Cost of Developmental Delay from Prenatal Exposure to Airborne Polycyclic Aromatic Hydrocarbons

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Abstract

Early life exposure to ambient polycyclic aromatic hydrocarbons (PAHs) can result in developmental delay. The negative health effects of PAHs have been well-documented but the cost of developmental delay due to PAH exposure has not been studied. The Columbia Center for Children's Environmental Health previously has reported the significant effect of prenatal exposure to ambient PAHs on delayed mental development at three years, using the Bayley Scales in a cohort of low-income women and children in New York City (NYC). Here we have used the cohort results to estimate the annual costs of preschool special education services for low-income NYC children with developmental delay due to PAH exposure using the Environmentally Attributable Fraction method. The estimated cost of PAH-exposure-related services is over $13.7 million per year for Medicaid births in NYC. This high cost supports policies to reduce level of PAHs in NYC air.

Keywords

PAH; developmental delay; costs

Polycyclic aromatic hydrocarbons (PAHs) are a group of approximately 10,000 organic compounds commonly found in the environment as a result of the incomplete burning of oil, wood, garbage, or coal. Several PAHs are widely believed to pose a major threat to human health, affecting outcomes ranging from lung and breast cancer[1] to developmental delay in

children.[2] Currently, seven PAHs are identified as probable carcinogens and 17 are identified as priority substances that pose a significant danger to public health.[1,3]

Polycyclic aromatic hydrocarbons are ubiquitous in urban areas. Motor vehicle emissions, particularly diesel exhaust, contribute as much as 90% of airborne particle-bound PAH mass.[4,5] The high traffic congestion and large concentrations of diesel vehicles traveling short distances further promote the emission of PAH in urban areas.[1] Benzo[a]pyrene (BaP), a representative PAH compound, has been found to be almost twice as high in urban areas as in rural areas.[6] Therefore, individuals in urban areas face greater risks of harm from PAH emissions than individuals in rural areas. There is increasing evidence that the fetus and young child are particularly susceptible to these environmental pollutants.[2,7]

The mounting evidence of the health concerns from PAH exposure, including from early-life exposure[7–13] and the widespread prevalence of PAH have led to increased efforts to implement policies that limit PAH emissions. At the same time, controversy surrounds the appropriate degree of regulation, in part stemming from lack of full knowledge of the societal costs associated with PAH exposure. In this paper, we attempt to fill some of the gap in understanding the benefits of such actions by providing estimates of just one of the monetizable costs of cognitive developmental delay associated with prenatal PAH exposure —preschool special education services.

Few studies have been conducted to evaluate the association between prenatal PAH exposure and cognitive development. The NYC cohort study that provides the basis for the present analysis found a negative impact of prenatal exposure to PAH on children's cognitive function (mental development index), as measured by the Bayley Scales, at three years of age. Infants who were exposed to the highest quartile of PAH levels *in utero* (relative to the three lowest quartiles) were more likely to be developmentally delayed, with an adjusted odds ratio of 2.89.[2] Studies in New York City, Krakow, Poland, and Telpice (Czech Republic) also found prenatal exposure to PAH to be associated with poorer birth outcomes, such as lower birthweight and smaller head circumference.[8,9,14]

Methods

Earlier work has demonstrated the significant association between prenatal exposure to PAH and child developmental delay in the Columbia Center for Children's Environmental Health (CCCEH) cohort in NYC.[2] In the present paper, we present estimates of the cost of preschool special education services to address developmental delays potentially attributable to prenatal exposure to PAHs. First, based on cohort findings, we compute the rate of developmental delay due to PAH exposure for the NYC Medicaid population. We multiply this by the prevalence of developmental delay, the size of the population at risk from exposure to PAH, and the cost per case for preschool special education to give the estimated cost of PAH-related preschool special education services.

PA H-related developmental delay and estimated costs

The CCCEH Cohort Study: Impacts of exposure to PAH on child development. The CCCEH cohort study enrolled Dominican and African American women and their children residing in Northern Manhattan and the Bronx, N.Y. Active smokers and women who had diabetes, hypertension, known HIV, or reported drug abuse were excluded from the study because of the well-documented associations between these factors and child health.[2,7] Women were enrolled during pregnancy in order to collect detailed, contemporaneous information about prenatal exposure to various potential health hazards. During their third trimester of pregnancy, women enrolled in the study wore a small backpack during the day for two consecutive days and placed the backpack near their bed at night. The personal air

sampling pumps collected vapors and particles of 2.5 μg in diameter on a pre-cleaned quartz microfiber filter and a pre-cleaned polyurethane foam cartridge backup. The samples were analyzed at Southwest Research Institute for eight carcinogenic PAHs.[2,7] Total PAH exposure was defined as the summation of exposure to benz[*a*]anthracene, chrysene, benzo[*b*]fluroanthene, benzo[*k*]fluroanthene, benzo[a] pyrene, indeno-[1,2,3-*cd*]pyrene, disbenz[*a,h*]anthracene, and benzo[*g,h,i*]perylene, all identified PAH carcinogens and potential developmental toxicants. Child development was measured using the Bayley Scales of Infant Development (second edition), which generate a Mental Development Index (MDI) and a corresponding Psychomotor Development Index (PDI), at 12, 24, and 36 months of age. The Bayley Scales are the most widely used development test for young children, with modest ability to predict subsequent intelligence and school performance when administered at three years of age overall[15] and in this cohort.[2]

Detailed measures of potential confounding variables were also collected. Cord blood and maternal blood were sampled at delivery for analysis of cotinine, heavy metals, and the pesticide chlorpyrifos (CPF). A prenatal questionnaire was administered to collect data on environmental tobacco smoke (ETS) as well as background characteristics of the mother, such as her age, education, and marital status. A measure of the quality and quantity of stimulation and support available to a child in the home environment was assessed by the Home Observation for Measurement of the Environment (HOME Inventory) at three years of age.[16]

The statistical analysis estimated the associations between prenatal PAH exposure and developmental delay using multiple logistic regression. As a clinical outcome, developmental delay was defined as a binary variable for children with scores one or more standard deviations below the national mean (MDI<85). Polycyclic aromatic hydrocarbon was dichotomized as a binary variable depending on whether levels of PAH exposure were in the highest quartile (greater than 4.16 ng/m^3, mean of 7.54) or lowest three quartiles (less than or equal to 4.16 ng/m^3, mean of 2.29). Regression models adjusted for race/ethnicity, gestational age, age of child when developmental assessments were administered, HOME inventory score, sex of the child, and prenatal exposure to ETS and CPF. The analysis involved 183 children who had reached three years of age with valid data for prenatal PAH, maternal and cord blood cotinine, CPF exposure level, and all three annual developmental assessments.

The CCCEH cohort study found a statistically significant negative impact of prenatal exposure to PAH on MDI at age three years.[2] Infants in the highest quartile of PAH levels *in utero* (relative to three lowest quartiles) were more likely to be developmentally delayed, with an estimated odds ratio of 2.89. The study did not, however, find a statistically significant relationship between PAH exposure and MDI at ages one or two years or PDI at any ages.

For this paper, we used the measure of developmental delay—1.5 standard deviations below the mean (MDI score < 77.5)—mandated by N.Y. State, to provide the basis for pricing the costs of developmental delay (described in detail below). We maintain the same specification for the logistic model in terms of covariates. After adjusting for race/ethnicity, gestational age, age of child when developmental assessments were administered, HOME inventory score, sex of the child, and prenatal exposure to ETS and the pesticide chlorpyrifos, the model yielded an odds ratio of 3.3 (p-value = .037, 95% confidence interval [1.07,10.17]).

Results

Calculation of cost of PAH-related preschool special education

Because the longer-term effects of early developmental delay are difficult to monetize, we focus on enrollment in preschool special education to measure the costs associated with PAH exposure. U.S. law requires that states provide preschool special education to all children between the ages of three and five years who exhibit significant delay or disorder related to cognition, communication, adaptive behavior, social-emotional development, or motor development.[17] If a child scores at least 1.5 standard deviations below the mean in two or more functional areas, he or she is eligible for preschool special education services. For our present analysis, children with a score of more than 1.5 SDs below the mean on MDI and one other test (the PDI, which reflects motor development, or the Child Behavior Checklist (CBCL), which reflects behavior problems) were deemed eligible for preschool special education. Both the PDI and CBCL are frequently used assessments approved by New York State for evaluating children referred to the committee on preschool special education.[18]

To calculate the costs of developmental delay due to prenatal exposure to PAHs, we employed the Environmentally Attributable Fraction (EAF) method:[19]

$$[\text{Costs attributable to PAH exposure} = \text{Disease rate} \times \text{EAF} \times \text{Population at risk} \times \text{Cost per case}] \quad [1]$$

The EAF was developed by the Institute of Medicine[19] and has been extensively used by researchers to calculate the environmentally attributable costs of pediatric cancer, asthma, lead poisoning, neuro-developmental disabilities and methylmercury exposure.[20] For our analysis, we calculate the environmentally attributable costs associated with being in the top quartile of PAH exposure (the exposed group) relative to the lowest three quartiles of exposure (the unexposed group).

The disease rate is the background rate of developmental delay, which is .023 in the unexposed group, shown in Table 1. We define this by using the criteria for preschool special education: more than 1.5 SDs below the mean in two functional areas. Since there was a significant impact of PAH on one functional area only (MDI), we define the disease rate by combining the rate of delay for MDI with an area not significantly affected by PAH, either the CBCL or PDI. Since these outcomes are comorbid, we compute the overall rate of disease by multiplying the rate of MDI delay by the conditional rate for the CBCL or PDI outcome, where we condition on having MDI delay. For the area not significantly affected by PAH, we use the rate of delay on either the PDI or CBCL in the unexposed group conditional on having cognitive delay. The rate of delay for MDI (<77.5) in the unexposed group is 6.47%. Of those with MDI<77.5, 36.2% had either PDI scores at least 1.5 standard deviations below the mean or CBCL scores 1.5 standard deviations above the mean (64). Therefore, the disease rate is the product of the two ($0.023 = 0.0647 * 0.362$).

The EAF is the product of the prevalence of PAH exposure (the top quartile ($= 0.25$)) and the percentage risk increase in MDI delay associated with exposure. We compute the percentage risk increase by evaluating the risk difference in MDI delay from being in the top quartile of PAH exposure and dividing it by the mean rate of developmental delay in the unexposed group (lowest three quartiles). We obtain the risk difference by subtracting the predicted cumulative distribution function based on the logistic regression results with the estimated PAH odds ratio set to 3.3 from the predicted cumulative distribution function with the PAH odds ratio set to one, with both cumulative distribution functions evaluated at the means of the other independent variable. This procedure yielded a percentage risk increase

in having MDI<77.5 at three years of age of 106. Multiplying by 0.25 yields an EAF of 0.266, or 26.6%.

To define the population at risk, our goal was to choose a population in NYC with both comparable demographics and PAH exposure as the CCCEH cohort. We are unaware of personal monitoring of PAH exposure for other segments of the population in the city, so we instead used a group with comparable demographic characteristics. Although PAH exposure may have comparable biological impacts for all segments of the population, psychological factors related to poverty may compound the impact of PAH, and 90% of mothers in the CCCEH cohort were on Medicaid. Therefore, to be conservative, we restricted our analysis to Medicaid births in NYC in 2000. Recognizing concerns about extrapolating results from a small, specialized cohort, Table 2 presents some basic characteristics of the CCCEH cohort and all Medicaid births in NYC. Mothers from the CCCEH cohort are younger and more likely to be unmarried, but are generally comparable to other Medicaid mothers in the City.

To compute the costs per case we use the cost of preschool special education attendance, which was defined as the average annual cost per child for preschool special education during the approximated period of enrollment in the cohort (provided by the New York State Department of Education, CCCEH personal communication). Citywide, the total cost for preschool special education services in 2000–2001 was approximately $303 million for 19,525 children, yielding an annual cost per case of $17,266 (provided by the N.Y. State Department of Education, CCCEH personal communication). Since these services generally last for 2 years from ages 3 to 5, we multiply the annual cost by two to obtain a cost per case of $34,532.

Using the EAF method, there were 396 additional cases of developmental delay per NYC Medicaid birth cohort associated with being in the top quartile of PAH exposure. The estimated cost of preschool special education services for these extra cases was approximately $13.7 million.

Discussion

This analysis estimates the potentially high costs of publicly-funded special education services associated with cognitive developmental delay that may be attributed to prenatal PAH exposure for Medicaid births in a single year in New York City. We estimate the costs of providing preschool special education services to children with developmental delays due to PAH exposure in the highest quartile relative to the three lowest quartiles at $13.7 million per birth cohort or $68.5 million for 5 years. While there is substantial evidence of the association between PAH exposure and a wide range of health outcomes, to the best of our knowledge this is the first study to compute the health costs of preschool special education services associated with PAH exposure.

Although our results suggest quantifiable benefits to reducing PAH exposure in terms of reducing the costs of preschool special education, reducing PAH levels could have much larger health benefits to society. While preschool special education helps mitigate some of the impacts from developmental delay, cognitive delays may persist beyond preschool so that costs accrue throughout these children's lives. Furthermore, we have defined our population at risk as one with similar demographics, but given that PAHs are ubiquitous in urban environments, benefits may accrue to other groups as well, though their impacts may be smaller than what we demonstrate here. Moreover, PAH and diesel exposure has been linked to other outcomes, such as various cancers and childhood asthma,[14,21] so reductions in PAH may lead to improvements in other health outcomes as well.

A notable limitation of our analysis is that unmeasured confounding factors may explain some of the estimated relationship between PAH exposure and developmental delay, which would invalidate the assumption of the EAF approach that our estimates of the association between PAH exposure and developmental delay can be interpreted as a causal effect of PAH. For example, children exposed to high levels of PAH may also be exposed to high levels of other air pollutants that originate from motor vehicles and may impair development as well. Two factors may alleviate this concern. First, our estimates control for several important confounding factors, including measures of the mother's intelligence and child's health status at birth. Second, related findings from other observational analyses discussed above and experimental animal studies of PAHs also find evidence to support negative neurodevelopmental impacts from exposure to PAH.[22–26] Whether we have isolated a causal estimate, however, cannot be definitively ascertained from the observational analysis conducted.

An additional limitation of our analysis is the level of uncertainty involved in making cost calculations. Although we demonstrate that children from the Medicaid population have comparable baseline characteristics as those from the CCCEH cohort, we cannot ascertain whether they face comparable levels of PAH exposure city-wide. Furthermore, although we find a statistically significant relationship between PAH exposure and developmental delay, our estimate of an EAF of 26.6% is considerably larger than previous national estimates of EAF due to environmental exposure though part of the significant effect in our cohort could be due to high rates of social disadvantage in this population and the wide confidence intervals due to small sample size.[27] For example, if we use the lower confidence interval of our odds ratio of 1.17, we obtain a considerably lower EAF of 1.6%, which translates into 25 extra cases of developmental delay from exposure to the highest quartile of PAH and total costs of $849,714 in preschool special education services. Moreover, the CCCEH sample excludes smokers and those with other serious medical conditions, so if there is an interactive relationship between PAH exposure and any of these factors, our findings may not readily generalize to the entire Medicaid population. Given the paucity of data available to address these issues more effectively, we view these findings as a useful starting point for future researchers as well as policymakers who may need to act in the face of this uncertainty.

On the other hand, subsequent follow-up of CCCEH cohort children has shown persistent longer-term impacts of prenatal PAH exposure on cognitive (IQ) and behavioral/emotional development.[28] Our estimates of the potential damage from prenatal PAH exposure, as well as other evidence of adverse health impacts associated with exposure, support policies to address the high levels of ambient PAHs. One potential approach for reducing the levels of ambient PAH is through government regulation of ambient levels comparable to national ambient air quality standards as set forth in the Clean Air Act Amendments. Although the Clean Air Act lists several PAHs as hazardous substances, there are currently no ambient standards for PAHs in the U.S. However, the Occupational Safety and Health Administration has set a limit of 0.2 ng/m^3 of PAHs for occupational settings.[1,3] Several European countries have set non-mandatory ambient standards for PAHs. For example, the Netherlands has introduced the goal of reducing the annual levels of the PAH B[a]P to .5 ng/m^3, Belgium has set the ambient standard for BaP at 1 ng/m^3 and a guide value of .5 ng/m^3, and France and Sweden have set a guide value of PAH at 0.1 ng/m$^{3.29,30}$ In the CCCEH cohort study, personal air monitoring of pregnant women showed a mean PAH concentration of 3.3 ng/m^3, with a range of .3 ng/m^3 to 36.5 ng/m^{3,9} which is considerably higher than these standards.

There are several approaches for reducing PAH emissions: fuel efficiency and conservation, innovations in emissions technology, and use of alternative fuels that emit less PAH. For

example, biofuels made from vegetable oil significantly reduce PAH emissions by as much as 80%.[30–32] Although these technologies exist to reduce PAH emissions, such interventions may pose costs to society as well. Studies such as this suggest the need to factor health costs into calculations of the societal costs of energy alternatives.

This paper demonstrates significant economic costs from one aspect of cognitive developmental delay (reduced MDI on the Bayley Scales) associated with low level prenatal PAH exposure. We estimated the cost of preschool special education for children with PAH-related cognitive developmental delay to be over $13.7 million per birth cohort in New York City. The associated impacts are likely to be similar in other urban areas with comparable PAH levels. Our analysis supports the role of policies aimed at reducing the level of PAHs in the air in order to reduce the health impacts associated with PAH exposure.

Abbreviations

PAH	Polycyclic Aromatic Hydrocarbons
EAF	environmentally attributable fraction
MDI	mental development index
PDI	psychomotor development index
CBCL	child behavior checklist
CPF	chlorpyrifos
ETS	environmental tobacco smoke
CPSE	Committee on Preschool Special Education

Notes

1. Ravindra K, Sokhia R, Grieken RV. Atmospheric polycyclic aromatic hydrocarbons: source attribution, emission factors and regulation. Atmos Environ. 2008 Apr; 42(13):2895–921.

2. Perera FP, Rauh V, Whyatt RM, et al. Effect of prenatal exposure to airborne polycyclic aromatic hydrocarbons on neurodevelopment in the first 3 years of life among inner-city children. Environ Health Perspect. 2006 Aug; 114(8):1287–92. [PubMed: 16882541]

3. Agency for Toxic Substances and Disease Registry. ToxFAQs for polycyclic aromatic hydrocarbons. Atlanta, GA: Agency for Toxic Substances and Disease Registry; 2009. Available at: http://www.atsdr.cdc.gov/tfacts69.html

4. Dunbar JC, Lin CI, Vergucht I, et al. Estimating the contributions of mobile sources of PAH to urban air using real-time PAH monitoring. Sci Total Environ. 2001 Nov; 279(1–3):1–19. [PubMed: 11712588]

5. Nielsen T. Traffic contribution of polycyclic aromatic hydrocarbons in the center of a large city. Atmos Environ. 1996; 30(20):3481–90.

6. Rogge WF, Hilldemann LM, Mazurek MA, et al. Sources of fine organic aerosol. 2. Noncatalyst and catalyst-equipped automobiles and heavy-duty diesel trucks. Environ Sci Technol. 1993; 27:636–51.

7. Perera FP, Rauh V, Tsai WY, et al. Effects of transplacental exposure to environmental pollutants on birth outcomes in a multiethnic population. Environ Health Perspect. 2003 Feb; 111(2):201–5. [PubMed: 12573906]

8. Dejmek J, Solansky I, Benes I, et al. The impact of polycyclic aromatic hydrocarbons and fine particles on pregnancy outcome. Environ Health Perspect. 2000 Dec; 108(12):1159–64. [PubMed: 11133396]

9. Choi H, Jedrychowski W, Spengler J, et al. International studies of prenatal exposure to polycyclic aromatic hydrocarbons and fetal growth. Environ Health Perspect. 2006; 114:1744–50. [PubMed: 17107862]

10. Choi H, Perera FP, Jedrychowski W, et al. Relationship between personal, indoor, and outdoor level of PAHs. Environ Health Perspect. 2008; 117:1125–37.

11. Perera FP, Li T-y, Zhou Z-j, et al. Benefits of reducing prenatal exposure to coal burning pollutants to children's neurodevelopment in China. Environ Health Perspect. 2008 Oct; 116(10):1396–400. Epub 2008 Jul 14. [PubMed: 18941584]

12. Perera FP, Tang D, Tu YH, et al. Biomarkers in maternal and newborn blood indicate heightened fetal susceptibility to procarcinogenic DNA damage. Environ Health Perspect. 2004; 112(10): 1133–6. [PubMed: 15238289]

13. Tang D, Li T-y, Liu JJ, et al. Effects of prenatal exposure to coal-burning pollutants on children's development in China. Environ Health Perspect. 2008 May; 116(5):674–9. [PubMed: 18470301]

14. Miller RL, Garfinkel R, Horton M, et al. Polycyclic aromatic hydrocarbons, environmental tobacco smoke, and respiratory symptoms in an inner-city birth cohort. Chest. 2004 Oct; 126(4):1071–8. [PubMed: 15486366]

15. Bayley, N. Bayley scales of infant development. San Antonio, TX: Psychological Corp; 1993.

16. Caldwell, BM.; Bradley, RH. Home observation for measurement of the environment. Little Rock, AK: University of Arkansas Press; 1979.

17. New York City Department of Education. Standard operating procedures manual for the committees on preschool special education. New York, NY: New York City Department of Education; 2007. Available at: http://schools.nyc.gov/NR/rdonlyres/1107B798-2CB4-4CFF-8FB6-9C7CBB45EC5B/0/SOPM2001FINAL.pdf

18. New York State Education Department. State performance plan—indicator #7 related to early childhood outcomes. New York, NY: New York State Education Department; 2007. Available at: http://www.vesid.nysed.gov/specialed/spp/earlychild.pdf

19. Institute of Medicine. Costs of environmental-related health effects: a plan for continuing study. Washington, DC: National Academy Press; 1981.

20. Landrigan P, Sonawane B, Mattison D, et al. Chemical contaminants in breast milk and their impacts on children's health: an overview. Environ Health Perspect. 2002 Jun; 110(6):A313–5. [PubMed: 12055061]

21. Boström CE, Gerde P, Hanberg A, et al. Cancer risk assessment, indicators, and guidelines for polycyclic aromatic hydrocarbons in the ambient air. Environ Health Perspect. 2002 Jun; 110(Suppl 3):451–88. [PubMed: 12060843]

22. Wormley DD, Chirwa S, Nayyar T, et al. Inhaled benzo(a)pyrene impairs long-term potentiation in the F1 generation rat dentate gyrus. Cell Mol Biol (Noisy-le-grand). 2004 Sep; 50(6):715–21. [PubMed: 15641162]

23. Saunders CR, Ramesh A, Shockley DC. Modulation of neurotoxic behavior in F-344 rats by temporal disposition of benzo(a)pyrene. Toxicol Lett. 2002 Mar; 129(1–2):33–45. [PubMed: 11879972]

24. Wormley DD, Ramesh A, Hood DB. Environmental contaminant–mixture effects on CNS development, plasticity, and behavior. Toxicol Appl Pharmacol. 2004 May; 197(1):49–65. [PubMed: 15126074]

25. Saunders CR, Shockley DC, Knuckles ME. Fluoranthene-induced neurobehavioral toxicity in F-344 rats. Int J Toxicol. 2003 Jul-Aug;22(4):263–76. [PubMed: 12933321]

26. Saunders CR, Das SK, Ramesh A, et al. Benzo(a)pyrene-induced acute neurotoxicity in the F-344 rat: role of oxidative stress. J Appl Toxicol. 2006 Sep-Oct;26(5):427–38. [PubMed: 16858674]

27. National Research Council. Toxicological effects of methylmercury. Washington, DC: National Academy Press; 2000.

28. Perera FP. Prenatal polycyclic aromatic hydrocarbon exposure and child intelligence at age 5. Pediatrics. 2009 Jul 20; 124(2):195–202.

29. Nielsen, T.; Feilberg, A.; Binderup, M., et al. Impact of regulations of traffic emissions on PAH level in the air. Strandgade, Denmark: Danish Environmental Protection Agency; 2008.

30. Yang H-H, Chien S-M, Lo M-Y, et al. Effects of biodiesel on emissions of regulated air pollutants and polycyclic aromatic hydrocarbons under engine durability testing. Atmos Environ. 2007; 41:7232–40.

31. Pan, J.; Quarderer, S.; Smeal, T., et al. Comparison of PAH and Nitro-PAH emissions among standard diesel fuel, biodiesel fuel, and their blend on diesel engines. San Antenio, TX: Southwest Research Institute; 2008.

32. Correa SM, Arbilla G. Aromatic hydrocarbons emissions in diesel and biodiesel exhaust. Atmos Environ. 2006; 40:6821–6.

Table 1

COSTS DUE TO INCREASED PRESCHOOL SPECIAL EDUCATION SERVICES IN THE NEW YORK CITY MEDICAID POPULATION FROM ELEVATED POLYCYLIC AROMATIC HYDROCARBON EXPOSURE

Disease (delay) rate	0.023
EAF	0.266
Population at risk	63642
Extra # of cases	396
Cost per case	$34,532
Cost:	$13,674,672

EAF = Environmentally Attributable Fraction

Table 2

CHARACTERISTICS OF ALL NEW YORK CITY MEDICAID BIRTHS (IN 2000) AND CCCEH (COLUMBIA CENTER FOR CHILDREN'S ENVIRONMENTAL HEALTH) COHORT [a]

	2000 Medicaid births in New York City	CCCEH cohort
Maternal characteristics		
Age (years)	26.35	24.85
No High School degree	0.39	0.36
Unmarried	0.65	0.86
Hispanic	0.59	0.54
African American (non-Hispanic)	0.41	0.46
Infant characteristics		
Birthweight (grams)	3251.29	3400.37
Gestation (weeks)	38.74	39.35
Male	0.51	0.46
Sample size	63630	183

[a]Hispanic (African-American) in Medicaid sample is % of Hispanics (African Americans) relative to Hispanics and African Americans. We thank Ted Joyce for providing aggregate data on characteristics of Medicaid births.

CCCEH = Columbia Center for Children's Environmental Health