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Traffic-related air pollution and sleep in the Boston Area Community Health Survey

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Abstract

Little is known about environmental determinants of sleep. We investigated the association between black carbon (BC), a marker of traffic-related air pollution, and sleep measures among participants of the Boston Area Community Health Survey. We also sought to assess the impact of sociodemographic factors, health conditions, and season on associations. Residential 24-h BC was estimated from a validated land-use regression model for 3821 participants and averaged over 1-6 months and 1 year. Sleep measures included questionnaire-assessed sleep duration, sleep latency, and sleep apnea. Linear and logistic regression models controlling for confounders estimated the association between sleep measures and BC. Effect modification was tested with interaction terms. Main effects were not observed between BC and sleep measures. However, in stratified models, males experienced 0.23 h less sleep (95% CI: –0.42, –0.03) and those with low SES 0.25 h less sleep (95% CI: –0.48, –0.01) per IQR increase in annual BC (0.21 $\mu g/m^3$). In blacks, sleep duration increased with annual BC ($\beta = 0.34$ per IQR; 95% CI: 0.12, 0.57). Similar findings were observed for short sleep (≤ 5 h). BC was not associated with sleep apnea or sleep latency, however, long-term exposure may be associated with shorter sleep duration, particularly in men and those with low SES, and longer sleep duration in blacks.

Keywords

black carbon; particulate matter; sleep apnea; community; epidemiology; environmental

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Introduction

Sleep is an integral part of human functioning and chronic sleep problems, including poor sleep quality, sleep deprivation, and sleep-disordered breathing affects an estimated 50–70 million people in the United States.[1] Short sleep duration and long sleep duration have both been linked with adverse health outcomes such as total mortality, diabetes, cardiovascular disease, and obesity.[2–5] Sleep-disordered breathing has been linked with mortality and cardiovascular disease.[6,7] Such sleep problems do not impact the population equally. Disparities in sleep duration have been documented by race/ethnicity[8,9] and socioeconomic status (SES),[10,11] with minorities and those with low SES being more likely to have sleep durations (i.e., shorter and longer) that are associated with adverse health. Noise[12] and neighborhood quality,[13] long working hours, and being unmarried are associated with shorter sleep duration.[10] Lifestyle habits such as alcohol consumption and smoking can also impact sleep,[10] including sleep duration[14] and exacerbation of sleep-disordered breathing.[15] Although a number of risk factors for sleep disturbances are known, little is known about environmental factors such as particulate matter (PM) air pollution.

Particulate air pollution, which differentially impacts population subgroups, with those living in poorer less-advantaged areas tending to have greater exposures from both mobile and non-mobile sources, has been linked with adverse health outcomes such as cardiovascular disease, respiratory disease, and mortality,[16–18] and may also be linked with sleep. Indeed, second hand smoke (SHS), which contains PM, has been associated with sleep outcomes.[19–23] Only two studies to date, however, have specifically investigated the association between air pollution and sleep, specifically PM_{10}, with one in US adults[24] and one in Egyptian children,[25] both finding some evidence of associations. The association between sleep parameters and other types of particulate air pollution has not been investigated.

Traffic is a predominant source of PM in urban areas. Black carbon (BC), a component of PM formed by incomplete combustion, is a widely used marker of traffic-related air pollution.[26] The objective of this study was to investigate the association between traffic-related air pollution, as measured by $PM_{2.5}$ BC, and sleep parameters in a large cohort of men and women in the Boston area. Residential exposure to BC was estimated from a validated land-use regression model.[27] We investigated risk of sleep apnea, sleep duration, and sleep latency with respect to BC in the previous 1–6 months and 1 year. We hypothesized that elevated levels of BC would be associated with poorer sleep outcomes. Importantly, because sleep outcomes vary by sociodemographic factors and exposure may also vary by subgroup, we expected that the association would vary by gender, race/ethnicity, and SES and also investigated the modifying effects of season and health conditions.

Materials and Methods

Study population

The study population consisted of participants from the Boston Area Community Health Survey (BACH), a longitudinal study of a random sample of 5502 residents aged 30–79

years from three racial/ethnic groups (black, Hispanic, and white) in Boston, MA, originally designed to assess racial/ethnic disparities in urologic and gynecological symptoms. The current analysis uses data from the second survey conducted between 2006 and 2010. A total of 4144 individuals participated in BACH II. Only participants who resided in MA at follow-up were included in the analysis, leaving 3821 participants. Participants completed an in-person interview, including questions on demographics, sleep outcomes, co-morbidities, and lifestyle factors. Further details on methods have been previously published.[28] All participants provided written informed consent and the study was approved by the Institutional Review Board of the New England Research Institutes.

Exposure Assessment

Addresses for each participant at the time of participation in BACH II were geocoded and used to obtain predictions of residential BC concentrations from a validated spatiotemporal land-use regression model. Details of this model are presented elsewhere.[27,29,30] In brief, BC was measured using an aethalometer at >80 representative locations in the greater Boston area, of which three quarters were residential; the rest were commercial or government facilities. The data consist of >6021 pollution measurements from 2127 unique exposure days. These measurements were used to calibrate a model predicting concentrations based on BC measurements at a central location, land-use terms at each of the calibration monitors, weather parameters, height of the planetary boundary layer, and interactions of these parameters. Penalized splines were used to capture non-linearities in dependence, and thin-plate splines of latitude and longitude were used to capture remaining spatial variability. The R^2 of the model was 0.82, and the cross-validated R^2 between the daily measurements taken outside the residential locations and corresponding predictions obtained from fitting the model to the data, excluding data from a particular residential location, was 0.36. This is in contrast to an R^2 of 0.09 for the association between the central site and each residential reading.

The exposure model predicted 24-h measures of BC for each individual going back 1 year prior to the date of study participation. Daily BC measurements, presented as micrograms per cubic meter, were averaged monthly up to 6 months, as well as 1 year before the visit date.

Sleep Outcomes

Sleep parameters were measured with self-reports, as widely used in epidemiological studies and linked with adverse health outcomes. Participants reported usual sleep patterns in the previous month. Sleep duration was assessed as a continuous variable in hours to the tenth decimal place with an open-ended question, “How much do you usually sleep?”. Sleep latency was also assessed as a continuous variable, in minutes, as determined by the open-ended question: “How long does it usually take for you to fall asleep at bedtime?”. In addition, sleep apnea was measured by the Berlin sleep questionnaire, which categorizes individuals into high- and low-risk groups based on self-reported presence and frequencies of snoring and fatigue-related symptoms and presence or absence of high blood pressure and/or obesity.[31] The Berlin questionnaire has been shown to have good internal consistency and is validated against the respiratory disturbance index, which is used to clinically

diagnose sleep apnea. For a respiratory disturbance index >5, the Berlin questionnaire has a sensitivity of 86% and a specificity of 77%.[31]

Statistical Analysis

To preserve the maximum available sample size, multiple imputation was used to impute missing values by gender and race/ethnicity. A total of 15 multiple imputations were performed in SAS 9.1.3 (SAS Institute, Cary, NC, USA). Details of the BACH study design can be found elsewhere.[28]

Sleep duration and sleep latency were treated as continuous variables. We also dichotomized sleep duration to indicate short sleep as ≤ 5 h.[3] Forty individuals with responses of ≤ 2 h of sleep/night were excluded from analysis of sleep duration, as these were viewed as outliers. Sleep latency was skewed and therefore log transformed after adding 0.1 to improve the normality of the residuals. Short sleep and sleep apnea was treated as dichotomous outcomes. Linear and losgistic regression models were used to estimate the cross-sectional associations between BC averages and sleep outcomes. For each outcome, associations with BC averages (1-, 3-, 6-months and 1 year) were investigated in separate models. A priori selected potential confounders included in models were the categorical variables age (< 40, 40–49, 50–59, 60–69, 70+ years), gender, race (black, Hispanic, white), education (< 12th grade, completed high school/GED, some college/AA degree, college graduate, post-graduate degree), smoking status (current *vs* never/former), alcohol consumption (0, <1, 1 to <3, ≥3 drinks/day), anti-depressant use (yes/no), use of sleep medications (yes/no), body mass index (<25, 25–29.9, ≥30 kg/m^2), and physical activity (low, medium, high), and continuous variables for total caffeine intake and average temperature in the last 2 days (measured at Logan Airport). We chose to model age and body mass index categorically because of non-linear relationships between age and sleep outcomes and for ease of interpretation with body mass index. Select sensitivity analyses using quadratic terms for age and body mass index did not appreciably alter findings for BC effect estimates. We considered long-term time trends in sleep outcomes by including a continuous term for day of study, as well as seasonal effects by including harmonic regression terms for day of the calendar year, but found effect estimates were unaltered so did not include these to maintain parsimony of the models. We also considered nocturia (defined as two or more urinations at night after falling asleep) and lower urinary tract symptoms, as assessed using the American Urologic Association Symptom Index, as potential confounders separately but did not retain these in the final models because parameter estimates were largely unaltered. For sleep duration, we estimated the absolute change in hours and 95% confidence intervals (CIs). For sleep latency, we estimated the % change and 95% CIs. For dichotomous outcomes (sleep apnea, subscales, and short sleep), we estimated the odds ratio (OR) and 95% CIs. All effect estimates were reported for an increase in the exposure equal to the average interquartile range (IQR) across the BC exposure window concentrations (0.21 $\mu g/m^3$). To evaluate effect modification by demographic variables (gender, race/ethnicity, SES), categorical season, and health conditions (type 2 diabetes, CHD, obesity, asthma, COPD), separate models were constructed that included an interaction term between the effect modifier and BC metric. Nocturia and LUTs were also considered as effect modifiers. Statistical significance for all testing was considered at the $\alpha = 0.05$ level.

Because BACH participants were selected using a stratified sampling scheme, observations were weighted inversely to their probability of selection, and weights were post-stratified to the Boston population in 2000. Percents presented are weighted. To accommodate use of multiple imputation data sets and survey weights, analyses were performed with SUDAAN 11.0.0 (RTI, Research Triangle Park, NC, USA).

Results

The study population of 3821 individuals was 61.5% female, with a mean age of 53.8 years (range: 31–87 years), and racially/ethnically diverse (65.5% non-white) (Table 1). Close to one-quarter of the weighted population were current smokers (24.5%). The median BC concentration did not vary greatly across averaging periods (1-year BC = 0.63 $\mu g/m^3$; range: 0.14–1.94) (Table 2). Sleep duration was normally distributed with an average of 6.6 h (SE 0.04) (Table 3), whereas sleep latency was skewed with a median of 20 min (range: 0–180). Approximately 30% of the participants had high risk of sleep apnea.

Effect estimates for the BC averages overlapped the null in both univariate- and multivariable-adjusted main effects models for each sleep outcome (Table 4). However, negative-effect estimates were consistently observed for sleep duration, and positive-effect estimates were consistently observed for sleep latency. In addition, effect estimates consistently increased with increasing duration of exposure for both these outcomes, peaking with annual exposure. Including the 40 individuals with ≤ 2 h sleep did not appreciable alter findings. When sleep duration was dichotomized as ≤ 5 h or > 5 h, similarly, no associations were observed with any of the BC averages. For sleep apnea, ORs were also positive but not statistically significant. Although ORs for sleep apnea tended to increase with increasing exposure in univariate models, the effect estimate peaked with the 3-month exposure in multivariable models.

Because, in general, annual BC showed the strongest associations in main effects models, we investigated interactions between modifying variables (excluding season) and annual BC in separate models for primary outcomes. Statistically significant interaction terms were not found in sleep latency on the additive scale or sleep apnea models on the multiplicative scale. For sleep apnea, we also investigated interaction with the 3-month BC average, but did not find significant interactions. When sleep duration was treated as a continuous variable, associations with annual BC were modified by gender (P-interaction<0.01), race (P-interaction <0.01), and SES (P-interaction=0.04) on the additive scale (Figure 1, top panel), but not health conditions (type 2 diabetes, CHD, obesity, asthma, COPD, nocturia, or lower urinary tract symptoms). Among men, sleep duration decreased 0.23 h (i.e., 13.8 min) per IQR increase in BC (95% CI: – 0.42, – 0.03), whereas no association was observed in women. Likewise, individuals with low SES experienced decreasing sleep duration with increasing annual BC: per IQR increase in BC, those with low SES had on average 0.25 h less sleep (95% CI: –0.48, –0.01), whereas those with medium and high SES showed no associations with annual BC. Among males with low SES, the effect estimate was –0.42 per IQR increase in BC (95% CI: –0.42, –0.76). Blacks, however, experienced increasing sleep duration with increasing BC (β = 0.34 per IQR; 95% CI: 0.12, 0.57), which persisted when further restricted to males and low SES (β = 0.42 per IQR; 95% CI: –0.01, 0.85). No

associations between annual BC and sleep duration were observed in whites or Hispanics, although a negative association in whites was marginally significant ($P = 0.08$). The additive interactions by gender and SES were further supported on the multiplicative scale in logistic regression models evaluating short sleep duration (≤5h) (Figure 1, bottom panel). The odds of short sleep duration among men were 1.7 times greater per IQR increase in annual BC (95% CI: 1.1, 2.6) and the odds ratio for low SES was 1.6 (95% CI: 1.1, 2.3). In race-stratified models, there was an increased risk of short sleep duration with respect to BC exposure among Hispanics (OR = 1.4; 95% CI: 1.1, 1.8).

Discussion

This is the first study to investigate the association between sleep and BC, a marker of traffic-related air pollution. In a large cohort of 3821 individuals, modeled residential BC, a marker of traffic-related pollution, in the previous 1–6 months or 1 year was not associated with sleep apnea risk as measured by the Berlin scale, or with self-reported measures of sleep duration or sleep latency in the entire cohort, although for sleep duration and sleep latency there was a trend of increasing magnitude of association with increasing duration of exposure. Using annual BC exposure, we observed additive interactions with gender, SES, and race for sleep duration. Among males and those with low SES, sleep duration decreased with increasing levels of average annual BC exposure while controlling for potential confounders. Unexpectedly, we observed a positive association between sleep duration and BC exposure in blacks. Use of short sleep as an outcome supported the inverse associations observed between sleep duration and BC for males and those with low SES, and further suggested an inverse association among Hispanics.

Previous studies have not specifically investigated sleep duration or sleep latency with particulate air pollution. However, sleep efficiency, which is the percentage of time in bed actually asleep, was reduced in relation to short-term elevations in PM_{10} in a cross-sectional study using objective measures of sleep.[24] Although not directly comparable, this may be consistent with our findings of reduced sleep duration, as sleep efficiency may be reduced by either increasing sleep latency or decreasing sleep duration. Contrary to our findings, however, the authors did not find an association with long-term exposure over a year. Differences in exposure patterns and assessment may explain this difference. In particular, that study relied on the use of a single central site monitor in each city, whereas ours relied on a validated spatiotemporal model, providing exposure at the individual address-specific level. In that same study, the authors also observed an association between sleep-disordered breathing and short-term PM_{10}, which is contrary to our null findings between BC and risk of sleep apnea. In another study of Egyptian children, however, PM_{10} was not associated with sleep-disordered breathing, but associated with disorders of initiating and maintaining sleep, although it was unclear what exposure duration of PM_{10} was studied.[25]

Associations between SHS and sleep further support our findings. In a large study of 3963 Japanese workers, SHS exposure at work was associated with short sleep duration (≤ 6h) in men, but not women,[19] similar to our findings in which we observed decreasing sleep duration with increasing BC exposure among men, but not women. In addition, SHS was not associated with sleep latency, consistent with our findings. PM present in SHS may have a

role in the association with sleep duration, however, effects of nicotine or other compounds present in SHS cannot be ruled out. Using data from the BRFSS, SHS exposure was also associated with insufficient sleep among non-smokers.[20] Using NHANES data, SHS exposure was associated with increased odds of self-reported sleep disorders and sleep symptoms, although effect estimates were not statistically significant.[21] In children with asthma, exposure to SHS was associated with sleep onset delay (i.e., sleep latency), sleep-disordered breathing, parasomnias, daytime sleepiness, and overall sleep disturbance.[23]

Although differences in study population and design (e.g., objective *vs* subjective measures of sleep) may account for some of these conflicting findings, differences in exposure type may also have a role. Specifically, PM_{10} are coarse particles that mostly deposit in the upper airways and can cause irritation and breathing problems. PM_{10} has been linked with respiratory symptoms, asthma, and other respiratory problems.[18,32] Thus PM_{10} may impact sleep-disordered breathing through direct mechanical and inflammatory effects on the upper respiratory system. Smaller-sized particles such as that contained in SHS[33] and $PM_{2.5}$ BC penetrate deep into the alveolar regions of the lungs, where they are able to interact directly with receptors in the lung altering autonomic nervous system activity, initiate local inflammatory responses that may lead to systemic inflammation, and translocate directly into the systemic vasculature.[16] A portion of these particles also deposit in the nasopharyngeal region, and the ultrafine particles ($PM_{0.1}$) can translocate from the nose up the olfactory nerve into the brain,[34] potentially impacting the central nervous system. Controlled exposure to diesel exhaust in humans demonstrated changes in brain activity by electroencephalography.[35] Further evidence of central nervous system effects are provided by an association between long-term BC exposure and decreased cognitive function in older men.[36] Particle deposition in the brain has also been associated with neural inflammation,[37–39] which may influence sleep-wake cycles.[40,41]

The finding that BC differentially impacted sleep in the study population is consistent with a growing number of epidemiologic studies that have identified low-SES populations to be especially vulnerable to the health effects of air pollution.[42–45] Reasons may include compromised health from material deprivation and psychosocial stress, greater environmental exposure, and the combination of greater exposure and susceptibility.[45] Lower access to health care and poorer health behaviors and lifestyle habits can further promote susceptibility. Variations by gender have also been observed for air pollution-associated health outcomes, such as cardiovascular health.[46] The reason why BC may impact sleep duration in men but not women is not clear. As a large proportion of women were peri- or post-menopausal (55.6%), we also controlled for menopausal status in gender-stratified models but found that this did not change findings for women. Racial differences observed in this cohort were also striking. Blacks report both shorter and longer sleep durations as compared with whites,[9] and it is thought that longer sleep durations in blacks may be caused by poorer health. When we further control for self-reported health status and chronic health conditions, the association between BC and sleep duration in blacks became even stronger while remaining the same for Hispanics and whites. The complexity of sleep duration in blacks underscores that there are many factors impacting sleep duration in blacks, some of which may be correlated with BC exposure.

Although consistent associations with sleep duration and annual exposure were observed in models of interactions by sociodemographic factors, results should be interpreted with caution given that we assessed multiple windows of exposure and several outcomes, including a number of different interactions. Further limitations include use of estimated exposures. The use of exposure estimates based on residential address may misclassify personal exposure levels. However, the majority of the participants remained at the same address as the baseline assessment, an average of 5 years prior to BACH II, and any misclassification would be expected to be due to occupational and commuting exposures. Whether this misclassification would be differential or non-differential with respect to sleep outcome is unclear. Further, use of spatial-temporal predictions of exposures are uncertain quantities and the bias in health effect estimates is unclear; however, simulation studies suggest that in logistic regression settings in which the true effect is relatively small, the bias is negligible.[47] Confounding by noise, ambient temperature, and co-pollutants are of particular concern. We did not have direct measurements of noise, or estimates of noise exposure. However, the land-use regression model used to estimate BC exposures included proximity to major roads, which is often used as a proxy for noise exposures. Thus traffic noise may account for some of the associations observed with BC. Ideally, we would have investigated the independent and confounding effects of indoor temperature, particularly night-time temperature, but lacked these data. We also did not evaluate outdoor temperature, as this varied little for our participants within Boston. We did include average temperature over the past 2 days as a covariate in the model, however, because recent temperature effects on sleep may have influenced participants' recall of usual sleep patterns in the past month. Although we were able to control for a large number of potential confounders, we cannot rule out residual confounding and confounding by other unmeasured covariates such as stress. Importantly, we lacked the data on co-pollutants, such as different size fraction PM or gaseous co-pollutants.

Another potential limitation is that we relied on a self-report of usual sleep parameters, which is subject to measurement error. Such measurement error in the outcome in a linear regression would decrease power, but does not bias the effect estimate for exposure. Subjective sleep parameters and symptoms are widely used in epidemiological studies, however, and self-reported outcomes are comparable in our study population to others.[9,48] Although we know of no data on sleep apnea risk in the general US population as assessed by the Berlin questionnaire, as would be expected, however, our estimate of 30% was slightly lower than that of those undergoing the sleep study (37%).[31] Although further research using objective measures of sleep apnea and long-term measures of BC may more accurately estimate exposure–response relationships, subjective reports of sleep are also important in terms of understanding the burden of symptom complaints associated with air pollution. It is also important to note that the directionality of our observed associations cannot be certain owing to the cross-sectional nature of this study. Finally, our study was conducted among a resictricted geographical region, and findings may not neccesarily generalize to non-urban areas or other geographical regions.

Despite the limitations of this study, we were able to investigate for the first time the association of source-specific $PM_{2.5}$ (i.e., traffic), as measured by individual level predictions of BC, with sleep outcomes in a large cohort while controlling for a wide range

of potential confounders. Findings suggest a possible link between long-term exposure to BC and decreased sleep duration in men and those with low SES. The clinical implications of the small decerase in sleep duration we observed in this study are unclear but with increasing recognition of the chronic health consequences of too much or too little sleep, including cardiovascular disease, and the accepted epidemiological link between particulate air pollution and cardiovascular disease and mortality, the associations between traffic-related particulate air pollution, and $PM_{2.5}$ in general, and sleep deserve further investigation.

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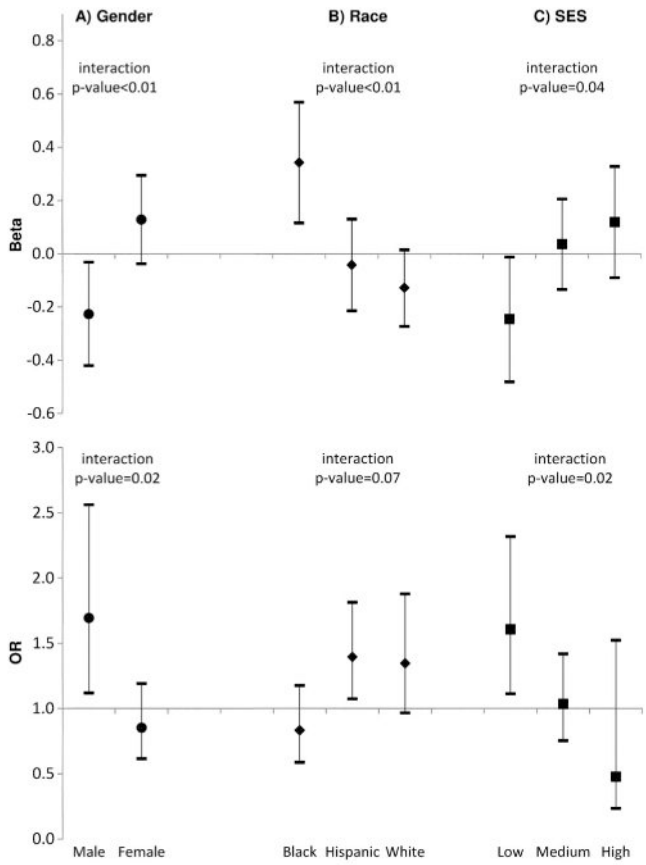

Figure 1.

Change in sleep duration in hours (top panel) and odds ratio for short sleep (r5 h) (bottom panel) per inter-quartile range increase (0.21 $\mu g/m^3$) in annual residential black carbon by (A) gender, (B) race, and (C) socioeconomic status (SES).

Table 1

Characteristics of study population (n = 3821).

	No.	Weighted %
Female gender	2351	61.5[a]
Age (years), mean (SE)	53.9	0.48
Race/ethnicity		
Black	1237	32.4[a]
Hispanic	1265	33.1
White	1319	34.5
Education		
<12th grade	919	12.3
Completed high school/GED	1249	29.8
Some college or AA degree	590	15.4
College graduate	558	21.5
Post-graduate degree	504	21.1
SES		
Low	2183	39.8
Middle	1329	46.5
High	309	13.7
Current smoker	953	24.5
Alcohol use, drinks per day		
0 drinks	1795	37.8
<1 drink/day	1275	37.8
1 to <3 drinks/day	561	19.8
3+ drinks/day	189	4.6
Caffeinated drinks/day, mean (SE)	2.69	0.07
Physical activity		
Low	1387	30.2
Medium	1801	49.4
High	633	20.4
BMI (kg/m^2), mean (SE)	30	0.2
Use of anti-depressants	716	17.7
Sleep medication use	641	17.0
Health conditions		
Obese (30+ kg/m^2)	1731	41.7
Diabetes (type 2)	652	11.9
CHD	517	11.5
Asthma	807	20.1
Chronic obstructive pulmonary disease	455	9.8
Nocturia	1451	32.4
Lower urinary tract symptoms	828	20.7

Abbreviations: BMI, body mass index; CHD, coronary heart disease; GED, general educational development; SES, socioeconomic status.

[a] Unweighted percents.

Table 2

Residential Black Carbon Exposure Among Study Participants ($\mu g/m^3$) (n = 3821).

	Mean	SD	Median	Min	Max	IQR[a]
1 Month	0.67	1.43	0.65	0.09	3.42	0.24
3 Months	0.66	1.32	0.65	0.10	3.07	0.23
6 Months	0.64	1.16	0.63	0.14	2.07	0.19
1 Year	0.65	1.10	0.63	0.14	1.94	0.18

[a] Average IQR = 0.21 $\mu g/m^3$.

Table 3

Distribution of sleep outcomes (n = 3821).

Sleep apnea (high risk), no. (%)	1196	29.6
Sleep duration (hours), mean (SE)	6.6	0.04
Sleep latency (min), median (range)	20.0	(0–180)
Short sleep (≤ 5 h), no. (%)	962	21.6

Table 4

Association between sleep outcomes and inter-quartile range change (0.21 $\mu g/m^3$) in Residential Black Carbon.

Outcome	*BC averaging period*	*Univariate models*			*Multivariable models*[a]		
		β	***95% CI***	**P-*value***	***β***	***95% CI***	**P-*value***
Sleep duration (h)	1 Month	0.00	(– 0.11, 0.10)	0.94	0.01	(– 0.08, 0.09)	0.8
	3 Months	–0.02	(– 0.14, 0.10)	0.70	–0.02	(– 0.12, 0.09)	0.73
	6 Months	–0.04	(– 0.18, 0.11)	0.60	–0.04	(– 0.17, 0.10)	0.59
	1 Year	–0.08	(– 0.23, 0.07)	0.32	–0.06	(– 0.19, 0.08)	0.40
		% Change	*95% CI*	P-*value*	*% Change*	*95% CI*	P-*value*
Sleep latency (minutes)	1 Month	6.3	(– 2.5, 16.1)	0.16	3.0	(– 4.1, 10.6)	0.42
	3 Months	8.8	(– 1.7, 20.3)	0.10	4.5	(– 4.1, 13.9)	0.31
	6 Months	12.0	(– 0.6, 26.3)	0.06	6.7	(– 4.1, 18.8)	0.23
	1 Year	11.5	(– 2.7, 27.9)	0.11	8.3	(– 3.5, 21.6)	0.18
		OR	*95% CI*	P-*value*	*OR*	*95% CI*	P-*value*
Short sleep (≤ 5 h)	1 Month	1.04	(0.85, 1.27)	0.74	0.97	(0.78, 1.21)	0.81
	3 Month	1.14	(0.91, 1.43)	0.25	1.10	(0.88, 1.38)	0.41
	6 Months	1.22	(0.94, 1.58)	0.250.14	1.17	(0.89, 1.55)	0.27
	1 Year	1.30	(0.98, 1.72)	0.07	1.26	(0.94, 1.69)	0.12
Sleep apnea (high risk)	1 Month	1.10	(0.94, 1.28)	0.23	1.06	(0.90, 1.24)	0.50
	3 Months	1.12	(0.94, 1.33)	0.21	1.10	(0.93, 1.30)	0.27
	6 months	1.07	(0.85, 1.34)	0.55	1.04	(0.83, 1.31)	0.72
	1 Year	1.13	(0.88, 1.44)	0.34	1.04	(0.82, 1.33)	0.58

[a] Adjusted for age, gender, race, education, smoking status, alcohol consumption, body mass index, physical activity, antidepressants, sleep medication, and average temperature in last 2 days.