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Prenatal Exposure to Airborne Polycyclic Aromatic Hydrocarbons and IQ: Estimated Benefit of Pollution Reduction

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Abstract

Outdoor air pollution, largely from fossil fuel burning, is a major cause of morbidity and mortality in the U. S., costing billions of dollars every year in health care and loss of productivity[1]. The developing fetus and young child are especially vulnerable to neurotoxicants, such as polycyclic aromatic hydrocarbons (PAH) released to ambient air by combustion of fossil fuel and other organic material. Low income populations are disproportionately exposed to air pollution. Based on the results of a prospective cohort study in a low-income population in New York City (NYC) that found a significant inverse association between child IQ and prenatal exposure to airborne PAH, we estimated the increase in IQ and related lifetime earnings in a low income urban population as a result of a hypothesized modest reduction of ambient PAH concentrations in NYC of .25 ng/m^3. For reference, the current estimated annual mean PAH concentration is~ 1 ng/m^3. Restricting to NYC Medicaid births and using a 5% discount rate, we estimated the gain in lifetime earnings due to IQ increase for a single year cohort to be $215 million (best estimate). Using much more conservative assumptions, the estimate was $43 million. This analysis suggests that a modest reduction in ambient concentrations of PAH is associated with substantial economic benefits to children.

Keywords

PAH; IQ; cost-benefit analysis; neurodevelopment; prenatal; children

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Introduction

The economic impact of environmental pollutants in U.S. children is estimated to be $76.6 billion [2] and for neurobehavioral disorders related to environmental pollutants, $9.2 billion [3]. Here we have estimated the economic cost of prenatal exposure to a component of air pollution, polycyclic aromatic hydrocarbons (PAH), and the economic benefit of a modest reduction in ambient concentrations of the pollutants. The estimate is based on a damage function derived from a prospective cohort study in NYC, the Columbia Center for Children's Environmental Health (CCCEH) cohort study.

PAH are widespread urban pollutants released to the air during incomplete combustion of fossil fuel, tobacco, and other organic material. Urban minority populations represent high-risk groups both for disproportionate exposure to air pollution and for adverse health and developmental outcomes [4–8]. Environmental exposures during the prenatal and early postnatal stages are of particular concern because of the heightened susceptibility of fetuses and infants to diverse environmental pollutants, including neurotoxicants such as PAH [9–12]. PAH, such as benzo[a]pyrene (BaP), have been shown to be neurodevelopmental toxicants in experimental and epidemiological studies involving prenatal exposure [13, 14].

In the CCCEH cohort study, prenatal exposure to airborne PAH was associated with developmental delay at 3 years of age[15], reduced child IQ at age 5 [16]and behavioral problems at age 7[17]. The present analysis is based on CCCEH results for the association between prenatal PAH exposure and IQ. Change in IQ has been used by Grosse et al. to estimate gains in lifetime earnings for each year's U.S. cohort as a result of reduced exposure to lead (from $110 billion to $319 billion per year) and by Trasande et al. to estimate the lost economic productivity of U.S. children attributable to mercury emissions from American power plants ($1.3 billion, range, $0.1–6.5 billion) each year [18, 19]. Here, by focusing on a prevalent environmental exposure in a low income population of pregnant women and their children, this paper provides new policy-relevant data.

Methods

Data from the CCCEH Cohort Study provided the damage function for PAH and IQ

As previously described, the cohort comprised children of nonsmoking African American or Dominican American women and their children residing in Washington Heights, Harlem, or the South Bronx in NYC who were followed from *in utero* to 5 years of age [16]. Prenatal personal air monitoring of 8 nonvolatile PAH, including benzo[a]pyrene (B[a]P) was carried out during the third trimester of pregnancy over a 48-hour period as described [16]. Total PAH exposure was defined as the summation of these 8 PAH. Prenatal and postnatal questionnaires were administered to the mothers to obtain demographic, residential, history, health, and environmental data. At age 5, 249 children were assessed using the Wechsler Preschool and Primary Scale of Intelligence-Revised (WPPSI-R). Multiple linear regression models were used to test the associations between prenatal PAH levels (using a dichotomous measure or continuous (Ln) transformed PAH) and IQ, accounting for factors other than PAH exposures that are known to affect intellectual development. The associations between the continuous measure of PAH exposure and IQ scores were significant (full-scale IQ: beta

−3.00; *P* = .009; verbal IQ: beta −3.53; P = .002). The observed decrease in full-scale IQ in the CCCEH cohort among children above the median PAH exposure level compared to less exposed children was similar to that reported for children with lifetime average blood lead concentrations between 5 and 9.9 μg/dL compared with children with less exposed children[20].

Calculation of the cost of PAH-related IQ reduction

To derive estimates of the cost of IQ reduction attributable to prenatal PAH exposure, we followed the method used by Grosse et al. and Trasande et al. in their assessments of lead and mercury, respectively [18, 19]. We first calculated the estimated gain in IQ corresponding to the hypothesized reduction in ambient PAH exposure of .25 ng/m^3. For context, the mean ambient PAH level measured in several studies of outdoor/ambient urban air in the U.S. was ~1 ng/m^3. Data on ambient levels of PAH in Los Angeles (LA)(1998−2002) [21] and recent data (unpublished, courtesy of R. Miller) from a single stationary air monitor at West 168th and Broadway, NYC, showed average ambient levels to be about 1 ng/m^3 in both cities. Subtracting the mean indoor level of PAH monitored over several weeks in the homes of women participating in the CCCEH cohort study (2.08 ng/ m^3) [22] from the women's personal monitored mean level (3.13 ng/m^3) gives an estimated outdoor exposure concentration of ~1.00 ng/m^3. However, the mean personal air concentration of nonvolatile PAH in our cohort study (3.13 ng/m^3) reflected both indoor and outdoor exposures to PAH, not only because the daily activities of the women took place in both environments, but also because the indoor concentration of nonvolatile PAH resulted in large part from the transport of outdoor air into the indoor environment [23, 24].

To estimate the size of the population at risk in NYC, we followed Weiland et al. [25] and selected Medicaid births in NYC which shared basic socioeconomic characteristics of the CCCEH cohort. In the absence of data from personal monitoring of PAH in other segments of the population in the city, we were unable to select a population known to have the same personal exposure to PAH. However, the analysis is broadly relevant to policy on air pollution because PAH exposure is ubiquitous in the urban environment from fossil fuel burning, especially traffic. City-wide monitoring data showed variability in concentrations across the area, with pollution levels generally higher in lower income areas, [26] supporting the selection of the Medicaid population as the population at risk.

The total number of births in NYC in 2002 was 122,937 of which 63,462 were births to women on Medicaid [27]. We assumed comparable exposure to PAH during pregnancy as in the CCCEH cohort. For a single NYC birth cohort on Medicaid, we multiplied the estimated gain in IQ per hypothesized reduction in PAH exposure (above) by the estimated gain in earnings per IQ point (see Table 1).

For our “best case” (Case 1) estimate of the benefit of reducing PAH levels, we assumed that one IQ point results in a 1.1% increase in earnings based on Grosse [28] who reviewed all studies on IQ points and annual earnings through 2007. For our Case 2 (lower bound) estimate, we applied the lower end value of a .85% increase in earnings based on Heckman et al. [29]. We used the published estimate of discounted lifetime earnings (in 2000$) for a 2-year-old member of the birth cohort [18]. This is reasonable since most of the CCCEH cohort

was enrolled over a period of several years spanning 2000. Our primary analysis used a 5% discount rate; we have also provided an analysis using a 3% discount rate.

Results

Based on our prior study [16], a one LnPAH unit decrease is associated with a 3 point increase in IQ. Assuming log linearity of dose-response (based on our data), the effect on IQ of a specified reduction in PAH exposure can be calculated as:

$$IQ_2 - IQ_1 = \hat{\beta}\left(log\left(PAH_2\right) - log\left(PAH_1\right)\right) = \hat{\beta} log\left(\frac{PAH_2}{PAH_1}\right) where\ \beta = -3.$$

The IQ regression model is IQ = alpha + beta*log(PAH) = alpha −3.0*log(PAH). With two levels of PAH and two levels of IQ:

$$IQ_1 = \text{alpha} - 3.0^* log\left(\text{PAH}_1\right) \text{ and } IQ_2 = \text{alpha} - 3.0^* log\left(\text{PAH}_2\right).$$

So:

$$IQ_2 - IQ_1 = -3.0^*\left[log\left(\text{PAH}_2\right) - log\left(\text{PAH}_1\right)\right] = -3.0^* log\left(\text{PAH}_2/\text{PAH}_1\right)$$

For a reduction of .25ng/m^3, the best estimate of the gain in IQ is .86 point (−3*log[1/.75]). The lower bound of the effect of PAH on IQ is $-0.77\left[\hat{\beta} + 1.96^* se\left(\hat{\beta}\right), \text{where } \hat{\beta} = -3, se\left(\hat{\beta}\right) = 1.14\right]$. Using the lower bound estimate of the PAH effect, the gain in IQ corresponding to a reduction of .25ng/m^3 is .22 point (−0.77*log[1/.75]).

Based on Grosse et al. [18], we estimated the effect of one IQ point on lifetime earnings in our selected population. Grosse et al. used an estimate of the present value (PV) of future lifetime earnings (2000$) for a 2-year-old child, calculated using a 3% discount rate ($723,300), a 5% discount rate ($353,400), and a 0% discount rate. We have provided results using 5% and 3% discount rates as recommended by an expert panel convened by the U.S. Public Health Service [30]. Our primary analysis (see Table 1) was based on the 5% discount rate; in the text we also summarize the results based on the 3% discount rate.

The lifetime economic gain for each year's birth cohort as a result of the specified decrease in PAH exposure is given in Table 1. For a reduction of .25 ng/m^3 PAH in air, for each annual cohort of NYC Medicaid births, the Case 1 estimate is $215 million; and the Case 2 estimate is $43 million. The Case 1 estimate corresponds to a .96% increase in total earnings per person (3,382/353,400); the Case 2 estimate corresponds to a .18% increase in total earnings per person (671/353,400).

With a 3% discount rate, the PV of lifetime earnings was calculated to be $723,300 [18]. Using a 3% discount rate, the Case 1 estimate of the benefit per cohort of a reduction of .25ng/m^3 PAH (in 2000$) is $441 million; the Case 2 estimate is $87 million.

Discussion

This analysis estimates substantial gains in earnings associated with a modest reduction in ambient PAH concentrations, hence in prenatal exposure, for each annual cohort of Medicaid births in NYC. Given the paucity of data available on the cost of environmentally-related neurodevelopmental effects, we view our findings as a useful departure point for future researchers as well as policymakers, who often need to act in the face of uncertainty.

The study has several limitations. We have defined our population at risk as one with similar demographic characteristics as our study cohort; i.e., the NYC Medicaid population. However, the CCCEH cohort excludes smokers and those with other serious medical conditions, so if these factors interact with PAH on risk of IQ loss, our findings may underestimate the effect of exposure on IQ. As PAH are ubiquitous in urban environments, IQ and related earnings benefits are likely to accrue to other groups as well, although their impacts may be smaller than estimated here for a low income population. Further, the present analysis does not consider other potential benefits of exposure reduction such as reduction of asthma and cancer risk which have been associated with PAH and diesel exposure [31, 32]. Another limitation is that, although the present estimates of the association between prenatal PAH exposure and IQ are based on a peer-reviewed study that controlled for a number of important confounding factors, we cannot rule out the possibility of unmeasured confounding. Further, the models used to assess PAH impacts did not account for exposures to mercury and lead which may contribute to overestimation.

WPPSI-R verbal and full scale IQ scores during the preschool period have been shown in many studies to be predictive of subsequent elementary school performance [33, 34] and lifetime earnings [28]. However, while IQ provides the best documented relationship with economic criteria, there are a number of limitations to IQ as an outcome, including the difficulty in estimating the full impact of cognitive ability on earning potential [18]. Neurotoxins can influence both cognitive and non-cognitive ability; so economic analyses of the effects of reducing neurotoxin exposures on cognitive ability alone can understate the overall economic benefit.

There are a number of approaches for reducing PAH emissions including fuel efficiency and conservation, innovations in emissions control technology, and use of alternative fuels. However, an assessment of the costs of controlling emissions of PAH from diverse sources is beyond the scope of this analysis.

To the best of our knowledge, this is the first study to estimate the costs of IQ loss associated with PAH exposure and the corresponding benefits of PAH exposure reduction. It adds to the prior literature on economic costs of particulate air pollution that estimated sizable costs of the attributable morbidity and mortality [2, 35–38].

Our analysis suggests that a small reduction in ambient PAH would be associated with substantial economic benefits as measured by increased lifetime earnings. Next steps in research include a more comprehensive accounting of the impacts of air pollution, going beyond the narrow outcome of reduction of IQ to include broader neurotoxic, respiratory,

and carcinogenic effects and also considering the effects of early exposure over the lifecourse.

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Biography

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Table 1

Assumptions and Results of Calculations of the Economic Benefit to the 2000 Birth Cohort from a Hypothesized Reduction in PAH Exposure of .25 ng/m^3 (Adapted from Grosse et al., 2002)

Parameter	**Lower estimate (Case 2)**	**Best estimate (Case 1)**
A. Reduction in Ln(PAH) (ng/m^3)	.29[1]	.29[1]
B. IQ Ln(PAH)Slope	.77	3.0
C. Earnings- IQ Slope (%)	.85	1.1
D. Present value of earnings of 2-year-old (in 2000$) (5% discount rate)	353,400	353,400
E. Size of 2-year-old cohort at risk	63,462	63,462
Value of one IQ point (in 2000$) (C × D)	3,004	3,887
Benefit of reduction of .25 ng/m^3 PAH in terms of lifetime earnings per individual[2] (in 2000$) (A × B × C × D)	671	3,382
Benefit per cohort (in 2000$) (A × B × C × D × E)	42,583,002	214,628,484

[1] .29=log(l)–log(0.75)

[2] Gain in IQ for a reduction of .25ng/m^3: Case 1=.86; Case 2=.22 (see text for detail)