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Ambient polycyclic aromatic hydrocarbons and pulmonary function in children

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Abstract

Few studies have examined the relationship between ambient polycyclic aromatic hydrocarbons (PAHs) and pulmonary function in children. Major sources include vehicular emissions, home heating, wildland fires, agricultural burning, and power plants. PAHs are an important component of fine particulate matter that has been linked to respiratory health. This cross-sectional study examines the relationship between estimated individual exposures to the sum of PAHs with 4, 5, or 6 rings (PAH456) and pulmonary function tests (forced expiratory volume in one second (FEV_1) and forced expiratory flow between 25% and 75% of vital capacity) in asthmatic and non-asthmatic children. We applied land-use regression to estimate individual exposures to ambient PAHs for averaging periods ranging from 1 week to 1 year. We used linear regression to estimate the relationship between exposure to PAH456 with pre- and postbronchodilator pulmonary function tests in children in Fresno, California (N =297). Among non-asthmatics, there was a statistically significant association between PAH456 during the previous 3 months, 6 months, and 1 year and postbronchodilator FEV_1. The magnitude of the association increased with the length of the averaging period ranging from 60 to 110 ml decrease in FEV_1 for each 1 ng/m^3 increase in PAH456. There were no associations with PAH456 observed among asthmatic children. We identified an association between annual PAHs and chronic pulmonary function in children without asthma. Additional studies are needed to further explore the association between exposure to PAHs and pulmonary function, especially with regard to differential effects between asthmatic and non-asthmatic children.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

Keywords

air pollution; polycyclic aromatic hydrocarbons; pulmonary function; children; California; asthma

INTRODUCTION

Numerous epidemiologic studies have reported adverse effects of short- and long-term traffic-related air pollution on pulmonary function in children. The majority of previous studies has focused on regularly monitored air pollutants (carbon monoxide, nitrogen oxide, nitrogen dioxide, sulfur dioxide, ozone, and particulate matter (PM) with aerodynamic diameter <10 and <2.5 μm)[1–4] and to a lesser extent proximity to traffic.[5,6] To our knowledge, only one previous study has examined the relationship between exposure to polycyclic aromatic hydrocarbons (PAHs) and pulmonary function in children.[7]

PAHs are a class of chemicals defined by two or more fused aromatic rings that are products of incomplete combustion of fossil fuels, wood, coal, and tobacco. Major sources of ambient PAHs are vehicular emissions, home heating, wildland fires, agricultural burning, and power plants.[8] PAHs are important components of $PM_{2.5}$ and PM_{10}, both of which have been linked to respiratory health.[9] PAHs have received particular attention because of their potential to cause oxidative stress and related cytotoxicity.[10]

Exposure to PAHs has been linked to several adverse outcomes in children, including asthma symptoms,[11,12] biomarkers of asthma,[13,14] respiratory health,[15,16] and cognitive development.[17,18] PAHs have also been associated with lower pulmonary function and wheezing in adults.[19] One study of community-level air pollution compared the average pulmonary function across two communities with different concentrations of ambient air pollutants, including PAHs,[7] but no previous study of individual PAH exposure and pulmonary function in children has been reported.

Estimates of exposures to ambient air pollutants are typically based on measurements from single fixed-site monitors that can capture temporal variability, but not often spatial variability.[20] For PAHs, this poses two problems. First, ambient concentrations of PAHs are not routinely measured at air quality monitoring sites.[21] Second, PAHs are highly dependent on local sources that have a high degree of spatial variability within an urban environment.[21–24]

Exposure assessment for spatially heterogeneous air pollutants is best performed with a model of exposure that accounts for both the temporal and spatial distributions. Land-use regression models can estimate pollution exposure by exploiting the relationship between the measurement site and local environmental variables, although they are typically cross-sectional and thought to represent long-term concentration gradients.[25] This relationship, formalized with regression equations, can incorporate small area variation and be used to assign estimated exposures for all participants in a cohort study. When repeated air pollution measures are available, mixed-effects regression can be used to model shorter time periods by accounting for both short-term temporal variability and spatial variability.[22,23]

Fresno is located in California's San Joaquin Valley and is one of the fastest-growing areas of California.[26–28] During the years 2005 to 2007, the population of Fresno was exposed to an annual average $PM_{2.5}$ concentration that often exceeded the federal annual standard by over 40%.[26,29–31] Motor vehicles account for one-third of PAH emissions in the United States,[32] and motor vehicles and residential wood combustion are the major sources of air pollution in Fresno.[33–36] The burden of asthma is also very high in Fresno. The 2009 lifetime prevalence of asthma in children 5 to 17 years in Fresno County was 20% (95% confidence interval (CI): 13–27) compared with 16% (95% CI: 15–18) for the state of California.[37] For these reasons, Fresno was selected for a study of the differential effects of air pollution on the respiratory health of asthmatic and non-asthmatic children.

The current study examines the relationship between estimated personal exposures to the sum of PAHs with 4, 5, or 6 rings (PAH456) and pre- and postbronchodilator pulmonary function tests in asthmatic and non-asthmatic children in Fresno, California.

MATERIALS AND METHODS

Study Population

The subjects in this study are a subset of those enrolled in the Children's Health and Air Pollution Study (CHAPS) that includes 467 children, with and without asthma, living in Fresno, California. Approximately half of the children with asthma had been followed for up to 8 years as part of the Fresno Asthmatic Children's Environment Study (FACES). The other participating children, asthmatic and non-asthmatic, were recruited from the Fresno Unified School District through questionnaires and fliers. All participating children were screened at the CHAPS Fresno field office.

The subset (n =309) of screened children with valid spirometry was included in this analysis. All participants were children whose residence was within 20 km of the California Air Resources Board air quality monitoring site in Fresno (Fresno-First Street). Participants answered a detailed respiratory health and general history questionnaire, performed spirometry, and underwent skin-prick testing. Children were classified as having active asthma when both a history of physician diagnosis of asthma and use of bronchodilator medications within the previous 12 months were reported. Non-asthmatic children had no history of diagnosis of asthma. The study was conducted under protocols approved by the University of California, Berkeley and Stanford University Committees for the Protection of Human Subjects. Written informed consent for all procedures was obtained from parents/legal guardians.

Spirometry

Spirometry was performed between 2007 and 2012 using an EasyOne spirometer (Medical Technologies, Chelmsford, MA, USA) that met American Thoracic Society 1994 spirometry standards[38] and a standard protocol that conformed to American Thoracic Society performance guidelines.[39] Spirometry was performed both pre- and postbronchodilator administration. A pulmonologist (JB) reviewed all forced expiratory curves. Forced expiratory volume in one second (FEV_1) and forced expiratory flow between 25% and 75%

of vital capacity ($FEF_{25–75}$) were obtained only from curves that met the American Thoracic Society acceptability criteria. The prebronchodilator pulmonary function tests were considered as a measure of acute effects of short-term exposure to PAHs. We were primarily interested here in the postbronchodilator pulmonary function tests as a measure of chronic effects of long-term exposure to PAHs.

Exposure Assessment

We applied a mixed-effects regression model to estimate individual-level residential exposures to PAH456. This spatiotemporal model used directly measured, daily particle-bound PAH concentrations from the Fresno-First Street site, also an EPA Supersite at the time of sampling, as measured by the PAS2000 (EcoChem Analytics, League City, TX, USA) and filter-based integrated 24-h measurements at homes in the FACES substudy (N =83) for estimates of temporal and spatial trends. PAH456 24-h mean concentration at subject homes was estimated from the particle-bound PAH concentrations measured for the relevant time periods in 2006–2012 at the Fresno-First Street site, distance to and intensity of source (minor collector roads, highway length within 500 m from the know residence locations), meteorological characteristics (wind direction, 24-h wind recirculation, 24-h relative humidity), neighborhood characteristics (home heating type by census block group), and season. The model estimates the sum of PAHs with 4-, 5-, and 6-rings, including fluoranthene, benz[*a*]anthracene, chrysene, benzo[*a*]pyrene, benzo[*b*]fluoranthene, benzo[*k*]fluoranthene, benzo[*ghi*]perylene, indeno[1,2,3-*cd*]pyrene, and dibenz [*a,h*]anthracene. These PAHs represent semivolatile PAHs of the original 16 priority PAHs that were identified by the US EPA. This model explained 81% of the between-house variability and 18% of the within-house variability as measured by mixed-effects model variance components. More detail on field collection, measurement results, and modeling can be found in Noth et al.[23]

Estimates for PAH456 were then calculated for each day and averaged over the following periods when at least 75% of the days had valid exposure estimates: previous year, 6 months, 3 months, 1 month, and 1 week. PAH456 exposure was estimated for all of these time periods before pulmonary function testing.

Statistical Analyses

Linear regression was used to evaluate the associations between annual mean PAH456 exposures and maximum pre- and postbronchodilator FEV_1 and $FEF_{25–75}$. The analysis was restricted to participants with acceptable spirometry and non-missing covariates.

The results were adjusted for age, race, sex, height, and socioeconomic status (as measured by parental-reported income and residing in a rented *versus* owned home).

Maternal and paternal education was considered as a covariate, but its inclusion did not change the estimates. As a sensitivity analysis, season and secondhand smoke were explored as potential confounders.

We stratified the analyses by sex to assess whether the relationship between PAH456 exposure and pulmonary function was different between boys and girls.

RESULTS

The entire study population consists of 467 children, of which 309 had acceptable postbronchodilator FEV_1 and/or $FEF_{25–75}$. We were able to estimate PAH456 exposure for 297 of those children (96% of those with acceptable postbronchodilator spirometry).

The ages of the children ranged from 9 to 18 years (Table 1). The majority of the population is Hispanic and about half of the study population lives in family-owned homes. Asthmatic children were more likely to be African American and have slightly more parental education, but otherwise there was little difference in demographic characteristics between asthmatic and non-asthmatic participants.

The distributions of exposures for each of the averaging times and by asthmatic status are presented in Table 2a. The correlation matrix of the exposure periods is in Table 2b.

Tables 3a and b show the distribution of FEV_1 and $FEF_{25–75}$ values stratified by asthmatic status. The tables include the raw values by sex and the percent predicted values using NHANES reference equations.[40] As expected, girls had lower pulmonary function compared with boys and, in general, asthmatic children had lower pulmonary function than non-asthmatic children with one exception among boys for prebronchodilator FEV_1. The tables are split by prebronchodilator (Table 3a) and postbronchodilator (Table 3b) values.

Tables 4a and b present the main results of the final analysis of the adjusted association of PAH456 during each of the averaging periods with maximum pre- and postbronchodilator FEV_1 and $FEF_{25–75}$, respectively, stratified by asthma status. For the prebronchodilator pulmonary function tests, the estimates were in mixed directions and only one estimate was statistically significant. A 1 ng/m^3 increase in 1-week PAH456 was associated with a 0.29 l increase in FEV_1 (95% CI: 0.07, 0.51) among asthmatics (Table 4a). As expected, after adjusting for season for this short-term exposure, the result was no longer statistically significant (data not shown).

For the postbronchodilator pulmonary function tests that represent a more chronic status of lung function, the associations between PAH and pulmonary function were more consistent. Among non-asthmatics, there was a statistically significant association between PAH during the previous 3 months, 6 months and 1 year and FEV_1. The magnitude of the association increased with the length of the averaging period.

Among non-asthmatic children, a 1 ng/m^3 increase in PAH456 was associated with a 0.11 l (110 ml) decrease in FEV_1 (95% CI: −0.20, −0.01) (Table 4b).

After adjustment for season, the postbronchodilator estimates were essentially unchanged and were borderline significant. The results adjusted for secondhand smoke were not considerably different among non-asthmatics. The effect estimates were larger among asthmatics compared with the primary analysis, although not statistically significant (Appendix Tables A1a and b). When the analysis was stratified by sex, no significant differences between boys and girls were found (data not shown). The results stratified by use of controller medication is presented in the (Appendix Tables A2a and b) for both pre- and

postbronchodilator pulmonary function tests by PAH exposure periods. As with the other results, they are largely null, especially among those on controller medications. There are subtle, but not convincing signals among those without controller medication use, but in opposite directions for FEV_1 and $FEF_{25–75}$.

DISCUSSION

We found a statistically significant association between exposure to PAH456 during the previous 3 months, 6 months and 1 year and a decrease in FEV_1 among non-asthmatic children. Among asthmatic children, there was little evidence for an effect of exposure to PAH456 on FEV_1. To our knowledge, this is the first study of the effect of individual-level PAH exposures on lung function in children.

Relatively few studies have evaluated the effects of short- or long-term exposure to PAHs on respiratory health, despite the potential biological impacts of this class of organic compounds.[41] In a study of longer-term exposure to air pollutants in Southern California, variants of the PAH-metabolizing enzyme, microsomal epoxide hydrolase, were associated with an increased risk of asthma and, in addition, the risk increased with proximity of residences to freeways.[42] Children living in a town in the Czech Republic with two- to threefold higher air pollution levels (including PAHs) had lower pulmonary function tests compared with children living in a town with lower air pollution.[7]

An additional study found prenatal exposure to PAHs, as measured by personal monitors during the third trimester of pregnancy, was not associated with respiratory symptoms of children at age 12 and 24 months; however, an interaction of PAH exposure with secondhand tobacco smoke to increase risk of respiratory symptoms was found.[11]

We showed in a previous analysis of the FACES that short-term increases in PAH456 were associated with risk of wheeze. Odds ratios ranged from 1.01 (95% CI, 1.00–1.02) to 1.10 (95% CI, 1.04–1.17) for multiple lags and moving averages out to 9 days.[12] In a second study that included a subset of 71 FACES participants, we demonstrated an association between PAH exposure and suppression of regulatory T-cell function through methylation of the *FoxP3* gene that regulates the function of these cells.[14] The suppression of regulatory T cells also was significantly associated with enhancement of the Th-2 phenotype, asthma symptoms, and lower FEV_1 in the 71 FACES children. The finding that PAH exposure was associated with methylation of *FoxP3* is consistent with the observation that exposure to traffic-related air pollution can lead to DNA methylation.[43] The association of PAH with *FoxP3* methylation is also consistent with the known effect of a common PAH, phenanthrene, to enhance IgE synthesis.[44] In another more recent analysis of 256 CHAPS subjects, results demonstrated that increased ambient PAH exposure was associated with impaired systemic immunity and epigenetic modifications in two key genes involved in atopy: *FoxP3* and *IFNγ*, with a higher impact on atopic children.[45] These FACES and CHAPS data, taken together with the other studies described above, provide evidence that PAH exposure may contribute to asthma morbidity.

Somewhat surprisingly, we found a statistically significant effect of PAH exposure on lung function in non-asthmatic children, but not in asthmatic children in the current study. One possible explanation for these results is that lung function is more variable with asthma such that a relatively small effect of PAH exposure could not be detected against the background variability. Thus, we might not have had the statistical power to detect a difference in pulmonary function among asthmatic participants. Another potential explanation may lie in the results of controlled exposure studies of diesel exhaust (containing multiple PAHs), which show that subjects with asthma have less acute inflammatory responses than those without asthma.[46] The results stratified by controller medication use among asthmatics did not provide any clear explanation to our results; however, there was little power to detect such a difference.

The role of season in the study of air pollution and pulmonary function is complex. The adjustment for season is more reasonably justified for the shorter averaging periods, but less so for the longer ones, particularly the 1-year averages, during which all the seasons are represented. In our study, adjustment for season did not substantially change the effect estimates.

Our study has several strengths. It is the first study of pulmonary function in children to employ individual daily estimates of PAH exposure. The exposure assessment is based on a well-developed regression model of PAH exposure that captures spatial variability in a region of the United States with high concentrations of PAHs. An additional strength is the phenotypically well-characterized study population with individual covariate data and careful quality control of spirometry.

Several limitations need to be considered, however, in the interpretation of these data. Because the spirometric data were cross-sectional, we were not able to assess change in pulmonary function over time and, therefore, could not examine the association between PAHs and growth of lung function.

We did not estimate individual-level exposures for other pollutants, so we did not evaluate other components of PM, secondhand smoke, or gaseous pollutants that are correlated with PAHs. (Although we explored family and home smoking, they were not associated with pulmonary function.) Thus, we cannot estimate the degree to which the associations we report here are independent of other pollutants. On the basis of the correlation structure of the other pollutants collected at the central site, the PAHs in this analysis are correlated with $PM_{2.5}$, carbon monoxide, nitrogen dioxide, and elemental carbon. These strong correlations suggest that the PAHs we measured in Fresno are likely from traffic emissions primarily.

The spatiotemporal regression model accounts for some of the spatial variability in the data, but the health effects associated with model estimates of PAH exposure are likely attenuated and biased toward the null. The PAH exposure estimates contain both classical and Berkson’s errors. However, we did not incorporate errors attached to the spatiotemporal model.[23]

In conclusion, this study identifies an association between PAH456 and pulmonary function in children without asthma. Additional studies are needed to further explore the association

between exposure to PAHs and pulmonary function, especially differential effects between asthmatic and non-asthmatic children. Future studies would benefit from improved individual-level assessment of PAH exposures and longitudinal pulmonary function follow-up.

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ABBREVIATIONS

ATS	American Thoracic Society
CHAPS	Children's Health and Air Pollution Study
CI	confidence interval
FACES	Fresno Asthmatic Children's Environment Study
$FEF_{25–75}$	forced expiratory flow between 25% and 75% of vital capacity
FEV_1	forced expiratory volume in one second
PAH456	PAHs with 4, 5, or 6 rings
PAHs	polycyclic aromatic hydrocarbons
PM	particulate matter

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APPENDIX

Table A1a

Adjusted[a] association of PAH and maximum, pre- BD PFTs, stratified asthma status (estimate/95% CI) for CHAPS study population.

Pre-BD PFT	PAH exposure period	Asthmatics		95% CI	Non-asthmatics		95% CI
		N	Estimate[b]		N	Estimate[b]	
FEV_1	1 Week	94	**0.30**	0.09, 0.52	91	0.05	−0.01, 0.11
	1 Month	103	**0.27**	0.02, 0.53	91	0.03	−0.04, 0.09
	3 Months	105	0.15	−0.27, 0.57	99	−0.01	−0.08, 0.06
	6 Months	108	0.10	−0.32, 0.51	102	−0.08	−0.18, 0.03
	1 Year	110	−0.02	−0.35, 0.66	102	−0.08	−0.20, 0.04
$FEF_{25–75}$	1 Week	89	−0.02	−0.12, 0.08	79	0.10	−0.02, 0.22
	1 Month	98	−0.04	−0.17, 0.08	79	0.06	−0.08, 0.19
	3 Months	100	0.05	−0.13, 0.24	87	−0.10	−0.27, 0.06
	6 Months	101	−0.03	−0.23, 0.17	89	−0.16	−0.37, 0.05
	1 Year	102	0.01	−0.23, 0.26	89	−0.16	−0.40, 0.09

Abbreviations: BD, bronchodilator; CI, confidence interval; CHAPS, Children's Health and Air Pollution Study; FEV_1, forced expiratory volume in one second; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of vital capacity; PAH, polycyclic aromatic hydrocarbon; PFT, pulmonary function tests; stratified asthma status.

[a] Adjusted for age, sex, race/ethnicity, height, and socioeconomic status (as measured by parental-reported family income < \$15,000 $year^{-1}$ and residing in a rented *versus* owned home) and secondhand smoke exposure.

[b] Results are presented in l (FEV_1) and l/s ($FEF_{25–75}$) for a 1 ng/m^3 change in PAH.

Bold values are statistically significant ($P<0.05$).

Table A1b

Adjusted[a] association of PAH and maximum, post-BD PFTs, stratified asthma status (coefficient/95% CI) for CHAPS study population.

Post-BD PFT	PAH exposure period	Asthmatics		95% CI	Non-asthmatics		95% CI
		N	Estimate[b]		N	Estimate[b]	
FEV_1	1 Week	111	−0.02	−0.06, 0.03	147	−0.004	−0.05, 0.04
	1 Month	122	−0.02	−0.07, 0.04	149	−0.01	−0.06, 0.03
	3 Months	125	0.02	−0.07, 0.10	158	−0.04	−0.10, 0.02
	6 Months	128	−0.01	−0.10, 0.08	161	**−0.10**	−0.18, −0.02
	1 Year	130	−0.001	−0.11, 0.11	161	**−0.11**	−0.20, −0.01
$FEF_{25–75}$	1 Week	101	−0.03	−0.12, 0.06	127	0.003	−0.09, 0.10
	1 Month	110	−0.04	−0.15, 0.07	129	−0.03	−0.12, 0.07
	3 Months	112	−0.01	−0.17, 0.18	138	−0.08	−0.23, 0.07
	6 Months	114	−0.03	−0.23, 0.17	140	−0.16	−0.33, 0.01
	1 Year	116	−0.03	−0.27, 0.21	140	−0.17	−0.36, 0.03

Abbreviations: BD, bronchodilator; CI, confidence interval; CHAPS, Children's Health and Air Pollution Study; FEV_1, forced expiratory volume in one second; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of vital capacity; PAH, polycyclic aromatic hydrocarbon; PFT, pulmonary function tests; stratified asthma status.

[a] Adjusted for age, sex, race/ethnicity, height, and socioeconomic status (as measured by parental-reported family income < \$15,000 $year^{-1}$ and residing in a rented *versus* owned home) and second hand smoke exposure.

[b] Results are presented in l (FEV_1) and l/s ($FEF_{25–75}$) for a 1 ng/m^3 change in PAH.

Bold values are statistically significant ($P<0.05$).

Table A2a

Adjusted[a] association of PAH and maximum, pre-BD, PFTs, stratified controller medication use among asthmatics (estimate/95% CI) for CHAPS study population.

Pre-BD PFT	PAH exposure period	Controller medication use		95% CI	No controller medication use		95% CI
		N	Estimate[b]		N	Estimate[b]	
FEV_1	1 Week	41	−0.01	−0.09, 0.06	52	**0.49**	0.09, 0.89
	1 Month	45	−0.01	−0.09, 0.07	57	0.40	−0.10, 0.90
	3 Months	39	0.03	−0.08, 0.14	42	0.13	−0.92, 1.18
	6 Months	49	0.02	−0.11, 0.14	58	0.06	−0.76, 0.87
	1 Year	49	−0.04	−0.21, 0.13	60	0.16	−0.76, 1.08
$FEF_{25–75}$	1 Week	39	0.02	−0.14, 0.19	49	−0.09	−0.23, 0.05
	1 Month	43	0.03	−0.15, 0.21	54	**−0.18**	−0.35, −0.001
	3 Months	37	0.01	−0.13, 0.36	41	−0.06	−0.38, 0.27
	6 Months	45	0.12	−0.17, 0.42	55	−0.23	−0.52, 0.05
	1 Year	45	0.05	−0.37, 0.46	56	−0.11	−0.45, 0.23

Abbreviations: BD, bronchodilator; CI, confidence interval; CHAPS, Children's Health and Air Pollution Study; FEV_1, forced expiratory volume in one second; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of vital capacity; PAH, polycyclic aromatic hydrocarbon; PFT, pulmonary function tests; stratified asthma status.

[a] Adjusted for age, sex, race/ethnicity, height, and socioeconomic status (as measured by parental-reported family income < \$15,000 $year^{-1}$ and residing in a rented *versus* owned home).

[b] Results are presented in l (FEV_1) and l/s ($FEF_{25–75}$) for a 1 ng/m^3 change in PAH.

Bold values are statistically significant ($P<0.05$).

Table A2b

Adjusted[a] association of PAH and maximum, post-BD PFTs, stratified controller medication use among asthmatics (coefficient/95% CI) for CHAPS study population

Post-BD PFT	PAH exposure period	Controller medication use		95% CI	No controller medication use		95% CI
		N	Estimate[b]		N	Estimate[b]	
FEV_1	1 Week	49	−0.01	−0.08, 0.05	61	−0.02	−0.08, 0.04
	1 Month	54	−0.03	−0.11, 0.05	67	−0.02	−0.10, 0.07
	3 Months	43	0.003	−0.10, 0.11	48	0.04	−0.10, 0.18
	6 Months	58	0.03	−0.11, 0.16	69	−0.07	−0.20, 0.06
	1 Year	58	0.05	−0.13, 0.23	71	−0.07	−0.22, 0.08
$FEF_{25–75}$	1 Week	46	−0.06	−0.19, 0.07	54	0.003	−0.12, 0.12
	1 Month	51	−0.11	−0.26, 0.04	58	−0.02	−0.18, 0.13
	3 Months	40	0.03	−0.21, 0.27	42	0.01	−0.29, 0.30
	6 Months	54	0.09	−0.19, 0.37	59	−0.22	−0.48, 0.05
	1 Year	54	0.14	−0.23, 0.51	61	−0.21	−0.53, 0.12

Abbreviations: BD, bronchodilator; CI, confidence interval; CHAPS, Children's Health and Air Pollution Study; FEV_1, forced expiratory volume in one second; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of vital capacity; PAH, polycyclic aromatic hydrocarbon; PFT, pulmonary function tests; stratified asthma status.

[a] Adjusted for age, sex, race/ethnicity, height, and socioeconomic status (as measured by parental-reported family income < \$15,000 $year^{-1}$ and residing in a rented *versus* owned home).

[b] Results are presented in l (FEV_1) and l/s ($FEF_{25–75}$) for a 1 ng/m^3 change in PAH.

Bold values are statistically significant ($P<0.05$).

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Table 1

Distribution of characteristics of CHAPS study population, stratified by asthma status.

Characteristic[a]	Total (N = 297)		Asthmatic (N =135)		Non-asthmatic (N =162)	
	Mean	SD	Mean	SD	Mean	SD
Age (years)	13.7	2.2	13.9	2.1	13.5	2.3
Height (cm)	158.7	12.2	159.2	11.7	158.3	12.7
	N	*%*	*N*	*%*	*N*	*%*
Male	154	51.9	70	51.9	84	51.9
Race						
White	95	32.0	43	31.9	52	32.1
Hispanic	167	56.2	70	51.9	97	59.9
African-American	33	11.1	21	31.9	12	7.4
Other	2	1.2	1	0.7	1	0.6
Low income[b]	83	28.2	38	28.6	45	28.0
Rented home	144	49.0	62	47.0	82	50.6
Mother's education						
<High school	86	29.3	34	25.4	52	32.7
≥High school	207	70.7	100	74.6	107	67.3
Secondhand smoke exposure[c]	65	21.9	31	23.0	34	21.0

Abbreviations: CHAPS, Children's Health and Air Pollution Study.

[a] Missing covariates: low income (n =3), rented home (n =3), and mother's education (n =3).

[b] Parental-reported family income ≤$15,000 $year^{-1}$.

[c] Parental-reported smoker (responder/caregiver) or if someone else who spends time with child smokes.

Table 2a

Distribution of different duration PAH (ng/m^3) exposure estimates stratified by asthma status (*N*/median/IQR) for CHAPS study population.

Time before test	Total			Asthmatic			Non-asthmatic		
	N	Median	IQR	N	Median	IQR	N	Median	IQR
1 week	269	3.09	2.16	120	3.24	2.01	149	3.00	2.19
1 month	282	2.96	1.93	131	3.14	1.71	151	2.72	2.21
3 months	293	2.97	1.64	132	3.86	1.65	161	3.12	1.71
6 months	297	3.05	1.10	135	3.11	1.01	162	2.93	1.22
1 year	297	2.99	1.03	135	3.25	1.06	162	2.90	0.84

Abbreviations: CHAPS, Children's Health and Air Pollution Study; IQR, interquartile range; PAH, polycyclic aromatic hydrocarbon.

Table 2b

Spearman's correlation coefficients of PAH456 by averaging periods.

PAH exposure	1 Week	1 Month	3 Months	6 Months	1 Year
1 week	1.00				
1 month	0.90	1.00			
3 months	0.65	0.80	1.00		
6 months	0.53	0.64	0.89	1.00	
1 year	0.42	0.51	0.74	0.89	1.00

Abbreviation: PAH, polycyclic aromatic hydrocarbon.

All *P*-values <0.01.

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Table 3a

Mean and SDs[a] of maximum, prebronchodilator pulmonary function tests, stratified by sex and asthma status for CHAPS study population (*N* =237).

		Asthmatic (N =115)			Non-asthmatic (N =122)		
		N	**Mean**	**SD**	**N**	**Mean**	**SD**
Boys	FEV_1	63	3.36	2.34	56	3.28	0.97
(raw values)	$FEF_{25–75}$	60	2.91	1.10	48	3.36	1.21
Girls	FEV_1	52	2.70	0.61	46	2.76	0.63
(raw values)	$FEF_{25–75}$	47	2.94	1.02	41	3.06	0.83
Percent predicted[b]	FEV_1	114	99.6	58.5	101	97.5	12.3
(boys and girls)	$FEF_{25–75}$	106	80.2	23.2	88	89.3	20.6

Abbreviations: CHAPS, Children's Health and Air Pollution Study; FEV_1, forced expiratory volume in one second; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of vital capacity; NHANES, National Health and Nutrition Examination Survey.

[a] Means and SDs are presented in l (FEV_1) and l/s ($FEF_{25–75}$).

[b] NHANES reference equations.

Table 3b

Mean and SDs[a] of maximum, postbronchodilator pulmonary function tests, stratified by sex and asthma status for CHAPS study population (*N* =297).

		Asthmatic (N =135)			Non-asthmatic (N =162)		
		N	**Mean**	**SD**	**N**	**Mean**	**SD**
Boys	FEV_1	70	3.25	0.92	84	3.32	1.00
(raw values)	$FEF_{25–75}$	65	3.39	1.13	74	3.79	1.34
Girls	FEV_1	65	2.74	0.62	78	2.74	0.64
(raw values)	$FEF_{25–75}$	56	3.32	0.96	67	3.44	0.96
Percent predicted[b]	FEV_1	134	97.6	13.3	161	99.6	13.3
(boys and girls)	$FEF_{25–75}$	120	91.8	20.5	140	102.1	24.0

Abbreviations: CHAPS, Children's Health and Air Pollution Study; FEV_1, forced expiratory volume in one second; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of vital capacity; NHANES, National Health and Nutrition Examination Survey.

[a] Means and SDs are presented in l (FEV_1) and l/s ($FEF_{25–75}$).

[b] NHANES reference equations.

Table 4a

Adjusted[a] association of PAH and maximum, pre-BD PFTs, stratified asthma status (estimate/95% CI) for CHAPS study population.

Pre-BD PFT	PAH exposure period	Asthmatics		95% CI	Non-asthmatics		95% CI
		N	Estimate[b]		N	Estimate[b]	
FEV_1	1 Week	94	**0.29**	0.07, 0.51	91	0.05	−0.02, 0.11
	1 Month	103	0.24	−0.01, 0.50	91	0.02	−0.04, 0.09
	3 Months	105	0.08	−0.24, 0.40	99	−0.03	−0.11, 0.05
	6 Months	108	0.06	−0.35, 0.47	102	−0.06	−0.16, 0.04
	1 Year	110	0.12	−0.38, 0.62	102	−0.07	−0.19, 0.04
$FEF_{25–75}$	1 Week	89	−0.007	−0.11, 0.09	79	0.09	−0.03, 0.21
	1 Month	98	−0.03	−0.15, 0.09	79	0.04	−0.09, 0.18
	3 Months	100	0.05	−0.11, 0.20	87	−0.11	−0.26, 0.05
	6 Months	101	−0.03	−0.22, 0.17	89	−0.12	−0.33, 0.08
	1 Year	102	0.03	−0.22, 0.27	89	−0.13	−0.37, 0.10

Abbreviations: BD, bronchodilator; CHAPS, Children's Health and Air Pollution Study; CI, confidence interval; FEV_1, forced expiratory volume in one second; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of vital capacity; PAH, polycyclic aromatic hydrocarbon; PFT, pulmonary function tests.

[a] Adjusted for age, sex, race/ethnicity, height, and socioeconomic status (as measured by parental-reported family income <$15,000 $year^{-1}$ and residing in a rented *versus* owned home).

[b] Results are presented in l (FEV_1) and l/s ($FEF_{25–75}$) for a 1 ng/m^3 change in PAH.

Bold value is statistically significant ($P<0.05$).

Table 4b

Adjusted[a] association of PAH and maximum, post-BD PFTs, stratified asthma status (coefficient/95% CI) for CHAPS study population.

Post-BD PFT	PAH exposure period	Asthmatics		95% CI	Non-asthmatics		95% CI
		N	Estimate[b]		N	Estimate[b]	
FEV_1	1 week	111	0.02	−0.06, 0.03	147	0.02	−0.05, 0.04
	1 month	122	0.03	−0.07, 0.04	149	0.02	−0.06, 0.03
	3 months	125	0.004	−0.06, 0.07	158	**−0.06**	−0.11, −0.004
	6 months	128	−0.02	−0.11, 0.07	161	**−0.09**	−0.17, −0.01
	1 year	130	−0.008	−0.11, 0.10	161	**−0.11**	−0.20, −0.01
$FEF_{25–75}$	1 week	101	−0.03	−0.11, 0.06	127	0.002	−0.09, 0.09
	1 month	110	−0.04	−0.15, 0.06	129	−0.03	−0.12, 0.07
	3 months	112	−0.02	−0.16, 0.12	138	−0.09	−0.20, 0.02
	6 months	114	−0.06	−0.25, 0.13	140	−0.14	−0.30, 0.03
	1 year	116	−0.05	−0.29, 0.19	140	−0.16	−0.36, 0.03

Abbreviations: BD, bronchodilator; CHAPS, Children's Health and Air Pollution Study; CI, confidence interval; FEV_1, forced expiratory volume in one second; $FEF_{25–75}$, forced expiratory flow between 25% and 75% of vital capacity; PAH, polycyclic aromatic hydrocarbon; PFT, pulmonary function tests.

[a] Adjusted for age, sex, race/ethnicity, height, and socioeconomic status (as measured by parental-reported family income <$15,000 $year^{-1}$ and residing in a rented *versus* owned home).

[b] Results are presented in l (FEV_1) and l/s ($FEF_{25–75}$) for a 1 ng/m^3 change in PAH.

Bold values are statistically significant ($P<0.05$).