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Long-term Metal $PM_{2.5}$ Exposures Decrease Cardiac Acceleration and Deceleration Capacities in Welders

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Abstract

Objective—To clarify if long-term metal particulates affect cardiac acceleration capacity (AC), deceleration capacity (DC) or both.

Methods—We calculated Chronic Exposure Index (CEI) for $PM_{2.5}$ over the work life of 50 boilermakers and obtained their resting AC and DC. Linear regression was used to assess the associations between CEI $PM_{2.5}$ exposure and each of AC and DC, controlling for age, acute effects of welding exposure, and diurnal variation.

Results—Mean (SD) CEI for $PM_{2.5}$ exposure was 1.6 (2.4)mg/m^3-workyears and ranged from 0.001 – 14.6mg/m^3-workyears. In our fully adjusted models, a 1 mg/m^3-workyear increase in CEI for $PM_{2.5}$ was associated with a decrease of 1.03 (95% CI: 0.10, 1.96)msec resting AC, and a decrease of 0.67 (95% CI: −0.14, 1.49)msec resting DC.

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Conflicts of Interest: None

Conclusion—Long-term metal particulate exposures decrease cardiac accelerations and decelerations.

Keywords

Deceleration; Heart Rate; Welding; Electrocardiography; Occupational Exposure

INTRODUCTION

Fine particulate ($PM_{2.5}$) exposure has been documented as a major environmental factor contributing to increased cardiovascular morbidity and mortality(1, 2). Fine particulates have also been shown to have cardio-pulmonary effects, especially among welders with large occupational exposures(3, 4).

Based on 2012 estimates from the Bureau of Labor Statistics (BLS), 357,400 workers in the United States are employed as welders, cutters, solderers, and brazers (5). Boilermaker construction workers (boilermakers) are welders who work in power plants and are exposed to large amounts of metal-rich particulate fumes. Occupational exposure to welding fumes occurs during the metal joining process. Boilermakers are also exposed to the residual fly ash left following the combustion of the fuel in the power plant. While $PM_{2.5}$ is highly ubiquitous in the ambient environment, exposures in these work settings where there is increased generation of particulates may be even more substantial. For example, epidemiologic studies have shown that boilermakers involved in welding activities are exposed to over ten times ambient levels of particulate matter in weld fumes(3).

Several studies have linked ambient $PM_{2.5}$ exposure with cardiovascular disease(6, 7), but there are few studies that have examined this association in an occupational setting with much higher airborne exposures from work processes. According to our knowledge, no study has examined fine particulate exposures over the course of the work-life among workers who have been in these occupational settings for long periods, and have the potential for a cumulative multiplier effect from $PM_{2.5}$ exposure over the years(8). The effect of these exposures in the long term on cardiac autonomic function remains unclear.

Autonomic changes in the heart from exposures to particulates have traditionally been measured using heart rate variability (HRV) indices(9), which have an inherent potential for misclassification because of their inability to account for accelerations and decelerations, which may be more predictive of morbidity and mortality among post-ischemic coronary artery disease patients than traditional HRV(10). Baeur et al described the phase-rectified signal averaging (PRSA) method for calculating the heart's acceleration capacity (AC) and deceleration capacity (DC), which are measures of the responsiveness of the heart like HRV during speeding up and slowing down of the heart respectively (10).. Therefore, using sensitive indices - Acceleration Capacity and Deceleration Capacity - this study aims to investigate the potential for cardiac autonomic dysfunction from long-term metal $PM_{2.5}$ exposure. We hypothesize that an increase in long-term particulate exposure will decrease AC and DC. We will also characterize and describe $PM_{2.5}$ exposures during the work-life of boilermakers using a chronic exposure index; this will be measured by obtaining typical work shift $PM_{2.5}$ exposure in these workers, and using their previous detailed work shift

schedules over the years to compute their long-term $PM_{2.5}$ exposure distribution patterns during their work life(11, 12).

METHODS

Subject Recruitment

The 50 study participants were among 72 boilermakers who were part of an ongoing "Harvard Boilermaker Cohort" initiated in 1999 to study the cardio-pulmonary effects of particulates(13). We recruited 52 of the 72 male boilermakers in the Harvard Boilermakers Cohort, through an outreach to them by phone and email, between January 2010 and June 2012 from the boilermaker union in Quincy, Massachusetts for our study. We were able to analyze data from 50 boilermakers for our study. They constituted 70% of the participants in the existing cohort whom we were able to retrieve detailed work histories from when they became boilermakers, obtain $PM_{2.5}$ exposure measures, as well as record resting digital ECG in sampling periods between 2010 and 2012. We restricted our study to the 52 participants who were monitored at least once on a welding day to estimate their shift $PM_{2.5}$ exposure. Of these 52, we lost data for 2 participants due to failed initialization of Holter and poor data retrieval of ECG data. We recorded their most recent ECG during winter or summer when 85% of the 50 study participants had not actively welded two weeks prior to our data collection. The Institutional Review Board at the Harvard T. H. Chan School of Public Health approved the study protocol. We obtained written, informed consent from all study participants.

Data Collection

We collected shift $PM_{2.5}$ exposure and resting ECG data of study participants at the boilermaker union welding school in Quincy, Massachusetts. Boilermakers practiced welding in booths designed for training apprentices at the welding school. We obtained from the boilermakers' union a detailed previous work schedule over their work life since they became boilermakers after we had obtained consent from the participants.

Participants completed a self-administered questionnaire on demographics, medical history and medication use information, lifestyle information including smoking, typical diet, and occupational history. We collected information on heart and blood vessel problems: myocardial infarction, angina, arrhythmia, hypertension, use of blood pressure medications such as beta blockers or ACE inhibitors, congestive heart failure, heart/chest surgery, or other heart problems diagnosed by a physician.

Shift $PM_{2.5}$ Assessment

We measured $PM_{2.5}$ concentrations during welding shifts of study participants using personal DustTrak™ Aerosol Monitor (TSI, Inc., St. Paul, MN). The DustTrak™ tube monitor was strapped to the participant's shoulder close to their breathing zone. DustTrak™ has a $PM_{2.5}$ inlet impactor to measure continuously and record at 1-minute intervals average concentrations of fine particulates during the welding shifts. Kim et al had validated DustTrak™ continuous readings of $PM_{2.5}$ with gravimetric methods in welders(14). We calculated mean concentrations of $PM_{2.5}$ exposure during each work shift for each

participant. Subsequently, we calculated the typical shift exposure for each participant by taking the average of $PM_{2.5}$ exposures for all work shifts for that participant.

Chronic ($PM_{2.5}$) Exposure Index

We computed a Chronic Exposure Index (CEI) in mg/m^3-workyears by multiplying the average $PM_{2.5}$ (in mg/m^3) during a typical shift and the work life – summation of all the days (expressed in workyears) they had ever worked as a boilermaker. A work year was defined as 260 days based on a five day work week for 52 weeks in a year. We used the sum of all days worked over the years because most boilermakers have variable off-work periods between workdays in a year. To express CEI in mg/m^3-workyears, we converted days to years by dividing by 260.

Work Life (workyears) = Σ workdays ever since boilermaker / 260

CEI (mg/m^3-workyears) = Shift $PM_{2.5}$ (mg/m^3) * work life (workyears)

ECG recording and processing

Study participants wore a standard five lead ECG Holter monitor after a thirty minute rest period in the morning on arrival at the union hall. The rest period was to allow us record their unbiased resting ECG free from acute changes resulting from commuting to the study site. To ensure the leads of the ECG were well secured and remained secured on the chest of participants, we shaved their skin if necessary, cleaned with an alcohol wipe after slightly abrading the skin, and research staff checked them intermittently(3). Trained technicians blinded to the exposure status of the participant from whom the ECG reading was obtained, cleaned and exported data for analysis using the Physionet toolkit(15). To remove artifacts from the data, they used only beats with an RR interval within 5% difference of adjacent beats. They used an automated process described by Bauer to create 5-minute segments with anchors for the Phase-Rectified Signal Averaging (PRSA) method of computing the acceleration capacity (AC) and deceleration capacity (DC) (10). In brief, to compute the DC, this involves identifying heartbeat intervals longer than the preceding interval as anchors (for AC, beats shorter than preceding beats were anchors). Overlapping segments of interval data were then automatically generated from the ECG such that all segments are aligned at the anchors in the center and averaged. The PRSA method then quantifies the signals within aligned segments using the Haar wavelet analysis with a scale of 2 by a computer processing of the ECG with visual and digital outputs. Thus, AC and DC were calculated separately as a quarter of the difference between two sums, that is, the sum of the averaged anchor points RR intervals (X_0) with the succeeding RR intervals (X_1) and the sum of the two averaged RR intervals preceding anchor points (X_{-2}, X_{-1}) (16).

Acceleration Capacity (AC) and Deceleration Capacity (DC)

Using the digital ECG data in the time domain, we computed the average AC and average DC for the resting 10-minute period by taking the mean of the first two 5-minute segments of the ECG using the automated output.

Data Analysis

We calculated summary measures of potential covariates of our exposure and outcome to further understand their distribution. We considered age, race, smoking status, time of day when ECG was obtained, season of study, acute effects of welding, and presence of heart problems as potential covariates. We then explored the inter-relationships between them by using spearman's correlations for continuous variables and t-tests for binary variables. We used α=0.10 level for these correlations and t-tests between covariates and outcome/ exposure, so as not to miss any potential inter-relationships between them.

We ran linear regression model to assess the crude association between $PM_{2.5}$ Chronic Exposure Index (CEI) as a continuous measure and AC, and a separate model for CEI and DC. Because age was correlated with CEI, we first ran a linear regression model of age versus CEI, and used the residuals (variation in age not accounted for by CEI) to adjust for the age. This represents the biological age of the heart independent of exposure. We then ran age-adjusted models to control for age – using the biological heart age independent of CEI. Furthermore we used backward selection method of model building using a p-value of 0.1 each for remove and stay to select our final model. We considered controlling for age, smoking status, and time of day and season when ECG reading was obtained. Our final model had only age in the model when we constrained CEI to remain in the model. We also ran a model that included age and time of day ECG was taken to control for diurnal variations in ECG, and another model that included age, time of day ECG was taken, and last weld day to additionally control for acute effects of particulates. Statistical significance was assessed at α=0.05 level in two sided tests for our final model. All analyses were performed using PROC REG in SAS version 9.4 (Cary, NC).

RESULTS

There were 50 participants, all males with a mean age of 39 years and were retrospectively followed for a median of 4 years (range 0.25 – 21 years). The study population were predominantly white (44 [88%]) non-smokers (32 [64%]) (Table 1). Ten (10) participants reported heart problems and possible heart-related problems including arrhythmias (n=3), sinus (sinoatrial node) problems (n=3), palpitations (n=2), and complicated diabetes (with palpitations) that may affect autonomic control of the heart (n=2), for which they were taking insulin. No participant reported the use of beta-blockers or ACE inhibitor drug use. Most (72%) of the resting ECG were taken in the morning, and each participant had $PM_{2.5}$ measurements taken in 3–6 typical work shifts of 4 – 6 hours.

The mean work life was 4 years (range 0.3 – 14.8 workyears). The mean $PM_{2.5}$ during a typical work shift was 0.4mg/m^3 (range 0.01 – 1.40 mg/m^3), and the mean CEI was 1.36 mg/m^3-workyears (range 0.001 – 14.56 mg/m^3-workyears).

The mean (range) AC was −7.1msec (−0.7 to −16.9) on the negative scale with the middle half of the participants ranging between −4.2 to −9.2msec (Table 2). The mean (range) DC was 8.7msec (1.8 to 18.5) on the positive scale with the middle half of the participants ranging 6.4 to 10.3msec.

Spearman's correlations coefficients (and p-value) for correlation between CEI and AC were 0.26 (*0.07*), and −0.30 (*0.03*) for that between CEI and DC. Among the covariates considered, only age was correlated with both exposure and outcome. Coefficients (and p-value) for correlations between age and CEI, AC, and DC, were 0.44 (*<0.01*), 0.30 (*0.03*), and −0.28 (*0.05*) respectively.

There was a marginal statistically significant difference between the exposure (CEI) indices of participants whose ECG were taken in the morning (1.26mg/m^3-workyears) versus afternoon (2.58 mg/m^3-workyears) (*p=0.08*), but not with the outcomes (AC and DC). There were no differences in categories of other covariates in terms of measures of CEI, AC and DC.

The linear regression analyses revealed that elevated CEI $PM_{2.5}$ levels were associated with AC and marginally associated with DC after adjusting for age (Table 3). A 1mg/m^3-workyear increase in CEI resulted in a decrease of 1.46msec (95% CI: 0.23, 2.69; *p=0.02*) resting AC and a decrease of 0.99msec (95% CI: −0.08, 2.06; *p=0.07*) resting DC. When we further controlled for the time of day ECG was taken and last day welded, the negative age-adjusted association between CEI and AC remained consistent.

We evaluated distributional assumptions of our linear regression model by examining the residual versus predictor plots and using the Cook's distance. We identified a potential outlier using Cooks' distance (criterion>0.5). We therefore evaluated these associations with and without this observation. Without this outlying observation, these associations were consistent.

We further explored the data by adding a quadratic term for CEI in our models. The associations remained the same, but the quadratic term for CEI was not significant. Therefore, our final model for all 50 participants was the linear regression model that was adjusted for age which was the variable retained in our model selection process when CEI was constrained in the model. Our sensitivity analyses for 40 participants without heart disease showed similar findings (Table 4).

DISCUSSION

The goal of this study was to investigate the associations between acceleration capacity and deceleration capacity with long-term occupational $PM_{2.5}$ exposures. True to our hypotheses, long-term occupational $PM_{2.5}$ exposure was associated with acceleration capacity and deceleration capacity. We found significant negative exposure–response relationships between long-term $PM_{2.5}$ exposure index with acceleration capacity and deceleration capacity. There may therefore be a reduction in the capacity of the heart to accelerate and decelerate over time with repeated injury from these exposures. These data imply that there may be changes in sympathetic and parasympathetic modulation and/or non-autonomic control of the heart over time with repeated exposure to particulates in the long term. Few studies have documented associations between particulate exposures and heart rate variability in the short term(1, 3, 17–19). Yet fewer studies have observed associations between fine particulate exposure and deceleration capacity, also in the short term(20). This

study is the first to demonstrate declines in acceleration capacity following occupational particulate exposure over a long-term period.

Due to the differences in the length of follow up period, exposure characteristics, and study population, we cannot directly compare our study results with those of prior studies. Although, we did find a trend towards a decline in the deceleration capacity similar to declines reported following acute $PM_{2.5}$ exposures, we also did observe declines in acceleration capacity which has never been reported even in studies in the short term. This result highlights the possibility of differences in effect among our study population (active welders) compared to other studies (acute myocardial infarction patients) that were mostly conducted in clinical settings. Ten of our study participants reported known or possible heart problems, but none reported angina, myocardial infarction or other symptoms of ischemia. This suggests that there may be modification of effects of particulates in acutely ischemic versus non-acutely ischemic conditions.

Furthermore, the different constituents of the particulate exposures may have varying effects on the electrical activity of the heart. Welding fumes contain fine particles and vaporized metals from the metal alloy being welded and the electrodes used. Common metal constituents include aluminum, chromium, copper, cadmium, iron, lead, manganese, magnesium, nickel, silica, titanium, and zinc(3, 14). While organic and elemental carbons have been implicated as suspects, metals have also been shown to play a role(4, 20–22). Welders are exposed to metal-rich fumes, and metal-rich $PM_{2.5}$ may be responsible for the effects on acceleration capacity we found in this study.

Acceleration capacity is a measure of the responsiveness of the heart to speed up when stimulated, and it is under both autonomic (sympathetic) and non-autonomic control. Deceleration capacity on the other hand, describes the behavior of the heart when the heart is slowing down, and it reflects a measure of parasympathetic modulation of the heart(20). While we found a significant effect with the acceleration capacity, the effect of particulates on the deceleration capacity was only marginally significant. We would therefore hypothesize that over a work life of exposure to particulates, the non-autonomic control may be playing a more prominent role in the response of the heart. We also cannot conclude if there is a significant change in the nervous control from exposure to particulates in the long term from our results.

Deceleration capacity has been shown to be predictor of mortality following acute myocardial infarction even among patients with preserved acceleration capacity. This is because there is a rapid decline in the parasympathetic innervation of the heart following ischemia(10, 23). However, the clinical usefulness of the acceleration capacity is still unknown. This may be explained by the multi-factorial influences on the acceleration capacity. Therefore, whereas deceleration capacity specifically measures vagal output to the heart, sole sympathetic innervation to the heart cannot be captured by acceleration capacity directly.

Age is a significant predictor of both acceleration and deceleration capacities. Ageing results in declines in both the ability of the heart to speed up and slow down. In our models, age

was a more significant predictor than our exposure index. This may suggest that declines in biological functions with aging may be more important in determining autonomic responses of the heart than exposure to fine particulates. However, there are studies that have demonstrated that there are changes in the automaticity of the heart (using heart rate variability indices) even in the young on exposure to fine particulates(17, 24).

The exposure distribution of our chronic $PM_{2.5}$ index in this study was log-normal, like most occupational exposures, with the mean about twice the median across all subjects. We used repeated shift $PM_{2.5}$ real time dust track measures to derive average exposures of each participant. The $PM_{2.5}$ surprisingly followed a near-normal distribution and we would have missed the accurate exposure distribution pattern if we used only shift $PM_{2.5}$ as our exposure measure. Work-life days (workyears) in our data also mirrored our chronic exposure index distribution pattern, and may be a fair exposure assessment in situations where shift $PM_{2.5}$ is unavailable to compute the chronic exposure index(8).

Based on our study results, reducing exposure and encouraging recovery activities following exposure would be a major goal of health protection for these workers. Even with the potential for a healthy worker effect, we still found an association with long-term exposure to particulates. The use of personal protective equipment such as a respirator has been shown to reduce exposures to particulates and improve heart rate variability(25). Furthermore, activities such as exercise, adequate sleep and good nutrition may allow for recovery of the heart after exposure to weld fumes during the day(26).

We recognize limitations in our study. Although, we were able to capture exposure to particulates in the long-term using a chronic index measure, this may have resulted in non-differential misclassification of the exposure. This would bias the results towards the null, and our results may therefore be slightly attenuated and the true effects may even be greater. There is a potential for selection bias in our study as we were only able to retrieve work records for 70% of our participants. However, there were no differences in characteristics of the welders whom we were not able to retrieve their records and those in our study. In spite of the retrospective nature of our study, we used recent repeated shift $PM_{2.5}$ measured between 2010 and 2012, and retrieved work records to estimate their average exposure patterns over their work life. This would have reduced the potential for recall bias in our exposure. These $PM_{2.5}$ measurements were obtained in comparable work settings at the union hall and may reflect what their typical exposures would have been if they were followed up prospectively.

Importantly, we captured ECG tracings digitally that were parsed into phases of accelerations and decelerations accounting for differences in heart rate. Our results are therefore not confounded by heart rate. We also attempted to adjust for confounding by potential confounders, but most were not retained in our final model. We were able to account for short-term effects of welding by restricting our study population to those who had not welded two weeks prior to our study, and adjusted for ‘last weld day’ (to control for acute effects of welding) in our models. Our results are robust and consistent with and without outlying observations.

In conclusion, we found that increasing long-term particulate exposure resulted in a decrease in both acceleration capacity and deceleration capacity. Although there may be potential differences in physiological mechanisms of control on acceleration and deceleration capacities, the effect of long term fine particulate exposure on the regulatory pathways for these indices may be inter-related. Efforts need to be concerted towards protecting cardiorespiratory health of these welders.

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Table 1

Demographics and characteristics for all 50 study participants.

Individual Characteristics	***N***	***%***
Male	50	100
Whites	44	88
[1]Smokers	18	36
Other Characteristics		
[2]Time of ECG (AM)	36	72
[3]Season of ECG (winter)	21	42
[4]Heart problems	10	20
	Mean	**s.d.**
Age at ECG measurement (years)	39	11
Age at becoming a boilermaker (years)	32	9
Length of follow up (years)	7	6
[5]Last Weld Day before study (days)	36	37

[1]Current Smokers vs Non-smokers and Previous smokers

[2]Morning (AM) vs Afternoon(PM)

[3]Winter vs Summer

[4]Heart problems include reported previous history of arrhythmia, cardiac sinus problems, or palpitations

[5]One participant had not welded for 3 years and was not included in this statistic

Table 2

Across subject distribution of $PM_{2.5}$ Chronic Exposure Index (CEI) in mg/m^3-workyears and acceleration (AC) and deceleration (DC) capacities in msec for 50 participants.

		Percentiles		
	Mean (SD)	**25th**	**50th**	**75th**
Exposure				
[1]CEI	1.6 (2.4)	0.5	0.8	1.7
[2]Shift $PM_{2.5}$	0.4 (0.3)	0.3	0.4	0.5
[3]Work Life	4.0 (3.7)	1.4	2.0	6.3
Outcome				
[4]AC(msec)	−7.1 (3.9)	−4.2	−6.4	−9.2
DC(msec)	8.7 (3.4)	6.4	8.4	10.3

[1]Cumulative Exposure Index (mg/m^3-years) = $PM_{2.5}$(mg/m^3) × Work Life (workyears)

[2]This is the across subject average for typical shifts sampled

[3]Work Life (workyears) = Σ Work Days ever since boilermaker/260

[4]Acceleration Capacity is measured on a negative scale.

Table 3

Linear regression coefficients (b1) of main effect of Cumulative Exposure Index (mg/m^3-workyears) on acceleration capacity (msec) and deceleration capacity(msec) for all 50 participants.

Parameter	[a]*Acceleration Capacity(AC)* Beta CEI (95% C.I.)	*Deceleration Capacity(DC)* Beta CEI (95% C. I.)
Models		
Model 1	0.43 (−0.03, 0.89)	−0.39 (−0.78, 0.01)
Model 2	*1.04 (0.16, 1.92)	−0.70 (−1.47, 0.06)
Model 3	*1.02 (0.12, 1.91)	−0.69 (−1.47, 0.09)
Model 4	*1.03 (0.10, 1.96)	−0.67 (−1.49, 0.14)

[a] Acceleration Capacity is measured on a negative scale, therefore positive estimates connote a decrease in AC on a negative scale.

* indicate significant associations ($p<0.05$)

Model 1(crude), Model 2(age-adjusted), Model 3 (adjusted for age and time of day), Model 4 (adjusted for age, time of day and last weld day)

CEI=Cumulative Exposure Index (mg/m^3-workyears)
Age=The residual of age on CEI was used to represent the heart age (variation in age not explained by the exposure) (years)
TOD=Time of Day ECG was taken (AM vs PM)
LWD=Last Weld Day (acute effects)

Table 4

Linear regression coefficients (b1) of main effect of Cumulative Exposure Index (mg/m^3-workyears) on acceleration capacity (msec) and deceleration capacity(msec) for 40 participants without heart disease.

Parameter	[a]*Acceleration Capacity(AC)* Beta CEI (95% C.I.)	*Deceleration Capacity(DC)* Beta CEI (95% C. I.)
Models		
Model 1	0.48 (−0.02, 0.97)	*−0.44 (−0.86, −0.01)
Model 2	*1.00 (0.11, 1.88)	−0.70 (−1.47, 0.09)
Model 3	*0.94 (0.03, 1.85)	−0.66 (−1.46, 0.14)
Model 4	0.93 (−0.02, 1.88)	−0.63 (−1.47, 0.21)

[a] Acceleration Capacity is measured on a negative scale, therefore positive estimates connote a decrease in AC on a negative scale.

* indicate significant associations ($p<0.05$)

Model 1(crude), Model 2(age-adjusted), Model 3 (adjusted for age and time of day), Model 4 (adjusted for age, time of day and last weld day)

CEI=Cumulative Exposure Index (mg/m^3-workyears)
Age=The residual of age on CEI was used to represent the heart age (variation in age not explained by the exposure) (years)
TOD=Time of Day ECG was taken (AM vs PM)
LWD=Last Weld Day (acute effects)