Published in final edited form as:

Environ Int. 2018 June ; 115: 188–195. doi:10.1016/j.envint.2018.02.040.

Indoor Black Carbon of Outdoor Origin and Oxidative Stress Biomarkers in Patients with Chronic Obstructive Pulmonary Disease

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Abstract

Objectives—We assessed relationships between indoor black carbon (BC) exposure and urinary oxidative stress biomarkers, 8-hydroxy-2'-deoxyguanosine (8-OHdG) and malondialdehyde (MDA), in participants with chronic obstructive pulmonary disease (COPD).

Methods—Eighty-two participants completed in-home air sampling for one week prior to providing urine samples up to four times in a year. Weekly indoor and daily outdoor concentrations were used to estimate indoor daily lags and moving averages. There were no reported in-home BC sources, thus indoor levels closely represented outdoor BC infiltration. Mixed effects regression models with a random intercept for each participant were used to assess relationships between indoor BC and 8-OHdG and MDA, adjusting for age, race, BMI, diabetes,

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Declarations of Interest: None.

heart disease, season, time of urine collection, urine creatinine, and outdoor humidity and temperature.

Results—There were positive effects of BC on 8-OHdG and MDA, with the greatest effect the day before urine collection (6.9% increase; 95% CI 0.9-13.3%, per interquartile range: 0.22 $\mu\text{g}/\text{m}^3$) for 8-OHdG and 1 to 4 days before collection (8.3% increase; 95% CI 0.03-17.3% per IQR) for MDA. Results were similar in models adjusting for $\text{PM}_{2.5}$ not associated with BC and NO_2 (10.4% increase, 95% CI: 3.5-17.9 for 8-OHdG; 8.1% increase, 95% CI: -1.1-18.1 for MDA). Effects on 8-OHdG were greater in obese participants.

Conclusions—We found positive associations between BC exposure and 8-OHdG and MDA, in which associations with 8-OHdG were stronger in obese participants. These results suggest that exposure to low levels of traffic-related pollution results in lipid peroxidation and oxidative DNA damage in individuals with COPD.

Keywords

urinary oxidative stress; pollution; COPD

1. Introduction

A number of studies conducted in human subjects have shown associations between acute exposures to air pollution and increases in various blood, urine, and exhaled breath biomarkers attributable to oxidative stress pathways.(Barregard et al. 2008; Ceylan et al. 2006; Neophytou et al. 2014; Romieu et al. 2008; Zhang et al. 2013) Oxidative stress refers to an imbalance of the cellular redox system whereby reactive oxidative species, including free radicals, are generated due to poorly controlled oxidative reactions.(Autrup et al. 1999; Betteridge 2000; Go and Jones 2010; Jones 2006) It is hypothesized that oxidative stress may lead to the development of disease when free radicals react with biomolecules, such as DNA and lipids. DNA ultimately regulates numerous cellular processes, and lipids are essential components of cell membranes. Reactions between these biomolecules and free radicals result in DNA oxidation and lipid peroxidation, which then can induce cellular damage.(Autrup et al. 1999; Betteridge 2000; Jones 2006; Kelly 2003) There is also evidence that lipid peroxidation products may result in secondary damage to DNA.(Ayala et al. 2014) The potential deleterious effects of oxidative damage to DNA and lipids are primarily studied by quantifying the concentrations of byproducts since free radicals are short-lived.(Dalle-Donne et al. 2006; Zinellu et al. 2016)

An example of a widely studied byproduct is 8-hydroxy-2'-deoxyguanosine (8-OHdG), a biomarker of DNA oxidation that has been associated with subsequent risk of cancer and cardiovascular diseases.(Chuang et al. 2014; Cooke et al. 2005; Cooke et al. 2002; Delfino et al. 2011; Tagesson et al. 1995; Tagesson et al. 1996) Malondialdehyde (MDA), a byproduct of lipid peroxidation, has also been associated with subsequent risk of cancer and atherosclerosis.(Delfino et al. 2011; Lykkesfeldt 2007; Shamberger et al. 1974; Tagesson et al. 1996) Examining the relationships of 8-OHdG and MDA with particulate air pollution can provide insight into mechanisms whereby particulate pollution results in cellular damage and potentially disease. Several studies have examined effects of ambient traffic related

particulate matter (Di et al. 2017; Gong et al. 2013; Huang et al. 2012; Li et al. 2016; Ren et al. 2011; Zhang et al. 2013); however, less is known on the effects of these traffic particles that infiltrate indoors.

There are few studies assessing effects of pollution on oxidative stress in populations with clinical respiratory disease, particularly those with chronic obstructive pulmonary disease (COPD), a leading cause of mortality worldwide. (Ceylan et al. 2006; Mathers and Loncar 2006; Wang and GBD 2015 Mortality and Causes of Death Collaborators 2016; Zanobetti et al. 2000; Zhang et al. 2017) Exposures to traffic related particles (as assessed by black carbon (BC) or elemental carbon), have been associated with hospitalization and mortality from COPD and other respiratory diseases. (Bell et al. 2009; Gan et al. 2013; Peng et al. 2009; Zanobetti and Schwartz 2006) Additionally, there is a growing body of literature indicating that biomarkers of oxidative stress increase during COPD exacerbations, which also contribute to increased hospitalizations, morbidity, and mortality. (Cheng et al. 2007; Kelly 2003; Tramuto et al. 2011) As a result, there is rationale for studying oxidative biomarkers associated with COPD severity.

2. Methods

2.1 Study Sample

Participants were recruited at the VA Boston Healthcare System between November 2012 and December 2014 as part of a COPD cohort examining associations between indoor air quality and health. Potential participants were identified by medical record review of VA Boston pulmonary, primary care, and pulmonary function clinic encounters using ICD-9 codes 490-493 and 496. Identified potentially eligible participants were sent recruitment mailings. In addition, recruitment flyers were placed at VA Boston clinics and Boston area civilian hospitals. Participants who expressed interest in the study were invited to attend a clinic visit to confirm eligibility and obtain consent.

Participants were eligible if they were at least 40 years old, smoked at least 10 pack years, had physician-diagnosed COPD, and had a $FEV_1/FVC < 0.70$ on post-bronchodilator spirometry or emphysema on a CT scan reported in their medical record. Participants were ineligible if they had a history of any malignancies other than stable skin or prostate cancer at the time of entry and could not be current smokers or live with smokers, use a wood stove or fireplace, or have other major sources of in-home pollution exposure.

Participants were asked to attend four in-person visits scheduled approximately three months apart over one year. Participants who underwent therapy for a COPD exacerbation were assessed at least two weeks after completion of treatment in order to be considered clinically stable. Urine samples and questionnaires on health and home environmental features were collected at each in-person clinic visit. The study protocol was approved by Institutional Review Boards at VA Boston and Harvard Medical School and informed consent was obtained from all participants prior to study procedures.

2.2 Pollution Assessment

Prior to each clinic visit, subjects were provided with an in-home micro-environmental sampler that collected data on in-home exposures for one week. Samplers were used to collect data in the room where participants reported spending the most time, excluding the kitchen. Each sampler was returned at the clinic visit or by express shipping. The in-home sampler included a pump set to a flow rate of 1.8 LPM, using a size-selective impactor to collect particles less than 2.5 μm in diameter ($\text{PM}_{2.5}$). An EEL M43D Smokestain Reflectometer was used to determine BC mass, using each filter as its own blank (measured before and after sampling), yielding the net weight of BC. Subsequently, BC concentrations were calculated by dividing the net measurement of each filter (μg) by the total volume of air sampled (m^3). The limit of detection for BC was 0.03 $\mu\text{g}/\text{m}^3$ based on 3 times the standard deviation of blank filters; about 10% of the samples were below the limit. All values were included to preserve the distribution of concentration levels. Nitrogen dioxide (NO_2) was measured over the same weekly sampling period using an Ogawa passive sampling badge attached to the micro-environmental sampler.

In addition to collecting measurements on weekly indoor BC, we estimated measurements of daily indoor BC. In the absence of indoor sources, the integrated indoor measurement of BC represented outdoor infiltration; therefore, we expected daily indoor levels to be proportional to outdoor levels.(Gryparis et al. 2007; Suglia et al. 2008) Daily outdoor BC averages were calculated using central site data (Francis A. Countway Library, Boston, MA) measured by an aethalometer (Magee Scientific Company, model AE-16, Berkeley, CA); methods for central site data collection have been described elsewhere.(Kang et al. 2010) Daily indoor BC for each home on each sampling day was estimated by further averaging the daily central site data according to the days reported by the participant and measuring in-home multi-day integrated values using the following equation, where i represents the number of days prior to urine collection (lag day 1 to day 8):

$$\text{Indoor day } BC_i = (\text{Outdoor day } BC_i / \text{Outdoor week } BC) \times \text{Indoor week } BC \quad (1)$$

We constructed daily moving averages starting the day before (lag day 1) to eight days before urine collection (lag day 8). Each of the moving averages was examined in a separate model for comparison among time periods. Since BC is a component of $\text{PM}_{2.5}$, we estimated the effects of $\text{PM}_{2.5}$ other than BC by constructing similar indoor $\text{PM}_{2.5}$ moving averages and regressing each $\text{PM}_{2.5}$ moving average on its corresponding BC moving average. We then included the residuals as an additional variable in each BC-biomarker regression model. This is a suggested approach since including directly measured $\text{PM}_{2.5}$ concentrations in the model may result in over-adjustment of BC.(Mostofsky et al. 2012)

2.3 Oxidative Stress Assessment

Urinary concentrations of 8-OHdG were measured using a method described previously using a HPLC-ESI-MS system.(Commodore et al. 2013) The limit of detection was 0.133 ng/mL, and the recovery of the sample treatment was >80%. We measured MDA in two forms, free (protein unconjugated) and total (sum of protein unconjugated and conjugated),

as there is conflicting evidence regarding the form that best reflects lipid peroxidation.(De Vecchi et al. 2009; Del Rio et al. 2005) Concentrations of free MDA were measured using a HPLC system with fluorescent detection, as described previously.(Gong et al. 2013) The detection limit, extraction recovery and analytical precision of this method were 1.8 nM, 75.9%, and 2.2% measured as the relative standard deviation (RSD) from 8 replicate injections, respectively. Total MDA was analyzed in a similar fashion as free MDA with the exception of an added alkaline hydrolysis step prior to sample extraction procedures. Concentrations of creatinine were measured for all urine samples using a colorimetric method.(Gong et al. 2013)

2.4 Covariates

Several variables were chosen *a priori* as potential confounders and effect modifiers as factors associated with oxidative stress or BC exposure. Race, heart disease requiring treatment in the past 10 years, diabetes, and smoking history were available from the demographic and health questionnaires completed at baseline. Age, statin use, non-steroidal anti-inflammatory (NSAID) medication use, percent-predicted post-bronchodilator forced expiratory volume in one second (%-predicted FEV₁)(Hankinson et al. 1999), and time of urine collection were collected at each clinic visit. Body mass index (BMI) was calculated from measured height and weight.

Participant home addresses were geocoded and daily outdoor temperature at each home was estimated by a validated model using a combination of satellite remote sensing of surface temperature (daily on 1 km × 1 km grids), land use (e.g., greenness), and ground level weather stations measuring air temperature.(Kloog et al. 2014) Relative humidity was measured at the Boston Logan International Airport Weather Station.(Kang et al. 2010) A season variable was created by categorizing the month of the clinical visit into one of four categories: winter (December, January, February), spring (March, April, May), summer (June, July, August), or fall (September, October, November).

2.5 Statistical Methods

Linear mixed effects regression models were used with a random intercept for each participant (PROC MIXED, SAS 9.4; SAS Institute, Inc., Cary, NC). Each urine biomarker was natural log-transformed to meet model assumptions. Beta and 95% CI values were exponentiated to interpret the results as percent changes of each outcome, based on a 0.22 µg/m³ increase in BC (integrated sample interquartile range; IQR). Model assumptions were confirmed by examination of residuals, and there were no deviations from linearity as determined using penalized splines (R mgcv package, R, version 3.1.2, Vienna, Austria). We assessed effect modification by diabetes, heart disease, statin and NSAID use, COPD severity (above and below median %-predicted FEV₁), and obesity status (BMI ≥30 versus BMI <30) in stratified models and assessed the statistical significance of interactions using multiplicative interaction terms. Lastly, we conducted a sensitivity analysis to consider possible confounding by other pollutants, specifically NO₂ and PM_{2.5} residuals.

3. Results

Between November 2012 and December 2014 we recruited 96 participants with COPD eligible for participation (Figure A1). There were 3 participants excluded who were found to be smoking and 8 participants who could not provide at least one urine sample. Since the study was designed to measure acute effects, participants with exposure samples collected more than nine days before the clinic visit were excluded ($n=3$). The final analytic dataset included 82 participants with 237 observations. There were 32 participants with four visits; 19 with three visits, 21 with two visits, and 10 with one visit. The mean age of participants was 72.9 yrs (Table 1); they were all male and predominantly white (89%). At study entry, most participants reported spending the majority of their time at home, with 17 hrs on average indoors on both weekdays and weekends which was similar across all study visits ($n=237$, also 17 hrs on average).

Participants ran the microenvironmental particle sampler over an average of 7.6 days (range 4 to 10 days) (Table 2). The median concentrations of home indoor and central site outdoor BC were 0.18 and 0.54 $\mu\text{g}/\text{m}^3$, respectively.

Final regression models included adjustment for season, outdoor temperature and relative humidity 24 hrs before urine collection, age, BMI, race, diabetes, heart disease, and time of collection. Urinary creatinine concentrations were included to adjust for urinary dilution. We observed positive associations between all BC measures (multi-day integrated values and moving averages) and oxidative stress markers (Figure 1, Table A1).

There were consistent positive effects of BC on 8-OHdG, ranging from 2.8% to 6.9% per IQR; the strongest effect was observed on the day before testing (day 1), yielding a statistically significant association (6.9%, 95% CI: 0.9-13.3). When examining effects in free and total MDA, there was a 0.8% (95% CI: -5.4-7.4) increase in free MDA and 5.4% (95% CI: -2.4-14.0) increase in total MDA per IQR of multi-day integrated BC. Additionally, effects on total MDA were statistically significant with exposure averaged days 1-4 before urine collection (8.3% increase; 95% CI: 0.03-17.3).

Diabetes, heart disease, statins, NSAIDs, COPD severity, and obesity were explored as effect modifiers between BC and oxidative stress using interaction terms. Only obesity was statistically significant with p-interactions of 0.04 and 0.02 on moving average days 1-3 and 1-4, respectively (Figure 2, Table A2). In participants who were classified as obese (BMI 30) at each visit, effect estimates were as large as a 15.4% increase (95% CI: 3.8-28.4) for every 0.22 $\mu\text{g}/\text{m}^3$ of BC on days 1-4 before urine collection. No statistically significant effects of 8-OHdG were observed in participants who were classified as non-obese at each visit. There was no evidence of effect modification with MDA.

In our sensitivity analysis, we explored NO_2 and $\text{PM}_{2.5}$ residuals as confounders of our associations; some observations were excluded since we did not have valid corresponding NO_2 and $\text{PM}_{2.5}$ measurements (Figure 3, Tables A3 – A5). BC was not correlated with $\text{PM}_{2.5}$ residuals as expected and weakly correlated with NO_2 (Spearman correlation coefficient = 0.33). After adjustment in separate and combined models including $\text{PM}_{2.5}$ residuals and NO_2 , we observed BC effect sizes that were positive and similar to effect sizes

in unadjusted models (Figure 3). Effects of BC on 8-OHdG remained significant on day 1 in all models (for example, increase of 10.4% per IQR, 95% CI: 3.5-17.9, adjusted for PM_{2.5} residuals and NO₂). Effects of BC on total MDA were still greatest with exposure averaged over days 1-4, but with wider confidence intervals (increase of 8.1% per IQR, 95% CI: -1.1-18.1, adjusted for PM_{2.5} residuals and NO₂) compared to unadjusted BC. Effects of NO₂ and PM_{2.5} residuals varied by model, but there was no consistent positive effect of either on MDA or 8-OHdG (data not shown).

4. Discussion

In this study we found positive relationships between BC moving averages up to eight days before urine collection and two oxidative stress markers, 8-OHdG and MDA, which represent measures of DNA oxidization and lipid peroxidation, respectively. In single and multipollutant models (NO₂ and PM_{2.5} residuals) we observed positive statistically significant effects of 8-OHdG with BC exposure the day before collection and positive effects for other moving averages. For total MDA, effects were also consistently positive and greatest on days 1-4, but effects became more imprecise adjusting for NO₂ and PM_{2.5}. Our analysis supports effects of BC on the selected oxidative stress biomarkers in COPD patients. Additionally, our findings are notable for demonstrating effects of outdoor BC infiltrating indoors since we made efforts to exclude BC sources of indoor origin.

We examined MDA in two forms, protein conjugated (free) and the sum of protein conjugated and unconjugated (total). Effects were greater in analyses that examined total MDA. Our finding regarding free MDA is likely explained by the binding of MDA to protein molecules in the urine; thus total urinary MDA is a better indicator of lipid peroxidation.

To account for potential confounding by other pollutants, we adjusted our BC-oxidative stress models for PM_{2.5} and NO₂ in sensitivity analyses. We regressed PM_{2.5} on BC moving averages and used the residuals of each of these associations to represent “non-BC” PM_{2.5} in our oxidative stress models. Although we did not have equivalent central site data for NO₂, we included the integrated sample of NO₂ in our models. Effects of BC on 8-OHdG and MDA were similar in unadjusted and adjusted models. For MDA, the confidence intervals slightly widened. There were no consistent positive effects of either NO₂ or residuals of PM_{2.5} on 8-OHdG or MDA across all models.

Our results are consistent with findings from other studies studying air pollution and oxidative stress in other populations. Huang and colleagues (2012) found significant effects of ambient elemental carbon (EC) exposures in the 24 hrs before urine collection and 8-OHdG in a population of healthy medical professionals during the Beijing Olympics.(Huang et al. 2012) Similarly, they found positive associations between EC four to five days before collection with 8-isoprostane, a measure of lipid peroxidation, in exhaled breath condensate. (Huang et al. 2012) We observed similar findings in urinary MDA with the largest effects in the moving averages four days before collection. Vinzents *et al* conducted personal monitoring of ultrafine particles (UFPs) in a small sample of healthy participants in Copenhagen; they found significant associations between UFPs and DNA base damage the day before collection.(Vinzents et al. 2005) In healthy trucking company workers exposed to

traffic particles we previously reported suggestive associations between ambient EC and urinary 8-OHdG.(Neophytou et al. 2013)

Other studies examining the associations between air pollution and oxidative stress have also been conducted in Boston. Li *et al* examined ambient BC obtained from the same central monitoring site (Countway) and a lipid peroxidation measure, plasma myeloperoxidase, in a cohort of healthy participants from Eastern Massachusetts in the Framingham Heart Study. They found positive associations indicating acute effects of ambient BC on myeloperoxidase.(Li et al. 2016) Conversely, Ren *et al* found no significant associations between ambient BC, measured at the Countway site, and urinary 8-OHdG in an elderly Boston VA cohort; they did however find associations between PM_{2.5} and organic carbon (OC) with 8-OHdG.(Ren et al. 2011) Our levels of BC (mean = 0.21 µg/m³) were almost four times lower than those previously reported (0.84 µg/m³ in Li *et al* and 0.88 µg/m³ in Ren *et al*). This suggest that individuals with COPD may be more susceptible to the effects of pollution.(McGuinness and Sapey 2017)

We also explored effect modification based on obesity and found greater effects of BC on 8-OHdG in obese individuals. Other studies have noted stronger effects of air pollution on inflammatory biomarkers in obese individuals compared to non-obese individuals but have not assessed oxidative stress biomarkers.(Alexeeff et al. 2007; Baja et al. 2010; Madrigano et al. 2010) It is possible that the effects of oxidative stress are enhanced in obese individuals since obesity is associated with inflammatory and oxidative stress biomarkers compared to non-obese individuals.(Furukawa et al. 2017; Madrigano et al. 2010) Antioxidants may represent a protective factor against oxidative damage, as studies have shown that non-obese individuals have increased levels of antioxidants compared to obese counterparts.(Wallstrom et al. 2001)

4.1 Limitations

A limitation of this study is that indoor BC was not directly measured on a daily basis; thus daily lags were estimated using only the integrated multiday indoor concentrations and the daily central site data. Additionally, it is possible that participants did not report indoor sources contributing to BC, thus some of the BC may have come from sources other than traffic. However, we limited this possibility by excluding homes with major sources of indoor BC. Given that all participants did not spend 24 hrs a day indoors at home, our measures of exposure capture a varying amount of each participant's total personal BC exposure. Another limitation is the generalizability of our results. Participants were predominantly white males, representing the Boston VA population, and had to be healthy enough to attend study visits. Those who may have been most susceptible to the effects of traffic related particles may have been excluded from these analyses. Finally, due to sample size considerations, our assessment of effect modification can be considered exploratory.

4.2 Strengths

The participants sampled all have well documented COPD as their disease diagnoses were confirmed by medical record review and in-person spirometry. We note associations even at low BC levels compared to previous studies which may be in part due to the greater

susceptibility of these patients with COPD. This is the first study that we are aware of assessing repeated measures of indoor BC with urinary biomarkers of oxidative stress.

Our results are novel as we directly measured BC levels seasonally inside the home, which was where participants spent most of their time during sampling (average of 17 hrs per day). Our reported time inside a residence is comparable to estimates reported in the literature (Klepeis et al. 2001), i.e., approximately two-thirds of a day as in the National Human Activity Pattern Survey.

5. Conclusion

We observed associations between BC, a marker of traffic-related particulate pollution measured in the home with two different measures of oxidative stress indicating an imbalance in oxidative metabolic pathways in COPD patients. These results are consistent with those reported by previous studies and are indicative of systemic effects even at low-level exposures. These studies support efforts by public health policies to control traffic emissions and protect susceptible individuals, such as those with COPD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

The authors would like to thank the study participants for their dedicated participation as well as Mike Wolfson, Choong-Min Kang, Denise Lee, Anisa Khadraoui, and Daniel Bernard for their assistance in collecting and managing data for this study.

Funding

This publication was supported by the National Institutes of Environmental Health Sciences, NIH Grant R01 ES019853, and by resources and the use of facilities at the VA Boston Healthcare System. The views expressed in this article are those of the authors and do not necessarily reflect the position or policy of the Department of Veterans Affairs or the United States Government.

This publication was made possible by USEPA grant RD-83479801 and RD-83587201. Its contents are solely the responsibility of the grantee and do not necessarily represent the official views of the USEPA. Further, USEPA does not endorse the purchase of any commercial products or services mentioned in the publication.

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HIGHLIGHTS

- Black carbon samples in participant homes were collected up to four times a year.
- Indoor black carbon represents infiltration of outdoor traffic-related particles.
- Participants provided urine samples after each environmental sampling.
- Indoor exposures are positively related to urinary oxidative stress biomarkers.

Figure 1. Percent increase in 8-OHdG, free MDA, and total MDA per interquartile range of black carbon (BC)

Percent increases (mean, 95% confidence interval) in 8-OHdG, free MDA, and total MDA are shown for the moving averages of BC starting the day before urine collection through days 1 through 8 and for the integrated filter sample per interquartile range ($0.22 \mu\text{g}/\text{m}^3$) of BC.

Figure 2. Associations between BC and 8-OHdG, free MDA, and total MDA in obese and non-obese participants

Percent increases (mean, 95% confidence interval) in 8-OHdG (panel A), free MDA (panel B), and total MDA (panel C) are shown for moving averages of BC starting the day before urine collection through days 1 through 8 and for the integrated filter sample per interquartile range ($0.22 \mu\text{g}/\text{m}^3$) of BC.

Figure 3. Associations between BC and 8-OHdG, free MDA, and total MDA unadjusted and adjusted for NO₂ and PM_{2.5} not associated with BC

Comparison of four models displaying percent increases (mean, 95% confidence interval) per interquartile range (0.22 µg/m³) of BC for 8-OHdG (panel A), free MDA (panel B), and total MDA (panel C). Unadjusted and adjusted models yielded similar effects for each biomarker.

Table 1

Participant Characteristics at Study Entry, n = 82 men

Variable	Mean (SD)	Range
Age (yrs)	72.7 (8.4)	46.7 – 90
Education (yrs)	13 (2)	8 – 20
BMI (kg/m ²)	29.7 (5.6)	19.5 – 50.8
Smoking history (pack-yrs)	63 (41)	10 – 212
Post bronchodilator percent-predicted FEV ₁	67.1 (20.6)	17.2 – 105.7
Post bronchodilator percent-predicted FVC	88.4 (19.5)	53.2 – 148.4
Weekday time inside at home (hrs)	17 (4)	6 – 24
Weekend time inside at home (hrs)	17 (5)	2 – 24
	<i>n</i>	%
Asthma	17	20.7
Diabetes	20	24.4
Heart disease	41	50.0
Obese (BMI ≥ 30)	39	47.6
Statin use	64	78.1
Non-steroidal anti-inflammatory drug (NSAIDs) use	62	75.6
GOLD Spirometric Severity		
Preserved Ratio Impaired Spirometry (PRISm)	1	1.2
0	6	7.3
1	19	23.2
2	42	51.2
3	9	11.0
4	5	6.1
Race		
White	73	89
Non-White *	9	11
Urinary Biomarkers (n = 237)	Median (IQR)	Range
8-OHdG (ng/mL)	4.3 (2.6 – 7.1)	0.5 – 28.1
Free MDA (μM)	1.0 (0.7 – 1.6)	0.2 – 7.3
Total MDA (μM)	12.6 (8.9 – 19.7)	1.2 – 275.6
Creatinine (mg/dL)	112.9 (72.6 – 157.4)	10.1 – 626.8

* Includes 8 Black/African American participants.

Table 2

Environmental Sample Characteristics (n = 237)

Variable	Home
Duration of sampling (days)	
Mean (SD)	7.6 (0.7)
Range	4 – 10
Season	
Winter, n (%)	48 (20.3%)
Spring, n (%)	52 (21.9%)
Summer, n (%)	71 (29.9%)
Fall, n (%)	66 (27.9%)
Central site relative humidity (%)	
Median (IQR)	64.0 (58.3 – 70.3)
Range	41.2 – 80.5
Indoor black carbon, $\mu\text{g}/\text{m}^3$	
Median (IQR)	0.18 (0.10 – 0.31)
Range	–0.42 – 1.39
Central site black carbon, $\mu\text{g}/\text{m}^3$	
Median (IQR)	0.54 (0.42 – 0.76)
Range	0.27 – 1.20
Temperature at the home ($^{\circ}\text{C}$)	
Median (IQR)	13.5 (5.0 – 20.0)
Range	–8.0 – 25.7