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# Associations between $PM_{10}$ with Sleep and Sleep-Disordered Breathing in Adults from Seven U.S. Urban Areas

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At a Glance Commentary.

Sleep-disordered breathing (SDB) and air pollution have separately been linked to increased cardiovascular diseases and mortality, but the influence of pollution on SDB is poorly understood. To our knowledge, the effects of pollutants on sleep architecture and SDB have not been previously studied. Our analyses add to the field evidence for two novel findings: In the Sleep Heart Health Study (SHHS), 1) increases in the respiratory disturbance index are associated with increases in short term temperature; 2) increases in SDB, as measured by both the respiratory disturbance index and the overnight hypoxemic index, and decreases in the percent of sleep efficiency, are associated with increasing levels of daily particulate matter in summer months. Our findings suggest that elevation in air pollution may increase the risk of poor sleep and may be partially be responsible for sleep health disparities. Follow-up work should evaluate whether reduction in air pollution exposure decreases severity of SDB and nocturnal hypoxemia and consequently improves cardiac risk.

This article has an online data supplement, which is accessible from this issue's table of content online at www.atsjournals.org"

Keywords: particulate matter, sleep disorder breathing, sleep architecture

**Abstract**

Rationale: Sleep-disordered breathing (SDB), the recurrent episodic disruption of normal breathing during sleep, affects as much as 17 percent of US adults, and may be more prevalent in poor urban environments. SDB and air pollution have been linked to increased cardiovascular diseases and mortality, but the association between pollution and SDB is poorly understood.

Objectives: We used data from the Sleep Heart Health Study (SHHS), a US multi-center cohort study assessing cardiovascular and other consequences of SDB, to examine whether $PM_{10}$ was associated with SDB among persons 39 years and older.

Methods: Using baseline data from SHHS urban sites, outcomes included: the respiratory disturbance index (RDI); percent of sleep time < 90% $O_2$ saturation; and sleep efficiency, measured using overnight in-home polysomnography. We applied a fixed effect model containing a city effect, controlling for potential predictors. In all models we included both the 365 day moving averages of $PM_{10}$ and temperature (long term effects) and the differences between the daily measures of these two predictors and their 365 day average (short term effects).

Main Results: In summer, increases in RDI or percent of sleep time at < 90% $O_2$ sat, and decreases in sleep efficiency, were all associated with increases in short term variation in $PM_{10}$. Over all seasons, we found that increased RDI was associated with an 11.5% (95% CI: 1.96, 22.01) increase per inter-quartile range (IQR) increase (25.5 degrees) in temperature.

Conclusions: Reduction in air pollution exposure may decrease severity of SDB and nocturnal hypoxemia and may improve cardiac risk.

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**Introduction**

Sleep-disordered breathing (SDB), the recurrent episodic disruption of normal breathing during sleep, affects as much as 17 percent of US adults (1), and may be more prevalent in poor urban environments (2). SDB and air pollution have each been linked to increased risk of autonomic dysfunction (3), pulmonary and systemic inflammation (4), elevated blood pressure (5), paroxysmal atrial fibrillation (6), ventricular arrhythmias (6), myocardial infarction, and cardiovascular mortality (7), but the influence of pollution on SDB is poorly understood. Many of the adverse cardiac effects of SDB are thought to be due in part to clinically significant apnea/hypopnea- induced hypoxemia and respiratory acidosis (6). Elevated particle pollution levels have been linked to far more modest, though statistically significant, reductions in oxygen saturation, (8) interpreted as resulting from ventilation:perfusion mismatch, but not from apnea/SDB. It is biologically plausible that elevations in ambient pollution might also increase the risk of more clinically relevant SDB and SDB-associated oxyghemoglobin desaturation, through effects on upper or lower airway inflammation, autonomic dysfunction, or oxidative stress (4). Our hypothesis was that elevation in ambient pollution would be associated with an increased risk of SDB and nocturnal hypoxemia, as well as with reduced sleep quality.

Any study of air pollution effects needs to take into account temperature, which may have independent effects on respiratory outcomes. Recent studies of European heat waves have demonstrated associations of high temperature with increased respiratory

mortality. Our secondary hypothesis was that there exists independent effects of elevated temperature on SDB and sleep efficiency (9).

To examine the associations of pollution with sleep quality and indices of SDB, we used data from the urban sites of the Sleep Heart Health Study (SHHS; http://www.jhucct.com/shhs/), a US multi-center cohort study designed to assess the cardiovascular and other consequences of SDB.

**Data and Methods**

The SHHS is a multicenter longitudinal study of 6,441 participants recruited from existing cohorts, aged >39 yr, designed to evaluate the cardiovascular consequences of SDB. The study design of SHHS, and detailed descriptions of its member cohorts, protocols, and quality-control procedures, have been published (6, 10, 11). The baseline examination for the SHHS was conducted between 1995 and 1998. Of the nine study locations, seven were urban locations for which regional U.S. Environmental Protection Agency (EPA) air pollution monitoring data were available: Phoenix, AZ; Tucson, AZ; Sacramento, CA; Framingham, MA; Minneapolis, MN; New York City, NY; Pittsburgh, PA.

Polysomnography was performed over a single night with use of a portable, unattended monitor set up in the home (Compumedics P Series system, Abbotsville, Victoria, Australia). Multiple channels were recorded, including electroencephalogram ($C_3/A_2$ and $C_4/A_1$), electroocculogram (bilateral), electrocardiogram, chin electromyogram, abdominal and thoracic excursions (by impedance plethysmography), oxyhemoglobin saturation (finger pulse oximetry; Nonin, Minneapolis, MN), airflow

(oral-nasal thermistor; Protec, Woodsville, Washington), body position (by mercury gauge), and ambient light. These leads were connected to a small monitor worn in a vest pocket.

A respiratory event was defined as a decrease in airflow or chest wall movement to an amplitude of less than 75 percent (apnea) or 30 percent (hypopnea) of the baseline breathing signal. A qualifying event was defined as one that lasted at least 10 seconds in association with an oxyhemoglobin desaturation of greater than or equal to 3 percent. The Respiratory Disturbance Index (RDI) was computed as the ratio of the count of all apneas and hypopneas to the total sleep time expressed in hours. Percent of sleep time with oxyhemoglobin saturation below 90% (hypoxemia index) was computed as the time with oxygen saturation under 90% to the total sleep duration. Sleep Efficiency was calculated as the proportion of time scored as sleep during the sleep period; i.e., between "lights off" and "lights on". During the home visit, a study technician interviewed the participant according to a standardized questionnaire, collecting information on medical history and health-related characteristics. Personal characteristics such as age, gender, and ethnicity were obtained from demographic data reported by the parent cohorts. These included educational level expressed as total number of years of school, (High School diploma = 12 years; 4-year college degree = 16 years; etc.), and smoking history. Alcohol consumption and consumption of coffee, tea and sodas data were derived from a sleep questionnaire completed the day after the sleep study and referred to the 24 hours prior to the study.

*Environmental Data*

We obtained data on levels of daily particulate air matter with aerodynamic diameter less than 10 μg ($PM_{10}$) from the U.S. EPA's Air Quality System Technology Transfer Network (U.S. EPA Technology Transfer Network, 2005). Daily meteorological data were available from the U.S. Surface Airways and Airways Solar Radiation sources. The 24-hour mean for temperature was calculated as degrees Fahrenheit.

Pittsburgh PA, Phoenix AZ, and Minneapolis MN had almost complete $PM_{10}$ data. In the other urban areas, $PM_{10}$ was available only for 1-3 out of every six days, reducing the number of participant observations that could be matched to pollution exposure measurements. To estimate regional pollution exposure for Framingham MA participants, we used data from the Boston U.S. EPA data set. Data from the Harvard central site in Boston MA were used to fill in occasional missing values in the Boston U.S. EPA data set.

Many of the urban areas had more than one monitoring location, requiring a method to average over multiple locations. We computed local daily mean $PM_{10}$ concentrations using an algorithm that accounts for the different monitor-specific means and variances. This process has been reported previously (12), (13), and used extensively in previous publications (13-17).

*Statistical methods*

We examined the association between $PM_{10}$ concentrations and the sleep outcomes in this cross-sectional study, using linear fixed effects regression models containing an indicator variable for city effect and controlling for seasonality (winter,

spring, summer, fall, plus linear day of the year), daily mean temperature, and factors found previously (18) to be predictive of SDB in this cohort or which were a priori identified as potential predictors: age, body mass index, gender, education, an age by gender interaction, smoking status, daily number of glasses of coffee, tea, and soda, and number of glasses of wine and beer four hours before going to sleep. All the outcomes were log-transformed after adding 0.1.

To examine the pollution effect by season, in model adjusted for all the potential predictors we added an interaction term between $PM_{10}$ and season.

To evaluate long-term effects of air pollution, we computed the 365 days moving average of $PM_{10}$ as the average of the exposure lags including the same day (lag 0), lag 1, 2, and so on, up to the 364th day before the polysomnography study. In all models we included both the 365 day moving average of $PM_{10}$ and the difference between the daily $PM_{10}$ concentration and the 365 day average. Previously used to assess pollution effects at the individual as well as the community level (19), this method enabled us to simultaneously estimate both long and short term effects of $PM_{10}$. In our study, the coefficient of the long term pollution average primarily reflects differences in outcome due to differences across communities in long term averages of pollution. To a lesser extent, the coefficient also reflects differences in outcome due to differences between individuals in long term averages of pollution. This is because, rather than using one 365-day average per community, we used a 365-day moving average that was dependent on when the individual was studied.

Our approach to estimation of long term vs short term $PM_{10}$ effects was also used for estimation of temperature effects. In the same model containing long term and short

term $PM_{10}$, we included both the 365 day moving average of temperature, and the difference between the daily temperature and the 365 day average.

The results are expressed as percent increase in each outcome for an inter-quartile (IQR) increase in exposure. An IQR for exposure was computed as the average IQR across the cities.

The data were examined using SAS for data management (SAS software release 9.2. 2001, SAS Institute, Cary NC), and R 2.10.1 for the analysis (R Development Core Team, 2010. R: A language and environment for statistical computing. R Foundation for Statistical Computing, Vienna, Austria. ISBN 3-900051-07-0, URL http://www.R-project.org.).

**Results**

The number of participants, number of missing values of personal characteristics, and number of $PM_{10}$ measurements available varied by city. The numbers of participants in each study site that were included in the analysis were: Phoenix, AZ (n=173); Tucson, AZ (n=575); Sacramento, CA (n=111); Framingham, MA (n=642); Minneapolis, MN (n=1031); New York City, NY (n=118); Pittsburgh, PA (n=380).

The methods used to recruit the SHHS participants, and participation rates have been previously described (1). SHHS aimed to enroll 36% of the parent cohort participants, overall. Compared to this overall goal of 36% enrollment, the seven cities used in this analysis achieved the following participation rates: 15% in Phoenix, AZ; 53% in Tucson, AZ; 74% in Sacramento, CA; 28% in Framingham, MA; 39% in Minneapolis, MN; 28% in New York City, NY; and 71% in Pittsburgh, PA.

Figure 1 presents the box plot for the RDI for each city. There were no significant differences among the city-specific RDI distributions. Figure 2 presents the box plots for actual daily $PM_{10}$ in each city and season; the median $PM_{10}$ concentrations were highest in summer and lower in winter, but in some cities the spring median concentrations were lowest.

Table 1 presents the summary statistics for the variables of interest across the seven cities. While there was a wide range of body-mass index (BMI), on average the population tended to be overweight. Over half the sample were either former smokers or current smokers. Table 2 presents the distribution of air pollution and temperature by city, together with the city-specific IQRs.

Over all seasons, elevations in short term temperature exposures, but not $PM_{10}$, were associated with increases in RDI. An IQR increase (25.5 degrees) in temperature predicted an 11.5% (95% CI: 1.96, 22.01) increase in RDI (Table 3). Short term effects of temperature were not modified by season, whereas short term effects of $PM_{10}$ were (Figure 3).

In the summer, increases in the short-term exposure $PM_{10}$ levels were associated with increases in SDB, as measured by both the RDI and percentage of sleep time with a oxyhemoglobin desaturation percentage <90%, and also with decreases in sleep efficiency. In summer we found a significant season by $PM_{10}$ interaction; for an inter-quartile increase in short-term $PM_{10}$ levels, we found a 12.9% increase (95% CI: 2.77, 24.09) in the RDI, a 19.4% increase (95% CI: 3.67, 37.5) in the percent of sleep time at < 90% oxygen saturation, and a 1.20% decrease (95% CI: -2.40, -0.004) in sleep efficiency. No statistically significant associations were found during the other seasons. With the

exception of Tucson, this summer-specific association between RDI and short-term ambient pollution exposure was consistent across cities. The city-specific effect was most precise for Minneapolis, MN, the city with the largest number of observations (Supplement Figure 1).

**Discussion**

In this large, geographically diverse community sample studied with standardized polysomnography we find novel evidence for pollution and temperature effects on sleep disordered breathing. Increases in apnea or hypopnea, measured as the respiratory disturbance index, were associated with increases in short-term temperature over all seasons, and with increases in particle pollution levels in the summer months. In the summer months, pollution levels were associated with increased percent of sleep time at < 90% oxygen saturation. Previously we have demonstrated that SDB in this population is a risk factor for cardiovascular morbidity (7). These data extend the growing literature demonstrating the contribution of air pollution to adverse pulmonary and cardiovascular health (13, 16).

Particles may influence sleep through effects on the central nervous system, as well as the upper airways. Particles have been shown to translocate from the nose up the olfactory nerve into the brain, including the striatum frontal cortex, and cerebellum (21, 22). This in turn is associated with increased brain inflammatory responses (23, 24) and changes in neurotransmitter levels (25). In humans, diesel exhaust exposure has been shown to alter EEG responses, with patterns indicative of cortical stress (26). Compared to less polluted cities, in polluted areas of Mexico City, dogs had more prefrontal lesions, neuroinflammation, gliosis, and particle deposition. In these polluted areas, brain imaging

studies of children showed more prefrontal lesions; autopsy studies of accident victims showed upregulation of cyclooxygenase-2 and CD14 (27-29).

Prior research in rodents (30) has shown that experimental exposure to increased ozone concentrations alters levels of serotonergic neurotransmitters in brainstem areas implicated in sleep-wake control (31, 32).

To our knowledge, the effects of pollutants on sleep architecture in humans have not been previously studied. However, environmental tobacco smoke exposure, which is a mix of particulate and gaseous pollution (33), has been reported to be associated with symptoms of disrupted sleep and insomnia (30). There is a growing literature that implicates low sleep efficiency, short sleep duration, and insomnia with adverse health outcomes (4-6, 34), with evidence that poor sleep may disproportionately afflict poor urban populations (35, 36). Our findings suggest that one mechanism for poor sleep and sleep health disparities may relate to environmental pollution levels.

In addition to effects on sleep architecture, during the summer, elevation in ambient pollution levels is associated with an increased risk of SDB as measured by the RDI as well as sleep-related hypoxemia in this urban SHH study cohort. To our knowledge, this is the first study to demonstrate a link between air pollution exposure and SDB. We have done so in a cohort where the prospective association of SDB with all-cause mortality and cardiovascular mortality has recently been demonstrated (37). Pollution may increase SDB through influencing central ventilatory control centers. Pollutants may directly contribute to nasal or pharyngeal inflammatory responses that increase upper airway resistance and reduce airway patency. Fine and ultrafine particles may alter ventilation:perfusion relations, exacerbating the hypoxia of SDB(4, 8). In

patients with asthma and SDB, elevated air pollution has been demonstrated to worsen lower airway inflammation and airflow obstruction through allergic and non-allergic mechanisms (19); this may also contribute to the propensity for desaturation with sleep-associated reductions in ventilation. In patients with hay fever, upper airflow obstruction may worsen on an allergic basis when air pollution particles also contain allergen fragments (4, 8).

Sudden Infant Death Syndrome (SIDS), which may occur because of brainstem ventilatory or autonomic control problems, abnormalities in cardiac function, or upper airway collapse, has been linked to ambient pollution levels in some (38) but not all studies (39). Familial aggregation studies suggest that there is an overlap of the etiologic factors for SIDS and for SDB (40). Thus, the mechanisms that increase risk of SIDS in association with ambient pollutants may be similar to the mechanisms that may underlie risk of SDB. These factors may include pollutant-associated effects on central or peripheral neurotransmitters that influence sleep state stability (and thus also explain the sleep efficiency findings), upper airway patency, and/or ventilatory control.

Several studies have reported that temperature changes predict mortality. These findings, whose mechanisms are poorly understood, are not restricted to extreme weather conditions, but are observed across the range of temperatures (41, 42). The association we found between short term temperature and RDI could represent one possible mechanism. Alternatively, temperature could be confounded by ozone, as the two often covary.

Fifteen to twenty years ago, when the first of hundreds of studies demonstrating the adverse cardiac effects of air pollution were published, the plausibility of these

associations was challenged because of the uncertain mechanistic links between the respiratory inhalation of air pollutants and subsequent cardiac morbidity or mortality (43). Subsequently, demonstration of the autonomic (44, 45), inflammatory (46, 47), oxidative stress (48), and pro-coagulant (49, 50) effects of particle pollution has lent biologic plausibility to the epidemiologic observations. Nevertheless, up until this study, there has been relatively weak evidence for hypoxemia as a potential link between pulmonary exposure to air pollution and adverse cardiac outcomes. Small but significant reductions in oxygen saturation during waking hours were associated with elevation in particulate pollution in elders panel studies from Utah (51) and Boston, MA (8); pollution effects on oxygen saturation during sleep were not evaluated in those studies.

The methodology for ascertainment of SDB outcomes in this cohort has been validated (10, 11). We present cross-sectional analyses; longitudinal analyses would be helpful to validate our findings. However the major limitations of our study design relate, for the most part, to estimation of pollution exposure. Estimates of home-specific exposures could not be made, as geo-coded addresses were not available. The number of observations was limited by the location of EPA monitoring stations. Detailed information on air conditioning was not available, but summer pollution effects were lower in cities known to be hot, with a large amount of air conditioner use (e.g. Tucson, AZ). In the cities we studied, oxidant gases such as ozone and other secondary emissions are higher in the summer, and it is likely that mixtures of pollution contributed to the summer PM effects on SDB. More detailed study of effects of season-specific mixtures on SDB is warranted. Another limitation of the study is due to possible measurement error. Our exposure $PM_{10}$ is based on an average concentration among several monitors,

which serves as a surrogate for location-specific exposure. However, the error generated by this approximation is likely not a major issue, as the within-community correlation among monitors is quite high, and in our data varies between 0.62 and 0.82. Classical measurement error could bias the coefficient of $PM_{10}$ in our analysis towards the null. On the other hand, Zeger and coworkers (52) showed that exposure measurement error for short term air pollution exposure is mainly Berkson. Therefore for our short term exposure, the error is likely to be predominantly Berkson, which will result in a loss of power but not bias our estimate of the association between $PM_{10}$ and sleep. Temperature levels vary geographically but their fluctuations are highly correlated, and hence measurement error is unlikely to be an issue. Other papers presented the magnitude of the potential bias due to measurement error (53, 54). Our study is also limited by the absence of data on ozone for Minneapolis, where we had the most outcome data. We were able to examine $NO_2$, and CO associations with sleep outcomes. The results were generally in the same direction as the $PM_{10}$ results, but were weaker (results not shown).

The prevalence of SDB among adults is high in the United States (approximately 17%) (1), and it may increase as the prevalence of obesity rises. While therapies are available for this disorder, the majority of adults with SDB are not being treated, and many people are resistant to therapy. Along with reduction in obesity, these new data suggest that reduction in air pollution exposure might decrease severity of SDB and nocturnal hypoxemia and may improve cardiac risk.

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**Figure legends**

Figure 1. City-specific distribution of Respiratory Disturbance Indices (RDI). RDI was computed as the ratio of the count of all apneas and hypopneas to the total sleep time expressed in hours.

Figure 2. City and season specific distribution of 24-hour (daily) $PM_{10}$.

Figure 3: Season specific percent change and 95% Confidence Intervals in sleep disordered breathing outcomes, for an interquartile increase in short term $PM_{10}$, adjusting for seasonality, daily mean temperature, age, body mass index, gender, education, an age by gender interaction, smoking status, daily number of glasses of coffee, tea, and soda, and number of glasses of wine and beer four hours before going to sleep and long term $PM_{10}$ averages.

**Table 1: Population characteristics**

| | N | 5% | 25% | Mean | 75% | 95% | % |
|---|---|---|---|---|---|---|---|
| Respiratory Disturbance Index (RDI) at 3% desaturation | 3030 | 0.6 | 3.6 | 13.5 | 18.0 | 43.5 | |
| Percent of sleep time < 90% desaturation | 3030 | 0 | 0 | 3.6 | 1.9 | 19.2 | |
| Sleep Efficiency (%) | 3030 | 62.6 | 77.6 | 82.9 | 90.7 | 95.3 | |
| Age (yrs) | 3030 | 45 | 55 | 63 | 70 | 80 | |
| Body Mass Index (kg/m$^2$) | 3030 | 21.2 | 24.6 | 28.0 | 30.7 | 37.4 | |
| Education (yrs) | 3030 | 10 | 12 | 15 | 17 | 20 | |
| Number of glasses of coffee, tea, and soda* | 3030 | 0 | 1 | 3 | 4 | 8 | |
| Number of glasses of wine and beer 4 hours before going to sleep* | 3030 | 0 | 0 | 0.2 | 0 | 2 | |
| Smoking: never | 1310 | | | | | | 43.2 |
| current | 326 | | | | | | 10.8 |
| former | 1394 | | | | | | 46.0 |
| Gender: Female | 1595 | | | | | | 52.6 |
| Male | 1435 | | | | | | 47.4 |

*Count in the day prior to testing

**Table 2: Distribution of $PM_{10}$ and temperature by city**

| City | Exposure * | N | 5% | 25% | MEAN | 75% | 95% | IQR |
|---|---|---|---|---|---|---|---|---|
| Phoenix, AZ | $PM_{10}$ long term | 173 | 41.9 | 42.5 | 42.9 | 43.3 | 44.0 | 0.9 |
| | $PM_{10}$ short term | | -19.4 | -7.7 | 0.7 | 9.7 | 26.7 | 17.4 |
| | Temperature long term | | 74.6 | 74.7 | 75.3 | 75.7 | 76.0 | 1.0 |
| | Temperature short term | | -27.5 | -14.5 | -4.5 | 6.6 | 18.0 | 21.0 |
| | Daily temperature | | 48.0 | 61.0 | 70.8 | 82.0 | 94.0 | 20.6 |
| Tucson, AZ | $PM_{10}$ long term | 575 | 25.5 | 26.1 | 27.2 | 28.2 | 29.2 | 2.1 |
| | $PM_{10}$ short term | | -14.6 | -7.1 | 0.3 | 6.1 | 20.8 | 13.2 |
| | Temperature long term | | 70.1 | 70.4 | 70.7 | 71.2 | 71.6 | 0.9 |
| | Temperature short term | | -22.4 | -8.7 | 2.2 | 14.4 | 19.5 | 23.0 |
| | Daily temperature | | 48.0 | 62.0 | 72.9 | 85.0 | 90.0 | 23.4 |
| Sacramento, CA | $PM_{10}$ long term | 111 | 23.7 | 24.6 | 27.3 | 29.2 | 29.5 | 4.6 |
| | $PM_{10}$ short term | | -19.6 | -13.0 | -4.7 | 3.6 | 9.9 | 16.6 |
| | Temperature long term | | 61.9 | 62.2 | 62.4 | 62.6 | 63.2 | 0.5 |
| | Temperature short term | | -13.9 | -10.1 | -0.9 | 7.3 | 16.4 | 16.0 |
| | Daily temperature | | 48.0 | 52.0 | 61.5 | 70.0 | 79.0 | 15.3 |
| Framingham, MA | $PM_{10}$ long term | 642 | 21.4 | 21.7 | 22.2 | 22.8 | 23.1 | 1.1 |
| | $PM_{10}$ short term | | -10.2 | -5.2 | 0.0 | 3.4 | 13.2 | 8.6 |
| | Temperature long term | | 50.7 | 51.1 | 51.3 | 51.5 | 51.7 | 0.4 |
| | Temperature short term | | -24.3 | -10.8 | 1.2 | 14.4 | 25.0 | 26.0 |
| | Daily temperature | | 27.0 | 40.0 | 52.5 | 66.0 | 76.0 | 25.9 |
| Minneapolis, MN | $PM_{10}$ long term | 1031 | 22.1 | 22.5 | 23.3 | 24.2 | 25.2 | 1.7 |
| | $PM_{10}$ short term | | -13.0 | -6.8 | 0.7 | 5.8 | 21.6 | 12.6 |
| | Temperature long term | | 42.9 | 43.2 | 43.6 | 43.8 | 45.1 | 0.6 |
| | Temperature short term | | -39.3 | -16.0 | 1.3 | 20.8 | 31.7 | 36.0 |
| | Daily temperature | | 4.0 | 28.0 | 44.9 | 64.0 | 75.0 | 36.8 |

| | | | | | | | | |
|---|---|---|---|---|---|---|---|---|
| New York City, NY | $PM_{10}$ long term | 118 | 25.3 | 27.3 | 28.7 | 30.1 | 30.8 | 2.7 |
| | $PM_{10}$ short term | | -12.2 | -5.7 | 2.7 | 7.1 | 25.4 | 12.8 |
| | Temperature long term | | 54.4 | 54.9 | 55.4 | 55.7 | 55.9 | 0.6 |
| | Temperature short term | | -19.8 | -13.3 | 1.9 | 16.6 | 26.3 | 27.0 |
| | Daily temperature | | 36.0 | 42.0 | 57.3 | 72.0 | 81.0 | 27.6 |
| Pittsburgh, PA | $PM_{10}$ long term | 380 | 26.5 | 27.1 | 27.9 | 28.9 | 29.9 | 1.8 |
| | $PM_{10}$ short term | | -17.9 | -11.7 | 0.7 | 8.4 | 34.9 | 20.1 |
| | Temperature long term | | 50.0 | 50.4 | 50.9 | 51.4 | 51.9 | 1.0 |
| | Temperature short term | | -27.5 | -13.2 | 0.5 | 15.7 | 24.3 | 28.9 |
| | Daily temperature | | 24.0 | 38.0 | 51.4 | 66.0 | 75.0 | 28.0 |

* “Long term” exposure is calculated as the 365 day moving average of the relevant exposure. “Short term” is the difference between the daily average exposure level and the 365 day average.

**Table 3: Association of $PM_{10}$ ($\mu g/m^3$) and temperature (F) with Sleep Disordered Breathing. Percent changes in outcomes are scaled to an inter-quartile increase in exposure level.**

| Outcome | Exposure * | % | 95% CI | P-value | IQR |
|---|---|---|---|---|---|
| RDI at 3% desaturation | $PM_{10}$ long term | 4.88 | (-5.77,16.73) | 0.39 | 2.1 |
| | $PM_{10}$ short term | -0.51 | (-5.22,4.44) | 0.84 | 14.5 |
| | Temperature long term | 0.20 | (-5.63,6.39) | 0.95 | 0.7 |
| | Temperature short term | 11.54 | (1.96,22.01) | 0.02 | 25.5 |
| Percent of sleep time < 90% desaturation | $PM_{10}$ long term | -3.10 | (-17.44,13.73) | 0.70 | 2.1 |
| | $PM_{10}$ short term | 2.67 | (-4.52,10.4) | 0.48 | 14.5 |
| | Temperature long term | 6.22 | (-2.89,16.2) | 0.19 | 0.7 |
| | Temperature short term | 5.12 | (-8.11,120.24) | 0.47 | 25.5 |
| Sleep Efficiency | $PM_{10}$ long term | 0.61 | (-0.77,2.0) | 0.39 | 2.1 |
| | $PM_{10}$ short term | -0.20 | (-0.82,0.42) | 0.53 | 14.5 |
| | Temperature long term | -0.33 | (-1.1,0.44) | 0.40 | 0.7 |
| | Temperature short term | 0.66 | (-0.49,1.84) | 0.26 | 25.5 |

* "Long term" exposure is calculated as the 365 day moving average of the relevant exposure. "Short term" is the difference between the daily average exposure level and the 365 day average.

Figure 1. City-specific distribution of Respiratory Disturbance Indices (RDI). RDI was computed as the ratio of the count of all apneas and hypopneas to the total sleep time expressed in hours.

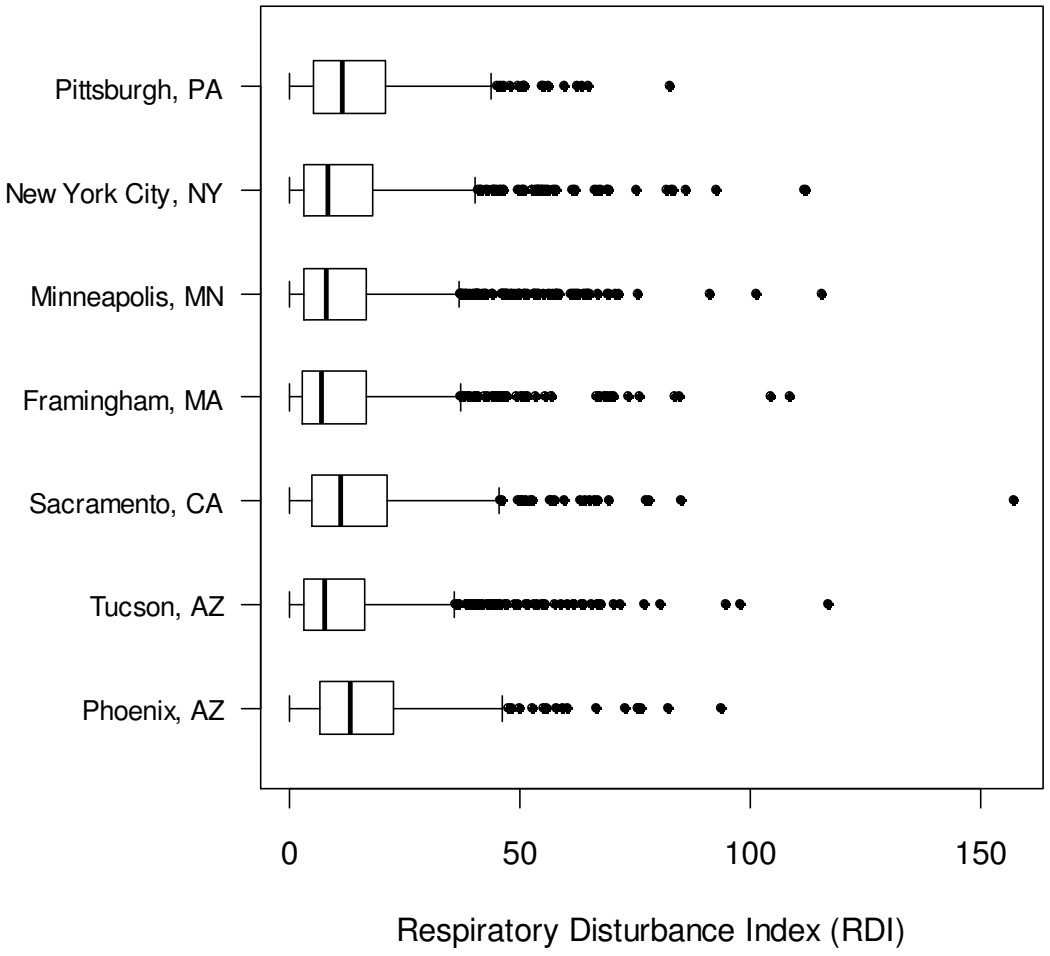


Figure 2. City and season specific distribution of 24-hour (daily) $PM_{10}$.

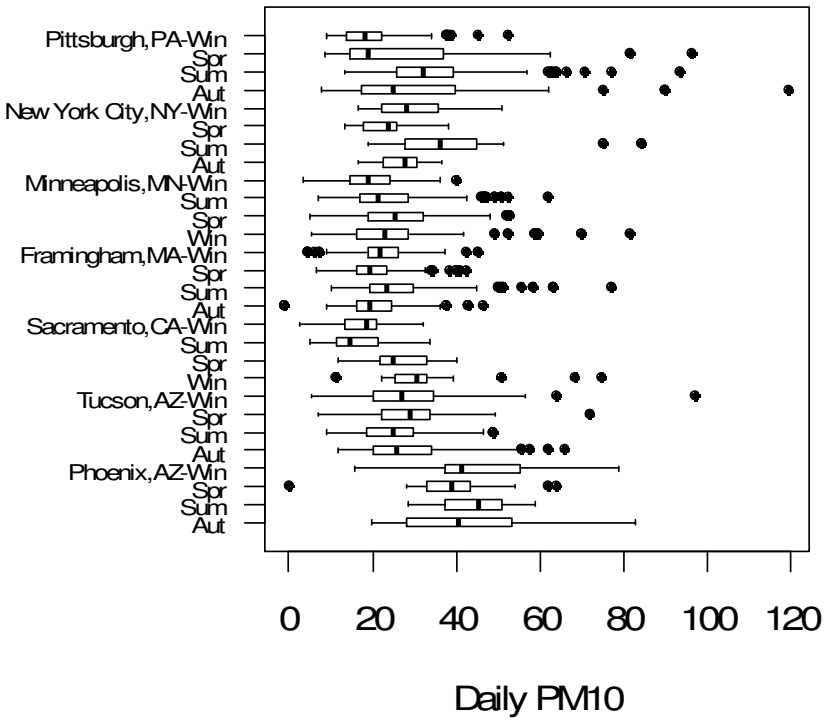
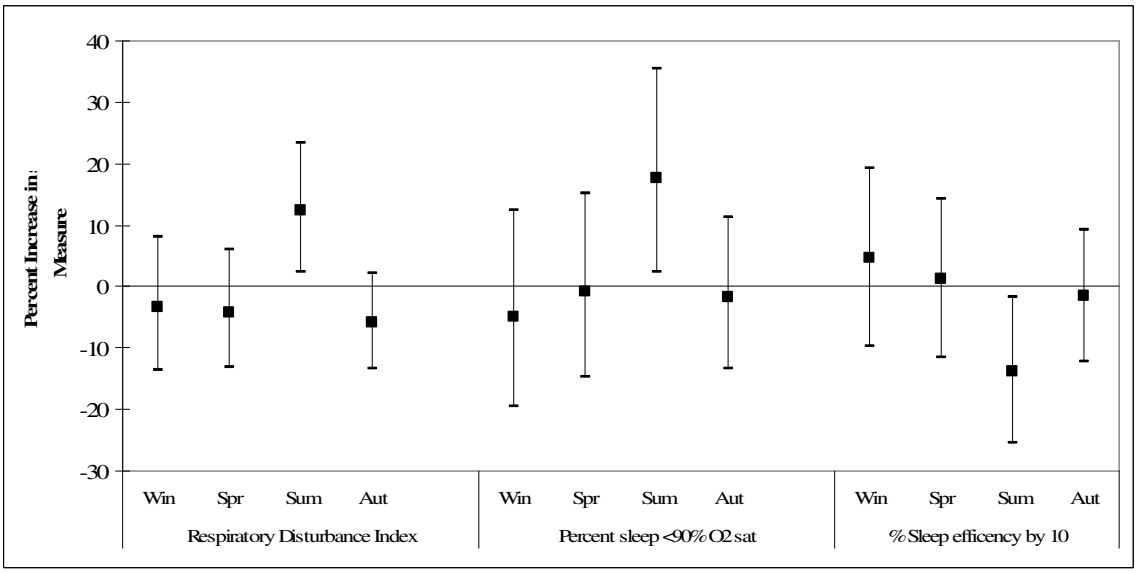


Figure 3: Season specific percent change and 95% Confidence Intervals in sleep disordered breathing outcomes, for an interquartile increase in short term $PM_{10}$, adjusting for seasonality, daily mean temperature, age, body mass index, gender, education, an age by gender interaction, smoking status, daily number of glasses of coffee, tea, and soda, and number of glasses of wine and beer four hours before going to sleep and long term $PM_{10}$ averages.



# Associations between $PM_{10}$ with Sleep and Sleep-Disordered Breathing in Adults from Seven U.S. Urban Areas

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## Online Data Supplement

Online Supplement

Figure 1. Season- and city-specific percent change in Respiratory Disturbance Index, for an interquartile increase in short term $PM_{10}$, adjusting for long term $PM_{10}$.