Long-Term Effects of Traffic Particles on Lung Function Decline in the Elderly

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Abstract

Rationale: Few studies have been performed on air pollution effects on lung function in the elderly, a vulnerable population with low reserve capacity, and even fewer have looked at changes in the rate of lung function decline.

Objectives: We evaluated the effect of long-term exposure to black carbon on levels and rates of decline in lung function in the elderly.

Methods: FVC and FEV₁ were measured one to six times during the period 1995–2011 in 858 men participating in the Normative Aging Study. Exposure to black carbon, a tracer of traffic emissions, was estimated by a spatiotemporal land use regression model. We investigated the effects of moving averages of black carbon of 1–5 years before the lung function measurement using linear mixed models.

Measurements and Main Results: A 0.5 $\mu\text{g}/\text{m}^3$ increase in long-term exposure to black carbon was associated with an additional rate of decline in FVC and FEV₁ of between 0.5% and 0.9% per year, respectively, depending on the averaging time. In addition, black carbon exposure before the baseline visit was associated with lower levels of both FVC and FEV₁, with effect estimates increasing up to 6–7% with a 5-year average exposure.

Conclusions: Our results support adverse effects of long-term exposure to traffic particles on lung function level and rate of decline in the elderly and suggest that functionally significant differences in health and risk of disability occur below the annual Environmental Protection Agency National Air Quality Standards.

Keywords: air pollution; black carbon; FEV₁

Short- and long-term effects of air pollutant exposures on lung function have been studied in children and adults (1–4). Although warranted (5), there are fewer studies in the elderly, a sensitive population, and even fewer looking at

changes in the rate of decline in lung function. By 2030, people older than 65 years will represent 19% of the US population (http://www.aoa.gov/aoaroot/aging_statistics/index.aspx). Changes in the lung structure and function during aging

alter remodeling phenomenon and respiratory mechanics, which makes the elderly more susceptible to environmental stressors, such as air pollutants. Lung function and accelerated lung function decline are known predictors of mortality

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At a Glance Commentary

Scientific Knowledge on the

Subject: Although there is compelling evidence that short- and long-term ambient air pollution exposure adversely affect lung function in children and young adults, altered respiratory mechanics in the elderly may put them at increased risk for experiencing toxicologic effects of air pollutants. Few studies have been performed in this increasing and vulnerable population with low reserve capacity, and even fewer have looked at changes in the rate of decline in lung function.

What This Study Adds to the

Field: We report robust results indicating lower lung function level and accelerated lung function decline in the elderly after long-term exposure to black carbon. Our findings suggest that levels of particles related to traffic, although within the Environmental Protection Agency annual air quality standards, may contribute to the significant differences in the rate of lung function decline.

risk (6), as is air pollution, especially particles. The mortality risk of particle exposures is greater in the elderly, and is much larger for chronic than for acute exposures. Hence, understanding how long-term air pollution exposure impacts lung function in the elderly is critical in determining whether this is a possible pathway for the mortality and morbidity associated with air pollution.

The use of fixed monitoring to assess exposures to air pollution is being replaced by the use of geocoded exposure to people's residences. These modeled exposures may be based on land use regression (7–9), dispersion models (10), kriging (11), and satellite remote sensing (12, 13). These allow the characterization of exposure contrasts within cities, offer reduced exposure error, and hence provide more precise and less biased estimates. These improvements are particularly important in the elderly, who no longer work and spend most of the time at home.

Adverse effects of short- to medium-term (4–28 days) exposure to black carbon

(BC), carbon monoxide, nitrogen dioxide, and particulate matter less than 2.5 μm ($\text{PM}_{2.5}$) on FVC and FEV_1 have been reported in the Normative Aging Study (14), a population of elderly subjects followed since the 1960s. BC is a traffic-related particulate pollutant and has been used as a surrogate of exposures to traffic-related particles in general, weighted toward diesel particles. We here examined the effect of long-term averaged BC on long-term levels and rates of decline in lung function in that same cohort.

Methods

More details on the study design, BC exposure, and statistical analysis can be found in the online supplement.

Study Population and Pulmonary Health

Our study included 858 elderly men living in the Boston area, enrolled in the Normative Aging Study cohort (15), who returned for examinations every 3–5 years between 1995 and 2011. During these examinations, height, weight, and medication use (sympathomimetic α and β , anticholinergics) were assessed. Pulmonary disorders confirmed by a physician (asthma, chronic bronchitis, emphysema) and smoking history were collected through the American Thoracic Society questionnaire. FVC and FEV_1 were assessed by spirometric tests performed following the American Thoracic Society guidelines. Methacholine challenge tests were also conducted, as previously reported (14). Participants provided written informed consent and the study was approved by the institutional review boards of all participating institutions.

BC Exposure

We have developed a spatiotemporal land use regression model for traffic particles based on BC in the greater Boston metropolitan area (9). This model for BC has been updated and revised to include data from 148 monitoring stations recording BC levels at some point between October 1995 and August 2011. Daily predictions at the geocoded address of each participant were used to create moving averages of 1–5 years before each examination. Other

pollutants were not examined, because we did not have land use regression models for them.

Statistical Analysis

We used separate linear mixed models for each lung function measurement:

$$\begin{aligned} \text{Log}(\text{FVC or FEV}_1)_{it} &= \beta_0 + u_i + \beta_1 \text{age_baseline}_i + \beta_2 \Delta \text{age}_{it} \\ &\quad + \beta_3 \text{black carbon}_{it} + \beta_4 \text{black carbon}_{it} \\ &\quad \times \Delta \text{age}_{it} + \beta_p Z_{pit} + \varepsilon_{it}, \quad (1) \end{aligned}$$

where u_i is the random intercept for participant i ; age_baseline is the participants age at the time of the first visit; BC is the average BC concentration at the home address of the participants for the preceding 365 days before the visit t , 730 days, and so forth; and Δage is a time trend term expressed in days since the baseline visit. Hence, in this model β_3 measures the cross-sectional effect of BC and β_4 measures the effect of BC on the rate of decline in lung function. Because BC exposures were centered on the mean (i.e., 0.5 $\mu\text{g}/\text{m}^3$), β_2 is the slope of lung function decline for participants with average exposure. Z represents the P covariates controlled for in the model.

All analyses were conducted controlling for a core set of covariates chosen based on biologic knowledge: race, height, weight, education level, smoking status, pack-years, and season. We then examined sensitivity to further control, specifically for asthma, emphysema, chronic bronchitis, methacholine responsiveness, and medication use (level 2); then for coronary heart disease, stroke, hypertension, and diabetes (level 3); and then for BC averaged over the previous week (level 4). We finally tested whether the BC–lung function decline association was stronger in participants older than 65 years versus less than or equal to 65 years, in participants with baseline FEV_1/FVC less than 0.7, and in ever versus never smokers.

Results

Of the 858 men, 24% had one visit, 23% had two visits, 18% had three visits, and 35% had four to six visits. Table 1 shows the characteristics of the participants at the first visit. At the first visit, 75% of the men were older than 68 years. Only 5% of the

Table 1. Characteristics of the 858 Men Participating in 2,410 Visits in the Context of the Normative Aging Study, 1995–2011

	Statistics
Participant characteristics at first visit	
Age, mean $\pm$ SD, yr	69.9 $\pm$ 7.2
Race, n (%)	
Black	18 (2.1)
White	840 (97.9)
Height, mean $\pm$ SD, cm	173.6 $\pm$ 6.6
Weight, mean $\pm$ SD, kg	84.6 $\pm$ 13.7
Education, n (%)	
<12 yr	41 (4.8)
12 yr	213 (24.8)
13–15 yr	265 (30.9)
>15 yr	339 (39.5)
Smoking status, n (%)	
Never	241 (28.1)
Current	46 (5.4)
Former	571 (66.5)
Pack-years,* mean $\pm$ SD	22.6 $\pm$ 28.6
Asthma, n (%)	50 (5.8)
Chronic bronchitis, n (%)	61 (7.1)
Emphysema, n (%)	29 (3.4)
Methacholine responsiveness, n (%)	87 (10.1)
Missing	138 (16.0)
Corticosteroids, n (%)	30 (3.5)
Sympathomimetic (α , β), n (%)	83 (9.7)
Anticholinergic, n (%)	11 (1.3)
Coronary heart disease, n (%)	237 (27.6)
Stroke, n (%)	46 (5.4)
Hypertension, n (%)	595 (69.3)
Diabetes, n (%)	88 (10.3)
FVC, mean $\pm$ SD, L	3.4 $\pm$ 0.7
FEV ₁ , mean $\pm$ SD, L	2.5 $\pm$ 0.6
Visit characteristics	
Season, n (%)	
Spring (March–May)	590 (24.5)
Summer (June–August)	637 (26.4)
Fall (September–November)	716 (29.7)
Winter (December–February)	467 (19.4)

*Among current or former smokers.

subjects had less than a high school education, and 70% had at least some college education. Only 5% were active smokers at the time of the first visit, although most of the participants were former smokers.

The 5th to 95th percentile ranges of the long-term BC exposure distribution were 0.9 or 0.8 $\mu\text{g}/\text{m}^3$ (vs. mean of 0.6), demonstrating the considerable variation in traffic particle exposure across the greater Boston area (Table 2).

Table 3 shows the results of the primary analysis. For each increment of 0.5 $\mu\text{g}/\text{m}^3$ in long-term BC exposure, we found an additional rate of decline in FVC of between 0.5% and 0.9% per year, depending on the averaging time, and an additional decline of FEV₁ of 0.5% per year, except for the 5-year average, where the decline was 0.8% per year. In addition, BC exposure before the baseline visit was associated with lower baseline levels of both FVC and FEV₁, with effect estimates increasing with averaging time. For the 5-year average before the baseline visit, FEV₁ was 6.7% (95% confidence interval [CI], 9.1–4.4%) lower for each increment of 0.5 $\mu\text{g}/\text{m}^3$. For FVC, the effect was -5.6% (95% CI, -7.5% to -3.6%). Figure 1 illustrates the expected lung function over time by exposure given this model.

Results were consistent when we further adjusted for asthma, emphysema, chronic bronchitis, methacholine responsiveness, medications, chronic heart disease, stroke, hypertension, and diabetes (Table 4). Spearman correlation between 1-year and 1-week BC exposure was 0.77. When we further adjusted for short-term exposure to BC, the cross-sectional effects of long-term exposure to BC were weaker, but the longitudinal effects were consistent. The effects of BC exposures were the same for participants younger and older than 65 years, and for persons with baseline FEV₁/FVC ratios above and below 0.7. When we compared the effect of BC on the rate of decline in lung function among never versus ever smokers, there was a suggestion of a higher effect on never smokers for FVC and a significant interaction for FEV₁. For a 0.5 $\mu\text{g}/\text{m}^3$ increase in the 5-year average BC exposure, the increased rate of FEV₁ decline was -1.5% per year in never smokers (95% CI, -2.2% to -0.8%) versus

Table 2. Black Carbon ($\mu\text{g}/\text{m}^3$) Exposure before Lung Function Assessment for 2,410 Visits Undergone by 858 Men Participating in the Normative Aging Study, 1995–2011

Exposure Window	Number of Observations	Statistics		
		Mean $\pm$ SD	5th, 95th Percentiles	Interquartile Range
1 wk	2,392	0.6 $\pm$ 0.4	0.2, 1.4	0.42
1 yr	2,410	0.7 $\pm$ 0.3	0.3, 1.2	0.39
2 yr	2,175	0.6 $\pm$ 0.3	0.3, 1.2	0.37
3 yr	1,943	0.6 $\pm$ 0.3	0.3, 1.1	0.37
4 yr	1,720	0.6 $\pm$ 0.3	0.3, 1.1	0.35
5 yr	1,508	0.6 $\pm$ 0.3	0.3, 1.1	0.34

Table 3. Longitudinal Association* between Chronic Black Carbon Exposure and Lung Function for 2,410 Visits Undergone by 858 Men Participating in the Normative Aging Study, 1995–2011

Outcome [†]	Number of Observations	Black Carbon Effect			
		Cross-Sectional		On Rate of Lung Function Decline	
		β (%)	95% CI	β (%)	95% CI
FVC					
1 yr	2,410	–1.0	–2.1 to 0.1	–0.9	–1.1 to –0.7
2 yr	2,175	–1.4	–2.8 to –0.01	–0.8	–1.0 to –0.5
3 yr	1,943	–3.1	–4.7 to –1.5	–0.7	–0.9 to –0.4
4 yr	1,720	–4.3	–6.1 to –2.5	–0.5	–0.8 to –0.2
5 yr	1,508	–5.6	–7.5 to –3.6	–0.7	–1.1 to –0.4
FEV ₁					
1 yr	2,410	–1.3	–2.6 to –0.1	–0.5	–0.7 to –0.3
2 yr	2,175	–1.9	–3.5 to –0.2	–0.5	–0.7 to –0.2
3 yr	1,943	–3.7	–5.6 to –1.7	–0.5	–0.8 to –0.2
4 yr	1,720	–5.2	–7.3 to –3.1	–0.5	–0.9 to –0.2
5 yr	1,508	–6.7	–9.1 to –4.4	–0.8	–1.2 to –0.4

Definition of abbreviation: CI = confidence interval.

Bold font indicates statistically significant results.

*Adjusted for race, ln(height), weight (linear and quadratic), education level, smoking status, pack-years, and season (sin[date], cos [date]).

[†]Times given in the “Outcome” column are exposure windows.

–0.5% per year in ever smokers (95% CI, –1.0% to 0.01%) with a significant interaction ($P = 0.02$). There were no significant differences in the baseline effect by smoking status.

Discussion

Our results show, for the first time, that exposure to traffic particles, as indexed by BC, is associated with lower baseline lung function and accelerated lung function decline in the elderly, a population sensitive to the effects of particles. These effects were the same for participants younger and older than 65 years at baseline, and remained stable with increasing level of control for other potential predictors of lung function.

In 1965, Holland and Reid (16) showed that after controlling for smoking, lung function in long-term postal workers in London was lower than in similar postal workers in the countryside, where BC levels were roughly half of those in London. Since that time considerable research has indicated that air pollution in general, and particles specifically, are associated with lower lung function. Lubinski and coworkers (17) reported lower FEV₁/FVC ratios in nonsmoking Polish men on military entrance examinations were associated with particle concentrations in their home cities. Fewer studies have examined changes in lung function over time and most of them have focused on

children. In Mexico City, reduced rates of lung function growth have been reported in children living in parts of the city with higher particle levels (18). The Southern California Children’s Health Study followed children older than 8 years and found that PM_{2.5} exposure was associated with clinically impaired lung function at 18 years of age (19). Avol and coworkers (20) identified 110 children from the University of Southern California Children’s Health Study who moved from the study area, and followed them in their new home with pulmonary function testing identical to that in the main cohort. Subjects who moved to locations with higher PM₁₀ concentrations had lower rates of annual growth in lung function, whereas children who moved to locations with lower PM₁₀ levels showed higher rates of growth in lung function. This effect was larger in subjects who lived in the new location for at least 3 years. Another study took advantage of a natural experiment—the collapse of communism. Sugiri and coworkers (21) examined lung function in children in East and West Germany repeatedly between 1991 and 2000. In 1991 the West German children had higher lung function, and much lower exposure to particles. By 2000, changes in East German environmental and socioeconomic conditions had eliminated the difference in particle exposure, and the difference in lung function also disappeared.

In our study focused on elderly males, we found 1.3–6.7% lower levels of baseline lung function associated with previous exposure to BC. For each 0.5 $\mu\text{g}/\text{m}^3$ increase in long-term (1–5 yr) BC exposure, we found an additional annual rate of decline of 0.5–0.9% for FVC and 0.5–0.8% for FEV₁. In SAPALDIA, Downs and coworkers (3) reported 9% lower annual rates of decline in FEV₁ associated with a 10 $\mu\text{g}/\text{m}^3$ decrease in the annual average PM₁₀ concentration between examinations. In ECRHS, Götschi and coworkers (22) found no significant effect of PM_{2.5} on FEV₁ level and decline. The authors acknowledge such results might be caused by methodologic issues. In women, Sekine and coworkers (23) compared annual mean change of FEV₁ for three exposure groups older than 8 years and found stronger

Figure 1. Rate of decline of FVC (and 95% confidence intervals [dotted lines]) comparing that seen at the mean black carbon (BC) in the study (0.5 $\mu\text{g}/\text{m}^3$) with what would have been expected in the counterfactual situation where BC was zero; 2,410 visits undergone by 858 men participating in the Normative Aging Study, 1995–2011.

Table 4. Sensitivity Analyses: Longitudinal Association between Chronic Black Carbon Exposure and Lung Function According to Confounders Adjusted for 2,410 Visits Undergone by 858 Men Participating in the Normative Aging Study, 1995–2011

Outcome*	Number of Observations	Adjustment Level											
		Level 2 Black Carbon Effect				Level 3 Black Carbon Effect				Level 4 Black Carbon Effect			
		Cross-Sectional		On Rate of Lung Function Decline		Cross-Sectional		On Rate of Lung Function Decline		Cross-Sectional		On Rate of Lung Function Decline	
		β (%)	95% CI	β (%)	95% CI	β (%)	95% CI	β (%)	95% CI	β (%)	95% CI	β (%)	95% CI
FVC													
1 yr	2,056	–0.9	–2.0 to 0.3	–1.0	–1.2 to –0.8	–0.8	–2.0 to 0.4	–1.0	–1.2 to –0.8	–0.2	–1.6 to 1.2	–1.0	–1.2 to –0.8
2 yr	1,859	–1.3	–2.7 to 0.2	–0.9	–1.1 to –0.6	–1.2	–2.7 to 0.2	–0.9	–1.1 to –0.6	–0.1	–1.8 to 1.6	–0.9	–1.2 to –0.7
3 yr	1,668	–2.8	–4.5 to –1.1	–0.7	–1.0 to –0.5	–2.7	–4.4 to –1.0	–0.8	–1.0 to –0.5	–0.9	–2.7 to 1.0	–0.8	–1.1 to –0.5
4 yr	1,471	–4.3	–6.1 to –2.5	–0.5	–0.9 to –0.2	–4.3	–6.1 to –2.5	–0.6	–0.9 to –0.3	–2.9	–4.9 to –0.8	–0.7	–1.0 to –0.4
5 yr	1,289	–5.7	–7.8 to –3.6	–0.8	–1.2 to –0.4	–5.8	–7.8 to –3.7	–0.8	–1.2 to –0.4	–4.6	–6.9 to –2.3	–0.9	–1.2 to –0.5
FEV ₁													
1 yr	2,056	–1.4	–2.7 to –0.1	–0.6	–0.8 to –0.4	–1.4	–2.7 to –0.1	–0.6	–0.9 to –0.4	–0.8	–2.3 to 0.8	–0.6	–0.9 to –0.4
2 yr	1,859	–2.0	–3.7 to –0.4	–0.6	–0.9 to –0.4	–2.0	–3.6 to –0.4	–0.6	–0.9 to –0.4	–1.0	–2.8 to 0.9	–0.7	–0.9 to –0.4
3 yr	1,668	–3.5	–5.4 to –1.6	–0.6	–0.9 to –0.3	–3.4	–5.3 to –1.5	–0.6	–0.9 to –0.3	–2.3	–4.4 to –0.1	–0.7	–1.0 to –0.3
4 yr	1,471	–5.2	–7.2 to –3.1	–0.6	–1.0 to –0.3	–5.3	–7.3 to –3.2	–0.6	–1.0 to –0.3	–4.2	–6.4 to –1.8	–0.7	–1.1 to –0.4
5 yr	1,289	–6.9	–9.2 to –4.6	–0.9	–1.2 to –0.5	–7.0	–9.3 to –4.7	–1.0	–1.4 to –0.6	–6.5	–9.0 to –3.9	–1.0	–1.4 to –0.6

Definition of abbreviation: CI = confidence interval.

Levels 2, 3, and 4 were adjusted for race, ln(height), weight (linear and quadratic), education level, smoking status, pack-years, season (sin[date], cos[date]).

Level 2 was further adjusted for chronic lung conditions (asthma, emphysema, chronic bronchitis), methacholine responsiveness, medication use (sympathomimetic α and β , anticholinergics).

Level 3 was adjusted for covariates of Level 2 and further adjusted for coronary heart disease, stroke, hypertension, and diabetes.

Level 4 was adjusted for covariates of Levels 2 and 3 and further adjusted for short-term exposure to black carbon.

Bold font indicates statistically significant results.

*Times given in the "Outcome" column are exposure windows.

FEV₁ decline in the group with the highest exposure to traffic. Comparison of results across studies is made difficult because of differences in exposure metrics and statistical methods used to study the relationship with lung function decline. In our study, the cross-sectional effects of BC were a bit stronger on FEV₁ compared with FVC, suggesting that BC exposure might lead to obstructive changes rather than restrictive. However, the longitudinal effects were slightly stronger on FVC compared with FEV₁. Further studies are needed to confirm whether traffic air pollutants affect lung function decline and to clarify whether such effect is more obstructive or restrictive.

We did not see any effect modification by FEV₁/FVC ratio, but we did see evidence of stronger effects in never smokers than in ever smokers, almost all of whom were former smokers. Former smokers often stop smoking because they become symptomatic, and it may be that the ability of air pollution to further damage already compromised lungs is limited, or that former smokers take action to limit their exposure. Further work is needed to first confirm this effect modification, and if confirmed, to identify the mechanism.

Hence, our main results are consistent with prior literature, but extend it to a key

susceptible population, the elderly, and to a key fraction of particulate air pollution, traffic particles. BC is a marker of harmful combustion particles (24). In Boston, BC is a marker of traffic particles associated with local traffic and long-range transported traffic particles. Epidemiologic studies are supported by toxicology. For example, one study exposed mice and their offspring to either ambient air or filtered air to remove only particles, not gases. Lung function in the particle-exposed mice (average particles concentrations of 16.8 $\mu\text{g}/\text{m}^3$) was lower than in the mice with filtered air (average particle concentrations of 2.9 $\mu\text{g}/\text{m}^3$) (25). The underlying mechanisms of the association between air pollution and lung function are not fully elucidated. Pulmonary inflammation has been shown after controlled exposure to diesel exhaust, rich in BC and nitrogen oxides (26). Oxidative stress is a key pathway for pulmonary diseases, and a continuous reduction in oxidative stress in the lung has been shown in animals first exposed to the Boston air and then to a filtered air (27). Aging involves complex biologic phenomena. Physiologic functions can be compromised, making the elderly more susceptible to inflammatory states and vulnerable to air pollution.

Particle clearance might be less efficient or impaired by other dysfunctions.

We acknowledge some limitations to our study. First, there is room for measurement error in BC exposure assessment, because we only accounted for home outdoor exposure as it is often the case in air pollution studies. By the time of our analysis, 75% of the men were older than 68 years, so essentially everyone was retired, making the home address a better location for assessing outdoor exposure than in a working population. Predictions from our land-use model have been merged to the geocoded address of each subject of the study and have been used to investigate the effect of traffic pollution on markers of inflammatory and endothelial response (28), blood pressure (29), cognitive function (30), and other outcomes. Because the elderly likely have reduced outdoor activities, indoor sources may contribute more to personal exposure than in other populations. Assuming that indoor and outdoor particles have the same composition and toxicity, Zeger and coworkers (31) have shown that errors caused by indoor exposures are of Berkson type, which is expected to increase standard errors. If indoor and outdoor particles are not

identical, then the effect would generally be underestimated.

Second, we cannot rule out residual or unmeasured confounding. However, we adjusted for a variety of potential predictors of lung function and our analyses indicated our results were robust. Furthermore, because we have seen associations of short-term exposure to traffic pollution with lung function (14), we controlled for BC averaged over the previous week (level 4) to ensure that we were really looking at an effect of long-term BC exposure. Cross-sectional effects of long-term exposure to BC were weaker when we adjusted for short-term exposure to BC, which means

that part of the short-term effects of BC was previously captured by the cross-sectional effect of long-term exposure. Third, study results were observed in a cohort of elderly men, which may limit the generalizability of our results to similar populations.

The magnitude of the difference in lung function decline in the elderly is not trivial, and points to increased levels of significant impairment caused by the substantially greater decline in lung function among elderly men with higher exposure to traffic particles. Such effects should not be underestimated because they might be well-tolerated by a healthy population, but

become life threatening for elderly or ill subjects (32). Moreover, particle pollution (PM_{2.5}) during the period of this study was always within the Environmental Protection Agency annual air quality standards. This indicates functionally significant differences in health and risk of disability occur within those standards. ■

Author disclosures are available with the text of this article at www.atsjournals.org.

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