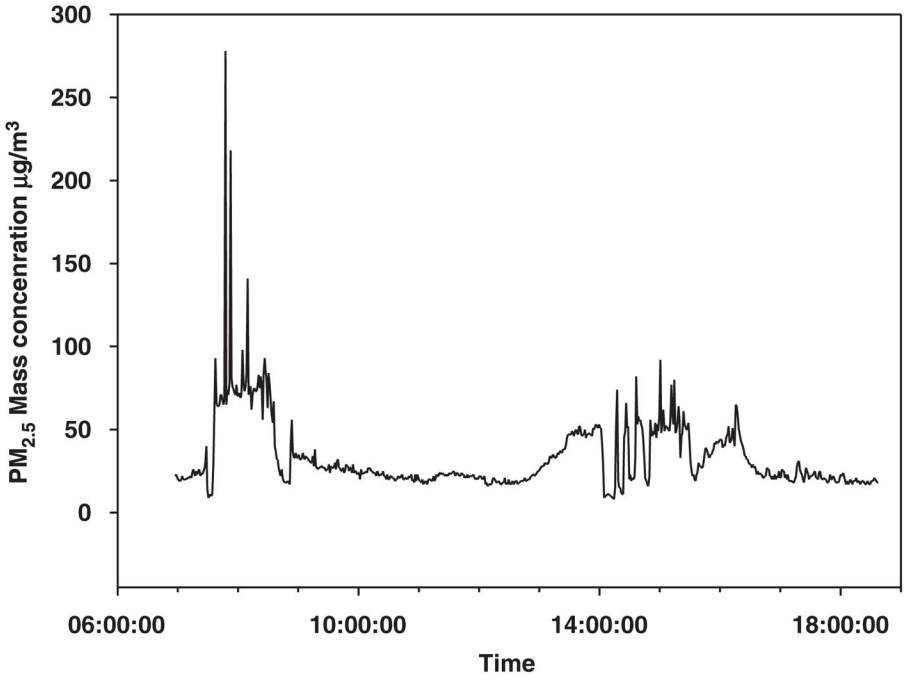

Figure 1.

The first 12 h $PM_{2.5}$ concentration over the 24-h monitoring period monitored by a real-time $PM_{2.5}$ monitor on 3/11/2005. The $PM_{2.5}$ was close to baseline between 1800 and 0700 hours in the next morning.

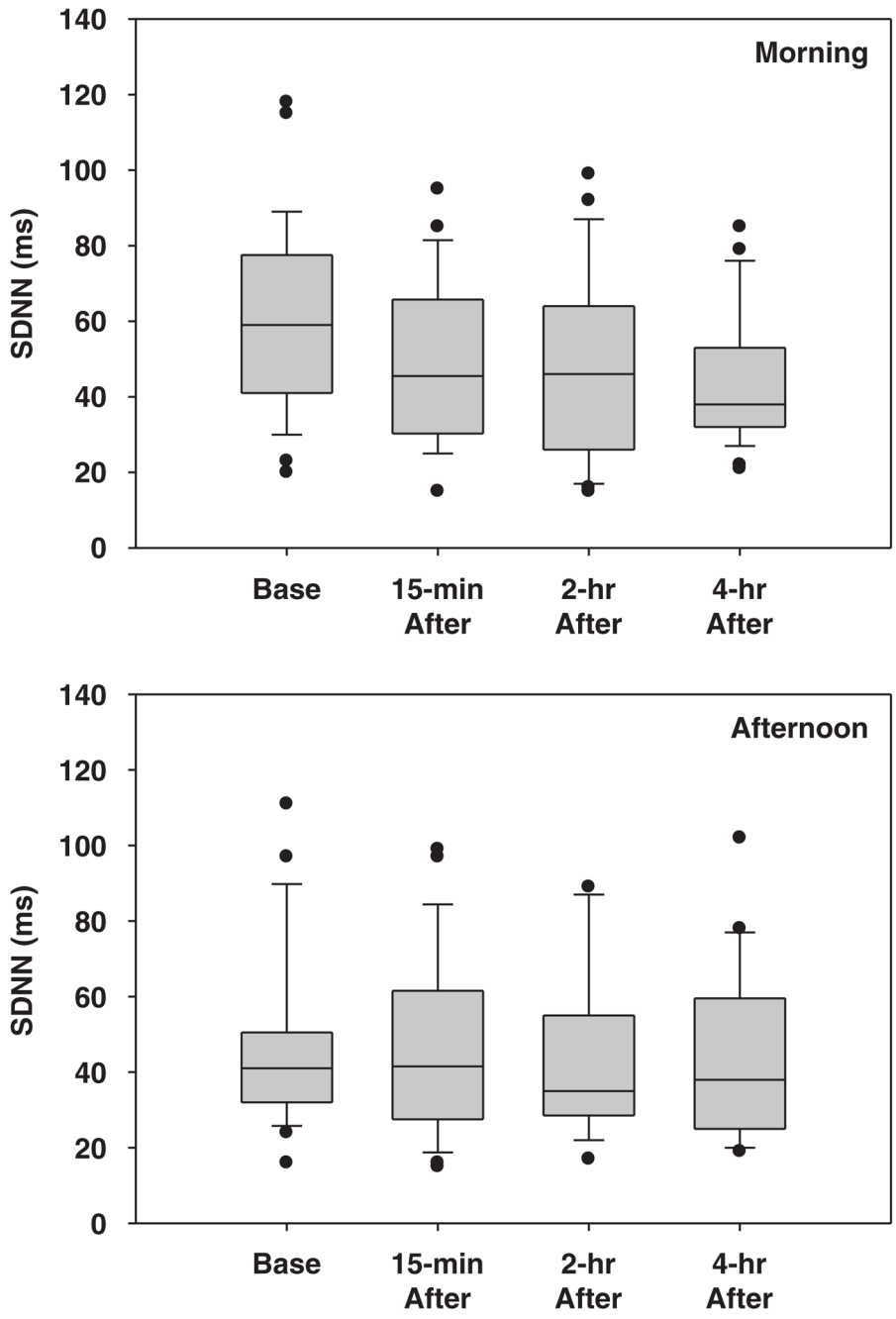

Figure 2.
The variable (SDNN) of heart rate variability measured before and after the morning (top) and afternoon work shift (bottom).

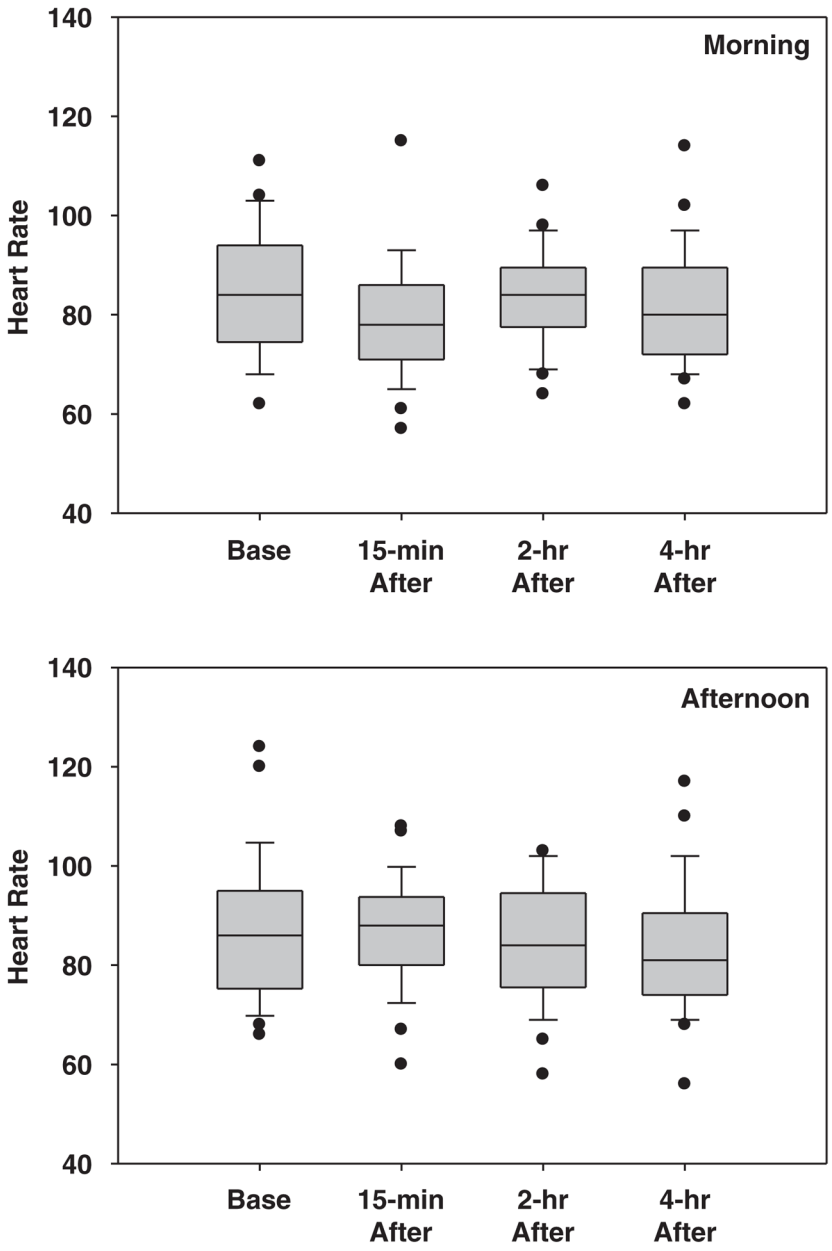

Figure 3.
The heart rate measured before and after the morning (top) and afternoon work shift (bottom).

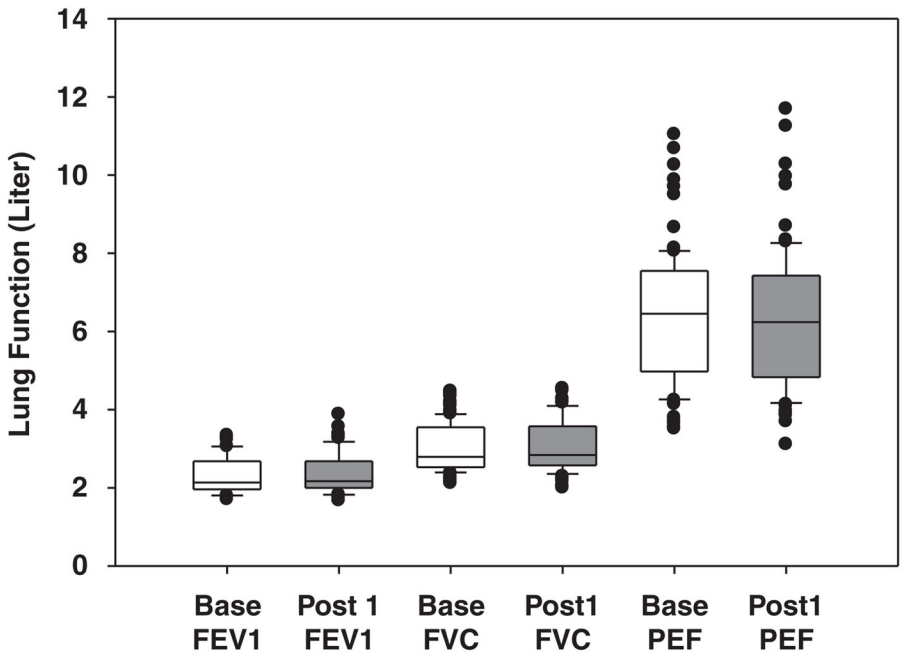

Figure 4.
The lung function (FEV_1, FVC, and PEF) measured before and after the morning shift.

Table 1

Characteristics of study subjects (n=11).

Characteristics	
Age (*years*)	61.2±13.7
<50	2
50–69	5
>70	4
Sex	
Male	6
Female	5
Race/ethnicity	
Black	6
Hispanic	5

No reports of bronchitis, respiratory illness, diabetics, or prior myocardial infarction. No subject taking β-blockers.

Table 2

Summary of the increases in $PM_{2.5}$ concentrations (1-h average and peak $PM_{2.5}$ concentrations, $\mu g/m^3$) during the morning and afternoon shifts.

Subject ID	$\Delta PM_{2.5\text{-ave}}$		$\Delta PM_{2.5\text{-peak}}$	
	Morning	Afternoon	Morning	Afternoon
01A	7.3	2.6	22	39
01B	35.1	4.4	89	26
01C	25.8	11.9	39	23
02A	62.4	13.9	146	120
02B	64.4	30.5	84	122
02C	86.8	47.2	148	101
03A	3.1	2.4	17	18
03B	16.1	1.2	24	3
03C	17.0	11.6	41	57
04A	84.7	23.7	126	45
04B	15.2	5.8	100	33
04C	1.1	2.2	4	4
05A	74.9	47.5	278	92
05B	47.9	37.6	73	120
05C	20.3	26.5	32	34
06A	22.1	11.2	62	67
06B	8.0	1.6	15	6
06C	65.9	63.8	77	95
07A	13.0	5.0	67	18
07B	40.8	24.9	110	53
07C	21.6	13.9	45	75
08A[a]	21.2	16.8	26	45
09A	32.7	21.2	50	28
09B	31.2	42.6	51	150
09C	28.8	22.0	63	105
10C[b]	80.0	51.0	107	72
11A[c]	15.8	25.0	46	114
11C[c]	41.6	33.3	54	60
Average±SD	35.2±25.9	24.1±22.1	71.3±56.1	64.3±43.5
Range	1.1–87	1.2–98	4.0–278	3.0–150

[a] Subject 08 withdrew from the study after one measurement.

[b] One valid exposure measurement for subject 10.

[c] Two valid exposures for subject 11.

Table 3

Traffic count and type recorded for 15 min during the morning shift at each school location.

Date	Site	Bus	Car	Large truck	Small truck
2/01/05	1	14	212	4	4
2/11/05	2	21	180	7	6
2/14/05	3	11	154	5	1
5/31/05	3	21	397	11	2
3/07/05	4	10	324	9	1
3/18/05	5	8	196	4	3
4/06/05	5	6	209	1	3
4/13/05	6	5	233	2	2
4/11/05	7	6	162	11	2
4/27/05	8	17	137	1	2
5/02/05	9	7	76	0	1
5/13/05	10	17	395	12	7

Table 4

Regression coefficients for various traffic counts controlled by wind speed (n=12)[a].

Variable	Slope for traffic counts (SE)	P-value	Slope for wind speed (SE)	P-value
All vehicles	0.018 (0.06)	0.78	−3.28 (1.52)	0.06
Diesel-powered vehicles	0.15 (0.68)	0.83	−3.44 (1.38)	0.04
Non-diesel-powered vehicles	0.019 (0.07)	0.78	−3.28 (1.53)	0.06

[a]Note: a multiple linear regression was used to predict the elevated average $PM_{2.5}$ ($\mu g/m^3$) during the recording time. Traffic counts and wind speed are two independent predictors.

Table 5

Regression coefficients of the changes in SDNN (ΔSDNN, in the unit of ms) and heart rate (ΔHR, in the unit of beat/ms) measured after the morning and afternoon shifts relative to increases in the average and peak $PM_{2.5}$ mass concentration during the 1-h work shift [$\ln(\Delta PM_{2.5})$, in the unit of $\mu g/m^3$] (N=25).

	ΔSDNN–$\Delta PM_{2.5}$			ΔHR–ΔPM	
	$\ln\_\Delta PM_{2.5}$	**Estimate (SE)**	**P-value**	**Estimate (SE)**	**P-value**
Morning					
15 min	$\ln\_\Delta PM_{ave\text{-}m}$	−14.5 (6.9)	0.06	1.2 (3.1)	0.71
2 h	$\ln\_\Delta PM_{ave\text{-}m}$	−18.9 (4.2)	0.0002	−5.5 (2.9)	0.08
4 h	$\ln\_\Delta PM_{ave\text{-}m}$	−2.5 (8.6)	0.78	−3.1 (4.6)	0.51
15 min	$\ln\_\Delta PM_{peak\text{-}m}$	−9.2 (11.2)	0.43	0.8 (4.4)	0.86
2 h	$\ln\_\Delta PM_{peak\text{-}m}$	−5.1 (13.8)	0.72	−7.2 (4.2)	0.11
4 h	$\ln\_\Delta PM_{peak\text{-}m}$	7.4 (12.0)	0.55	−7.1 (6.3)	0.28
Afternoon					
15 min	$\ln\_\Delta PM_{ave\text{-}a}$	−2.4 (7.6)	0.77	−2.0 (4.0)	0.62
2 h	$\ln\_\Delta PM_{ave\text{-}a}$	−20.2 (10.8)	0.10	0.9 (5.4)	0.87
4 h	$\ln\_\Delta PM_{ave\text{-}a}$	−0.7 (11.2)	0.95	8.2 (5.2)	0.14
15 min	$\ln\_\Delta PM_{peak\text{-}a}$	0.6 (8.9)	0.95	−5.6 (5.3)	0.31
2 h	$\ln\_\Delta PM_{peak\text{-}a}$	−19.2 (14.6)	0.23	3.1 (8.1)	0.71
4 h	$\ln\_\Delta PM_{peak\text{-}a}$	−6.8 (14.1)	0.64	11.1 (8.1)	0.20

Table 6

Regression coefficients (95% CI) of the changes in lung function (ΔFEV_1, ΔFVC, and ΔPEF, in the unit of L) measured after the morning shift relative to increases in the average and peak $PM_{2.5}$ mass concentration during the 1-h work shift ($ln\Delta PM_{2.5}$, in the unit of $\mu g/m^3$) (*N*=27).

Lung function	PM type	Estimate (SE)	P-value
ΔFEV_1	$ln\_\Delta PM_{ave\text{-}m}$	0.02 (0.04)	0.68
ΔFVC	$ln\_\Delta PM_{ave\text{-}m}$	−0.10 (0.09)	0.31
ΔPEF	$ln\_\Delta PM_{ave\text{-}m}$	−0.54 (0.62)	0.42
ΔFEV_1	$ln\_\Delta PM_{peak\text{-}m}$	−0.13 (0.08)	0.16
ΔFVC	$ln\_\Delta PM_{peak\text{-}m}$	−0.12 (0.17)	0.51
ΔPEF	$ln\_\Delta PM_{peak\text{-}m}$	−1.46 (1.12)	0.24

Abbreviations: FVC, forced vital capacity; FEV_1, forced expired volume in the first second; PEF, peak expired flow.