

Assessing the Health Benefits of Air Pollution Reduction for Children

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Benefit–cost analyses of environmental regulations are increasingly mandated in the United States. Evaluations of criteria air pollutants have focused on benefits and costs associated with adverse health effects. Children are significantly affected by the health benefits of improved air quality, yet key environmental health policy analyses have not previously focused specifically on children's effects. In this article we present a "meta-analysis" approach to child-specific health impacts derived from the U.S. Clean Air Act (CAA). On the basis of data from existing studies, reductions in criteria air pollutants predicted to occur by 2010 because of CAA regulations are estimated to produce the following impacts: 200 fewer expected cases of postneonatal mortality; 10,000 fewer asthma hospitalizations in children 1–16 years old, with estimated benefits ranging from \$20 million to \$46 million (1990 U.S.); 40,000 fewer emergency department visits in children 1–16 years old, with estimated benefits ranging from \$1.3 million to \$5.8 million; 20 million school absences avoided by children 6–11 years old, with estimated benefits of \$0.7–1.8 billion; and 10,000 fewer infants of low birth weight, with estimated benefits of \$230 million. Inclusion of limited child-specific data on hospitalizations, emergency department visits, school absences, and low birth weight could be expected to add \$1–2 billion (1990 US\$) to the \$8 billion in health benefits currently estimated to result from decreased morbidity, and \$600 million to the \$100 billion estimated to result from decreased mortality. These estimates highlight the need for increased consideration of children's health effects. Key needs for environmental health policy analyses include improved information for children's health effects, additional life-stage-specific information, and improved health economics information specific for children. *Key words:* air pollution, benefit, children, morbidity, mortality, risk assessment. *Environ Health Perspect* 112:226–232 (2004). doi:10.1289/ehp.6299 available via <http://dx.doi.org/> [Online 14 October 2003]

Ambient air pollution has been associated with a multitude of health effects, including mortality, respiratory and cardiovascular hospitalizations, changes in lung function, asthma attacks, and days lost from work (Bates 1995a; Pope 1996, 2000; Samet et al. 2000a, 2000b; Segala 1999). These studies have been performed in multiple cities around the United States and internationally using various designs and statistical methods.

Attempts have been made to consider the public health impacts of the criteria air pollutants [particulate matter (PM), ozone, carbon monoxide, sulfur dioxide, nitrogen dioxide, and lead]. The U.S. Clean Air Act (CAA) Amendments of 1990 (1990) included the provision (section 812) that the U.S. Environmental Protection Agency (U.S. EPA) perform periodic analyses of the benefits and costs of the CAA. A retrospective analysis of the benefits and costs from 1970 to 1990 compared the costs of implementation of the CAA and its regulations with the health and welfare effects avoided (benefits) because of decreases in criteria air pollutant concentrations and found that benefits outweighed costs between 11 and 95 times (U.S. EPA 1997). A prospective analysis examining the benefits and costs of criteria air pollutant reductions (excluding lead) from 1990 to 2010 found that benefits would outweigh costs by 4 to 1 in 2010 (U.S. EPA 1999a).

In retrospective and prospective analyses, the U.S. EPA attempted to analyze the effects of the criteria air pollutants on 20 health end points. Although some children's health effects were considered, these data were not comprehensive and were typically aggregated with estimates of impacts in adults. Lave and Seskin (1970) included infant mortality rates in their analysis of mortality attributable to air pollution in the 1960s. International and regional analyses have also been recently conducted but do not highlight child-specific impacts (Cifuentes et al. 2001; Hall et al. 1992; Murray and Lopez 1997).

In the United States, federal impetus has increased to include benefit–cost analyses when promulgating significant pieces of regulation (> \$100 million), or benefit–cost analyses may be included in new or amended legislation. Executive Order 12866 of 1993 (Clinton 1993) established principles for evaluating risks, benefits, and costs of proposed, existing, or final pieces of significant regulation. Similarly, U.S. Executive Order 13045 (Clinton 1997) has led to increased federal attention to the specific susceptibility of children. Regulatory analyses can be useful tools and provide valuable information for decision makers (National Research Council 2002).

Several studies examining the associations between ambient air pollution and health effects have focused exclusively on the health

effects of infants and children. This focus is important because children may be at higher risk than adults due to several factors, including differences in exposures, differences in age-specific activity patterns, and varying sensitivity during specific periods of development. Estimates based on U.S. surveillance data indicate that in 1999 asthma accounted for 658,000 emergency department visits, with children < 5 years old having the highest hospitalization and emergency department visit rates (Centers for Disease Control and Prevention 2002). Specific to children, associations with air pollution have been found for hospitalizations, increased symptoms, decreased lung function, low birth weight (LBW), and school absences (Bates 1995b). Associations with postneonatal mortality have been found for ambient pollution levels in the United States (Woodruff et al. 1997). Recent studies in the United States and internationally have found impacts on intrauterine death, birth outcomes, LBW, birth defects, and premature delivery (Bobak 2000; Bobak and Leon 1992; Bobak et al. 1999, 2001; Dejmek et al. 1999; Maisonet et al. 2000; Ritz and Yu 1999; Ritz et al. 2000, 2002; Rogers et al. 2000).

In this article, we expand an existing benefit–cost analysis framework to examine the impacts of the criteria air pollutants except lead on children's health and to quantify the health benefits associated with reductions in criteria air pollutants during the period 1990–2010. The U.S. EPA prospective (1990–2010) analysis has previously considered the following health end points specifically for children: postneonatal infant

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This study was funded by the Center for the Study and Improvement of Regulation at Carnegie Mellon University/University of Washington, the U.S. Environmental Protection Agency (EPA-R826886), and the National Institute of Environmental Health Sciences (NIEHS 1P01 ES09601). This article has not been reviewed by the U.S. EPA or NIEHS and should not be assumed to represent agency, department, or U.S. Government Accounting Office views.

The authors declare they have no competing financial interests.

Received 24 February 2003; accepted 14 October 2003.

mortality (not included in the U.S. EPA's main results); asthma hospitalizations; emergency department visits for asthma; acute bronchitis; upper and lower respiratory symptoms; and respiratory illnesses (U.S. EPA 1999a). We therefore performed our study to identify child-specific health end points that were not included in the prospective analysis, to update the literature where available, to estimate the national health impacts for children of reductions in the criteria air pollutants, and to conduct a preliminary estimation of the associated benefits. We used a model that was developed previously to follow the methodology of the U.S. EPA (1999a) while also considering inter- and intrastudy uncertainty, transparency, and best practice (Farrow et al. 2001). This model has been previously calibrated to and shown to approximate the U.S. EPA 1990–2010 analysis (Farrow et al. 2001).

Materials and Methods

To examine impacts of the criteria air pollutants on children, we surveyed the peer-reviewed air pollution literature (last search conducted April 2002) for studies focused exclusively on children or presenting results for children ≤ 18 years of age in the United States, and the impacts of one of the following: PM ≤ 10 μm and ≤ 2.5 μm in diameter (PM₁₀, PM_{2.5}), O₃, CO, NO₂, or SO₂. The effects of lead were not included because of low ambient concentrations during the time period 1990–2010. The methodology evolved from the U.S. EPA (1999a) and is briefly described below. More detail is presented in

Farrow et al. (2001). A literature search was performed in Medline (National Library of Medicine 2002) using the key words “child” (and all variants) and the pollutants of interest for studies examining the epidemiology-based exposure–response relationship between outdoor criteria air pollutant concentrations and children's health effects. All available studies were used in the analysis below except that of Rogers et al. (2000), which used an unusual exposure metric (total suspended particulates + SO₂). An important aspect of this meta-analysis type approach is the incorporation of exposure–response parameters and value estimates from multiple sources where possible. We modified results from published studies very little because our explicit intent was to capture the original authors' modeling and characterization. Information was extracted from each study on the study population and location, pollutants studied, exposure levels, statistical methods, exposure–response function used, lag structure, *International Classification of Diseases*, 9th revision, codes (U.S. Department of Health and Human Services 1991) or symptoms defining a case (if applicable), exposure–response parameter (β coefficient, relative risk, or percent change), and measure of error [standard error (SE) or confidence interval (CI)].

We included 23 original studies examining the association between a considered health effect and an air pollutant (Tables 1–3). In Table 1, health studies examining mortality and hospitalization outcomes are listed by health effect and pollutant considered.

Table 1 includes, for each study, summary information on key parameters and findings: the regression coefficient and SE describing the exposure–response relationship, exposure measure, and geographic locale considered. One Canadian study was also included because of geographic proximity and demographic similarity. Table 2 lists parameters and findings for respiratory ailments not requiring hospitalization. Table 3 details risk information for previously unconsidered child-specific health end points. All information presented in the tables was used in the analysis. Multiple values per pollutant presented for a single study may denote either differences in exposure measures or data sources. Data entry was validated through replication by a second member and quality assurance/quality control analysis by a third member of the group.

To estimate the expected cases averted for each health effect considered, we converted measures of risk to an exposure–response regression coefficient. Because most regressions were in a log-linear form, the term “regression coefficient” is used here and describes the general case. Regression coefficients reported in the study were used untransformed. Measures of risk reported as odds ratios or percent change were converted into a regression coefficient using the property that the natural logarithm of the odds ratio is the regression coefficient. For each study, the central tendency and variation of the exposure–response parameter were used. Study-specific exposure–response functions were used. Log-linear models were used by

Table 1. Data from studies analyzing children's health, mortality, and hospitalizations (ages denote those examined in the present study).

End points	Age (years)	Reference	Pollutant	Location, year	Risk coefficient (SE)	Exposure measure
Postneonatal mortality	1 month–1 year	Woodruff et al. 1997	PM ₁₀	Various U.S. cities, 1989–1991	0.00392 (0.0012) μg/m ³	Mean PM ₁₀ for first 2 months of life
Hospital admissions Respiratory	< 2, < 5	Pope 1991	PM ₁₀	Utah and Salt Lake Valleys, UT, 1985–1989	0.000149 (0.000068) μg/m ³ 0.000139 (0.00026) μg/m ³	Mean monthly PM ₁₀
	< 2	Burnett et al. 2001	O ₃	Toronto, Canada, 1980–1994	0.00661 (0.0014) ppb	Daily 1-hr maximum moving average
Asthma	1–16	Friedman et al. 2001	PM ₁₀	Atlanta, GA, 2000	-0.0223 (0.044) μg/m ³	3-day cumulative
			PM ₁₀		-0.0511 (0.041) μg/m ³	2-day cumulative
	O ₃		0.00 (0.0049) ppb	3-day cumulative		
	O ₃		0.00 (0.0045) ppb	2-day cumulative		
Emergency department visits for asthma	1–16	Sheppard et al. 1999	PM _{2.5}	Seattle, WA, 1987–1994	0.00250 (0.00095) μg/m ³	Daily average
			CO		0.0528 (0.019) ppm	Daily average
	1–16	Friedman et al. 2001	PM ₁₀	Atlanta, GA, 2000	0.0337 (0.029) μg/m ³	3-day cumulative
			PM ₁₀		0.0406 (0.026) μg/m ³	
			PM ₁₀		0.00953 (0.027) μg/m ³	2-day cumulative
			PM ₁₀		0.0262 (0.024) μg/m ³	
	1–16	Norris et al. 2000	O ₃		0.00673 (0.0033) ppb	3-day cumulative
			O ₃		0.00673 (0.0030) ppb	
O ₃				0.00673 (0.0031) ppb	2-day cumulative	
O ₃				0.00673 (0.0028) ppb		
1–16	Norris et al. 1999	PM ₁₀	Seattle, WA, 1995–1996	0.00892 (0.0035) μg/m ³	24-hr average	
		CO		0.180 (0.064) ppm	24-hr average	
1–16	Schwartz et al. 1993	SO ₂		0.0017 (0.0029) ppb	Maximum daily 1-hr average	
		PM ₁₀	Seattle, WA, 1995–1996	0.0113 (0.0035) μg/m ³	24-hr average	
1–16	White et al. 1994	PM ₁₀	Georgia, 1990	0.159 (0.066) ppm		
				0.00378 (0.0013) μg/m ³	4-day average	
				0.00198 (0.0042) μg/m ³	1-hr maximum	

many investigators and can be used to assess health impacts in the general form

$$\Delta \text{cases of health effect} = - \left(\text{baseline incidence} \times \left[\exp(-\beta \times \Delta PC) - 1 \right] \right) \times \text{population at risk,}$$

where β = study-specific regression coefficient and ΔPC = change in pollutant concentration. To allow comparability with previous studies, we used estimated expected average changes in annual air pollutant concentrations for the entire United States on a national level through 2010 based on our analysis of the U.S. EPA 1990–2010 study (Farrow et al. 2001). This led to estimated decreases in expected concentrations of criteria air pollutants (point estimates only): $PM_{10} = 2.85 \mu\text{g}/\text{m}^3$, $O_3 = 1.34 \text{ ppb}$, $CO = 1.68 \text{ ppm}$, $NO_2 = 9.4 \text{ ppb}$,

$SO_2 = 1.15 \text{ ppb}$ (Farrow et al. 2001; U.S. EPA 1999a). These changes in concentration are the estimated national average difference between projected criteria air pollutant concentrations in the year 2010 had the CAA not been in place versus those with the CAA in place.

The present analysis was estimated for a projected 2010 U.S. population ≤ 18 years old of 76,461,986 and projected age distribution of the population (U.S. Census Bureau 2002). Baseline rates were obtained from U.S. national sources (Adams and Marano 1995; Martin et al. 2002; U.S. EPA 1999a) or from individual studies (Gilliland et al. 2001; Ostro et al. 2001; Ware et al. 1986). For analyses of the health impacts, we combined regression coefficients from different studies of the same health outcome using inverse variance weighting methods to form a regression coefficient

specific to each end point and pollutant. Using the inverse variance weighting method, we weighted each study by its fractional contribution to the sum of the inverse variance of the considered studies (per end point, per pollutant). Results from multivariate regression models were used when available. To determine the impacts of all pollutants for each health outcome, we added together the impacts caused by separate pollutants. Many of the studies focused on children of a particular age or ethnic group, so the results presented below are for those specific groups. Pollutant-specific health impact estimates have two significant figures to assist with computation and to prevent growth in rounding errors; however, they are likely not significant to more than one figure. If the lower bound includes a negative number, estimates have not been truncated at 0,

Table 2. Data from studies analyzing impacts on children's health from nonhospitalization ailments (ages denote those examined in the present study).

End points	Age (years)	Reference	Pollutant	Location, year	Risk coefficient (SE)	Exposure measure
Upper respiratory symptoms	10–12	Pope et al. 1991	PM_{10}	Utah Valley, UT, 1989–1990	0.0036 (0.0015) $\mu\text{g}/\text{m}^3$	Same day
Lower respiratory symptoms	7–14	Schwartz et al. 1994	$PM_{2.5}$	6 U.S. cities, 1984–1988	0.0182 (0.0059) $\mu\text{g}/\text{m}^3$	Daily average
Acute bronchitis	8–12	Dockery et al. 1996	$PM_{2.1}$	24 U.S./Canadian cities, 1988–1990	0.0272 (0.017) $\mu\text{g}/\text{m}^3$	Annual average
Respiratory illness	6–7	Hasselblad et al. 1992	NO_2	Meta-analysis	0.0275 (0.013) ppb	Annual change
Moderate or worse asthma	8–13 AA	Ostro et al. 2001	PM_{10}	Los Angeles, CA, 1993	0.00534 (0.0022) $\mu\text{g}/\text{m}^3$	1-hr maximum
			O_3		0.00349 (0.0004) ppb	1-hr maximum
Shortness of breath, chest tightness, or wheeze	8–13 AA	Ostro et al. 2001	PM_{10}		0.00231 (0.0017) $\mu\text{g}/\text{m}^3$	24-hr average
			$PM_{2.5}$		0.00561 (0.0016) $\mu\text{g}/\text{m}^3$	
			O_3		0.00194 (0.0008) $\mu\text{g}/\text{m}^3$	12-hr average
			NO_2		0.000985 (0.0007) $\mu\text{g}/\text{m}^3$	
			O_3		–0.00155 (0.0008) ppb	1-hr maximum
			NO_2		–0.00181 (0.0008) ppb	
			NO_2		0.00154 (0.0006) ppb	1-hr maximum
			NO_2		0.000591 (0.0006) ppb	
	6–9	Peters et al. 1999	PM_{10}	Southern California, 1986–1990	0.00195 (0.0035) $\mu\text{g}/\text{m}^3$	24-hr average
			O_3		0.00192 (0.0028) ppb	1-hr maximum
			NO_2		0.00345 (0.0048) ppb	24-hr average
	6–9	Ware et al. 1986	PM_{10}	6 cities, 1979–1980	0.0101 (0.0018) $\mu\text{g}/\text{m}^3$	24-hr average
			PM_{10}		0.0103 (0.0046) $\mu\text{g}/\text{m}^3$	
			SO_2		0.0028 (0.0033) $\mu\text{g}/\text{m}^3$	
Shortness of breath	8–13 AA	Ostro et al. 1995	SO_2	Los Angeles, CA, 1992	0.0091 (0.003) ppb	24-hr average
			PM_{10}		0.00841 (0.0036) $\mu\text{g}/\text{m}^3$	24-hr average
			O_3		0.00420 (0.0013) ppb	1-hr maximum
	8–13 AA	Ostro et al. 2001	PM_{10}	Los Angeles, CA, 1993	0.00771 (0.0026) $\mu\text{g}/\text{m}^3$	24-hr average
			$PM_{2.5}$		0.00257 (0.0013) $\mu\text{g}/\text{m}^3$	12-hr average
			O_3		0.000249 (0.0011) ppb	1-hr maximum
			NO_2		0.00154 (0.0009) ppb	1-hr maximum

AA, African-American asthmatics.

Table 3. Data from studies analyzing children's school absences and birth impacts (ages denote those examined in the present study).

End points	Age (years)	Reference	Pollutant	Location, year	Risk coefficient (SE)	Exposure measure
School absences	9–10	Gilliland et al. 2001	PM_{10}	Los Angeles area, 1996	–0.00440 (0.018) $\mu\text{g}/\text{m}^3$	24-hr average
			O_3		0.0302 (0.014) ppb	8-hr average
			NO_2		0.0179 (0.032) ppb	24-hr average
	9–10, 6–11	Ransom and Pope 1992	PM_{10}	Utah Valley, UT, 1985–1986 to 1990–1991	0.0219 (0.0046) $\mu\text{g}/\text{m}^3$	28-day average
			PM_{10}		0.0212 (0.0046) $\mu\text{g}/\text{m}^3$	
	9–10, 6–11	Chen et al. 2000	PM_{10}	Washoe County, NV, 1996–1998	–0.0154 (0.0044) $\mu\text{g}/\text{m}^3$	24-hr average
			O_3		0.0132 (0.0049) ppb	1-hr maximum ^a
			CO		0.193 (0.072) ppm	1-hr maximum
LBW	Singleton, first birth	Maisonet et al. 2001	PM_{10}	U.S. cities, ^b 1994–1996	–0.00408 (0.0047) $\mu\text{g}/\text{m}^3$	Average for 3rd month of pregnancy
			CO		0.270 (0.11) ppm	
			SO_2		0.000995 (0.0085) ppb	
			CO	Southern California, 1989–1993	0.0289 (0.029) ppm	Average last trimester
Ventricular septal defect	Singleton	Ritz and Yu 1999	CO	Southern California, 1987–1993	0.122 (0.15) ppb	Average for 2nd month of pregnancy
		Ritz et al. 2002	O_3		0.285 (0.15) ppm	
			CO		0.285 (0.15) ppm	

^aPreceding 14 days. ^bBoston and Springfield, MA; Hartford, CT; Philadelphia and Pittsburgh, PA; and Washington, DC.

to provide a sense of the uncertainty associated with results. We used available data for each pollutant and health effect considered in a one-dimensional 10,000-run Monte Carlo simulation to determine the health impacts (Burmester and Anderson 1994; Thompson et al. 1992). Monte Carlo simulation is a statistical technique that allows for the propagation of uncertainty in an analysis. Use of this technique allows us to use information about the uncertainty around the point estimate of effect reported in the original, peer-reviewed studies. Analyses were conducted in Microsoft Excel (Microsoft Corp., Redmond, WA) and used Decisioneering Crystal Ball (Decisioneering Inc. 2000), an Excel add-on that allows an analyst to perform Monte Carlo simulations.

To consider the potential economic impacts, benefits were the present value benefits of considered health effects as estimated in published studies. Estimates of the reduced number of cases can be combined with existing economic valuation data to determine the benefits associated with reductions in these illnesses. We determined mortality estimates using existing recommendations for the value of a statistical life at \$4.8 (SD = 3.2) million in 1990 US\$ (U.S. EPA 1999a). Not all health end points considered have available valuation data that are specific for children. Limited child-specific information on the present value of asthma hospitalizations, emergency department visits for asthma, school absences, and LBW is available (Hall et al. 2002; Smith et al. 1997; U.S. EPA 1999b; Weiss et al. 1992). Smith et al. (1997) estimated a value of \$4,900 (SD = 1,300) in 1990 US\$ for each asthma hospitalization, \$220 (SD = 42) per emergency department visit, and \$42 (SD = 8) per school absence. Alternatively, point estimates of values associated with select health end points are available. U.S. EPA (1999b) estimates a value of \$18,000 (1990 US\$) for all medical costs associated with LBW in infancy, an average annual cost of \$34 per patient for emergency department visits, and \$2,000 per asthma hospitalization. Yet other alternative data from Weiss et al. (1992) for children \leq 17 years old value each case at \$2,200 (1990 US\$) per asthma hospitalization, \$150 per emergency department visit, and \$100 per school absence. Unlike the latter two studies, in Smith et al. (1997) CIs are used to represent uncertainty, but no information is presented that allows child-specific values to be estimated. Estimates from Weiss et al. (1992) and estimates of average hospitalization costs from U.S. EPA (1999b) were specific for children < 18 years old; however, only point estimates were presented. Although there are moderate differences in the estimates presented by the three sources, no one source can be considered preferable to the others. We adjusted valuation estimates from these studies to 1990 US\$ by

using the Consumer Price Index Medical Care (U.S. Department of Labor Bureau of Labor Statistics 2002) to facilitate comparison with prior analyses, particularly that of U.S. EPA (1999a), which reports values in 1990 US\$.

Results

In this study, we analyzed children's health impacts from changes in ambient concentrations of the criteria air pollutants (excluding lead) due to the CAA from 1990 through 2010 (Figure 1). Reductions in PM₁₀ in the year 2010 were estimated to lead to a median of 160 (5th–95th percentiles; 90% CI, 45–270) fewer expected cases of postneonatal mortality (from 1 month to 1 year of birth); 3,000 (500–6,000) fewer respiratory hospitalizations in children 0–2 years of age, or 10,000 (1,000–20,000) fewer in children 0–5 years of age; and 1,300 (480–6,600) fewer emergency department visits in children 1–16 years of age (Figure 1, Table 4). Approximately 2.5 (–1.8 to 3.5) million school absences may also be avoided by children 6–11 years old in the United States.

Small reductions in ambient O₃ concentrations may lead to 700 (400–1,000) fewer respiratory hospitalizations in children ages 0–2 and 1,600 (220, 3,000) fewer emergency department visits for asthma in children ages 1–16, or 750,000 (470,000–1,000,000) fewer school absences in children ages 6–11 (Figure 1, Table 4). Reductions in CO concentrations may lead to 9,400 (4,200–19,000) fewer asthma hospitalizations or 35,000 (8,800–66,000) emergency department visits in children ages 1–16 years. NO₂ reductions may lead to 2.2 (–8.4 to 7.9) million fewer school absences in children ages 9–10.

Combining the pollutant-specific estimates, the specified changes in criteria air pollutant concentrations would lead to a total of 10,000 (4,000–20,000) averted asthma hospitalizations and 40,000 (10,000–70,000) fewer emergency department visits in children ages 1–16. Approximately 20 (10–20) million fewer school absences in children ages 6–11 and 10,000 (–20,000 to 70,000) averted LBW infants may also be expected (Table 4).

Estimated reductions in air pollution concentrations in 2010 could lead to reduced postneonatal mortality with estimated benefits of \$590 (\$150–1,300) million (1990 US\$). Because we are aware of only three sources of valuation data, we report valuation results separately using each of the three studies (all 1990 US\$). Using estimates from Smith et al. (1997), benefits of \$46 (\$17–84) million in asthma hospitalizations, \$5 (\$2–11) million in emergency department visits for asthma, and \$700 (\$400–1,000) million in school absences are estimated (Table 5). Using estimates from U.S. EPA (1999b) results in benefits of \$20 (\$6–42) million for asthma hospitalizations,

\$1.3 (\$0.3–3) million for emergency department visits, and \$230 (–\$500 to 1,400) million for LBW costs during infancy (Table 5). Using estimates from Weiss et al. (1992) for children < 17 years old leads to benefits of \$22 (\$7–47) million in asthma hospitalizations, \$5.8 (\$1.3–12) million for emergency department visits, and \$1.8 (\$0.75–2.4) billion for school absences in children 6–11 years of age (Table 5).

Discussion

Given the currently available health literature on children's health effects associated with the criteria air pollutants (excluding lead) and the limited literature on the valuation of children's health, this analysis should be considered as a starting point and as identifying key research needs for examining a unique and susceptible population in benefit–cost or cost-effectiveness analyses of environmental policies. When additional information on health effects and detailed economic valuation data for children are available, the data can be combined and used in analyses of environmental policies. The results of the present analysis are not intended to and cannot provide absolute estimates of the benefits to children associated with decreases in ambient criteria air pollutant levels, but can provide information about the importance of children's health effects in aggregate studies based on an assessment of order of magnitude or ranges of expected health benefits.

The magnitude of omitting children's health impacts can be seen when comparing the mean impact results here with mean U.S. EPA (1999a) results. Inclusion of child-specific data on asthma hospitalizations, emergency department visits for asthma, school absences, and LBW could be expected to add between \$1 and \$2 billion (depending on source of valuation estimates) to the \$8 billion (1990 US\$) in mean health benefits from decreased morbidity currently estimated by U.S. EPA (1999a) for the U.S. population. Consideration of postneonatal mortality would add \$600 million to the present mean estimate of \$100 billion resulting from decreases in adult mortality (U.S. EPA 1999a). Decreases in adult mortality were the key driver in prior analyses of benefits and costs. Although the benefits to infants are a small percentage (< 1%) of the estimated benefits to adults, benefits were estimated for adults \geq 30 years old, whereas benefits estimated here are for infants only between 1 month and 1 year of age. Future research examining the susceptibility of children and young adults is needed.

Availability of data for additional children's health end points and consideration of other metrics (e.g., life-years) may be expected to increase estimates of children's health benefits.

Estimates of morbidity benefits were assessed using cost of illness methods and are considered lower bounds of the estimate (Krupnick 2003; U.S. EPA 1999b). Our current analysis presents results only for specific age or ethnic groups and does not extrapolate findings to the total population of U.S. children. Extrapolation to all children would be expected to lead to an increase in the number of cases averted. Thus, estimated health benefits in this analysis may be considered conservative. The "value of a statistical life" method has long been a subject of contention (Viscusi 1993). Recent attention has focused on using life-years lost or valuing a life without using an age adjustment. To provide comparisons with the U.S. EPA's estimates of the benefits and costs of the CAA, each estimated premature death of a child was valued at the same value as an adult. These approaches have recently been affirmed by the federal government in the face of opposition to the "senior

discount" implicit in valuing life-years saved (Graham 2003; Whitman 2003).

The above estimates for the child-specific impacts of the CAA are comparable with recent studies. Via the modified Delphi technique (an expert elicitation method), 30% of acute exacerbations of pediatric asthma were estimated to be environmentally related, for an estimated \$2 billion (1997 US\$) per year in environmentally attributable costs of pediatric asthma in the United States (Landrigan et al. 2002). U.S. EPA (1999a) estimated the benefits associated with 950,000 upper respiratory symptoms, 520,000 lower respiratory symptoms, and 330,000 respiratory illness cases to reach \$19 million, \$6 million, and \$6 million (1990 US\$) per year, respectively.

In the present analysis, benefits resulting from reduced numbers of LBW infants arose from the first year of birth only, but LBW may also have lifelong effects on health and productivity. Lifetime medical costs due to

LBW of \$436,000 (1996 US\$, undiscounted) have been estimated (U.S. EPA 1999b). Use of lifetime estimates rather than those from the first year of birth alone would lead to increased estimates of health benefits. The benefits of reduced birth defects were not explicitly calculated in the present analysis because of mismatches in health and economic end points. However, given available economic data on other types of cardiac defects, additional benefits in the hundreds of millions could be inferred.

Many assumptions were included in the present analysis. Prior analyses have focused on geographically detailed estimates of exposures, whereas we did not include modeling of ambient exposures, regional variation, or human activity patterns affecting exposure. Average nationwide ambient concentrations were estimated from U.S. EPA (1999a). Although this approximation neglects the seasonal and regional variation of ambient air pollution, our purpose was to generate a preliminary estimate of the impacts in children in the entire United States over time. The extent to which air pollutants are merely a marker for some other compound will also affect the findings of this analysis. Because of the complex interplay of copollutants found in the United States, it may be difficult to distinguish the role of each pollutant.

The results of this analysis depend on the available information in the peer-reviewed literature. Some of the studies used in the present analysis (e.g., Woodruff et al. 1997) were not included in the U.S. EPA (1999a) analysis, reportedly because of lack of confidence in the new end point of postneonatal mortality. Child-specific associations are the focus of this

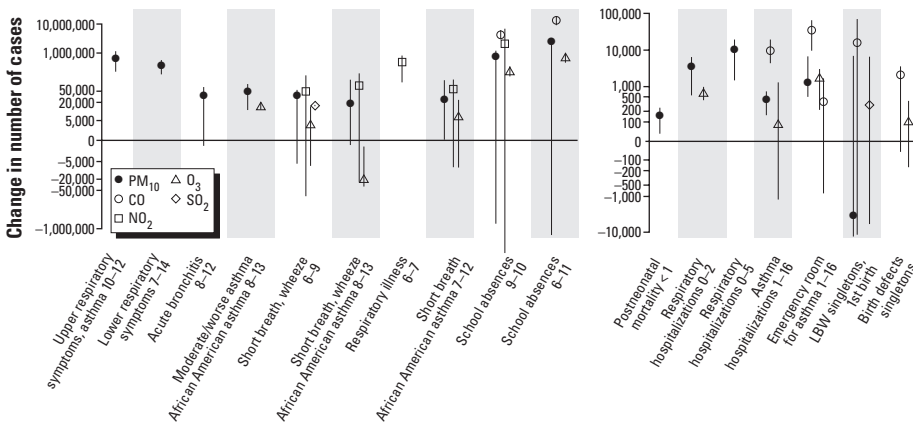


Figure 1. Estimated reductions in 2010 in health end points considered for specific age groups (years), by pollutant: 50th percentile presented with error bars indicating 5th–95th percentile.

Table 4. Estimated reduced numbers of select health end points by pollutant.

Pollutant	Postneonatal mortality ^a (1 month–1 year)	Asthma hospitalizations ^a (1–16 years)	Emergency department visits ^a (1–16 years)	School absences ^{a,b}		LBW ^a (singletons)
				(9–10 years)	(6–11 years)	
PM ₁₀	160 (45–270)	430 (150–700)	1,300 (480–6,600)	0.85 (–0.67 to 1.2)	2.5 (–1.8 to 3.5)	–3,300 (–13,000 to 7,200)
O ₃	—	–10 (–1,300 to 1,300)	1,600 (220–3,000)	0.26 (0.16–0.37)	0.75 (0.47–1.0)	—
CO	—	9,400 (4,200–19,000)	35,000 (8,800–66,000)	4.6 (3.0–6.4)	14 (8.5–19)	16,000 (–13,000 to 69,000)
NO ₂	—	—	—	2.2 (–8.4 to 7.9)	—	—
SO ₂	—	—	380 (–920 to 1,600)	—	—	300 (–6,000 to 6,500)
Total ^c	—	10,000 (4,000–20,000)	40,000 (10,000–70,000)	8.0 (–2.0 to 10)	20 (10–20)	10,000 (–20,000 to 70,000)

—, data unavailable. Note lack of available child-specific data for some end points and age groups.

^a50th percentile [5th–95th percentile (90% CI)]. ^bIn millions. ^cPollutant-specific estimates have two significant figures to assist with computation; totals may not add due to rounding.

Table 5. Values per case from the literature and estimated benefits associated with age-group-specific reductions in select health end points (1990 US\$).

End point	Value per case ^a			Estimated benefits (in millions) ^b		
	Smith et al. (1997)	U.S. EPA ^c (1999a, 1999b)	Weiss et al. (1992)	Smith et al. (1997)	U.S. EPA (1999a, 1999b)	Weiss et al. (1992)
Postneonatal mortality	—	4.8 ± 3.2 million	—	—	590 (150–1,300)	—
Asthma hospitalizations	4,900 ± 1,300	2,000	2,200	46 (17–84)	20 (6–42)	22 (7–47)
Emergency department visits	220 ± 42	34 ^d	150	5 (2–11)	1.3 (0.3–3)	5.8 (1.3–12)
School absence ^e	42 ± 8	—	100	700 (400–1,000)	—	1,800 (750–2,400)
LBW	—	18,000	—	—	230 (–500 to 1,400)	—

—, data unavailable.

^aMean ± SE. U.S. EPA (1999b) and Weiss et al. (1992) are point estimates only. ^bMonetized value based on each study. 50th percentile [5th–95th percentile (90% CI)]. ^cMortality estimates from U.S. EPA (1999a); morbidity estimates from U.S. EPA (1999b). ^dAnnual average cost per patient. ^eEstimate of cases averted in children 6–11 years old.

preliminary study, and all available evidence was used. Any choices or assumptions made by the original investigators (e.g., choice of exposure-response function, adjusting for key variables) are implicitly included in this analysis. Differences in study design, regional air pollutant characteristics, and sources of data used may affect the comparability of studies but should not significantly affect their aggregate interpretation. The results of this analysis may be affected by results of the effort to reanalyze results of several existing studies because of software errors (Health Effects Institute 2002). However, the impact on the present analysis is not expected to be significant because few studies used the affected statistical procedure.

Future quantitative analyses of children's health benefits may wish to evaluate effects separately for different ages in order to use other metrics such as years of life lost or quality-adjusted life-years (Fabian 1994). Increased data from cohort studies will allow estimation of life-years for use in these measures (National Research Council 2002). An age-specific analysis may better reflect higher-risk age groups. For example, Ransom and Pope (1992) reported absences by grade, with a higher effect seen in the younger grades. Similarly, Burnett et al. (1994) found that infants ≤ 1 year old had significantly more respiratory hospitalizations (15% of admissions) associated with air pollution than did the middle-aged and elderly groups (4%) examined.

For many of the children's health effects considered, the paucity of existing health effects data and economic data represents an opportunity for health researchers to present informative end points and data for economic analyses. Similarly, it presents an opportunity for health economists to provide key data for a susceptible population and an opportunity for increased collaboration between health risk assessors and economists for improved environmental health decision making. To take advantage of these opportunities, agencies need to consider funding priorities in children's health and health economics.

The estimates of health benefits are conservative. Estimates of the number of cases averted are presented only for specific age groups. Some health effects were unable to be included in the analysis. Estimates of the benefits are also low because we present data for only a subset of health effects considered (those with economic valuation information available on children's health effects). Cost-of-illness estimates do not include pain and suffering, altruism, or lost leisure time. However, the results of this analysis suggest that air pollution imposes a significant burden on U.S. children and have also allowed us to identify significant data gaps that impede our understanding of the full benefits of air pollutant reductions for children's health.

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