Night heart rate variability and particulate exposures among boilermaker construction workers

Citation

Published Version
doi:10.1289/ehp.10019

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Accessibility
BACKGROUND: Although studies have documented the association between heart rate variability (HRV) and ambient particulate exposures, the association between HRV, especially at night, and metal-rich, occupational particulate exposures remains unclear.

OBJECTIVE: Our goal in this study was to investigate the association between long-duration HRV, including nighttime HRV, and occupational PM$_{2.5}$ exposures.

METHODS: We used 24-hr ambulatory electrocardiograms (ECGs) to monitor 36 male boilermaker welders (mean age of 41 years) over a workday and nonworkday. ECGs were analyzed for HRV in the time domain; rMSSD (square root of the mean squared differences of successive intervals), SDNN (SD of normal-to-normal intervals over entire recording), and SDNN$_i$ (SDNN for all 5-min segments) were summarized over 24-hr, day (0730–2130 hours), and night (0000–0700 hours) periods. PM$_{2.5}$ (particulate matter with an aerodynamic diameter ≤ 2.5 µm) exposures were monitored over the workday, and 8-hr time-weighted average concentrations were calculated. We used linear regression to assess the associations between HRV and workday particulate exposures. Matched measurements from a nonworkday were used to control for individual cardiac risk factors.

RESULTS: Mean (± SD) PM$_{2.5}$ exposure was 0.73 ± 0.50 mg/m$^3$ and ranged from 0.04 to 2.70 mg/m$^3$. We observed a consistent inverse exposure–response relationship, with a decrease in all HRV measures with increased PM$_{2.5}$ exposure. However, the decrease was most pronounced at night, where a 1-mg/m$^3$ increase in PM$_{2.5}$ was associated with a change of –8.32 [95% confidence interval (CI), –16.29 to –0.35] msec nighttime rMSSD, –14.77 (95% CI, –31.52 to 1.97) msec nighttime SDNN, and –8.37 (95% CI, –17.93 to 1.20) msec nighttime SDNN$_i$, after adjusting for nonworking nighttime HRV, age, and smoking.

CONCLUSION: Metal-rich particulate exposures were associated with decreased long-duration HRV, especially at night. Further research is needed to elucidate which particulate metal constituent is responsible for decreased HRV.

responses to particulates. Workers were recruited 64 male boilermakers from a local approved the study protocol, and informed Board at the Harvard School of Public Health Subject recruitment.

Methods

time with low within-person day-to-day varia-

HRV on a day when particulate exposures are low compared with workday levels. We chose long-duration HRV measures, in part, because they have been shown to be less influenced by daily activities including physical activity (Task Force of the European Society of Cardiology and the North American Society of Pacing and Electrophysiology 1996) and more stable over time with low within-person day-to-day varia-

Kleiger et al. 1991). A priori, we hypothe-
sized that HRV at night would better capture the time course of the biological response, consistent with previous evidence of a lag (Pope et al. 1999). Night, a period dominated by sleep, is often free from physical activity, which is a strong predictor of HRV and a potential confounder. Studies suggest that sleep may be a condition in which HRV best identifies autonomic derangements because sleep is a condition in which cardiac autonomic activity can be studied in the absence of factors including physical activity and higher cortical factors (Vanoli et al. 1995). There is also clinical significance in understanding changes in HRV at night and during sleep. Despite the fact that sleep is a time when an individual is protected from known precipitating factors for cardiac events, 12–15% of all cardiac events and almost 36,000 deaths annually occur during sleep, resulting in a substantial health problem and raising the question of mechanism (Muller et al. 1997). We hypothesized that workday particulate exposures would be associated with declines in 24-hr, day and night HRV and that night would best capture this association among the boilermaker cohort.

Methods

Subject recruitment. The Institutional Review Board at the Harvard School of Public Health approved the study protocol, and informed consent was obtained from each adult prior to participation. From 1999 to 2006, we recruited 64 male boilermakers from a local union into a panel study to assess cardiac responses to particulates. Workers were recruited while training at an apprentice weld-

ing school and while working on the repair and maintenance of an oil-fired boiler. Recruitment at the welding school occurred in the winter and summer months, when the majority of workers were not actively working. We solicited a random subset of 36 partici-

pants for extensive ECG monitoring over two 24-hr periods on both a workday and a non-

workday. Workers participated in the study on multiple occasions over the 7-year sampling period; 26 (72%) were monitored on one occasion and 10 (28%) on multiple occasions. Workday and nonworkday monitoring occurred at each occasion.

Data collection. Workday monitoring occurred at a union welding school and at the overhaul of an oil-fired boiler within a power plant. The welding school consisted of a large, temperature-controlled room outfitted with 10 workstations with local exhaust ventilation where the boilermakers received instruction and practiced welding, cutting, and grinding techniques. The boiler overhaul entailed removing and replacing the boiler’s interior wall panels and water-circulating tubing. At both locations, boilermakers primarily performed shielded metal arc welding (stiek) and gas metal arc welding, most commonly using base metals of mild steel (manganese alloys) and stainless steel (manganese, chromium, and nickel alloys) with electrodes composed mainly of iron with variable amounts of manganese (1–5%). Plasma arc or acetylene torch cutting and grinding also occurred at both work sites.

Participants were also monitored over a 24-hr period on a nonworkday when they were not welding, gridding, or cutting. Although nonworkday monitoring occurred within 6 months of the workday monitoring, 80% of the observations occurred within the same week as the workday monitoring.

We used a self-administered questionnaire to collect information on medical history and medication use for each participation year. Participants were classified as cardiac compromised if they reported any of the following: heart trouble, congestive heart failure, angina, arrhythmia, myocardial infarction, heart/chest operation, or otherwise nonclassified heart problems. Hypertension was classified by reporting a doctor diagnosis or the use of hypertensive medications including ACE (angiotensin-converting enzyme) inhibitors or beta-blockers. We also collected demographic and lifestyle information, including smoking and occupational history.

Particulate exposure assessment. We continuously monitored personal, cross-shift PM$_{2.5}$ exposures using the light-scattering technology of a DustTrak Aerosol Monitor (TSI Inc., St. Paul, MN) fitted with a PM$_{2.5}$ inlet impactor. The monitor was placed in a padded pouch, with the inlet tubing secured to the participant’s shoulder in the breathing zone area. The DustTrak received yearly factory calibration and daily zero balance and flow checks. The monitor took PM$_{2.5}$ concentration readings every 10 sec and recorded 1-min averages. Participants were asked to keep a work log to record information on shift length and respirator use. We calculated 8-hr time-weighted average (TWA$_{8,hr}$) PM$_{2.5}$ exposures from the 1-min PM$_{2.5}$ concentrations over the reported shift length. A previous study of welding fume exposures (Kim et al. 2004) validated the use of the DustTrak to capture welding fume exposures.

Continuous Holter monitoring and tape processing. Participants were fitted with a standard 5-lead ECG Holter monitor. To facilitate good lead contacts, the participant’s skin was shaved (if necessary), cleansed with an alcohol wipe, and slightly abraded. At the workplace, study staff periodically checked leads. Each 24-hr tape was sent to Raytel Cardiac Services (Haddonfield, NJ) for processing and analysis using a StrataScan 563 (DelMar Avionics, Irvine, CA). Only beats with an RR interval between 0.6 and 1.5 sec and an RR ratio of 0.8–1.2 were included in the analysis. Trained technicians, blinded to work and nonwork periods and exposures, used standard criteria to accept or reject all normal or abnormal find-
ings. Tapes were analyzed in the time domain, and indexes including the square root of the mean of the sum of the squared differences between adjacent NN intervals (rMSSD), the SD of all NN intervals over the entire period (SDNN), and the mean of the SDNN intervals for all 5-min segments (SDNN$_{5}$) were calculated over the entire 24-hr recording, the 14-hr day period (0730–2130 hours), and the 7-hr night period (0000 to 0700 hours).

Data analysis. To account for correlated outcomes among subjects who participated on multiple occasions, we used mixed-effects regression models with random intercepts and unstructured covariance to investigate the association between each of the HRV outcomes and either dichotomous or continuous PM$_{2.5}$ exposure. For dichotomous exposure, the outcomes of interest were work and nonwork HRV measures, and the predictors included a dichotomous exposure variable (work/nonwork) and a random intercept for individuals. The p-value of the dichotomous exposure regression coefficient was used to assess statistically significant differences between work and nonwork HRV measures. Next, continuous workday PM$_{2.5}$ exposures were investigated by modeling each workday HRV outcome while adjusting for nonworkday HRV measured at the same time scale and during the same period as the workday outcome. Because baseline, nonwork HRV was included in each model, personal factors (including cardiac risk factors) that do not.
change between work and nonwork measurements were unlikely to introduce bias due to confounding. However, because age and smoking are strong predictors of HRV, they were included in each model as a continuous and dichotomous covariate, respectively. Each outcome measure (rMSSD, SDNN, and SDNNi) summarized over the three time periods (24-hr, 14-hr day, and 7-hr night) gave a total of nine exposure–response models.

We further investigated model results by restricting the cohort. Because of small sample size and unbalanced design, a formal investigation of effect modification was not feasible. However, we compared the effect estimates from the full cohort to a restricted sample of participants who did not report cardiac compromise or hypertension at the workday measurement to evaluate whether the exposure–response relationship varied by health conditions.

We assessed distributional assumptions by examining residuals plotted against predictors. We used Cook’s distance to identify potentially influential points using a criteria of Cook’s distance > 0.5. Statistical significance was confirmed model assumptions and showed no evidence of influential points for all models.

### Results

The study population consisted of 36 males, with a mean age of 41 years; 29 (81%) of the participants were white (Table 1). Of the 36 participants, 10 contributed more than one work/nonwork pair of observations. Eight were monitored twice, 1 was monitored on three occasions, and 1 was monitored on four occasions. There were 12 smokers at study enrollment, and 2 quit smoking by subsequent participation. Five participants reported cardiac conditions, including two myocardial infarctions, one stent, one murmur, and one arrhythmia. Three of these individuals participated only once, and two participated twice, reporting no cardiac condition on first participation.

Six participants reported either doctor diagnosis of hypertension or were using medication for hypertension. Of these six individuals, three participated once, one twice (hypertension reported both occasions), one three times (hypertension reported two occasions), and one four times (hypertension reported at one occasion). Two participants reported the use of statins, and three used ACE inhibitors; no one reported using beta-blockers, calcium channel blockers, or diuretics. TWAs, PM$_{2.5}$ exposures ranged from 0.04 to 2.70 mg/m$^3$ with a mean (± SD) of 0.73 ± 0.50 mg/m$^3$ (Table 1). Workers reported respirator use on 13 (26%) monitoring occasions. Forty-five (92%) of the 49 monitored workdays occurred at the apprentice school.

Ambulatory ECG data were collected for a total of 98 person-days and are summarized in Table 2. The mean levels of the 24-hr HRV measures were unremarkable and were comparable to previously reported levels of rMSSD (35 ± 11 msec), SDNN (146 ± 30 msec), and SDNNi (58 ± 18 msec) for men 40–49 years of age (Jensen-Urstad et al. 1997). We found a wide range of HRV within each period over both workdays and nonworkdays because of between-person heterogeneity. However, the mean work rMSSD and SDNN, measures were consistently lower than the mean nonwork HRV measures, and statistically significant differences were found between work and nonwork during all periods for rMSSD and SDNN, measures, with the exception of night rMSSD. SDNN measures across the three time periods did not follow this pattern, and in fact, there was no statistically significant difference between work and nonwork SDNN across all time periods.

The mixed model regression analyses revealed that elevated PM$_{2.5}$ levels were consistently associated with declines in rMSSD, SDNN, and SDNNi over the 24-hr, day, and night periods after adjusting for HRV during matched time-periods of nonworkdays, age, and smoking status (Table 3). When the 24-hr period was broken into day and night, the most pronounced decline in HRV was during the night, where statistically significant associations were observed for night rMSSD.

We further explored the data by performing a series of restricted analyses for each model after adjusting for nonwork nighttime HRV, age, and smoking status (Table 4). In the cohort restricted to participants reporting no cardiac compromise (model II) or among those who did not report or were not on medication for hypertension (model III), the declines in HRV at night subsequent to workday particulate exposures were comparable to results from the unrestricted cohort (model I). Examination of residual plots and Cook’s distance confirmed model assumptions and showed no evidence of influential points for all models.

### Discussion

Among a cohort of boilermakers, we observed statistically significant lower 24-hr, day, and night rMSSD and SDNN, on workdays compared with nonworkdays. Furthermore, we found an inverse dose–response relationship between workday PM$_{2.5}$ exposure and 24-hr, day and night HRV. When the 24-hr period was separated into the day and night periods, the most pronounced declines in HRV were at night. This association was statistically significant for night rMSSD; every 1-mg/m$^3$ increase in workday PM$_{2.5}$ TWAs was associated with a change of −8.32 [95% confidence interval (CI), −16.29 to −0.35] msec nighttime rMSSD after adjusting for nonwork nighttime rMSSD, age, and smoking status. Few studies have documented the association between particulate exposures and long-duration HRV (Brauer et al. 2001; Magari et al. 2002; Pope et al. 1999, 2004; Vallejo et al. 2006), and to date, the majority have investigated the effect of ambient particulate air pollution exposures. The present study is the first to demonstrate declines in HRV over the entire day and night subsequent to occupational particulate exposures.

### Table 1. Population and exposure characteristics for male boilermaker construction workers (n = 36).

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Mean ± SD or No. (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age (years)$^a$</td>
<td>41 ± 11</td>
</tr>
<tr>
<td>Range</td>
<td>22–63</td>
</tr>
<tr>
<td>Race</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>29 (81)</td>
</tr>
<tr>
<td>Black</td>
<td>3 (8)</td>
</tr>
<tr>
<td>Hispanic</td>
<td>3 (8)</td>
</tr>
<tr>
<td>Asian</td>
<td>1 (3)</td>
</tr>
<tr>
<td>Current smoker$^b$</td>
<td>12 (33)</td>
</tr>
<tr>
<td>Medical information$^b$</td>
<td>6 (17)</td>
</tr>
<tr>
<td>Hypertensive</td>
<td>5 (14)</td>
</tr>
<tr>
<td>Cardiac compromised</td>
<td>5.7 ± 0.50</td>
</tr>
<tr>
<td>PM$_{2.5}$ (mg/m$^3$)$^c$</td>
<td>0.73 ± 0.50</td>
</tr>
<tr>
<td>Range</td>
<td>0.04–2.70</td>
</tr>
<tr>
<td>Reported respirator use$^c$</td>
<td>13 (26)</td>
</tr>
</tbody>
</table>

$^a$At study entry. $^b$Ever reported condition at one or more study occasions. $^c$For 49 measurement occasions.

### Table 2. Summary and comparison of long-duration HRV by nonwork and work periods.

<table>
<thead>
<tr>
<th>HRV measure</th>
<th>Nonwork$^a$</th>
<th>Work$^a$</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean ± SD</td>
<td>Range</td>
</tr>
<tr>
<td>rMSSD</td>
<td>29.6 ± 13.9</td>
<td>7.6–71.1</td>
</tr>
<tr>
<td>SDNN</td>
<td>130.9 ± 36.1</td>
<td>32.9–213.9</td>
</tr>
<tr>
<td>SDNNi</td>
<td>56.3 ± 19.1</td>
<td>10.5–94.8</td>
</tr>
<tr>
<td>Day$^c$</td>
<td>rMSSD</td>
<td>25.3 ± 10.8</td>
</tr>
<tr>
<td></td>
<td>SDNN</td>
<td>98.8 ± 30.1</td>
</tr>
<tr>
<td></td>
<td>SDNNi</td>
<td>52.4 ± 18.1</td>
</tr>
<tr>
<td>Night$^c$</td>
<td>rMSSD</td>
<td>37.7 ± 19.7</td>
</tr>
<tr>
<td></td>
<td>SDNN</td>
<td>101.3 ± 31.9</td>
</tr>
<tr>
<td></td>
<td>SDNNi</td>
<td>85.0 ± 22.5</td>
</tr>
</tbody>
</table>

$^a$Thirty-six individuals and 49 measurements. $^b$Statistical comparison between work and nonwork measures using mixed regression models to account for multiple participation times. $^c$0730–2130 hours. $^d$0000–0700 hours.
Because of differences in exposure characterization and HRV measures, direct comparison of our study results with previous studies is not feasible. However, the direction of associations in the present study is consistent with the inverse exposure–response relationship found in studies of particulate air pollution. Among a panel of seven compromised individuals, elevated PM10 (particulate matter with an aerodynamic diameter ≤10 μm) levels were associated with decreased 24-hr SDNN and increased 24-hr rMSSD (Pope et al. 1999). A subsequent study among 88 elderly Utah residents confirmed the inverse association between 24-hr SDNN and ambient particulate exposure, but also reported a decline in 24-hr rMSSD with PM2.5 exposures (Pope et al. 2004). A similar association was observed with 24-hr SDNN and rMSSD in a population of elderly individuals with chronic obstructive pulmonary disease (COPD) (Brauer et al. 2001). In addition to elderly and compromised individuals, an inverse association (between 2-hr HRV and 2-hr PM2.5 exposure) was observed among a young cohort of nonsmoking, healthy adults (Vallejo et al. 2006). These results are consistent with the declines in 24-hr SDNN, SDNN, and rMSSD that we observed in the boilermaker cohort following PM2.5 exposure.

Likewise, the pattern of day and night long-duration HRV changes among this cohort is confirmed by air pollution studies. Pope et al. (1999) further analyzed the 24-hr rMSSD with PM2.5 exposures (Pope et al. 2004). A similar association was observed with 24-hr SDNN and rMSSD in a population of elderly individuals with chronic obstructive pulmonary disease (COPD) (Brauer et al. 2001). In addition to elderly and compromised individuals, an inverse association (between 2-hr HRV and 2-hr PM2.5 exposure) was observed among a young cohort of nonsmoking, healthy adults (Vallejo et al. 2006). These results are consistent with the declines in 24-hr SDNN, SDNN, and rMSSD that we observed in the boilermaker cohort following PM2.5 exposure.

Table 3. Effect estimates for change in long-duration HRV measures per 1-mg/m3 increase in workday PM2.5 exposures.

<table>
<thead>
<tr>
<th>HRV measure</th>
<th>β* (95% CI)</th>
</tr>
</thead>
<tbody>
<tr>
<td>24-hr</td>
<td></td>
</tr>
<tr>
<td>rMSSD</td>
<td>–3.29 (–7.66 to 1.09)</td>
</tr>
<tr>
<td>SDNN</td>
<td>–11.66 (–28.84 to 5.51)</td>
</tr>
<tr>
<td>SDNNi</td>
<td>–4.58 (–10.16 to 0.99)</td>
</tr>
<tr>
<td>Day</td>
<td></td>
</tr>
<tr>
<td>rMSSD</td>
<td>–0.59 (–4.18 to 3.01)</td>
</tr>
<tr>
<td>SDNN</td>
<td>–3.39 (–22.34 to 5.57)</td>
</tr>
<tr>
<td>SDNNi</td>
<td>–4.06 (–9.22 to 1.10)</td>
</tr>
<tr>
<td>Night</td>
<td></td>
</tr>
<tr>
<td>rMSSD</td>
<td>–8.32 (–16.29 to –0.35)</td>
</tr>
<tr>
<td>SDNN</td>
<td>–14.77 (–31.52 to 1.97)</td>
</tr>
<tr>
<td>SDNNi</td>
<td>–8.37 (–17.92 to 1.20)</td>
</tr>
</tbody>
</table>

*Adjusted for nonwork HRV measure over the same time period, age, and smoking status. p < 0.10. **p < 0.05.

Table 4. PM2.5 regression coefficients [95% confidence interval (CI)] for models of nighttime rMSSD, SDNN, or SDNNi, after restricting the data set.

<table>
<thead>
<tr>
<th>HRV measure (msec)</th>
<th>Model Ia,b</th>
<th>Model IIc</th>
<th>Model IIIa,d</th>
</tr>
</thead>
<tbody>
<tr>
<td>Night rMSSD</td>
<td>–8.32 (–16.29 to –0.35)**</td>
<td>–8.19 (–17.31 to 0.93)**</td>
<td>–6.71 (–16.17 to 2.75)</td>
</tr>
<tr>
<td>Night SDNN</td>
<td>–14.77 (–31.52 to 1.97)**</td>
<td>–13.87 (–32.95 to 4.61)</td>
<td>–18.72 (–39.89 to 2.22)**</td>
</tr>
<tr>
<td>Night SDNNi</td>
<td>–8.37 (–17.92 to 1.20)**</td>
<td>–8.78 (–19.76 to 2.21)</td>
<td>–9.29 (–20.84 to 2.26)**</td>
</tr>
</tbody>
</table>

*aAdjusted for nonwork nighttime HRV, age, and smoking status. Includes 36 individuals over 49 measurement occasions.

**Includes 33 individuals over 44 measurement occasions, including 2 individuals who reported no cardiac compromise.

*cIncludes 32 individuals over 44 measurement occasions, including 2 individuals who reported no hypertension.

dIncludes 32 individuals over 44 measurement occasions, including 2 individuals who reported no hypertension on initial participation, and excluding subsequent participation reporting cardiac compromise.

*p < 0.10. **p < 0.05.
concentrations were 1.14 mg/m³, as compared to
workday PM2.5 exposure; however, the differ-
ence between work and nonwork periods, such as caffeine or
alcohol consumption or noise levels. For the
comparison of dichotomous exposure (work
and nonwork), we were unable to account for
these factors. However, for the exposure–
response analysis, we hypothesize that neither
caffeine and alcohol consumption or noise are
related to the observed changes in HRV.

Conclusions

This panel study demonstrated decreases in
long-duration HRV subsequent to metal-rich
workday particulate exposure in a relatively
healthy cohort of boilermaker construction
workforce. Furthermore, we found that the
differences in HRV were most pronounced at
night; this is consistent with results seen in
studies of air pollution exposures (Pope et al.
1999). More research is needed to elucidate
which of the particulate metal constituents is
responsible for decreases in nighttime HRV.

Correction

The authors note that Yeatts et al. have recently published an article on the associa-
tion between particulate exposures and long-
duration HRV [Yeatts K, Svensden E,
Coarse particulate matter (PM2.5–10) affects
heart rate variability, blood lipids, and circu-
lating cosinophils in adults with asthma.
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