

Elemental carbon exposure and lung function in schoolchildren from Mexico City

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Abstract

Background: Though exposure to air pollution has a detrimental effect on respiratory health, few studies have examined the association between elemental carbon exposure and lung function among schoolchildren.

Objective: To determine the association between short-term elemental carbon exposure and lung function in schoolchildren from Mexico City.

Materials and methods: Fifty-five asthmatic and 40 non-asthmatic children were followed for an average of 22 weeks. A spirometry test was performed every 15 days during follow-up. Portable air samplers collected particulate matter onto Teflon filters. Gravimetric analysis was conducted and elemental carbon was quantified using transmission densitometry. The association between the main variables was analyzed using linear mixed effects models.

Results: The mean of elemental carbon light absorption was 94.7 Mn^{-1} (standard deviation 55.7). An increase of one interquartile range in the 24-hour average of elemental carbon (100.9 Mn^{-1}) was associated with a significant negative impact on FEV₁ [-62.0ml (95%CI -123.3ml, -1.2ml)] and FEV₂₅₋₇₅ [-111ml (95%CI -228.3, -4.1ml)] among asthmatic children—equal to 3.3% and 5.5%, respectively; and on FEV₁ [-95.0ml (95%CI -182.3ml, -8.5ml)] and FVC [-105.0ml (95%CI -197.0ml, -13.7ml)] among non-asthmatic children.

Conclusion: Exposure to elemental carbon resulted in an important negative effect on lung function in atopic schoolchildren, regardless of asthma status.

Keywords: Air pollution, Asthma, Epidemiology, Elemental carbon, Lung function, Schoolchildren

Introduction

Vehicular traffic is a major source of air pollutants and studies from different parts of the world have documented the relationship between exposure to air pollution and poorer respiratory health among children [1-5]. Children living in southern California exposed to traffic-related pollution have been reported to have lower lung function, with negative associations reported between the forced expired volume in one second (FEV₁) and nitrogen dioxide (NO₂), acid vapor, elemental carbon (EC) and particles with a mean aerodynamic diameter of less than 2.5 μ (PM_{2.5}) [1]. Furthermore, Kulkarni et al examined alveolar macrophages in induced sputum from children living in Leicester, UK and found a negative association between lung function and the amount of carbon seen in macrophages [6].

Experimental evidence from animals and humans has suggested that diesel exhaust particles may have a greater negative impact on respiratory health than other air pollutants [7, 8]. The metropolitan Mexico City area is one of the biggest and most polluted urban areas in the world, with close to 85% of air pollutants coming from motor vehicles. One of the main pollutants consists of fine particles (PM_{2.5}), 20% of which come from industry, 32% from diesel and 25% from gasoline [9]. The toxicity of the particles is a subject of interest for epidemiological research [10], and one of the most likely toxic components of the particles are elemental and organic carbon fractions of PM_{2.5} originating mostly from incomplete combustion of fossil fuels.[10]. We previously reported the association of ambient PM_{2.5} concentrations with an increased inflammatory response and decreased lung function in Mexican school children [11, 12]. The present study was undertaken to expand on our previous studies involving the association between exposure to traffic-related pollution and poorer respiratory health in children. We evaluated the association between exposure to EC (as a marker for diesel exhaust particles) and

lower lung function in said children. We hypothesized that short-term exposures to traffic-related EC was related to decrements in lung function in this population.

Material and methods

Study design

The children in the present study were a subset of the larger cohort of schoolchildren in Mexico City previously evaluated [11, 12]. The present report includes data obtained from 55 asthmatic and 40 non-asthmatic schoolchildren who were enrolled during the first 10 months of the study and followed for an average of 22 weeks (Figure 1). These children attended 37 of the total of 107 schools represented in the larger study. Lung function was measured and respiratory health assessed every 15 days during the follow-up period.

Location and population

The overall study population consisted of schoolchildren living in three Mexico City municipalities: Iztapalapa, Iztacalco and Netzahualcoyotl. These regions are characterized by high levels of traffic-related emissions. The fifty-five asthmatic children included in the present analysis were recruited from clinics at the Hospital Infantil de Mexico Federico Gomez, one of the largest pediatric hospitals in the city. The diagnosis and severity of their asthma was based on clinical criteria and response to treatment; severity was rated by a pediatric allergist as mild (intermittent or persistent), moderate or severe, according to the Global Initiative for Asthma (GINA) guidelines [13]. Forty non-asthmatic children were recruited by asking the asthmatic children to invite a schoolmate or a friend from their neighborhood. The children in both groups were between 6 and 14 years old. They lived in the study area, attended public schools located close to their home, were volunteers and were not selected using probability-based sampling. All

procedures were explained to the parents, who signed an informed consent form. The children also gave their informed assent. The study protocol was reviewed and approved by ethics committees at both the National Institute of Public Health and the Hospital Infantil de Mexico.

Data collection procedures

A general purpose questionnaire (adapted from existing survey instruments) was used to collect data on sociodemographic variables, past health history and potential indoor environmental exposures (tobacco smoke and pets in the home). Allergy test results, medication used and medical visits over the past 2 years were obtained from medical records. A respiratory symptom questionnaire was applied and lung function was measured at baseline and every 15 days during follow-up.

Spirometry

A total of 186 measurements of lung function were performed among asthmatic children and 122 among non-asthmatic schoolchildren. Measurements were repeated an average of 3 times (range 1–9) per subject during the study period using an Easy One spirometer (ndd Medical Technologies, Andover, Mass., USA), according to American Thoracic Society standards. Spirometric methods and the validation process have been detailed in our previous reports [11].

Exposure assessment

Local monitoring of PM_{2.5} (particulate matter < 2.5 µm in aerodynamic diameter) was undertaken for continuous periods of 15 days (2 weeks) at different times in the 37 public schools attended by the sub-group of children (n= 95) participating in the present study (Figure 1). PM_{2.5} concentration was measured at each school 14 times (range: 1-26), on average, during the period

June 2003 to June 2005. In addition, measurements were conducted of elemental carbon fractions of PM_{2.5} in 207 Teflon filters from a subsample of 20 schools. EC concentrations were measured at each school 3 times (range, 1–9), on average, during this period. Said schools were chosen for monitoring based on their distance (24 - 800 m) to the closest roadways with high traffic density and all schools are close to the children's homes. Five of the schools were in Iztapalapa-West, 6 in Iztapalapa-East, 4 in Iztacalco and 5 in Nezahualcoyotl. Each child's home and school was geo-referenced using a geographic information system (GIS). Exposure was estimated from the 24-h average local outdoor EC fractions of PM_{2.5} concentration at the child's school and the closest monitoring school was assigned to the child. Meteorological (temperature and humidity) and PM_{2.5} concentration data were also obtained from the Mexico City government from four fixed-site central monitoring (RAMA) locations within the study area for the study period.

Mini-Vol portable air samplers (version 4.2; Airmetrics, Eugene, Oregon, USA) with flows of 5 liters/min using 47 mm Teflon membrane filters (R2PJ047; Pall Gelman, Ann Arbor, MI, USA) were used to monitor local daily 24-hour outdoor PM_{2.5} concentrations. Mini Vols were located on school rooftops (height 3 m above the ground); care was taken not to place monitors within 90 cm of walls and windows or close to plants or trees. Gravimetric analysis of the 47 mm Teflon filters was performed at the air laboratory of the National Center for Environmental Research and Training (CENICA) in Mexico City. EC concentrations for visible light absorption (b_{abs}) (as a marker for diesel "soot") was measured using transmission densitometry (Tobias Associates, Inc., Ivyland, PA) at the Desert Research Institute (DRI), Nevada USA, according to the procedures previously described by Watson [14]. The transmission densitometry method measures optical density with an incandescent broadband lamp (400-650 nm, peaking at 575 nm) transmitted through a glass diffuser. Transmittance is measured before and after Teflon filter exposure, to

determine particle b_{abs} and the difference in the logarithms of the transmitted light is proportional to the absorption of the particle deposit [15]. $\text{PM}_{2.5} b_{\text{abs}}$ have been found to be highly correlated with collocate EC analysis by the thermal/optical reflectance method [16] and provides reliable information on the variability of EC levels related to traffic exhaust and these results can be used as a marker for diesel exhaust particles [17] .

Statistical analysis

The basic characteristics of the two groups of children were compared by bivariate analysis using the t-test, the Fisher exact test or the χ^2 test, depending on variable type.

In order to analyze the variation in lung function due to short-term exposure to local outdoor EC fractions of $\text{PM}_{2.5}$ concentration, we used linear mixed effects models with random intercept (xtreg command in stata software), with a continuous response for longitudinal data. The models were run to evaluate exposure effects on the same day. That is, the association was calculated by selecting the day that pulmonary function was performed and assigning the measurement of local elemental carbon for the same day, considering average EC concentrations during 24 hours. This enabled us to appreciate the variability within and between subjects. We ran models with both random intercept and random slope, and with random intercept only. As the coefficients were similar in both types of models, we present the results from the linear mixed model with random intercept only, as previously reported [11].

Models were adjusted for potential confounding factors, including gender, body mass index, previous day minimum temperature, outside physical activity and chronological time; data were

stratified by characteristics of the subjects studied. Other variables were not significant ($p>0.10$) and did not alter the results by $>1\%$, such as age, socioeconomic index (considering mothers' education and school type), outdoor activities, exposure to environmental tobacco smoke, use of anti allergic medicine and season. Pearson correlations were determined between $PM_{2.5}$ concentrations at the fixed-site monitoring stations and local monitoring of $PM_{2.5}$, and with local outdoor EC fractions of $PM_{2.5}$ concentration. Analyses were conducted using Stata software version 9.2 (Stata Corp, College, Station, TX, USA) [18].

Results

Only 95 of the 208 schoolchildren participating in the larger cohort study were included in the present report. The characteristics of the study population and main outcomes (participants vs. non-participants) are presented in Table 1; we did not observe significant differences between groups with respect to the main variables. The mean age of participants was 9.7 years (standard deviation (SD 1.9) for the asthmatic and 9.3 (SD 2.0) for the non-asthmatic children. Fifty-six percent of the asthmatic children were classified as having mild intermittent, 23.6 % as having mild persistent and 20.0 % as having moderate persistent asthma. Eighty-one percent of the asthmatic children and 70% of the non-asthmatic children had positive skin prick tests. The most common sensitivities were to house dust mite (*D. pteronyssinus*), cat (Fel D1) and cockroach (*American blatella*) allergens. A total of 186 measurements of lung function were made among the asthmatic children and 122 among the non-asthmatic children. Measurements were repeated an average of 3 times (range 1-9) per subject during the study period. Lung function was similar for both groups (Table 1).

Environmental exposure data

The 24-hr average of PM_{2.5} at the schools was 25.5 µg/m³ (SD = 13.7), with a range of 3.1 to 96.1 µg/m³ during the study period. The 24-hr average of EC was 92.7 Mm-1 (SD = 54.7) with a range of 11.1 to 210.5. Local measurements of PM_{2.5} conducted at the children's schools were correlated with levels at the central monitoring stations (r= 0.77); the correlation between EC and local measurements of PM_{2.5} in the school was 0.47.

Association between lung function and EC exposure

The associations between traffic-related carbon exposure and lung function are shown in Table 2. After adjusting for gender, body mass index, previous day minimum temperature, outdoor physical activity and chronological time, an average increase of 100.93 in EC levels (equivalent to one interquartile range (IQR)) was associated with an important negative effect on FEV₁ [-62.0ml (95%CI -123.3ml, -1.2ml)], on FVC [-48.0ml (95%CI -124.4ml, 26.6ml)] and on FEV₂₅₋₇₅ [-111ml (95%CI -228.3, -4.1ml)] among asthmatic children—equal to 3.2%, 2.1% and 5.5%, respectively. In non-asthmatic children, we also observed an important negative effect on FEV₁ [-95.0ml (95%CI -182.3ml, -8.5ml)], on FVC [-105.0ml (95%CI -197.0ml, -13.7ml)] and on FEV₂₅₋₇₅ [-142.0ml (95%CI -347.9ml, 63.8ml)]—equal to 5.0%, 5.0% and 6.2%, respectively. When the analysis was stratified by atopy, the association was only significant for atopic children. In the model for atopic children, the magnitude of the association was -64.1ml (95%CI -123.4ml, -4.6ml) for FEV₁ and -138.6ml (95%CI -241.5ml, -35.6ml) for FEV₂₅₋₇₅—equal to 3.3% and 6.0%, respectively, for an interquartile range increase in EC concentration (Table 3).

Discussion

The results of the present study show that lung function in Mexican school children was inversely associated with exposure to elemental carbon, with the main effect related to EC exposure on the day lung function was measured. This is one of the first prospective studies to report an association between exposure to EC—assessed in the child’s immediate environment—and lung function decrements in schoolchildren with and without asthma. This effect was more significant in atopic children, regardless of their asthma status.

In the present study, while we observed a decrease in all lung function variables analyzed in both asthmatic and non-asthmatic children, the most robust findings were between increased EC and a reduction in FEV₁ and FEF₂₅₋₇₅. These inverse associations are consistent with those reported in other studies relating traffic exposures to lung function, and suggest a short-term effect of this pollutant. Long-term effects on lung function have also been observed. Gauderman et al measured ambient air quality in 12 communities in southern California, USA and compared lung function in children living in the most polluted communities with those in the less polluted areas [1]. They reported an inverse association between FEV₁ (ml) and EC [-87.9 ml (95% CI -146.4, -29.4 (p=0.007))] for an increase of 1.2 ug/m³ in EC, corresponding to a decrease in FEV₁ of approximately 4.4%. Kulkarni et al assessed the carbon content of alveolar macrophages obtained by inducing sputum production with hypertonic saline in healthy school children in Leicester, UK and reported inverse associations between carbon content and lung function expressed as percent of predicted values [6]. For each increase of 1µm² in carbon content they found a 12.9% decrease in FVC, a 17% decrease in FEV₁ and a 34.7% decrease in FEF₂₅₋₇₅. Similar results have been reported for adults [4]. In a prospective study including 272 women, Franco et al reported a 1.1%

decrease (95%CI -2.5%, 0.3%) in FEV₁, a 0.6% decrease (95%CI -1.9%, 0.6%) in FVC and a 3.0% decrease (95%CI -5.8%, -0.2%) in FEF₂₅₋₇₅ for an interquartile range increase (22 µg/m³) in black carbon exposure [19].

The association we report here between EC and lower lung function was seen in both asthmatic and non-asthmatic children. And though asthmatic children are generally considered to be at increased risk of the adverse consequences of environmental exposures, our study design does not allow for a true assessment of this premise. We recruited asthmatic children from the clinics of the Hospital Infantil de Mexico Federico Gomez. These children then “invited” a friend who attended the same school or lived in the same neighborhood to participate as controls. While these groups had similar demographic characteristics (Table 1), there were more girls in the control group and they had higher FEF₂₅₋₇₅. A larger study recruiting a community-based sample of asthmatic and non-asthmatic children will be required to determine whether asthmatic children are indeed more susceptible to exposure to traffic-related pollution.

An important finding from the present study was that the inverse association between EC and lung function was mostly seen in atopic children, regardless of their asthma status. Exposure to diesel exhaust particles is associated with an increased risk of allergic sensitization [7]. Thus, it is possible that chronic exposure to higher levels of elemental carbon—as an index of exposure to traffic-related pollution in general, and to diesel exhaust particles, in particular—increased the risk of allergic sensitization in these children and had a short-term effect on lung function as a result of acute exposure. While our study is not ideal for assessing this possibility, the rate of allergic sensitization in the non-asthmatic group (70.0%) was higher than expected. In addition,

in a prior analysis we observed a stronger effect of PM_{2.5}, measured at fixed central monitoring stations, and lung function decrement among a larger group of atopic children [11], lending more support to our current results. Again, specific studies will need to be conducted to examine the interrelationships between exposure to traffic-related pollution, allergic sensitization and lung function in children.

Exposure assessment among children is difficult and individual exposure is not well represented by data obtained from fixed central monitoring stations. Thus, an important strength of the present study is that we were able to measure EC in filters collected locally within the schools attended by the children, thereby providing a better exposure assessment than using central monitoring sites [20-22]. In addition, few studies have used real-time local measurements that enable assessing the effects of elemental carbon, as presented in this study.

Some limitations must be taken into account when interpreting the results of this study. The temporal variations in each child's exposure were assumed to follow the same tendencies on days with and without EC measurements. To strengthen the validity of this assumption, for the entire period of the study we observed high correlations ($r=0.77$) between measurements of PM_{2.5} from fixed central monitoring sites and those from local monitoring conducted at the schools. Also, the correlation between EC and local PM_{2.5} particles was highly significant ($p<0.001$).

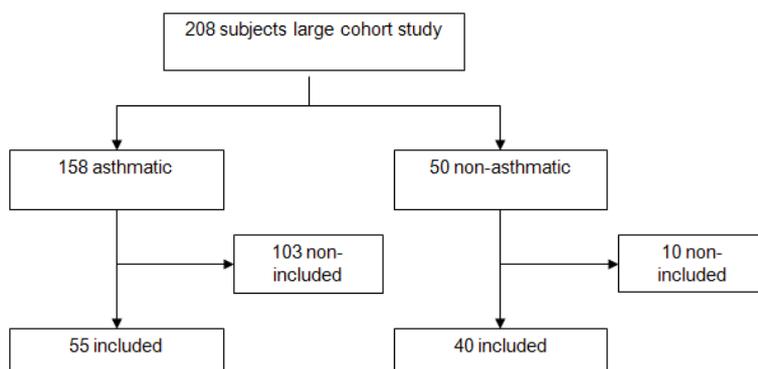
In summary, the data from the present study show adverse effects of EC exposure on lung function among atopic schoolchildren, regardless of their asthma status. These data show the detrimental effects of locating schools in areas exposed to heavy traffic.

Support Statement

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Figure legends

Figure 1. Flow chart of Study design



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Table 1. Demographic characteristics and main outcomes of the study population

| Variable | Participants | | | Non-participants | | | <i>Asthmatic vs</i> | <i>Non-asthmatic vs</i> |
|---|---------------------|------------------------------|----------------|----------------------|------------------------------|-------------------|--------------------------------------|---|
| | Asthmatic (n=55) | Non- asthmatics (n=40) | <i>p</i> value | Asthmatic (n=103) | Non- asthmatics (n=10) | <i>p</i> value | <i>Asthmatics</i> <i>p value*</i> | <i>Non-asthmatic</i> <i>p value*</i> |
| Sex, masculine (%) | 73.4 | 26.5 | 0.002 | 60.0 | 60.0 | 0.605 | 0.608 | 0.121 |
| Age, years (mean(SD)) | 9.7(1.9) | 9.3(2.0) | 0.344 | 9.5(2.3) | 10.3(2.4) | 0.214 | 0.530 | 0.151 |
| Weight, kg (mean(SD)) | 36.8(10.8) | 33.1(11.1) | 0.104 | 37.2(13.0) | 43.3(17.2) | 0.240 | 0.901 | 0.043 |
| Height, cm (mean(SD)) | 137.2(12.2) | 133.5(11.9) | 0.151 | 135.8(15.0) | 143.0(15.6) | 0.104 | 0.564 | 0.042 |
| Maternal schooling, years (mean(SD)) | 8.8(3.1) | 8.8(3.0) | 0.9231 | 10.3(2.8) | 10.6(2.7) | 0.799 | 0.004 | 0.062 |
| Paternal smoking at home, (%) | 54.6 | 38.2 | 0.353 | 54.88 | 83.33 | 0.600 | 0.285 | 0.091 |
| Maternal smoking at home, (%) | 36.8 | 21.9 | 0.105 | 43.48 | 50 | 0.361 | 0.013 | 0.033 |
| Pets at home, (%) | 54.6 | 65 | 0.306 | 57.69 | 93.33 | 0.009 | 0.738 | 0.045 |
| Carpet at home, (%) | 7.6 | 30 | 0.006 | 18 | 46.67 | 0.020 | 0.094 | 0.341 |
| Humidity at home, (%) | 41.8 | 44.7 | 0.78 | 42.86 | 33.33 | 0.582 | 1.000 | 0.544 |
| Prick test positivity, (%) | 81.8 | 70.0 | 0.653 | 89.13 | 66.67 | 0.055 | 1.000 | 0.219 |
| Moderate persistent asthma, (%) | 20 | | | 16.35 | | | 0.725 | |

| | | | | | | | | |
|---------------------------------------|----------|----------|-------|----------|----------|-------|-------|-------|
| Mild persistent asthma, (%) | 23.6 | | | 28.85 | | | | |
| Mild intermittent asthma, (%) | 56.4 | | | 54.81 | | | | |
| FEV ₁ (L/s) (mean(SD)) | 1.9(0.6) | 1.9(.6) | 0.76 | 1.9(0.7) | 2.2(0.6) | 0.760 | 0.437 | 0.146 |
| FVC (L/s) (mean(SD)) | 2.3(0.7) | 2.1(0.6) | 0.173 | 2.9(0.8) | 2.6(0.7) | 0.144 | 0.419 | 0.141 |
| FEF ₂₅₋₇₅ (L/s) (mean(SD)) | 2.0(0.7) | 2.3(0.9) | 0.051 | 2.0(1.0) | 2.5(0.9) | 0.087 | 0.937 | 0.571 |

Table 2. Air pollutants and climatic variables during the study period.

| Variables | Mean (SD) | Interquartile Range* | Min-Max |
|--|-------------|----------------------|--------------|
| EC (Mm-1) | | | |
| 24-hrs average | 92.7 (54.7) | 100.93 | 11.1 – 210.5 |
| Local PM_{2.5} (µg/m³) | | | |
| 24-hrs average | 25.5 (13.0) | 13.7 | 3.1 - 96.1 |
| Temperature | | | |
| 1 hr minimum (°C) | 11.4 (3.0) | 4.0 | 0.7 - 17.9 |
| Humidity | | | |
| 1 hr minimum | 34.8(11.6) | 18.1 | 5.9 - 70.5 |

SD: Standard Deviation; PM_{2.5} = Particles less than 2.5 µg/m³ in diameter.

Mm-1= mega meters

* IQR (Q25- Q75)

Table 3. Association between 24 hour average elemental carbon (coefficients per increase in interquartile range) and lung function parameters in schoolchildren living in Mexico City, 2003-2005.

| EC models | FEV ₁ (ml/s) | | FVC (ml/s) | | FEF _{25-75*} (ml/s) | |
|------------------------|-------------------------|-----------------------------|-------------|------------------------------|------------------------------|------------------------------|
| | Coefficient | 95%CI | Coefficient | 95%CI | Coefficient | 95%CI |
| Asthmatic [†] | -62.0 | (-123.3, -1.2) [‡] | -48.0 | (-124.4, 26.6) | -111.0 | (-228.3, -4.1) [‡] |
| Non-asthmatic* | -95.0 | (-182.3, -8.5) [‡] | -105.0 | (-197.0, -13.7) [‡] | -142.0 | (-347.9, 63.8) |
| Atopic§ | -64.1 | (-123.4, -4.6) [‡] | -46.0 | (-120.3, 28.1) | -138.6 | (-241.5, -35.6) [‡] |

CI: confidence interval. Coefficient was calculated for an interquartile range of 24-hr average elemental carbon: 100.93 (Mm-1).

*Non-asthmatic n=40 and 122 measurements model were adjusted for gender, body mass index, previous day minimum temperature and chronological time.

[†]Asthmatic model n=55 and 186 measurements were adjusted for gender, body mass index, previous day minimum temperature, outdoor physical activity and chronological time.

[§]Atopic n=65 and 213 measurements models were adjusted for gender, body mass index, previous day minimum temperature, outside physical activity and chronological time.

[‡]P< 0.05