Published in final edited form as:
Occup Environ Med. 2012 June ; 69(6): 422–427. doi:10.1136/oemed-2011-100268.

Association between long-term exposure to traffic particles and blood pressure in the Veterans Administration Normative Aging Study

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Abstract

Objectives—Particulate air pollution is associated with cardiovascular events, but the mechanisms are not fully understood. The main objective was to assess the relationship between long-term exposure to traffic-related air pollution and blood pressure (BP).

Methods—The authors used longitudinal data from 853 elderly men participating in the Veterans Administration Normative Aging Study, followed during 1996–2008. Long-term average exposures to traffic particles were created from daily predictions of black carbon (BC) exposure at the geocoded address of each subject, using a validated spatiotemporal model based on ambient monitoring at 82 Boston-area locations. The authors examined the association of these exposures with BP using a mixed model. The authors included the following covariates: age, body mass index, smoking, alcohol, fasting glucose, creatinine clearance, use of cardiovascular medication, education, census-level poverty, day of week and season of clinical visit.

Results—The authors found significant positive associations between 1-year average BC exposure and both systolic and diastolic blood pressure. An IQR increase in 1-year average BC exposure (0.32 $\mu g/m^3$) was associated with a 2.64 mm Hg increase in systolic blood pressure (95% CI 1.47 to 3.80) and a 2.41 mm Hg increase in diastolic blood pressure (95% CI 1.77 to 3.05).

Conclusions—Long-term exposure to traffic particles is associated with increased BP, which may explain part of the association with myocardial infarctions and cardiovascular deaths reported in cohort studies.

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An additional figure is published online only. To view this file please visit the journal online (http://oem.bmj.com/content/69/6.toc).

Competing interests None.

Ethics approval Human Subjects Committee.

Provenance and peer review Not commissioned; externally peer reviewed.

INTRODUCTION

Both short-term and long-term exposure to particulate air pollution has been associated with cardiovascular morbidity and mortality in numerous epidemiological studies.[1–6] The effect sizes of long-term exposure are substantially larger than those of short-term exposure, suggesting differences in the mechanisms may at play or differences in how the mechanisms are impacted by longer-term exposures. A number of pathways have been proposed to explain these associations, including, at the molecular level, increased oxidative stress,[78] systemic inflammation[910] and thrombotic potential.[11] At the functional level, potential pathways include changes in autonomic function, which may result in changes in blood pressure (BP).[12]

Elevated BP is an established risk factor for coronary heart disease and stroke and an important intermediate marker of cardiovascular health. The relationship between air pollution exposure and BP is still not well understood. Studies of short-term PM exposure and BP show mixed results, with some studies showing an inverse association or no association[13–15] and positive findings in other studies.[16–20] A key to understanding the mixed results in the observed health effects of PM is that PM is a complex mixture and the concentrations of its individual components vary regionally and seasonally.

Growing evidence suggests that traffic-related components of PM pollution contribute significantly to particle-related cardiovascular effects. For example, a recent chamber study examining the mechanisms of short-term effects of $PM_{2.5}$ on BP found that effects were much stronger for the samples collected from a high-traffic area.[21] A study of BP and short-term exposure to a number of air pollutants found the strongest association with organic carbon and its estimated fossil–fuel combustion fraction. [22] More research is needed to examine the relationship between traffic-related components of PM and BP, which will also help us understand the overall relationship between BP and PM.

Less is known about the relationship between long-term exposures to air pollution and BP, although mortality studies have found strong associations with long-term air pollution exposures.[2324] In particular, only one recent study has investigated the relationship between long-term average air pollution exposures and BP. This study in Taiwan found a strong association between BP and 1-year averages of $PM_{2.5}$.[25] Since traffic components of PM have been implicated as a key component in relation to cardiovascular disease, research is needed to address long-term exposure to traffic-related air pollution and BP.

We sought to address these research gaps by examining the relationship between BP and 1-year average exposures to traffic-related air pollution in a cohort study within the greater Boston area. An important tool for studying within-city variation in air pollution is the development of geographic-based exposure models. Black carbon (BC) is a traffic-related particle and a common surrogate for traffic particles in general, weighted towards diesel particles. We have developed and applied a land-use regression model for traffic particles based on BC in the greater Boston metropolitan area.[2627]

We hypothesised that estimated 1-year average BC at participants' addresses would be associated with elevated BP. We examined this in a longitudinal study in a closed cohort of elderly men in the greater Boston area with repeated measurements of BP taken roughly every 4 years.

MATERIALS AND METHODS

Study population

Our study participants were from the Veterans Administration Normative Aging Study (NAS), a longitudinal study established by the Veterans Administration in 1963.[28] The NAS is a closed cohort of male volunteers from the Greater Boston area aged 21–80 years at entry, who enrolled after an initial health screening determined that they were free of known chronic medical conditions. Participants were re-evaluated every 3–5 years using detailed on-site physical examinations and questionnaires. Air pollution data were collected from 1995 onward, so 1-year average BC concentrations were available starting in 1996. This analysis restricted the study population to subjects who were still participating in clinic visits after 1 January 1996, and subjects were followed through December 2008. Our analysis included 853 participants with complete information regarding BC concentrations and all covariates. These participants presented for a total of 2136 examinations during the study period. At each study visit, systolic blood pressure (SBP) and diastolic blood pressure (DBP) were measured once in each arm while the subject was seated, using a standard cuff, and the mean of right and left arm values was used in these analyses.

BC exposure estimation

BC exposures were estimated from a spatiotemporal model that we developed and validated previously, which has been described in detail previously.[27] Daily concentrations at the Boston central-site monitor were used as a predictor to reflect average concentration levels for a given day, serving as a direct estimate of the daily time effect. Data from 82 other stationary air monitors were used to fit the model and estimate the effect of each covariate in the land-use regression model. Covariates in the BC prediction model included measures of land use for each address (cumulative traffic density within 100 m, population density, distance to nearest major roadway and per cent urbanisation), geographic information system, location (latitude and longitude), daily meteorological factors (apparent temperature, wind speed and height of the planetary boundary layer) and temporal factors (day of week and day of season).

Separate models were fit for the warm and cold season. Interaction terms between the temporal meteorological predictors and land-use variables allowed for space–time interactions. Regression splines allowed main effect terms to non-linearly predict exposure levels, and thin-plate splines modelled the residual spatial variability additional spatial variability unaccounted for by the spatial predictors. A latent variable framework was used to integrate BC and EC exposure data, where BC and EC measurements were treated as surrogates of some true unobservable traffic exposure variable, see Gryparis *et al*[27] for further details.

Our BC model showed more than a threefold range of variation in long-term average exposure across the measuring sites, and the adjusted R^2 for this model was 0.83. A subsequent validation sample using monthly monitoring data collected at 30 additional locations found an average correlation of 0.59 between predicted values and observed BC levels.

All addresses of participants in the NAS have been geocoded and we used the model to generate daily predicted BC at the address of each participant. Daily BC predictions outside of the observed range of the monitored exposure measurements were excluded. Long-term average exposures were created by averaging the daily estimates at the participant's residential address or addresses for the 365 days before each clinical visit.

Statistical methods

We analysed associations between 1-year average BC exposure and BP using linear mixed effects models with a random intercept for each subject. We evaluated SBP and DBP as dependent variables. The models took the general form:[29]

$$Y_{it} = \beta_0 + u_i + \beta_1 X_{1it} + \cdots + \beta_k X_{kit} + \beta_{BC} BC_{it} + \varepsilon_{it},$$

where Y_{it} is the level of SBP or DBP in subject i at visit t; covariates for subject i at visit t are denoted by X_{1it} to X_{kit}. BC_{it} is the 1-year average BC concentration for subject i during the 365 days before visit t. Here, u_i represents a subject-specific intercept, reflecting unexplained heterogeneity in subjects' overall level of outcome. We assume that the u_i are generated from a normal distribution with common variance, yielding the compound symmetry variance structure. This model requires estimation of two variance components, which represent between- and within-subject variation. Models with unbalanced data (ie, varying numbers of repeated measurements on each subject) typically yield accurate estimates of within-subject variation, provided a sufficient number of repeated measurements contribute to the estimate.

To examine effect modification by a subgroup, we used an interaction term to fit separate pollution slopes for each subgroup and also controlled for group main effects. We tested for interactions with diabetes status, obesity and medication use.

The following covariates were chosen a priori based on established relationships with BP and air pollution and included in the regression models regardless of statistical significance: age, body mass index, cigarette smoking (never, current or former), pack years of smoking, alcohol intake (<2 drinks per day vs 2+ drinks per day), fasting glucose, use of antihypertensive medications (ACE inhibitors, β blockers, calcium channel blockers, angiotensin receptor blockers and diuretics), α blockers, creatinine clearance, weekday of clinical visit and season of clinical visit. In addition, to control for socioeconomic status (SES) at the individual level and the neighbourhood level, we included years of education for each subject and neighbourhood poverty level for each address as measured by per cent below poverty level of each census block group in the 1999 census. We considered additional potential confounders (short-term apparent temperature, median income level and per cent of population over 25 years of age without a high school diploma as measured by the 1999 census) and included them if effect estimates changed by more than 5% to ensure that all measured relevant confounders were included.

RESULTS

The characteristics of the population are shown in table 1. Subjects were older people, with a mean age of 70 at the first study visit. Less than 10% of our participants were current smokers, but more than half were former smokers. At the first study visit, participants had a mean body mass index of 28 kg/m^2. Average SBP and DBP at this visit were 137 mm Hg and 82 mm Hg, respectively. Fifty-three per cent of participants were initially antihypertensive medications users and that usage increased over the follow-up period. One-year average BC concentrations at participant addresses were 0.51 μg/m^3, and the IQR was 0.32 μg/m^3. Of the 853 subjects, 605 subjects (71%) were followed for two or more clinical study visits, with 29% of subjects having only one visit. Subjects who participated in only one study visit were 5 years older on average than those who were followed for two or more study visits, but there were no differences observed in other covariates. By the end of follow-up in 2008, 403 of the 853 subjects (43%) were deceased, 32 subjects (4%) dropped out and 23 subjects (3%) became too ill to participate.

We evaluated the association of SBP and DBP with geocoded long-term BC concentrations and expressed the results as the change associated with a 0.32 μg/m^3 increase in BC exposure, which was the IQR for this study (table 2). We observed significant associations between 1-year average BC exposure and both SBP and DBP, adjusting for confounders. In our evaluation of additional potential confounders, we found that the other neighbourhood SES measures and the measures of short-term apparent temperature had a negligible effect on the estimate of BC exposure and did not meet our criteria of at least a 5% change (data not shown). Table 2 also reports the association for the crude model, which includes only age and 1-year average BC exposure as covariates for comparison. The estimated effects are similar and estimated to be slightly higher than in the adjusted model, also with p values <0.001.

Because short-term exposure to air pollution has been associated with changes in BP in other studies, we wanted to ensure that our results were not driven solely by a relationship with short-term air pollution exposures. In figure 1, we compare the effects of average BC exposure over a range of exposure times from 24 h to 1 year. We see an upward trend across each time window of average BC exposure. This provides strong evidence that the effects of traffic particles on BP are not limited only to short-term effects, and long-term effects warrant further scientific investigation.

We examined the correlations between the short-term and long-term exposures and found that the 1-year average predicted BC exposure for each address was only moderately correlated with the predicted exposure 24 h before the study visit (r=0.53) and the predicted exposure 1 week before study visit (r=0.60). When adjusting for 24 h BC exposure, we observed a slight increase in the estimated effect of 1-yrea BC exposure on SBP, 3.22 mm Hg (95% CI 1.93 to 4.52), and on DBP, 2.63 mm Hg (95% CI 1.92 to 3.34). Adjusting for 1-week average BC exposure did not noticeably change the estimated effect of 1-year BC exposure on SBP nor on DBP (data not shown). Overall, the trend in figure 1 and our examination of the short-term correlations show that 1-year averages BC exposures have an effect that is not explained by shorter-term exposures.

We also assessed the linearity of the effect of long-term BC on BP by fitting a penalised spline for the 1-year BC exposures. For SBP, the effect was estimated to be linear with 1 df, and for DBP, the effect was estimated to have 4 df, although the overall trend is still roughly linear with no evidence of a threshold (supplementary figure 1).

Of the 853 subjects in the analysis, 30 subjects (3.5%) indicated having a seasonal residence and 63 subjects (7.4%) indicated a change in their primary address during the year prior a clinic visit. Of the subjects who moved, the mean number of days lived at the new address was 144 days. As a sensitivity analysis, excluding the subjects who had a seasonal residence or who had moved during the exposure period did not noticeably change the estimated effect of 1-year BC exposure on SBP, 2.68 mm Hg (95% CI 1.47 to 3.88), nor on DBP, 2.43 mm Hg (95% CI 1.77 to 3.09).

We examined interactions with diabetes, obesity and use of antihypertensive medications and considered an interaction significant if the p value was <0.05. These results are in table 3. Most of these interactions were not significant; the only significant interaction was for DBP with use of antihypertensive medications, where those taking antihypertensive medications were estimated to have a greater effect of BC exposure on BP. The interaction between antihypertensive medications and SBP was suggestive (p value=0.085). In addition, as a sensitivity analysis, we examined this interaction among the subgroup of participants who already had hypertension prior to the first visit.

The results were similar but not as strong, with a p=0.05 for DBP, while the interaction with SBP was not significant or suggestive, which may reflect reduced power from looking at only a subset of participants. Overall, these results are only suggestive and exploratory since our ability to examine the effect of medication usage in this observational epidemiological study is extremely limited and cannot be considered causal.

DISCUSSION

We found that 1-year average BC concentration estimated at each participant's home was positively associated with SBP and DBP in a cohort of elderly men. This association was quite strong for an epidemiological study of its kind, with p values all <0.001 for adjusted and crude models. Our interactions with obesity and diabetes were not significant. The interactions with antihypertensive medication use were suggestive, with a statistically significant association for DBP only.

Comparison to the literature

The cardiovascular effects of long-term exposure to particulate air pollution are largely unknown. A recent study in Taiwan of 1-year averages of several air pollutants and cardiovascular outcomes found a strong association between BP and 1-year averages of $PM_{2.5}$.[25] The 1-year average $PM_{2.5}$ exposures in that study were quite high, with mean 35.3 $\mu g/m^3$ (SD 15.9 $\mu g/m^3$). In the USA, the Environmental Protection Agency criteria level for annual average exposure to $PM_{2.5}$ is 15 $\mu g/m^3$, which is substantially lower than many of the levels measured in the Taiwan study. A rough rescaling of the effect in the Chuang study is a 3.1 mm Hg change in DBP per 2 $\mu g/m^3$ increase in $PM_{2.5}$. Since BC represents a variable fraction of $PM_{2.5}$, we cannot directly compare effect sizes, but effects appear to be of similar magnitudes relative to small changes in $PM_{2.5}$. Taken together, our results show no evidence of a thresholding effect in the relationship between BP and BC, the traffic component of $PM_{2.5}$, at these lower exposure levels observed in our study. In addition, our results suggest that traffic components of $PM_{2.5}$ may explain a large part of the association between BP and $PM_{2.5}$.

The literature on short-term to medium-term PM exposure and BP is mixed.[13–20] This may be due to differences in the composition of the particles across study sites. In particular, if the association is really with traffic particles, differing associations between $PM_{2.5}$ and BC across locations and over time within locations could obscure the true relation. For example, among recent studies examining the short-term and medium-term effects of $PM_{2.5}$ on BP, investigators found some evidence that $PM_{2.5}$ in higher traffic areas had stronger effects on BP compared with $PM_{2.5}$ in lower traffic areas.[16,21] More research is needed to examine the relationship between BP and traffic-related components of PM to understand its role in the overall relationship between BP and PM.

Exposure strengths and weaknesses

An important issue in the relationship between air pollution and cardiovascular disease is how short-term and long-term effects fit together. Our results show that 1-year average BC exposure is associated with increases in BP and that association has a larger effect size than short-term associations and cannot be explained by short-term effects. In a previous study of long-term air pollution exposure, lag time and mortality, we found the strongest association was with the most recent year's exposure in the Harvard Six City cohort.[30] Another recent mortality and air pollution study of the Nurses Health cohort showed little additional explanatory power for $PM_{2.5}$ exposures in the past 2 years.[23] Further research is needed to understand how exposures longer than 1 year affect BP and whether there is a point of attenuation in these effects.

Laden and coworkers[31] examined the association between particles from different sources and mortality and reported that the strongest effects (per micrograms per cubic metre of exposure) were for traffic particles. Moreover, these particles seemed to be particularly associated with cardiovascular deaths. This is consistent with our finding that traffic particles (in our case BC) are associated with increased BP. Traffic-related particles have also been associated with elevated homocysteine concentrations, [32] increased intercellular cell adhesion molecules (ICAM) and vascular cell adhesion molecules (VCAM),[33] decreased flow-mediated dilatation of the brachial artery,[34] reduced parasympathetic tone[35] and with acute effects on BP.[2236]

Another related study, by Hoffmann and coworkers,[37] reported an association between distance to roadway measures and ankle–brachial index as well as an increased risk for peripheral arterial disease among those living within 50 m of a major road compared with living >200 m away in a cohort in Germany. The study also examined long-term $PM_{2.5}$ exposure (average estimated 1-year $PM_{2.5}$ concentration at the home address) and found that it was not related to ankle–brachial index or peripheral arterial disease in that cohort nor was it correlated with distance to roadway in that cohort. The findings of the Hoffmann study support the hypothesis that traffic particles are a key component in the toxicity of $PM_{2.5}$ and may be the more relevant exposure with respect to cardiovascular health.

Mechanisms and interactions

BC may elevate BP by increasing oxidative stress, inducing endothelial dysfunction and promoting inflammatory activity. These pathways may interact. For example, oxidative stress may promote inflammatory activity, and systemic oxidative stress and inflammation may induce endothelial dysfunction by reducing levels of nitric oxide, a vasodilator important in maintaining vascular tone. Other mechanisms by which BC may elevate BP include activation of the sympathetic nervous system, alterations in blood coagulability and direct vasoconstriction.

It is important to note that research regarding mechanisms mediating BC's hypertensive effect is quite limited. Plasma markers of systemic oxidative stress and inflammation have been associated with BC exposure in epidemiologic studies. However, much of our discussion regarding potential mechanisms of BC cardiotoxicity is necessarily based on particulate matter research in general.

Previous studies have shown that diabetes status,[934] and sometimes obesity,[938] modify the effects of particles on cardiovascular outcomes. Our evaluation of whether the associations we found were modified by these factors was inconclusive. There was no evidence to support a differential effect for these subpopulations in this study. This could be because our population is so older people that there is too much comorbidity to examine these subgroups individually. Alternatively, the mechanisms at play in the short-term effects of air pollution may differ from the mechanisms involved in long-term effects.

Our finding that antihypertensive medications may interact with this effect of BC is suggestive but considered exploratory. In our sensitivity analysis restricting to those with hypertension at baseline, the interaction was not as strong, which may be due to lack of power from examining a smaller subgroup. With an observational study with participants often taking multiple medications, we cannot effectively evaluate the differential effects of any of these medications. In addition, the inclusion of antihypertensive medication use as a covariate is problematic because hypertension and subsequent medication use is a potential consequence of exposure and an important predictor of BP, so there is a potential for bias by including or not including this covariate.

Another important consideration of the findings is the clinical relevance of an increase of 2 mm Hg in DBP or SBP. While levels of 2 mm Hg may seem relatively small, some regions have higher levels of combustion particles and may see greater effects. Although the IQR of 1-year average BC was only 0.32 $\mu g/m^3$ in our cohort, the overall distribution was skewed and ranged from 0.02 to 1.90 $\mu g/m^3$. An extreme example would be the levels observed in Taiwan in the Chuang 2011 study, where the range of annual $PM_{2.5}$ exposures was 8.8–82.3 $\mu g/m^3$. A meta-analysis of BP to cause-specific mortality found that a 10 mm Hg decrease in DBP was associated with a notable decreased risk of ischaemic heart disease of 0.62 (95% CI 0.60 to 0.64) for those ages 70–79. Thus, the clinical relevance of a 2 mm Hg increase in DBP per 0.32 $\mu g/m^3$ increase in 1-year average BC depends on the regional exposure levels and the relationship between BP and cardiovascular disease.

Limitations and generalisability

Our estimates of long-term exposure are model based, rather than based on measurements. Our model is relatively rich and was validated on a large number of sampling sites, but prediction error is always a concern as measurement error in spatial models can sometimes lead to bias and/or CIs that are too narrow.[39] However, we note that this is a limitation in any air pollution study using stationary monitors or modelled exposures because the true exposure of interest is not measured exactly.

Although we did control for SES at the individual level and at the neighbourhood level, it is possible that the number of years of education does not provide strong enough control of individual-level SES; hence, there may still be some residual confounding by SES. In addition, there is potential for confounding by other exposures such as environmental tobacco smoke, as we only measured personal smoking and not whether there was another smoker in the home. Another possible unmeasured confounder is road noise, which also comes from heavy traffic and has been associated with cardiovascular outcomes. Road noise is particularly difficult to address since traffic pollution and road noise come from the same source. Overall, we have included most key confounders in our model so we do not expect that unmeasured confounding would have a strong influence on our results.

Our study population was homogeneous, consisting entirely of elderly men, 97% of whom were Caucasian. Thus, these results cannot be generalised to other populations without further research on how effects vary by age, gender and race. Additionally, the study population is a self-selected group of people who continued to participate in an ongoing study for many years and may not be representative of all elderly men in the USA. In particular, there may be survivor bias if the subjects who continue to participate are healthier than other older people, which would bias effect estimates towards the null, so the true effect in the general population may be stronger.

Conclusions

Long-term exposure to BC is associated with increases in BP in this older population, a finding that could explain part of the association of particulate air pollution with cardiovascular mortality. More research is needed to address the relation between traffic-related air pollution exposures and BP among diverse study populations, including women, other races and younger populations. Further research is also needed to study the role diabetes, obesity and antihypertensive medication use in modifying the effect and to clarify other mechanisms underlying the association between BC and BP.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

Funding This work was supported by the National Institute of Environmental Health Sciences grants ES015172-01 and ES00002 and the US Environmental Protection Agency grants RD-832416-01 and RD-834798-01. The Normative Ageing Study is supported by the Cooperative Studies Program/Epidemiology Research and Information Center of the US Department of Veterans Affairs and is a component of the Massachusetts Veterans Epidemiology Research and Information Center (MAVERIC), Boston, MA.

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What is known about this subject

Short-term air pollution exposures are associated with adverse cardiovascular effects, but studying the effects of longer-term exposures requires more complex exposure modelling.

What this paper adds

Long-term exposure to traffic-related air pollution is associated with increases in blood pressure, a finding that could explain part of the association of particulate air pollution with cardiovascular mortality.

Policy implications

This work impacts regulatory decisions about the level of traffic-related air pollution that affects cardiovascular health.

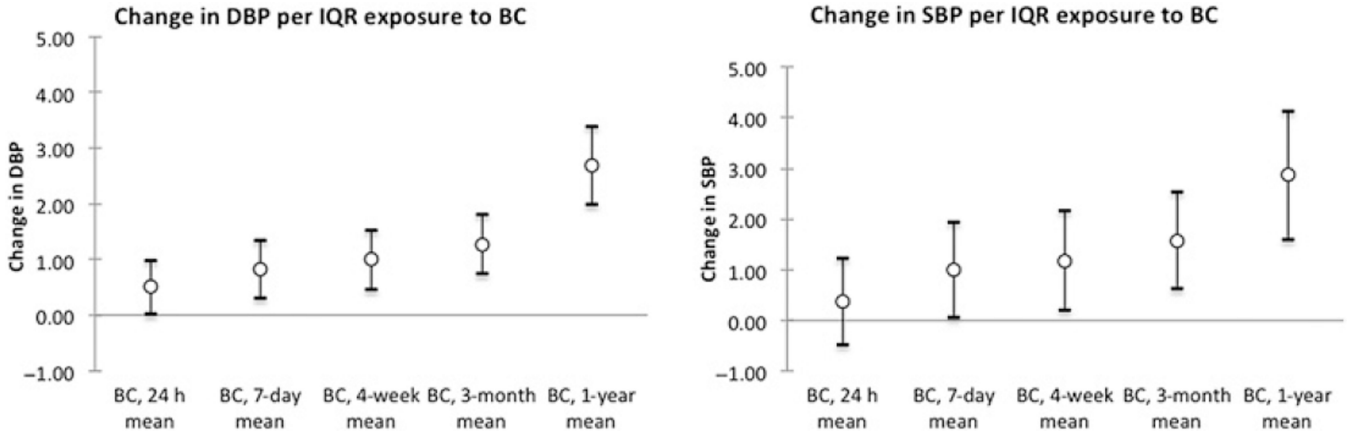

Figure 1.
Estimated change in blood pressure associated with an IQR increase in black carbon (BC) exposure. DBP, Diastolic blood pressure; SBP, systolic blood pressure.

Table 1

Descriptive statistics at first study centre visit (n=853) and over all visits (N=2136) given as mean (SD) or number (%)

Study variables	First visit (n=853)	All visits (N=2136)
Continuous variables	**Mean (SD)**	**Mean (SD)**
Age(years)	70.1(7.5)	72.6(7.4)
Body mass index (kg/m^2)	27.9(3.9)	28.0(4.2)
Lifetime smoking (pack-years)	31.8(29.4)	29.8(27.1)
Systolic blood pressure	136(17.9)	131(18.4)
Diastolic blood pressure	82(9.1)	77(10.9)
Fasting plasma glucose (mg/dl)	109.6(32.8)	108.3(28.1)
Creatinine clearance (mg/dl)	1.04(0.23)	1.09(0.29)
Years of education (individual)	14.4(2.7)	14.6(2.8)
Per cent below poverty level (census tract, 1999)	5.89(5.19)	5.72(4.96)
Black carbon 1-year average (μg/m^3)	0.61(0.29)	0.51(0.26)
Study variables	**First visit (n=853)**	**All visits (N=2136)**
Categorical variables	**N (%)**	**N (%)**
Smoking status		
Never-smoker	235(28%)	606(28%)
Former smoker	572(67%)	1444(68%)
Current smoker	46(5%)	86(4%)
Alcohol intake (2 + drinks per day)	178(21%)	408(19%)
Obese	222(26%)	559(26%)
Diabetes	94(11%)	280(13%)
Antihypertensive medication		
Any	407(48%)	1215(57%)
Calcium channel blockers	153(18%)	355(17%)
ACE inhibitors	141(17%)	515(24%)
Angiotens in receptor agonists	14(2%)	88(4%)
β blockers	204(24%)	720(34%)
Diuretics	116(14%)	424(20%)
αblockers	72(8%)	259(12%)

Table 2

Estimated change in blood pressure associated with a IQR increase (0.32 μg/m^3) in 1-year average black carbon (BC) levels for 2136 clinical visits (n=853 subjects)

	Crude model† Effect (95% CI)	Adjusted model‡ Effect (95% CI)
Systolic blood pressure (mm Hg)	4.00(2.90 to 5.10) ***	2.64(1.47 to 3.80) ***
Diastolic blood pressure (mm Hg)	3.19(2.58 to 3.81) ***	2.41(1.77 to 3.05) ***

*** $p < 0.001$.

† Crude regression models included only age and 1-year average BC level.

‡ Adjusted regression models controlled for age, cigarette smoking, pack-years of smoking, season of clinical visit, weekday of clinical visit, body mass index, fasting glucose level, use of antihypertensive medications (ACE inhibitors, angiotensin receptor agonists, Calcium channel blockers, β blockers and diuretics), use of a blockers, years of education, per cent below poverty level in the census tract, creatinine clearance and daily alcohol intake.

Table 3

Modification of the effects of 1-year average black carbon exposure on blood pressure by obesity, diabetes and antihypertensive medication use

Outcome	Effect (95% CI) No diabetes (N=1856)	Effect (95% CI) Diabetes (N=280)
SBP mm Hg	2.63(1.42 to 3.84)	2.83(−0.29 to 5.95)
DBP mm Hg	2.34(1.68 to 3.00)	2.38(0.69 to 4.08)
Outcome	**Effect (95% CI) Not obese (N=1577)**	**Effect (95% CI) Obese (N=559)**
SBP mm Hg	2.76(1.43 to 4.09)	2.35(0.39 to 4.30)
DBP mm Hg	2.59(1.86 to 3.32)	1.96(0.89 to 3.03)
Outcome	**Effect (95% CI) Not taking antihypertensive medicines (N=921)**	**Effect (95% CI) Taking antihypertensive medicines (N=1215)**
SBP mm Hg	1.78 (0.18 to 3.39)	3.32 (1.88 to 4.76)
DBP mm Hg	1.73 (0.85 to 2.60)	2.96 (2.17 to 3.75)

N is the number of observations in each analysis.
DBP, Diastolic blood pressure; SBP, systolic blood pressure.