Appendix E: Ecological Effects of Criteria Pollutants

Introduction

Benefits to human welfare from air pollution reductions achieved under the CAA can be expected to arise from likely improvements in the health of aquatic and terrestrial ecosystems and the myriad of ecological services they provide. For example, improvements in water quality stemming from a reduction in acid deposition-related air pollutants (e.g., SO_x and NO_x) could benefit human welfare through enhancements in certain consumptive services such as commercial and recreational fishing, as well as non-consumptive services such as wildlife viewing, maintenance of biodiversity, and nutrient cycling. Increased growth and productivity of U.S. forests could result from reduced emissions of ozone-forming precursors, particularly VOCs and NO_x, and thus may yield benefits from increased timber production; greater opportunities for recreational services such as hunting, camping, wildlife observation; and nonuse benefits such as nutrient cycling, temporary CO_2 sequestration, and existence value.

In this Appendix, the potential ecological benefits from CAA pollutant controls are discussed in the context of three types of ecosystems: aquatic, wetland, and forest. In describing the potential ecological benefits of the CAA, it is clearly recognized that this discussion is far from being comprehensive in terms of the types and magnitude of ecological benefits that may actually have occurred from the implementation of the CAA. Rather, this discussion reflects current limitations in understanding and quantifying the linkages which exist between air quality and ecological services, in addition to limitations in the subsequent valuation of these services in monetary terms. This discussion also does not cover potential benefits from improvements in other ecological services, namely agriculture and visibility, which are discussed and quantified in other sections of this report. This appendix is dedicated to a *qualitative* evaluation of ecological benefits. However, where possible, the existing body of scientific literature is drawn upon in an attempt to provide insights to the possible magnitude of benefits that may have resulted from CAA-related improvements of selected ecological services. It is important to note that the inability to fully value ecological services results in a significant undervaluation of the ecological benefits of air pollution reductions. This undervaluation should not be interpreted as a devaluation.

Benefits From Avoidance of Damages to Aquatic Ecosystems

Aquatic ecosystems (lakes, streams, rivers, estuaries, coastal areas) provide a diverse range of services that benefit the welfare of the human population. Commercially, aquatic ecosystems provide a valuable food source to humans (e.g., commercial fish and shellfish harvesting), are used for the transportation of goods and services, serve as important drinking water sources, and are used extensively for irrigation and industrial processes (e.g., cooling water, electrical generation). Recreationally, water bodies provide important services that include recreational fishing, boating, swimming, and wildlife viewing. They also provide numerous indirect services such as nutrient cycling, and the maintenance of biological diversity.

Clearly, these and other services of aquatic ecosystems would not be expected to be equally responsive to changes in air pollution resulting from the implementation of the CAA. The available scientific information suggests that the CAA-regulated pollutants that can be most clearly linked to effects on aquatic resources include SO_x and NO_x (through acid deposition and increases in trace element bioavailability), NO_x (through eutrophication of nitrogen-limited water bodies), and mercury (through changes in atmospheric deposition). Potential benefits from each of these processes (acid deposition, eutrophication, mercury accumulation in fish) are described separately in the following sections.

Acid Deposition

Background

Acid deposition refers to the depositing of strong acids (e.g., H_2SO_4, HNO_3) and weak acids ($(NH_4)_2SO_4$, NH_4NO_3) from the atmosphere to the earth's surface. Acid deposition can occur in the wet or dry form and can adversely affect aquatic resources through the acidification of water bodies and watersheds. Acidification of aquatic ecosystems is of primary concern because of the adverse effects of low pH and associated high aluminum concentrations on fish and other aquatic organisms. Low pH can produce direct effects on organisms, through physiological stress and toxicity processes, and indirect effects, mediated by population and community changes within aquatic ecosystems. Acidification can affect many different aquatic organisms and communities. As pH decreases to 5.5, species richness in the phytoplankton, zooplankton, and benthic invertebrate communities decreases.[1] Additional decreases in pH affect species richness more significantly, and may sometimes affect overall biomass.[2] Table E-1 presents descriptions of the biological effects of acidification at different pH levels. In evaluating the severity of biological changes due to acidification, the reversibility of any changes is an important consideration; biological populations and communities may not readily recover from improved water quality under certain circumstances. Researchers have addressed acidification effects through many different experimental protocols, including laboratory bioassays, particularly concerning pH, aluminum, and calcium; manipulative whole-system acidification studies in the field; and comparative, nonmanipulative field studies.

Although acidification affects phytoplankton, zooplankton, benthic invertebrates, fish, amphibians, and waterfowl, most acidification research has concentrated on fish populations.[3] Aluminum, which can be toxic to organisms, is soluble at low pH and is leached from watershed soils by acidic deposition.[4] Acidification may affect fish in several ways. The direct physiological effects of low pH and high aluminum include increased fish mortality, decreased growth, and decreased reproductive potential. The mechanism of toxicity involves impaired ion regulation at the gill.[5] Population losses occur frequently because of recruitment failure,[6] specifically due to increased mortality of early life stages.[7] Changes at other trophic levels may affect fish populations by altering food availability.[8] Fish in poorly buffered, low pH water bodies may accumulate higher levels of mercury, a toxic metal, than in less acidic water bodies, due to increased mercury bioavailability. The primary consequence of mercury accumulation appears to be hazardous levels to humans and wildlife who consume fish, rather than direct harm to aquatic organisms (discussed further below).

The CAA-regulated pollutants that are likely to have the greatest effect on aquatic ecosystems through acid deposition and acidification are SO_2 and NO_x. In the atmosphere, SO_2 and NO_x react to form sulfate and nitrate particulates, which may be dry-deposited; also the pollutants may react with water and be wet-deposited as dilute sulfuric and nitric acids. SO_2 is considered the primary cause of acidic deposition, contributing 75 to 95 percent of the acidity in rainfall in the eastern United States.[9]

Current Impacts of Acid Deposition

Effects on Water Chemistry

The effects of acid deposition and resulting acidification of water bodies was intensively studied as part of a 10-year, congressionally-mandated study of acid rain problems in the United States.[10] Based on the NAPAP study, it is estimated that 4 percent of the lakes and 8 percent of the streams in acid-sensitive

[1] J. Baker et al., NAPAP SOS/T 13, 1990; Locke, 1993.

[2] J. Baker et al., NAPAP SOS/T 13, 1990.

[3] NAPAP, 1991.

[4] J. Baker et al., NAPAP SOS/T 13, 1990.

[5] J. Baker et al., NAPAP SOS/T 13, 1990.

[6] Rosseland, 1986.

[7] J. Baker et al., NAPAP SOS/T 13, 1990.

[8] Mills et al., 1987.

[9] NAPAP, 1991.

[10] NAPAP, 1991.

Table E-1. Summary of Biological Changes with Surface Water Acidification.

pH Decrease	Biological Effects
6.5 to 6.0	Small decrease in species richness of phytoplankton, zooplankton, and benthic invertebrate communities resulting from the loss of some acid-sensitive species, but no measurable change in total community abundance or production. Some adverse effects (decreased reproductive success) may occur for acid-sensitive fish species (e.g., fathead minnow, striped bass).
6.0 to 5.5	Loss of sensitive species of minnows and dace, such as blacknose dace and fathead minnow; in some waters decreased reproductive success of lake trout and walleye. Distinct decrease in the species richness and change in species composition of the phytoplankton, zooplankton, and benthic invertebrate communities. Loss of a number of common invertebrate species from the zooplankton and benthic invertebrate communities, including zooplankton species such as *Diaptomus silicis, Mysis relicta, Epischura lacustris*; many species of snails, clams, mayflies, and amphipods, and some crayfish. Visual accumulations of filamentous green algae in the littoral zone of many lakes and in some streams.
5.5 to 5.0	Loss of several important sport fish species, including lake trout, walleye, rainbow trout, and smallmouth bass; as well as additional non-game species such as creek chub. Continued shift in the species composition and decline in species richness of the phytoplankton, periphyton, zooplankton, and benthic invertebrate communities; decreases in the total abundance and biomass of benthic invertebrates and zooplankton may occur in some waters. Loss of several additional invertebrate species common in oligotrophic waters, including *Daphnia galeata mendotae, Diaphanosoma leuchtenbergianum, Asplancha priodonta*; all snails, most species of clams, and many species of mayflies, stoneflies, and other benthic invertebrates. Inhibition of nitrification. Further increase in the extent and abundance of filamentous green algae in lake littoral areas and streams.
5.0 to 4.5	Loss of most fish species, including most important sport fish species such as brook trout and Atlantic salmon. Measurable decline in the whole-system rates of decomposition of some forms of organic matter, potentially resulting in decreased rates of nutrient cycling. Substantial decrease in the number of species of zooplankton and benthic invertebrates, including loss of all clams and many insects and crustaceans; measurable decrease in the total community biomass of zooplankton and benthic invertebrates in most waters. Further decline in the species richness of the phytoplankton and periphyton communities. Reproductive failure of some acid-sensitive species of amphibians such as spotted salamanders, Jefferson salamanders, and the leopard frog.

Source: Baker, J. et al. (NAPAP SOS/T 13, 1990), p. 13-173.

regions of the U.S. are chronically acidic due to natural and anthropogenic causes. NAPAP defines acidic conditions as occurring when the acid neutralizing capacity[11] (ANC) is below 0 µeq/L. Furthermore, approximately 20 percent of the streams and lakes in these regions are considered to be extremely susceptible to acidity (defined as ANC <50 µeq/L) and slightly more than half show some susceptibility to acidification (defined as ANC <200 µeq/L).

In terms of the role of acid deposition as a causal mechanism for the acidification of water bodies, it is estimated that 75 percent of the 1,181 acidic lakes and 47 percent of the 4,668 streams studied under

[11] ANC is expressed in units of microequivalents per liter (µeq/L), where an equivalent ANC is the capacity to neutralize one mole of H^+ ions. Generally, waters with an ANC < 0 have corresponding pH values of less than 5.5 (L. Baker et al., NAPAP SOS/T 9, 1990).

NAPAP receive their dominant source of acid anions from atmospheric deposition (see Table E-2). On a regional basis, the importance of acid deposition varies considerably, which is believed to result from regional differences in SO_x and NO_x emissions and differences in the biogeochemistry of individual watersheds. For acidic lakes (ANC <0), the regions that appear most likely to be influenced by acid deposition include the Adirondacks and Mid-Atlantic Highland region, with acid deposition cited as the dominant source of acidity in 100 percent of the acidic lakes studied (Table E-2). This is in stark contrast to the West region, where none of the acidic lakes studied were dominated by acid deposition (notably, the sample size of lakes for this region was small to begin with). For acidic streams, the Mid-Atlantic Highland region contains the greatest proportion of streams whose acidic inputs are dominated by acid deposition (56 percent). This contrasts with acidic streams of Florida, where the vast majority (79 percent) are acidic primarily due to organic acids, rather than acid deposition.

Effects on Fish Habitat Quality

By combining information on relevant water chemistry parameters (pH, aluminum, calcium), fish toxicity models, and historical and current distributions of fish populations in the lakes and streams included in the National Surface Water Survey (NSWS), NAPAP investigators estimated the proportion of water bodies with water chemistry conditions that are unsuitable for survival of various fish species.[12] In the Adirondack region, where the acidic lakes are dominated by acid deposition, it is estimated that ten percent of the lakes are unsuitable for the survival of acid-tolerant fish species such as brook trout; twenty percent of the lakes are estimated to be unsuitable for

Table E-2. Comparison of Population of Acidic National Surface Water Survey (NSWS) by Chemical Category[1]

Region	Number of Acidic Waters	Deposition Dominated (%)	Organic Dominated (%)	Acid Mine Drainage Dominated (%)	Watershed Sulfate Dominated (%)
LAKES					
New England	173	79	21	--	--
Adirondacks	181	100	--	--	--
Mid-Atlantic Highlands	88	100	--	--	--
Southeastern Highlands	--	--	--	--	--
Florida	477	59	37	--	4
Upper Midwest	247	73	24	--	3
West	15	--	--	--	100
All Lakes	1,181	75	22	--	3
STREAMS					
Mid-Atlantic Highlands	2,414	56	--	44	--
Mid-Atlantic Coastal Plain	1,334	44	54	--	2
Southeastern Highlands	243	50	--	50	--
Florida	677	21	79	--	--
All Streams	4,668	47	27	26	<1

[1] Source: NAPAP 1991 (Table 2.2-3, p. 28).

[12] NAPAP, 1991.

the survival of acid-sensitive species such as minnows. About two percent and six percent of the lakes in the New England region are estimated to be unsuitable for acid-tolerant and acid-sensitive fish species, respectively. A greater proportion of streams in the Mid-Atlantic Highland region are estimated to be unsuitable for acid-tolerant and acid-resistant fish species (18 percent and 30 percent, respectively); however, about 44 percent of streams surveyed in this region are thought to be heavily influenced by acid mine drainage (Table E-2).

Economic Damages to Recreational Fishing

In an effort to assess some of the impacts from *existing* levels of acid deposition to public welfare, NAPAP investigated the current economic damages associated with acid deposition to trout anglers of New York, Maine, Vermont, and New Hampshire. The general approach used consisted of linking the catch per unit effort (CPUE) for four species of trout at individual lakes (estimated using participation survey data) to the relevant water quality conditions at these lakes (namely, the acid stress index or ASI). Using historical water quality data, critical water quality conditions (i.e., the ASI values) were estimated for lakes in the absence of acid deposition and compared to current conditions reflecting the presence of acid deposition. Using two types of travel cost models, the Random Utility Model (RUM) and Hedonic travel-cost model (HTCM), estimates of the willingness to pay (WTP) per trip of sampled trout anglers were obtained. Aggregate estimates of the WTP were obtained across the populations of trout anglers using statistical weighting factors. Finally, the difference in total WTP between the current (acid deposition) scenario and the historical (acid deposition-free) scenarios was determined.

The resulting estimates of economic damages to trout anglers in the four state region are relatively small. Specifically, damage estimates range from $0.3 million to $1.8 million (in 1989 dollars) for the hedonic travel-cost and random utility models, respectively. By many accounts, these estimates can be considered to underestimate actual damages to anglers in these states. First, data limitations precluded the development of meaningful WTP estimates for brook trout anglers, which may be a significant component of trout fishing in these areas. Second, resource constraints necessitated exclusion of a large population of trout anglers (i.e., those residing in New York City). Third, the economic damage estimates were limited to trout anglers, thus excluding potentially similar if not greater economic damages to anglers fishing for other coldwater or warmwater fish species. In addition, the NAPAP analysis was performed in the context of recreational fishing in lakes, thereby excluding potentially important welfare impacts from recreational fishing in streams. Finally, these estimates do not address non-use values of lakes in this region.

Benefits From Acid Deposition Avoidance Under the CAA

It is currently estimated that in the absence of pollution reductions achieved under the Clean Air Act, total sulfur emissions to the atmosphere would have increased by nearly sixteen million tons by 1990, a 40 percent increase above 1990 levels estimated with CAA controls remaining in place.[13] Based on atmospheric transport and deposition modeling, this increase in sulfur emissions corresponds to an approximate 25 to 35 percent increase in total sulfur deposition (wet & dry) in large portions of the northeastern portion of the United States.[14] Given sulfur emission and deposition changes of this magnitude, and the importance of sulfur emissions in contributing to acid deposition, one would expect some benefits to human welfare to be achieved as a result of improved quality of aquatic ecosystems. To date, however, no formal benefits assessment of CAA-avoided acid deposition impacts has been conducted for aquatic ecosystems. Nevertheless, past benefit assessments involving acid deposition impacts on aquatic ecosystems provide some opportunity to gain insights into the relative magnitude of certain aquatic-based benefits that may be achieved through pollution reductions under the CAA.[15]

Recreational Fishing

NAPAP evaluated the impact of changes in acid deposition on use values of aquatic ecosystems (i.e., recreational fishing).[16] In their integrated assessment, NAPAP valued the impacts of three different sulfur-

[13] U.S. EPA, 1995; Table B-2.

[14] U.S. EPA 1995, p. 3-10.

[15] See, for example, NAPAP, 1991.

[16] NAPAP, 1991.

induced acid deposition scenarios to trout anglers from NY, VT, NH and ME.[17] The three scenarios evaluated were:

1. No change in acid deposition.
2. A 50 percent reduction in acid deposition.
3. A 30 percent increase in acid deposition.

As described above, equations were developed by NAPAP to estimate the catch per hour for species at each lake as a function of the ASI value for each lake and of the technique of the fishers. Baseline and predicted changes in CPUE were evaluated for all lakes modeled in the region. Willingness-to-pay estimates for CPUE per trip were derived for the baseline and sulfur emission scenarios using two travel-cost models, a random utility model and a hedonic travel cost model. These willingness-to-pay estimates were then combined with the results of a participation model that predicted the total number of trips taken by trout anglers. Total welfare changes were determined over a 50 year period (from 1990 to 2040).

At current levels of acid deposition, NAPAP estimates that trout anglers in these four states will experience annual losses by the year 2030 of $5.3 or $27.5 million (in 1989 dollars) for the random utility model and hedonic travel cost model, respectively (see Table E-3). If acid deposition *increases* by 30 percent, which roughly corresponds to the 25 to 35 percent increase predicted for the northeast U.S. in the absence of CAA sulfur controls,[18] the resulting economic losses to trout anglers in 2030 would range from $10 million to nearly $100 million annually (in 1989 dollars) for the RUM and HTCM, respectively. If deposition decreases by 50 percent, annual benefits to recreational anglers are estimated to be $14.7 million (RUM) or $4.2 million (HTCM).

While an estimation of CAA-related benefits to trout anglers based on the 30 percent increase in acid deposition scenario has some appeal, a strict transfer of these benefits to the section 812 retrospective analysis is hindered by several factors. First, the NAPAP benefits estimates are projected for future conditions (the year 2030). Therefore, the extent to which the NAPAP benefits reflect conditions and benefits in 1990 (the focus of the section 812 retrospective assessment) is unclear. Second, the NAPAP and CAA section 812 analyses operate from different baselines (1990 for the NAPAP study versus 1970-1990 for the section 812 study). However, the NAPAP estimates of annual benefits of $10 to $100 million provide a rough benchmark for assessing the likely magnitude of the avoided damages to an important and sensitive recreational fishery in a four-state area most impacted by surface water acidification from atmospheric deposition.

Table E-3. Results from Benefits Assessments of Aquatic Ecosystem Use Values from Acid Deposition Avoidance.

Study	Use Value	Scenario Modeled	Method	Annual Benefits
NAPAP (1991)	Trout Fishing (NY, ME, VT, NH)	No change in acid deposition	RUM HTCM	-$5.3 million -$27.5 million
		50% decrease in acid deposition	RUM HTCM	$14.4 million $4.2 million
		30% increase in acid deposition	RUM HTCM	-$10.3 million -$97.7 million
		No new emission reductions after 1985	RUM HTCM	-$5.5 million -$3.5 million
		10 million ton reduction of SO_2 from 1980 levels by 2000	RUM HTCM	$9.7 million $4.4 million

Eutrophication

Eutrophication is the process by which aquatic systems respond to nutrient enrichment. The most common nutrients involved in eutrophication are nitrogen and phosphorous (and related chemical species). When water bodies receive excessive amounts of nutrients, adverse impacts on their resident species and on ecosystem functions can occur from excessive algal growth and the reduction in dissolved oxygen caused by decaying algal biomass. Under highly eutrophic conditions, excessive nutrients can cause depleted oxygen levels that result in subsequent loss of economically important benthic organisms (shellfish), fish kills, and changes in phytoplankton, zooplankton, and fish community

[17] NAPAP, 1991; p. 383-384.

[18] U.S. EPA, 1995.

composition.[19] Nuisance algal blooms can have numerous economic and biological costs, including water quality deterioration affecting biological resources, toxicity to vertebrates and higher invertebrates, and decreased recreational and aesthetic value of waters.[20] Although severe eutrophication is likely to adversely affect organisms, especially fish, a moderate increase in nutrient levels may also increase fish stocks, by increasing productivity in the food chain.[21]

Atmospheric Deposition and Eutrophication

The deposition of NO_x in aquatic systems and their watersheds is one source of nitrogen that may contribute to eutrophication. The relative importance of NO_x deposition as a contributor to aquatic eutrophication depends on the extent to which the productivity of an aquatic ecosystem is limited by nitrogen availability and the relative importance of nitrogen deposition compared to other internal and external sources of nitrogen to the aquatic ecosystem. Furthermore, the vulnerability of aquatic ecosystems to eutrophication is known to vary seasonally and spatially, although these systems are affected by nutrient deposition throughout the year. In general, freshwater ecosystems appear to be more often limited by phosphorus, rather than nitrogen, and are not as likely to be heavily impacted by nitrogen deposition compared to some estuarine and coastal ecosystems.[22] In contrast to acidification of streams and lakes, eutrophication from atmospheric deposition of nitrogen is more commonly found in coastal and estuarine ecosystems, which are more frequently nitrogen-limited.[23]

Unfortunately, there is limited information with regard to the relative importance of atmospheric deposition as a nitrogen source in many estuarine and marine ecosystems. Estimates of the importance of atmospheric nitrogen deposition are difficult to make because of uncertainties in estimating deposition, especially dry deposition, as well as watershed nitrogen retention.[24] Paerl (1993) reviews the importance of atmospheric nitrogen deposition as a contributor to eutrophication of coastal ecosystems; he concludes that 10 to 50 percent of the total nitrogen loading to coastal waters is from direct and indirect atmospheric deposition. Estimates for the economically important Chesapeake Bay indicate that about 25 to 40 percent of the nitrogen loadings to the bay occur via atmospheric deposition.[25] Hinga et al. (1991) estimate that anthropogenic deposition provides 11 percent of total anthropogenic inputs of nitrogen in Narragansett Bay, 33 percent for the New York Bight, and 10 percent for New York Bay. Fisher and Oppenheimer (1991) estimate that atmospheric nitrogen provides 23 percent of total nitrogen loading to Long Island Sound and 23 percent to the lower Neuse River in North Carolina. Information on the importance of atmospheric nitrogen deposition for most other U.S. coastal ecosystems is not available in the literature. Episodic atmospheric inputs of nitrogen may be an important source of nitrogen to nutrient-poor marine ecosystems, such as the North Atlantic near Bermuda and the North Sea.[26]

Valuing Potential Benefits from Eutrophication Avoidance Under the CAA

It is currently estimated that in the absence of pollution reductions achieved under the Clean Air Act, total nitrogen emissions to the atmosphere would have increased by nearly 90 million tons by 1990, a two-fold increase above 1990 levels estimated with CAA controls remaining in place.[27] However, the ability to determine the potential economic benefit from such a reduction in nitrogen emissions is heavily constrained by gaps in our current biological and economic knowledge base of aquatic ecosystems.

One water body that has received much study in the area of nitrogen-induced eutrophication is Chesapeake Bay. As previously discussed, it is estimated that atmospheric deposition of nitrogen contributes approximately 25 percent to the total nitrogen load-

[19] Paerl, 1993.

[20] Paerl, 1988.

[21] Hansson and Rudstam, 1990; Rosenberg et al., 1990; Paerl, 1993.

[22] Hecky and Kilham, 1988; Vitousek and Howarth, 1991.

[23] U.S. EPA, 1993; Paerl, 1993.

[24] U.S. EPA, 1993.

[25] U.S. EPA, 1994.

[26] Owens et al., 1992.

[27] U.S. EPA, 1995; Table B-3.

ings to the bay.[28] In deposition terms, an estimated 15 to more than 25 percent increase in total nitrogen deposition has been forecast in the Chesapeake Bay watershed by 1990 in the absence of CAA pollution controls.[29] These results are based on an estimated 40,000 tons of atmospherically deposited nitrogen (as nitrate and ammonia) to Chesapeake Bay in 1985,[30] which means a 20 percent increase in atmospheric deposition would amount to approximately 8,000 additional tons.

One indirect method available to gauge the potential economic relevance of avoidance of such atmospheric nitrogen loadings to Chesapeake Bay is through the avoidance cost of nitrogen controls. However, such an assessment is difficult due to the site, facility, and treatment-specific variation in treatment costs. For example, Camacho (1993) reviewed nitrogen treatment costs for chemical treatment of water from important point sources (mostly public owned treatment works) and found that costs ranged from $9,600 to $20,600 per ton (annual costs, 1990 dollars), depending on the facility evaluated. Biological treatment of nitrogen from point sources was far more expensive, varying from $4,000 to $36,000 per ton. For control of non-point source loading, values of nitrogen removal practices ranged from $1,000 to $285,000 per ton.[31] Taking chemical addition as one possible example, the avoided costs of treatment of 8,000 tons of nitrogen would range from about $75 million to about $170 million annually (in 1990 dollars).

Mercury

Mercury, in the form of methyl mercury, is a neurotoxin of concern and can accumulate in tissue of fish to levels that are hazardous to humans and aquatic-feeding wildlife in the U.S. In relation to the section 812 CAA retrospective analysis, mercury is of interest for two reasons. First, potential benefits to human welfare may have occurred as a result of mercury emission controls implemented under EPA's National Emission Standards for Hazardous Air Pollutants (NESHAP). Second, experimental and observational evidence suggests that acidification of water bodies enhances mercury accumulation in fish tissues.[32] Therefore, CAA-mandated reductions in sulfur and nitrogen oxide emissions and subsequent acid deposition may have resulted in indirect benefits from a reduction in mercury accumulation in fish and subsequent improvements to human health and welfare.

The accumulation of mercury to hazardous levels in fish has become a pervasive problem in the U.S. and Canada. A rapid increase in advisories occurred during the 1980s, including a blanket advisory affecting 11,000 lakes in Michigan.[33] The Ontario Ministries of Environment and Natural Resources (1990) recommend fish consumption restrictions for 90 percent of the walleye populations, 80 percent of smallmouth bass populations, and 60 percent of lake trout populations in 1,218 Ontario lakes because of mercury accumulation. In many instances, mercury has accumulated to hazardous levels in fish in highly remote water bodies that are free from direct aqueous discharges of mercury.[34] Mass balance studies have shown that atmospheric deposition of mercury can account for the accumulation of mercury in fish to high levels in lakes of these remote regions.[35] The potential impacts of mercury on the health of humans and fish-eating (piscivorous) wildlife has lead EPA to recently establish water quality criteria to protect piscivorous species in the Great Lakes.[36]

Although mercury accumulation in fish via atmospheric deposition is now widely recognized as a potential hazard to human health and certain wildlife species, studies establishing quantitative linkages between sources of mercury emissions, atmospheric deposition of mercury, and subsequent accumulation in fish are lacking. Thus at the present time, we are unable to quantify potential benefits from CAA-avoided mercury accumulation in fish of U.S. water

[28] U.S. EPA, 1993.

[29] U.S. EPA 1995, Figure C-6.

[30] NERA, 1994.

[31] Shuyler, 1992.

[32] Bloom et al., 1991; Watras and Bloom, 1992; Miskimmin et al., 1992; Spry and Wiener, 1991; Wiener et al., 1990.

[33] Watras et al., 1994.

[34] Glass et al., 1990; Sorenson et al., 1990; Grieb et al. 1990; Schofield et al. 1994.

[35] Fitzgerald et al. ,1991.

[36] U.S. EPA, 1995.

bodies. Given the pervasiveness of the mercury problem with CAA-pollution controls, potential benefits to human health and welfare from avoidance of further mercury related damages to aquatic ecosystems could be substantial.

It should also be noted that atmospheric deposition is a major contributor to surface water loads of other toxic pollutants as well. For example, scientists believe that about 35 to 50 percent of the annual loadings of a variety of toxic chemicals to the Great Lakes may be from the air; for lead, atmospheric deposition currently accounts for an estimated 95 percent of the total load in the Great Lakes.[37] CAA-related reductions in air emissions of toxic pollutants (such as lead) undoubtedly reduced the loading of these chemicals to the Great Lakes and other water bodies; the magnitude of the benefits of reducing these exposures to humans and wildlife is not known.

Benefits from Avoided Damages to Wetland Ecosystems

Introduction

This review addresses the effects of air pollutants on wetland ecosystems; the focus is on acidification and nutrient loading. Valuable service flows of wetland ecosystems include flood control, water quality protection and improvement, wildlife and fish habitat, and biodiversity. The limited scientific evidence suggests that air pollutants may most affect biodiversity, in particular because of nutrient loading through nitrogen deposition.

Wetlands are broadly characterized as transitional areas between terrestrial and aquatic systems in which the water table is at or near the surface or the land is periodically covered by shallow water.[38] Types of wetlands include swamps (forested wetlands), marshes (herbaceous vegetation), and peatlands, which are wetlands that accumulate partially decayed vegetative matter due to limited decomposition.[39] Peatlands include bogs and fens. Bogs receive water solely from precipitation, are generally dominated by *Sphagnum* moss, and are low in nutrients. Fens receive water from groundwater and precipitation, contain more marsh-like vegetation, and have higher pH and nutrient levels than bogs.[40] Most of the limited work on the effects of atmospheric deposition on wetlands has been done in peatlands, specifically in Europe, where levels of atmospheric deposition are generally much higher than in the U.S.

The air pollutants of greatest concern with respect to effects on wetland ecosystems are oxides of nitrogen (NO_x) and oxides of sulfur (SO_x), primarily sulfur dioxide (SO_2). Air pollutants may affect wetland ecosystems by acidification of vulnerable wetlands and by increasing nutrient levels. Acidification in vulnerable wetlands may affect vegetation adversely, as appears to have occurred in Europe. In wetlands where nitrogen levels are low, increased nitrogen deposition may alter the dynamics of competition between plant species. Species adapted to low-nitrogen levels, including many endangered species, may decrease in abundance.[41]

Effects of Acidification

Limited evidence suggests that acidic deposition and decreased pH may harm certain wetland plants, alter competitive relations between wetland plants and cause changes in wetland drainage and water retention.

Work concerning the possible acidification of peatlands is inconclusive. Acidic deposition is unlikely to result in displacement of base cations from cation exchange sites in bogs, and therefore it will not cause a drop in pH.[42] Peatland sediments are low in Al^{3+}, so mobilization of toxic aluminum is not a concern as it is in forest soils and aquatic ecosystems.[43] Acidification might affect certain fen ecosystems. Gorham et al. (1984) have hypothesized that acidic deposition could leach base cations from mineral-poor fens and decrease pH levels. This could result in a

[37] U.S. EPA, 1994.

[38] Cowardin et al., 1979.

[39] Mitsch and Gosselink, 1986.

[40] Mitsch and Gosselink, 1986.

[41] U.S. EPA, 1993.

[42] Gorham et al., 1984.

[43] Turner et al., NAPAP SOS/T 10, 1990.

transition to bog vegetation such as *Sphagnum* and away from sedge meadow vegetation. At this time, this remains a hypothesis; however, pH did not decrease in a mineral-poor Ontario fen during a four-year period in which researchers experimentally increased acidic deposition.[44]

In European wetlands affected by high levels of deposition for many years, acidic deposition has seriously affected wetland vegetation. Roelofs (1986) reports that acidification of heath pools in the Netherlands has caused a change in species composition with *Sphagnum* and rushes replacing the original vegetation. Likewise, significant declines in *Sphagnum* in British bogs have occurred in areas affected by 200 years of atmospheric pollution, including nitrogen deposition.[45] It is unclear how such changes have affected wetland service flows apart from the effects on biodiversity; however, water retention has decreased and significant erosion has occurred in seriously perturbed British bogs near Manchester and Liverpool.[46]

Effects of Nutrient Loading

Atmospheric deposition may affect wetlands by increasing the level of nutrients, particularly nitrogen, in wetlands. Sulfur is not a limiting nutrient in peatlands,[47] but nitrogen commonly limits plant growth.[48] The effects of increased nitrogen levels in wetlands include an increased threat to endangered plant species and possible large-scale changes in plant populations and community structure. Endangered and threatened plant species are common in wetlands, with wetland species representing 17 percent of the endangered plant species in the U.S. (U.S. EPA, 1993). These plants are often specifically adapted to low nitrogen levels; examples include isoetids[49] and insectivorous plants.[50] In eastern Canadian wetlands, nationally rare species are most common in infertile sites.[51] When nitrogen levels increase, other species adapted to higher levels of nitrogen may competitively displace these species. Thus, NO_x emissions that increase nitrogen levels in nitrogen-poor wetlands may increase the danger of extinction for threatened and endangered species.

By changing competitive relations between plant species, increased nitrogen deposition may broadly affect community structure in certain wetlands. Common species that thrive in nitrogen-poor wetlands may become less abundant. Many nitrogen-poor bogs in the northern U.S. are dominated by *Sphagnum* species. These species capture low levels of nitrogen from precipitation. Increased nitrogen levels may directly harm *Sphagnum* and cause increased nitrogen to be available to vascular plants that may out compete *Sphagnum.*[52] Studies in Great Britain have documented large declines in *Sphagnum* moss because of atmospheric pollution;[53] nitrogen loading may play an important role in these declines. However, Rochefort et al. (1990) document limited effects of fertilization from experimentally-increased NO_3^- and SO_4^{2-} deposition on an Ontario mineral-poor fen over a four-year period, apart from initially increased *Sphagnum* growth. Thus, increased nitrogen loading might adversely or beneficially affect wetland plants depending on baseline nitrogen concentrations in the wetland, atmospheric nitrogen loading, and species requirements for and sensitivity to nitrogen.

Increases in nitrogen levels due to NO_x emissions will have the greatest effect on wetlands that are extremely nitrogen-limited and that receive small amounts of nitrogen naturally. Since bogs, including *Sphagnum* bogs, receive little surface water runoff, they get most of their nutrient and water loadings through precipitation. These bogs may receive a total of approximately 10 kg nitrogen per hectare per year (kg N/ha/yr), which is one to two orders of magnitude less nitrogen than other freshwater wetlands and

[44] Rochefort et al., 1990.

[45] Lee et al., 1986.

[46] Lee et al., 1986.

[47] Turner et al., NAPAP SOS/T 10, 1990.

[48] U.S. EPA, 1993.

[49] Boston, 1986.

[50] Moore et al., 1989.

[51] Moore et al., 1989; Wisheu and Keddy, 1989.

[52] Lee & Woodin 1988, Aerts et al., 1992.

[53] Ferguson et al., 1984; Lee et al., 1986.

saltmarshes receive.[54] As atmospheric deposition of nitrogen has been estimated to be at least 5.5 to 11.7 kg N/ha/yr,[55] changes in NO_x emissions would most likely affect these bogs. The results of a model by Logofet and Alexandrov (1984) suggest that a treeless, nutrient-poor bog may undergo succession to a forested bog because of the input of greater than 7 kg N/ha/yr.

As in freshwater wetlands, significantly increased nitrogen deposition to coastal wetlands will increase productivity and alter the competitive relationships between species.[56] However, studies showing this increased productivity have used 100 to 3000 kg N/ha/yr.[57] Therefore, limited changes in NO_x emissions may not affect coastal wetland productivity.

Summary of Wetland Ecosystem Effects

The effects of air pollutants on wetlands have received little attention, in contrast to the large body of work on the effects of acid rain on aquatic and forest ecosystems. Little evidence exists suggesting that acidification due to atmospheric deposition is a major threat to wetlands. In particular, peatlands are naturally acidic, although mineral-poor fens may be at risk from acidification. Nitrogen loading may alter community composition in wetlands naturally low in nutrients, such as bogs. Nitrogen loading may threaten rare species adapted to low nitrogen levels. In Britain and The Netherlands, heavy atmospheric deposition over a long period appears to have caused serious declines in *Sphagnum* in peatlands.

Air pollutants appear to most seriously threaten rare and endangered species, biodiversity, and community composition in wetlands, particularly bogs. These changes are difficult to associate with changes in economic value; even the qualitative nature of the effects is uncertain. Air pollutants may not significantly affect such important wetland service flows as flood control, water quality protection, and wildlife habitat in most wetlands, so the impacts on the more readily monetized aspects of the economic value of wetlands may be limited.

Benefits from Avoided Damages to Forests

Introduction

Forests occupy 33 percent of the land mass in the U.S. (some 738 million acres) and provide a wealth of services to the U.S. population.[58] Notable services provided by forests include timber production, recreational opportunities such as hunting, camping, hiking, and wildlife observation, water quality protection, nutrient removal and cycling, flood control, erosion control, temporary carbon sequestration, preservation of diversity, and existence values. In 1991, hunting participation alone accounted for 236 million recreation days that included 214 million person trips with estimated expenditures valued at $12.3 billion.[59]

The Clean Air Act-regulated pollutants of greatest concern with respect to effects on forest ecosystems are oxides of sulfur (SO_x), primarily sulfur dioxide (SO_2), oxides of nitrogen (NO_x), and volatile organic compounds (VOCs). While extremely high ambient concentrations of SO_2 and NO_x may directly affect vegetation, such effects are uncommon in the U.S.;[60] the indirect effects of these pollutants are of greater concern. Specifically, emissions of SO_2 and NO_x are known to contribute to acid deposition in portions of the United States, with SO_2 contributing 75 to 95 percent of the acidity in rainfall in the eastern U.S.[61] Acid deposition is of concern to forests primarily from the acidification of soils (i.e., by reducing seed germination, altering nutrient and heavy metal availability). Direct foliar damage can occur from precipitation with extremely low pH levels (i.e., 3.0-3.6 and below), although these levels are lower than ambient levels in the U.S.[62] VOCs and NO_x are

[54] U.S. EPA, 1993.

[55] U.S. EPA, 1993.

[56] U.S. EPA, 1993.

[57] U.S. EPA, 1993.

[58] Powell et al. 1993.

[59] U.S. DOI, 1993.

[60] Shriner et al., NAPAP SOS/T 18, 1990.

[61] NAPAP, 1991.

[62] Shriner et al., NAPAP SOS/T 18, 1990.

important precursors to ozone formation, which can affect leaf photosynthesis and senescence and decrease cold hardiness, thereby causing deleterious impacts on tree growth, survival and reproduction. Deposition of NO_x may also alter the nutrient balance of forest soils, which in turn might alter the competitive relationships between tree species and affect species composition and diversity.[63]

Current Air Pollutant Effects on Forests

Acid Deposition Impacts

In 1985, NAPAP organized the Forest Response Program (FRP) to evaluate the significance of forest damage caused by acidic deposition, the causal relationships between air pollutants and forest damage, and the dynamics of these relationships regionally. Research was focussed on four forest regions: Eastern Spruce-Fir, Southern Commercial Forests, Eastern Hardwoods, and Western Conifers. With the exception of high-elevation spruce-fir forests, the available evidence suggests that acidic deposition does not currently affect these forests and that observed declines in sugar maple and southern pines are not due to acidic deposition.[64]

Circumstantial evidence suggests that acidic deposition may affect high-elevation spruce-fir forests in the northeastern U.S. These forests have extensive contact with acidic cloud water.[65] Experimental evidence suggests that acidic deposition may affect cold hardiness in red spruce, an important component of the spruce-fir forest. Significant declines in red spruce growth and in its importance in the forest have occurred in New York and northern New England. The proximate cause of death of red spruce in the region is pathogens and insects; acidic deposition may interact with these biological stresses and with weather-induced stress to produce adverse effects in red spruce. Ozone may also play a role in red spruce decline in this region.[66] Available evidence suggests that soil aluminum and soil pH levels have not affected red spruce adversely.[67]

Ozone Impacts

Experimental Evidence

For practical reasons, the majority of experimental evidence linking ozone exposure to damage to tree species has been derived from studies of individual plants, especially seedling and branch studies.[68] Results from these studies suggest that ozone exposure can reduce photosynthesis and increase senescence in leaves. Subsequently, such effects from ozone may alter the carbohydrate allocation to plant tissues such as roots, which may affect plant growth and cold hardiness. Decreases in cold tolerance may be particularly important for trees in northern latitudes and high elevations. Recent work on quantifying the relationship between ozone exposure and plant responses suggest that seedlings of aspen, ponderosa pine, black cherry, tulip poplar, sugar maple, and eastern white pine seedlings may experience biomass reductions of approximately 10 percent at or near ambient ozone exposures.[69] Because trees are perennials, the effect of even a 1-2 percent per year loss in seedling biomass (versus 10 to 20 percent yield loss in crops), if compounded over multiple years under natural field conditions of competition for resources, could be severe.

Although indicative of short-term relative response to ozone exposure, results from these experiments are unable to provide reliable information on the long-term effects of ozone on forests. This limitation arises because the effects of ozone on forests will depend on both the response of individual plants to ozone exposure and the response of populations of plants, which interact with their environment. Population response will be altered by the varying intraspecific genetic susceptibility to ozone. Individual plant response will also be affected by many environmental factors, including insect pests, pathogens, plant symbionts, competing plants, moisture, temperature, light, and other pollutants. Consistent evidence on the interaction of ozone with other environmental factors is lacking. Furthermore, most experimental stud-

[63] U.S. EPA, 1993.

[64] Barnard et al., NAPAP SOS/T 16, 1990; NAPAP, 1991.

[65] Barnard et al., NAPAP SOS/T 16, 1990.

[66] Shriner et al., NAPAP SOS/T 18, 1990.

[67] Barnard et al., NAPAP SOS/T 16, 1990.

[68] U.S. EPA, 1996a.

[69] Hogsett et al. , 1995.

ies have only studied exposure for one growing season; effects on forest species may occur over decades.[70] Therefore, considerable uncertainties occur in scaling across individuals of different ages, from individuals to populations and communities, and across time.

Observational Evidence

Studies of the forests of the San Bernardino Mountains provide the strongest case for linking ozone exposure to damages to an entire forest ecosystem. These forests have been exposed to extremely high ambient ozone levels over the past 50 years due to their proximity to the Los Angeles area. The area has been extensively studied regarding the effects of ozone, as described in U.S. EPA (1996a). The ecosystem has been seriously affected by ozone pollution, with the climax-dominant, but ozone-sensitive ponderosa pine and Jeffrey pine declining in abundance, replaced by more ozone-tolerant species. These sensitive species have experienced decreased growth, survival, and reproduction, and susceptibility to insects. The effects of ozone on these species have resulted in other ecosystem effects, including the buildup of a large litter layer, due to increased needle senescence. The decline of the fire-tolerant ponderosa and Jeffrey pines may seriously affect the fire ecology of the ecosystem, with fire-sensitive species becoming more common. Ozone concentrations have been declining in recent decades, and crown injury of ponderosa and Jeffrey pine has decreased. However, the two species have continued to decline in abundance, as measured by total basal area, compared with other species over the period 1974 to 1988.[71] The nature of community dynamics, particularly in mixed species, uneven aged stands, indicates that subtle long-term forest responses (e.g., shifts in species composition) to elevated levels of a chronic stress like exposure to ozone are more likely than wide-spread community degradation.[72]

Limited field studies have been completed in other forest ecosystems. Foliar injury has been observed in the Jefferson and George Washington National Forests and throughout the Blue Ridge Mountains, including areas of the Shenandoah National Park.[73] In the Great Smoky Mountains National Park, surveys made in the summers from 1987 through 1990 found 95 plant species exhibited foliar injury symptoms consistent with those thought to be caused by ozone.[74] Foliar ozone injury has also been documented in National Parks and Forests in the Sierra Nevada mountains.[75]

Growth and productivity of seedlings have been reported to be affected by ozone for numerous species in the Blue Ridge Mountains of Virginia. In the Shenandoah National Park, Duchelle et al. (1982, 1983) found that tulip poplar, green ash, sweet gum, black locust, as well as several evergreen species (e.g., Eastern hemlock, Table Mountain pine, pitch pine, and Virginia pine), common milkweed, and common blackberry all demonstrated growth suppression of seedlings. Except for the last two species mentioned, almost no visible injury symptoms accompanied the growth reductions. Studies of mature trees in the Appalachian Mountains also indicate that injury associated with exposure to ozone and other oxidants has been occurring for many years.[76] Researchers have also found that major decreases in growth occurred for both symptomatic and asymptomatic trees during the 1950s and 1960s in the Western U.S.[77] The adverse response of a number of fruit and nut trees to ozone exposure has been reported.[78]

Monitoring by the USDA Forest Service shows that growth rates of yellow pine in the Southeast have been decreasing over the past two decades in natural stands but not in pine plantations.[79] Solid evidence linking this growth reduction to air pollutants is lack-

[70] U.S. EPA, 1996a.

[71] Miller et al., 1989 and Miller et al., 1991.

[72] Shaver et al., 1994

[73] Hayes and Skelly, 1977; Skelly et al., 1984

[74] Neufeld, et al., 1992

[75] Peterson and Arbaugh, 1992

[76] Benoit et al., 1982

[77] Peterson et al., 1987; Peterson and Arbaugh, 1988, 1992; Peterson et al., 1991

[78] McCool and Musselman, 1990; Retzlaff et al., 1991, 1992a, b

[79] NAPAP, 1991.

ing, although ozone, in particular, may be a factor.[80] Ambient ozone levels in the region are high enough to damage sensitive tree species, including pine seedlings during experimental exposure.[81] Due to the commercial importance of yellow pine, the economic impacts of ozone on forest ecosystems in this area could be significant if ozone is affecting growth.

Although the ecosystem effects occurring in the San Bernardino forest ecosystem have occurred at very high ozone exposures, lower ozone exposure elsewhere in the U.S. may still affect forests. The EPA Ozone Staff Paper[82] assessed the risk to vegetation, including forests, under current ambient air quality. Using a GIS approach, it was found that under the base year (1990) air quality, a large portion of California and a few localized areas in North Carolina and Georgia have seasonal ozone levels above those which have been reported to produce greater than 17 percent biomass loss in 50 percent of studied tree seedling species. A broader multistate region in the east is estimated to have air quality sufficient to cause 17 percent biomass loss in seedlings, while at least a third of the country, again mostly in the eastern U.S., most likely has seasonal exposure levels which could allow up to 10 percent yield loss in 50 percent of studied seedlings. The Staff Paper did not present monetized benefits because of lack of exposure-response functions.[83]

Even small changes in the health of ozone-sensitive species may affect competition between sensitive and tolerant species, changing forest stand dynamics.[84] Depending on the sensitivities of individual competing species, this could affect timber production either positively or negatively, and affect community composition and, possibly, ecosystem processes.

Endangered species

Ozone effects may also reduce the ability of affected areas to provide habitats to endangered species. For example, two listed endangered plant species, the spreading aven and Roan Mountain bluet, are currently found at a small number of sites in eastern Tennessee and western North Carolina — forested areas where ozone-related injury is of concern.[85] In addition, ozone-related effects on individual ozone-sensitive species that provide unique support to other species can have broader impacts. For example, one such species is the common milkweed, long known for its sensitivity to ozone and usefulness as an indicator species of elevated ozone levels, as well as being the sole food of the monarch butterfly larvae. Thus, a major risk associated with of the loss of milkweed foliage for a season is that it might have significant indirect effects on the monarch butterfly population. A large number of studies have shown that ozone-sensitive vegetation exists over much of the U.S., with many native species located in forests and Class I areas, which are federally mandated to preserve certain air quality related values.

Valuation of Benefits From CAA-Avoided Damages to Forests

Background

To quantitatively assess the economic benefits of avoided damages of relevant CAA pollutants to forests, it is necessary to link estimated changes in air pollution to measures of forest health and conditions that can be readily quantified in economic terms. For commercial timber production, this would require quantifying the relationship between atmospheric deposition and measures of forest productivity such as timber yield. For assessing recreational benefits, linkages would have to be drawn between air pollution and vulnerable factors that influence forest-based recreation (e.g., site-characteristics such as canopy density, type of tree species, degree of visible tree damage, etc.). While important strides have been made in establishing these linkages (e.g., NAPAP modeling of air pollution effects on forest soil chemistry and tree branch physiology), critical gaps in our ability to predict whole tree and forest responses to air pollution changes have precluded the establishment of such quantitative linkages.[86] Critical knowl-

[80] NAPAP, 1991.

[81] NAPAP, 1991.

[82] U.S. EPA, 1996b

[83] U.S. EPA, 1996b.

[84] U.S. EPA, 1996a.

[85] U.S. EPA, 1996b

[86] NAPAP, 1991.

edge gaps exist in our ability to extrapolate experimental results from seedling and branch studies to whole tree and forest responses, to account for key growth processes of mature trees, to integrate various mechanisms by which air pollution can affect trees (e.g., soil acidification, nitrification, and direct foliar damage, winter stress, etc.), and to account for the interaction of other stressors on forest health and dynamics (susceptibility to insect damage, drought, disease, fire, nutrient and light competition, etc.).

Despite these constraints to quantifying economic benefits from air pollution reductions on forest ecosystems, relevant studies that have attempted to value air pollution damages on forests are reviewed and summarized below. In some cases, the relationship between air pollution and forest response is estimated using expert judgement (e.g., for NAPAP assessment from various growth scenarios). In other cases, damage estimates reflect current impacts of air pollution on forests, and the dose-response relationship is absent. In the aggregate, this summary provides some insight into possible CAA-related benefits from avoided damages to a select and narrowly focussed group of forest services, but, because of severe data constraints, does not provide an estimate of the overall range of forest-based benefits possible under the CAA.

Commercial Timber Harvesting

The economic impact of hypothetical growth reductions in northeastern and southeastern trees (both hardwood and softwood species) was intensively studied under NAPAP.[87] Growth reductions ranging from 5 to 10 percent over a 5 to 10 year period, depending on the species and location, were assumed to occur as a result of all forms of air pollution based on expert opinion derived from a survey by deSteigner and Pye (1988). Timber market responses to these hypothesized growth declines were modeled until the year 2040 using a revised version of the Timber Assessment Market Model (TAMM90) and the Aggregate Timberland Assessment System (ATLAS), which was used to simulate timber inventories on private timberland in the United States. Economic welfare outputs included changes in consumer and producer surplus and changes in revenue to southeast stumpage owners. Results indicate that annualized reductions in consumer and producer surplus would total $0.5 billion by the year 2000 and $3 billion by the year 2040 (in 1967 dollars). Simulated effects on stumpage owners' revenues were minimal ($10 to $20 million).

In an attempt to estimate the net economic damages from ozone effects on selected U.S. forests, NAPAP studied the effect of various *assumed* reductions in growth rates of commercial southeastern pine forests (both natural and planted).[88] For both planted and natural plus planted pines, the following changes in growth rates were assumed to occur: a two percent increase, no change, a two percent decrease, a five percent decrease, and a ten percent decrease. The two to five percent growth reductions were considered as possible outcomes from current ozone induced damage to southeastern forests, although no quantitative linkage between ozone exposure and damages was established. The ten percent growth reduction scenario was primarily included for evaluating model sensitivity to severe changes in growth and was considered out of the range of likely ozone damage estimates. The TAMM and ATLAS models were again used to simulate timber market responses under baseline and hypothesized growth change scenarios from 1985 to 2040. Results indicate that *annual* changes in total economic surplus (i.e., the sum of consumer and producer surplus and timber owner revenues in 1989 dollars) would range from an increase of $40 million (for the two percent increase in growth scenario) to a decrease of $110 million (for the ten percent decrease in growth scenario) for planted and natural pine model simulations.

In the context of estimated benefits from avoidance of other damages in the absence of the Clean Air Act from 1970 to 1990,[89] the magnitude of economic damages estimated to the commercial timber industry are comparatively small. For example, economic damage estimates range up to $3 billion annually for five to ten percent growth rate reductions in northeast and southeast forests, and just $110 million for southeastern pines. However, in the context of damages to forest-based services as a whole, the NAPAP-derived commercial timber damage estimates should be viewed as representing a lower bound estimate for a variety of reasons. First, these damage estimates exclude other categories of possible forest-based ben-

[87] Haynes and Kaiser, NAPAP SOS/T 27 Section B, 1990.

[88] NAPAP, 1991.

[89] Most notably avoided human health effects, which are estimated on the order of $300 to $800 billion annually.

efits, including recreational and non-use values. Second, even within the context of timber-related damages, the NAPAP forest-damage studies focused on a portion of U.S. forests (northeastern and southeastern U.S.); a much greater geographic range of forests could become susceptible to timber-related damages in the absence of CAA controls. Finally, the NAPAP damage estimates consider only two types of tree species: planted and naturally grown pines, although these species are economically important. Damages to other commercially harvested tree species, such as mixed pine and hardwood forests, are therefore excluded.

Non-marketed Forest Services

In an effort to address the potential benefits resulting from avoidance of acid deposition-induced damages to non-marketed forest-based services (e.g., recreation use, existence value), an extensive review of the economic literature was conducted under the auspices of NAPAP.[90] From their review, NAPAP could not identify any single study or model that could be reliably used to quantify economic benefits from avoided acid deposition-caused damages to non-marketed forest services (such as recreational use) on a regional or national basis. The primary limitation in many of the studies reviewed was the absence of a quantitative linkage between the value of a recreational user day and important site characteristics which could be tied to air pollution effects. In addition, most studies were narrowly focused geographically to specific sites and did not attempt to value system-wide (larger scale) damages that could result from acid deposition over an entire region. Since the availability of nearby substitution sites will affect the recreational value for a given site, the benefits from such site-specific studies may not reflect actual economic damages incurred from wide-scale air pollution impacts on forests. The inability of studies to consider additional crowding at unaffected sites in addition to changes in recreational participation rates as a function of air pollution damages was also recognized as an important limitation.

Despite not being able to quantitatively assess the benefits from avoided acid deposition-induced damages to nonmarket forest services, several important concepts emerge from NAPAP's review of recreational benefits, that bear relevance to the section 812 retrospective analysis. First, several studies were identified that established a relationship between key forest site characteristics and the value of recreational participation. For example, Brown et al. (1989) used contingent valuation to evaluate the relationship between scenic beauty ratings and willingness of recreationalists to pay at pictured sites. Based on their interviews with over 1400 recreationalists at ten different sites in Arizona, positive correlations were established between scenic beauty rankings determined from one group of recreationalists and willingness to pay to recreate determined by a separate group of recreationalists (r^2 ranged from 0.27 to 0.98 depending on ranking). In another study, Walsh et al. (1989) developed a functional relationship between reduction of recreational benefits and tree density changes that reflected varying levels of insect damage at six campgrounds in the Front Range of the Colorado Rockies. By using both contingent valuation and travel cost models, Walsh et al. (1989) were able to show that 10 percent, 20 percent, and 30 percent decreases in tree densities reduces the total recreational benefits at their sites by 7 percent, 15 percent and 24 percent, respectively. Although results from these studies are limited to the sites from which they were derived, they do support the intuition that the degree of visible damage to forests is to some extent correlated with the magnitude of damages to forest-based recreation expected. This finding supports the notion that the avoidance of damages to forest ecosystems from CAA-induced pollution controls (albeit currently unquantified) have likely benefited forest-based recreation in the U.S.

In addition to establishing relationships between recreational value and visible damage to forest sites, there is evidence linking air pollution (ozone) effects on forests to economic damages to non-use values of forests. For example, D.C. Peterson et al. (1987) valued ozone-induced damages to forests surrounding the Los Angeles area. Using contingent valuation methods, D.C. Peterson et al. (1987) surveyed recreationalists (a random survey of households in the San Bernardino, Los Angles and Orange counties) and residents (a sample of property owners within the San Bernardino and Angeles national forests) for their willingness to pay to prevent forest scenes from degrading one step on a "forest quality ladder" depicting various levels of ozone-induced damages. The mean willingness to pay to protect further degradation was $37.61 and $119.48 per household for recreationalists and residents, respectively. Annual damages to Los Angeles area residences from a one-step drop on the forest quality ladder were estimated between $27 million and $147 million.

[90] Rosenthal, NAPAP SOS/T 27 Section B, 1990.

These estimates cannot be directly translated into a rough estimate of the potential non-use values of avoided forest damages. Considering the limited size of the population generating the estimated benefits of forest degradation, however, they do provide evidence that the recreational and non-use benefits may substantially exceed the commercial timber values.

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Appendix F: Effects of Criteria Pollutants on Agriculture

Introduction

One potential impact of air pollutants on economic welfare is their effect on agricultural crops, including annual and perennial species. Pollutants may affect processes within individual plants that affect growth and reproduction, thereby affecting yields of agricultural crops. Possible physiological effects of pollutants include the following: decreased photosynthesis; changes in carbohydrate allocation; increased foliar leaching; decreased nutrient uptake; increased sensitivity to climatic stress, pests, and pathogens; decreased competitive ability; and decreased reproductive efficiency. These physiological effects, in conjunction with environmental factors and intraspecies differences in susceptibility, may affect crop yields.

Primary air pollutants that might damage plants include SO_2, NO_x, and volatile organic compounds (VOCs). These pollutants may have direct effects on crops, or they may damage crops indirectly by contributing to tropospheric (ground-level) ozone, peroxyacetyl nitrate (PAN), and/or acid deposition, all of which damage plants. Tropospheric ozone is formed by photochemical reactions involving VOCs and NO_x, while SO_2 and NO_x cause acidic deposition.

While all of these air pollutants may inflict incremental stresses on crop plants, in most cases air pollutants other than ozone are not a significant danger to crops. Based primarily on EPA's National Acid Precipitation Assessment Program (NAPAP) conclusions,[1] this analysis considers ozone to be the primary pollutant affecting agricultural production.

This analysis estimates the economic value of the difference in agricultural production that has resulted due to the existence of the CAA since 1970. The analysis is restricted to a subset of agricultural commodities, and excludes those commodity crops for which ozone response data are not available. Fruits, vegetables, ornamentals, and specialty crops are also excluded from this analysis. To estimate the economic value of ozone reductions under the CAA, agricultural production levels expected from control scenario ozone conditions are first compared with those expected to be associated with ozone levels predicted under the no-control scenario. Estimated changes in economic welfare are then calculated based on a comparison of estimated economic benefits associated with each level of production.

Ozone Concentration Data

To estimate the nationwide crop damages as a result of ozone exposure, the first step is to estimate the nationwide ozone concentrations under the control and no-control scenarios. This section describes the methodology used to estimate ozone concentrations for each county in each of these two scenarios.

First, historical ozone concentration data at the monitor level were compiled from EPA's AIRS system. Differences between the modeled control and no-control scenario ozone concentrations were then used to scale historical data to derive no-control scenario ozone air quality profiles.[2] Next, the ozone index used in the exposure response evaluation was calculated and applied at the monitor level. For this analysis, the W126 index, a peak-weighted average of cumulative ozone concentrations, was selected to conform with the index currently being used by EPA in ozone NAAQS benefits analysis. The W126 index is one of several cumulative statistics, and may correlate more closely to crop damage than do unweighted indices.[3] EPA has not yet made a final determination of the appropriate index to use in agricultural benefits analy-

[1] Shriner et al., 1990; NAPAP, 1991.

[2] Derivation of these ozone air quality profiles for the control and no-control scenario is summarized in the following section and described in detail in Appendix C.

[3] Lefohn et al., 1988.

sis; thus this analysis should be viewed only as an indicator of the magnitude of potential benefits.

The third step in ozone concentration estimation involved the use of triangulation and planar interpolation to arrive at a W126 statistic at the county, rather than at the monitor, level. For each county centroid, the closest surrounding triangle of monitors is located and the W126 is calculated for that county using a distance-weighted average of the ozone concentration at each of these monitors.

Control and No-control Scenario Ozone Concentration Data

The initial estimation of ozone concentrations in the control and no-control scenarios was performed by Systems Applications International (SAI). To create the control scenario, SAI compiled ozone data from the EPA's Aerometric Information and Retrieval System (AIRS).[4] SAI summarized these data by fitting gamma distributions to them and providing the alpha and the beta parameters to these distributions. Each of these distributions describes a set of ozone concentration levels, and the distributions are categorized by year, season, and averaging time. SAI defines six distinct "seasons," each composed of a two month period in the year. This analysis uses those distributions which describe 1-hour average ozone concentrations taken from 7 AM to 7 PM and separated into seasons. The analysis utilizes only those monitor records that were modeled in both the control and no-control scenarios.

To determine the ozone concentrations for the no-control scenario, SAI utilized the Ozone Isopleth Plotting with Optional Mechanisms-IV (OZIPM4) model. The input data required for OZIPM4 includes air quality data, surface and upper-air meteorological data, and estimates of anthropogenic and biogenic emissions of volatile organic compounds, NO_x and CO.[5] To create these inputs, SAI used (among other sources) outputs from the Regional Acid Deposition Model (RADM) and the SJVAQS/AUSPEX Regional Modeling Adaptation project (SARMAP). Additional detail concerning the development of ozone concentration data is available in Appendix C and in the SAI report to EPA.[6]

Calculation of the W126 Statistic

Using the SAI ozone concentration distributions, we calculated a sigmoidally weighted ozone index for each monitor. The generalized sigmoidal weighting function used in calculating such indices is presented in Lefohn and Runeckles (1987) as:

where:

$$w_i = 1 / [1 + M \cdot \exp(-A \cdot i)] \quad (1)$$

w_i = weighting factor for concentration$_i$ (unitless)
c_i = concentration$_i$ (ppm)
M = an arbitrary constant
A = an arbitrary constant

The constants M and A are chosen to give different weights to higher or lower concentrations. The index used in this analysis is the W126 statistic, which is calculated as follows:[7]

$$w_i = 1 / [1 + 4403 \cdot \exp(-126 \cdot c_i)] \quad (2)$$

and

$$W126 = \sum w_i \quad (3)$$

Missing values are accounted for by multiplying the resulting W126 statistic by the ratio of the number of potential observations to the number of actual observations (i.e., total hours in period/hours of data in period).

To calculate W126 indices from the monitor level gamma distributions, we used an inverse cumulative density function to calculate a separate representative air concentration for each hour in the two month season. These values are then used in the above equation to obtain a monitor-level W126 statistic.

To ensure that the interpretation of the gamma distributions in this manner does not generate errors, we tested our gamma-derived control-scenario W126s

[4] SAI, ICF Kaiser, 1995.

[5] SAI, ICF Kaiser, 1995.

[6] SAI, ICF Kaiser, 1995.

[7] Lefohn et al., 1988.

against W126s calculated directly from the AIRS database. We found that insignificant error resulted from the utilization of the gamma distributions to create W126 statistics.

Aggregating Ozone Data to the County Level

Because crop production data are available at the county level, the lowest level of aggregation that could be used for ozone indices is also the county level. Therefore, monitor level data needed to be aggregated to a county level. For each county, we first located the monitors from which we would be interpolating data. To identify these monitors, we searched for the three monitors which formed the closest triangle around the centroid of the county.[8] The closest triangle was defined as that triangle in which the sum of the distances from the three monitors to the county centroid was the least. The algorithm stopped searching for closest triangles of monitors when it had searched all monitors within 500 km of a given county centroid (an arbitrary distance, selected to reduce computational requirements).

For coastal counties and some rural counties in some years, monitor triangles around the county centroid do not exist. We assigned the W126 value from the monitor closest to the centroid to these counties. Approximately 15 percent of all county-years (36,973 of 248,880 records) were assigned W126s in this manner.

For the remaining 85 percent, after the closest triangle of monitors was found, a "planar interpolation" was used to calculate the W126 at that county for that year. One way to picture this process is to plot each of the three monitors as a point in space. For each monitor, the x axis represents longitude, the y axis represents latitude and the z axis represents the W126 statistic. A plane can then be drawn through these three points in space. Furthermore, using the equation for the plane, and given the x and y values (latitude and longitude) for the county centroid, the county centroid's z value (W126 statistic) can be calculated. In essence, this procedure calculates a distance-weighted average of three monitors' W126 index values and assigns this value to the county centroid.

The result of this data manipulation is a monthly W126 statistic for each county in the continental United States for the years 1971-1990. From these data, yield change estimates were generated, and economic impacts were estimated.

Yield Change Estimates

There are several steps involved in generating yield change estimates. The first is the selection of relevant ozone exposure-response functions (minimum and maximum) for each crop in the analysis. Ozone data, triangulated to the county level, are transformed into an index suitable for use in the selected function(s) to estimate county level predicted yield losses for both the control and no-control scenarios. In the next step, the proportion of each county to the national production of each crop is calculated to permit national aggregation of estimated yield losses. Finally, the control scenario percentage relative yield loss (PRYL) is compared to the minimum and maximum PRYL for the no-control scenario. Each step is discussed in more detail below.

Exposure-Response Functions

To estimate yield impacts from ozone, exposure-response functions are required for each crop to be analyzed. This analysis was restricted to estimating changes in yields for those commodity crops for which consistent exposure-response functions are available and that are included in national agricultural sector models. To maintain consistency with the current ozone NAAQS benefits analysis being conducted by OAQPS, NCLAN-based exposure-response functions using a Weibull functional form and a 12-hour W126 ozone index were used.

Several crops included in the NCLAN research program were not evaluated in this analysis. Non-commodity crops that are not modeled in national agricultural sector models were not included in this analysis: lettuce, tomatoes, potatoes, alfalfa, tobacco, turnips, and kidney beans. In addition, one commodity crop, spring wheat, was excluded because the NCLAN exposure-response function was only developed for winter wheat.

[8] The vast majority of monitors had latitude and longitude data available through AIRS. 1,528 of 1,536 monitors were located in this manner. For the remaining 8 monitors, if in a given year of monitor data another monitor exists in the county of the unfound monitor, we discarded the unlocated monitor's data. Otherwise, we located that monitor at the county's centroid. We located 5 of the remaining 8 monitors in this fashion.

Minimum/Maximum Exposure-Response Functions

Estimated responsiveness of a given crop to ozone varies within the NCLAN data. This range of response is partially explained by the program's evaluation of several cultivars for some crops; ozone sensitivity varies across cultivars. In addition, the conditions for different experiments varied due to variations in location, year, and additional treatments included in some experiments. No one exposure-response function can be assumed to be representative of all cultivars in use, or of all environmental conditions for crop production. To develop a range of benefits estimates that reflects this variation in responsiveness, a minimum responsiveness and a maximum responsiveness function were selected for each crop. In actuality, a number of different cultivars are planted by producers, and so ozone response will be a weighted average of the responsiveness of each cultivar to its ozone condition and its proportion of total acreage. It is important to note that these values do not necessarily bound the analysis, since the number of cultivars evaluated by NCLAN is small relative to the number grown for many crops.

For the crops used in this study, CERL conducted an analysis to identify the ozone concentration required to reduce yields by 10 percent for each crop cultivar using its 12-hour W126 exposure-response function. For each crop, the function demonstrating the lowest ozone concentration at a 10 percent yield loss represents the maximum response, and the function with the highest concentration at 10 percent yield loss represents the minimum response. Table F-1 reports the minimum and maximum exposure-response functions for each crop. Two crops, peanuts and sorghum, did not have multiple NCLAN experiments on which to base a comparison of the responsiveness of different cultivars or the variation in response with different experimental conditions.

Calculation of Ozone Indices

Each NCLAN ozone exposure-response experiment exposed each studied crop over a portion of the crop's growing season. The duration of the NCLAN experiments was provided by CERL and was rounded to the nearest month. The W126 index is cumulative, and so is sensitive both to the duration over which it is calculated and to the specific month(s) within a growing season that are included in it. Because cropping seasons vary across the U.S., the ozone index used to calculate county-level changes in yield due to ozone must reflect the local season for each crop. To determine which portion of the growing season a particular exposure period pertains to (in order to calculate an exposure index), we developed state-specific growing seasons based on planting and harvesting data developed by USDA.[9] The W126 index was calcu-

Table F-1. Agriculture Exposure-Response Functions.

Crop	Cultivar	Equation Type	Yield Function (PRYL, ppm)	Duration (days)
Barley	CM-72	Both	$1-\exp(-(W126/6998.5)^{1.388}$	95
Corn-Field	PAG 397	Min	$1-\exp(-(W126/94.2)^{4.176}$	83
Corn-Field	Pioneer 3780	Max	$1-\exp(-(W126/92.7)^{2.585}$	83
Cotton	McNair 235	Min	$1-\exp(-(W126/113.3)^{1.397}$	125
Cotton	Acala SJ2	Max	$1-\exp(-(W126/74.6)^{1.066}$	98
Grain Sorghum	DeKalb A28+	Both	$1-\exp(-(W126/205.3)^{1.957}$	85
Peanuts	NC-6	Both	$1-\exp(-(W126/96.8)^{1.890}$	112
Soybeans	Corsoy-79	Min	$1-\exp(-(W126/476.7)^{1.113}$	93
Soybeans	Davis	Max	$1-\exp(-(W126/130.1)^{1.000}$	93
Wheat	ART	Min	$1-\exp(-(W126/76.8)^{2.031}$	54
Wheat	VONA	Max	$1-\exp(-(W126/24.7)^{1.00)}$	61

Source: EPA/CERL (unpublished) for all functions.

[9] USDA, 1984. Some states did not have explicit growing seasons reported for certain crops due to the low production in these states. In these cases a proxy state growing season was used. In most of these cases the proxy growing season was taken from a state with an adjoining boundary within the same geographic region.

lated using the county level ozone data developed in the prior section, summed for the number of months of NCLAN experimental duration, with the exposure period anchored on the usual harvest month for each crop.[10]

Calculations of County Weights

Because the benefits analysis did not require a regional level of disaggregation and to minimize computational burdens the economic analysis was conducted at a national level. Ozone data and estimated yield responses, however, were developed at a county level. To conduct a national analysis, the county level yield change estimates were weighted to develop a single national percent relative yield loss for each crop relative to the control scenario, for both the minimum and the maximum yield responses. As a part of calculating a percent change in yield at the national level, weights for each county and crop were created for 1975, 1980, 1985, and 1990. The weights for these four years were used to represent the year itself and the four years immediately following it (e.g., 1975 weights were also used for 1976, 1977, 1978, and 1979). Although weather and other conditions may change the proportion of counties' production to the total national production in each year, five year weights should reflect stable periods of agricultural policy between each Farm Bill, and are sufficient for the level of precision needed for this analysis. The weights were calculated by dividing the production level of a crop in a county[11] by the sum of all states' reported production for that crop.[12] These county weights were applied to the percent relative yield loss results for each county, as discussed below.

Calculation of Percent Change in Yield

Ozone exposure-response functions are expressed in terms of percent relative yield loss (PRYL); the ozone level being analyzed is compared with "clean" (charcoal filtered/zero ozone) air. To calculate a percent change in yield between the control and no-control scenarios, we first calculate a PRYL based on the county-level control scenario W126 ozone index, and a PRYL based on the no-control scenario W126 index. Next, the county weights are applied to the PRYLs. The change in yield, measured relative to the hypothetical zero-ozone crop production, is then:

$$(PRYL_C - PRYL_{NC}) \qquad (4)$$

To obtain the change in terms of our (non-zero) baseline yield, we divide by that yield, and get:

$$(PRYL_C - PRYL_{NC}) / (100 - PRYL_C) \qquad (5)$$

To create the national percent change in yield for each crop, the results of this equation are summed for each scenario (maximum and minimum) and for each year. Tables F-2 and F-3 present the resulting percent yield changes that were used as inputs to the economic model.

Economic Impact Estimates

To estimate the economic benefits of controls on ozone precursor pollutants under the Clean Air Act, changes in yields due to those controls need to be evaluated in terms of their effect on agricultural markets. To do this, yield changes can be incorporated into an economic model capable of estimating the associated changes in economic surpluses within the agricultural economy, preferably one that reflects changes in producers' production decisions and demand substitution between crops. This type of dynamic analysis is needed because even small changes in yield or price expectations can cause large shifts in the acreage allocated to specific crops, and the degree to which alternative crops will be substituted (particularly for feed uses).

Agricultural Simulation Model (AGSIM)

The modeling approach used in this analysis is an econometric model of the agricultural sector, which estimates demand and supply under different production technologies and policy conditions. The AGricultural SImulation Model (AGSIM) has been

[10] This analysis required "rounding" some months: if a harvest date was specified to be from the 15th to the end of a month, the W126 index was calculated using that month's data; if the harvest date was specified to be from the first to the 14th of a month, the W126 index was calculated using the prior month's data as the final month in the exposure period.

[11] USDA, 1995.

[12] The national total does not include USDA areas designated "other counties". These areas are groups of counties that for one reason or another (disclosure rules, low amount of production, etc.) are not individually listed. Because we did not have ozone values for these groups, we did not use their production levels in the calculation of the total national production.

Table F-2. Relative No-control to Control Percent Yield Change (harvested acres) for the Minimum Scenario.

	Crop						
Year	**Barley**	**Corn**	**Cotton**	**Peanuts**	**Soybeans**	**Sorghum**	**Winter Wheat**
1975	-0.000020	-0.000171	-0.011936	-0.006635	-0.001166	-0.000717	-0.005631
1976	-0.000013	-0.000329	-0.017505	-0.024048	-0.002171	-0.001841	-0.004841
1977	-0.000013	-0.000169	-0.013114	-0.015150	-0.001562	-0.001118	-0.005464
1978	-0.000019	-0.000291	-0.018692	-0.017606	-0.002480	-0.001844	-0.005894
1979	-0.000027	-0.000100	-0.017217	-0.013067	-0.001898	-0.001389	-0.004998
1980	-0.000019	-0.000200	-0.021315	-0.022761	-0.002397	-0.002222	-0.005385
1981	-0.000016	-0.000071	-0.018552	-0.014269	-0.001951	-0.000802	-0.003964
1982	-0.000020	-0.000070	-0.017295	-0.014200	-0.002107	-0.001050	-0.004773
1983	-0.000023	-0.000617	-0.020842	-0.028601	-0.003901	-0.002366	-0.005904
1984	-0.000027	-0.000111	-0.023552	-0.019225	-0.002919	-0.002881	-0.006121
1985	-0.000025	-0.000132	-0.020947	-0.017965	-0.002645	-0.001726	-0.007316
1986	-0.000029	-0.000158	-0.027968	-0.031605	-0.002899	-0.001564	-0.007597
1987	-0.000033	-0.000358	-0.034584	-0.043854	-0.003776	-0.001812	-0.009669
1988	-0.000027	-0.000662	-0.035069	-0.038085	-0.004563	-0.002922	-0.019873
1989	-0.000024	-0.000150	-0.031245	-0.022094	-0.003769	-0.001359	-0.007605
1990	-0.000024	-0.000210	-0.037988	-0.047395	-0.003819	-0.001567	-0.006449

Note: There is only one scenario for barley, peanuts, and sorghum, because there was only one exposure-response function..

Table F-3. Relative No-control to Control Percent Yield Change (harvested acres) for the Maximum Scenario.

	Crop						
Year	**Barley**	**Corn**	**Cotton**	**Peanuts**	**Soybeans**	**Sorghum**	**Winter Wheat**
1975	-0.000020	-0.001139	-0.021059	-0.006635	-0.005808	-0.000717	-0.034803
1976	-0.000013	-0.002281	-0.032063	-0.024048	-0.010298	-0.001841	-0.040303
1977	-0.000013	-0.001232	-0.025773	-0.015150	-0.007764	-0.001118	-0.049636
1978	-0.000019	-0.002015	-0.033075	-0.017606	-0.011803	-0.001844	-0.050308
1979	-0.000027	-0.001052	-0.031433	-0.013067	-0.009592	-0.001389	-0.052211
1980	-0.000019	-0.001537	-0.037278	-0.022761	-0.011845	-0.002222	-0.054128
1981	-0.000016	-0.000923	-0.035058	-0.014269	-0.009902	-0.000802	-0.053470
1982	-0.000020	-0.000974	-0.034101	-0.014200	-0.010815	-0.001050	-0.058409
1983	-0.000023	-0.003888	-0.040405	-0.028601	-0.018597	-0.002366	-0.063556
1984	-0.000027	-0.001443	-0.043890	-0.019225	-0.014502	-0.002881	-0.067612
1985	-0.000025	-0.001377	-0.040845	-0.017965	-0.013384	-0.001726	-0.072177
1986	-0.000029	-0.001451	-0.052426	-0.031605	-0.014754	-0.001564	-0.081225
1987	-0.000033	-0.002565	-0.061295	-0.043854	-0.018578	-0.001812	-0.089042
1988	-0.000027	-0.004318	-0.061660	-0.038085	-0.021767	-0.002922	-0.120703
1989	-0.000024	-0.001987	-0.059573	-0.022094	-0.018739	-0.001359	-0.086958
1990	-0.000024	-0.002056	-0.071659	-0.047395	-0.018670	-0.001567	-0.082309

Note: There is only one scenario for barley, peanuts, and sorghum, because there was only one exposure-response function.

used extensively to evaluate air pollution impacts, as well as a number of other environmental policy analyses. AGSIM is an econometric-simulation model that is based on a large set of statistically estimated demand and supply equations for agricultural commodities produced in the United States. The model is capable of estimating how farmers will adjust their crop acreages between commodities when relative profitability changes as a result of crop yield and production cost changes. Acreage and yield changes from various scenarios will affect total production of crops, which then affects commodity prices and consumption. The commodity price changes, in turn, affect profitability and cropping patterns in subsequent years. Federal farm program and conservation reserve effects are also incorporated into the model.

The initial version of AGSIM (which went through various acronym revisions) was developed in 1977.[13] The model was developed to permit estimation of aggregate impacts associated with relatively small changes in crop yields or production costs, which might result from various policy conditions such as changes in pesticide input availability, or in this case, changes in crop exposure to ozone. Subsequent revisions to the model as well as the current specification are described in Taylor (1993a).[14] Several policy applications of AGSIM were tested and reported in Taylor (1993b)[15] to provide a comparison to the results of several alternative agricultural sector models. These tests included an expansion of Conservation Reserve acreage, reduced target prices, elimination of agricultural programs in the U.S. other than the Conservation Reserve Program (CRP), and a tax on nitrogenous fertilizer use in the U.S. The model has been used to evaluate the effects of changes to the CRP,[16] changes in agricultural price support programs,[17] and evaluations of policies concerning pesticide availability.[18]

AGSIM is designed to estimate changes in the agricultural sector resulting from policies that affect either the yields or the costs of crop production. Changes in economic variables are computed by comparing a policy simulation of the model with a baseline simulation of the model. For this retrospective evaluation, the baseline reflects actual farm programs, prices, and other parameters since 1970. The model's author, Dr. C. Robert Taylor, modified AGSIM for this analysis to reflect production conditions and policies as they changed through the 20-year span of the Clean Air Act, from 1970 to 1990. During this period, U.S. farm policy parameters changed every five years with the enactment of each Farm Bill, and producer participation varied significantly over the period. Over this time, due to policy, weather, technological development, and other variations, production levels and prices have varied, as have production technologies, costs of production, and relevant cultivars. To reflect these changes, Dr. Taylor re-estimated demand relationships for three periods (1975-79; 1980-84; and 1985-89) based on the farm policies in effect in each period, and structured the model to run on a national level rather than a regional level. The period from 1970-1975 was not modeled because of data limitations and because there was limited impact from the CAA on ozone levels during that period.

The AGSIM baseline production and price data serve as the control scenario baseline. Percent relative yield losses (PRYLs) between the control and no-control scenarios are the relevant input parameter for this analysis, from which AGSIM calculates new yield per planted acre values. Based on these values (as well as on lagged price data, ending stocks from the previ-

[13] Taylor, C.R., R.D. Lacewell, and H. Talpaz. 1979. Use of Extraneous Information with the Econometric Model to Evaluate Impacts of Pesticide Withdrawals. Western J. of Ag. Econ. 4:1-8.

[14] Taylor, C.R. 1993a. AGSIM: An Econometric-Simulation Model of Regional Crop and National Livestock Production in the United States. In: C.R. Taylor, K.H. Reichelderfer, and S.R. Johnson (Eds) Agricultural Sector Models for the United States: Descriptions and Selected Policy Applications. Ames Iowa: Iowa State University Press.

[15] Taylor, C.R. 1993b. Policy Evaluation Exercises with AGSIM. In: C.R. Taylor, K.H. Reichelderfer, and S.R. Johnson (Eds) Agricultural Sector Models for the United States: Descriptions and Selected Policy Applications. Ames Iowa: Iowa State University Press.

[16] Taylor, C.R. 1990. Supply Control Aspects of the Conservation Reserve. In: T.L. Napier (Ed) Implementing the Conservation Title of the Food Security Act of 1985. Ankeny, Iowa: Soil and Water Conservation Society; Taylor, C.R., H.A. Smith, J.B. Johnson, and T.R. Clark. 1994. Aggregate Economic Effects of CRP Land Returning to Production. J. of Soil and Water Conservation 49:325-328.

[17] Talyor, C.R. 1994. Deterministic vs. Stochastic Evaluation of the Aggregate Effects of Price Support Programs. Agricultural Systems 44:461-474.

[18] Taylor, C.R., G.A. Carlson, F.T. Cooke, K.H. Reichelderfer, and I.R. Starbird. Aggregate Economic Effects of Alternative Boll Weevil Management Strategies. Agricultural Econ. Res. 35:19-19;Taylor, C.R., J.B. Penson Jr., E.G. Smith, and R.D. Knutson. 1991. Impacts of Chemical Use Reduction in the South. S. J. Of Ag. Econ. 23:15-23.

ous year, and other variables), AGSIM predicts acreage, production, supply, and price parameters for each crop for each year, as well as calculating yield per harvested acre. From these results and the demand relationships embedded in the model, AGSIM calculates the utilization of each crop (i.e., exports, feed use, other domestic use, etc.), as well as the change in consumer surplus, net crop income, deficiency payments and other government support payments. Net surplus is calculated as net crop income plus consumer surplus, less government payments. The first year of results is 1976 because AGSIM must have one year (1975) of lagged data.

Table F-4 presents the net *changes* in economic surpluses (in 1990 dollars) annually and as a cumulative present value (discounted at 5 percent) over the period 1976-1990 due to the Clean Air Act. The positive surpluses exhibited in almost all years are a result of the increase in yields associated with lower ozone levels than those predicted to occur under the no-control scenario. The present value of the estimated agricultural benefits of the CAA ranges between $7.8 billion in the minimum response case to approximately $37 billion in the maximum response case. This range represents the impacts that would occur if all of the acreage planted to a given crop had an ozone response function similar to either the minimum *available* response function or the maximum *available* response function. The available response functions do not necessarily bracket the true range of potential crop responses, and it is unrealistic to anticipate that all acreage will be planted in cultivars with a uniform response to ozone exposure. These considerations notwithstanding, these values do indicate the likely magnitude of agricultural benefits associated with control of ozone precursors under the CAA, but not the precise value of those benefits. In addition to estimating the present value of net surplus at a discount rate of five percent, two alternative discount rates were used. At a three percent discount rate, the range of net surplus is between $6.7 billion and $32 billion; at seven percent discount rate, the range is between $9 billion and $43 billion. For more detail on AGSIM intermediate model outputs, see Abt Associates (1996).

Table F-4. Change in Farm Program Payments, Net Crop Income, Consumer Surplus, and Net Surplus Due to the CAA (millions 1990 $).

	Change in Farm Program Payments		Change in Net Crop Income		Change in Consumer Surplus		Change in Net Surplus	
Year	**Minimum**	**Maximum**	**Minimum**	**Maximum**	**Minimum**	**Maximum**	**Minimum**	**Maximum**
1976/77	0	0	243	486	236	993	477	1,479
1977/78	0	0	-97	-259	349	1,557	253	1,297
1978/79	43	345	30	298	392	1,646	379	1,597
1979/80	0	0	-140	-406	449	2,000	309	1,594
1980/81	0	0	8	-178	392	2,049	400	1,870
1981/82	112	518	-99	-406	440	2,594	231	1,670
1982/83	168	981	64	107	377	2,730	273	1,856
1983/84	153	1,009	231	958	316	1,969	395	1,917
1984/85	-182	808	82	560	-279	1,686	-14	1,437
1985/86	289	1,291	181	879	616	2,054	509	1,644
1986/87	270	1,356	230	966	462	2,265	422	1,875
1987/88	469	2,033	320	1,405	708	2,990	558	2,361
1988/89	557	2,023	316	1,508	796	2,943	556	2,428
1989/90	329	1,401	161	614	527	2,572	358	1,785
1990/91	414	1,927	180	473	618	3,047	384	1,593
Cumulative Present Value of Net Surplus at 5 percent ($ 1990)							7,763	37,225

Conclusions

Agricultural benefits associated with control of ozone precursors under the Clean Air Act are likely to be fairly large. Because it is possible that over time producers have adopted more ozone-resistant cultivars, it may be appropriate to consider the lower end of the range of predicted benefits to be more indicative of the likely total benefits. The estimates developed in this analysis, however, do not represent all of the likely benefits accruing to agriculture, in that many high-value and/or ozone sensitive crops could not be included in the analysis due to either exposure-response data limitations or agricultural sector modeling limitations. The second consideration implies that benefits will likely be larger than estimated. The minimum case may be the most appropriate starting point, however, due to the first consideration: the current crop mix is probably biased toward lower ozone responsiveness. Therefore, we anticipate that cumulative total agricultural benefits from the Clean Air Act are on the order of ten billion dollars (real terms).

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Appendix G: Lead Benefits Analysis

Introduction

The scientific understanding of the relationship between lead and human health is rapidly expanding. This expansion is documented in numerous EPA studies on the health effects associated with lead exposure. In a pioneering study, Schwartz et al. (U.S. EPA, 1985) quantified a number of health benefits that would result from reductions in the lead content of gasoline. The work was extended by EPA's analysis of lead in drinking water (U.S. EPA, 1986a) and by an EPA-funded study of alternative lead National Ambient Air Quality Standards (U.S. EPA, 1987).

Despite this substantial research, much uncertainty remains. While the health effects of very high levels of blood lead (PbB) are quite severe (including convulsions, coma and death from lead toxicity) and have been known for many years, the effects of lower lead doses continue to be the subject of intensive scientific investigation. Dose-response functions are available for only a handful of health endpoints associated with elevated blood lead levels. Other known or strongly suspected health endpoints cannot be quantified due to a lack of information on the relationship between dose and effect. Table G-1 presents the health effects that are quantified in this analysis, as well as important known health effects that are not quantified.

Some of the health effects that are quantified in this analysis have not been estimated in previous EPA analyses. This is largely due to more recent information about the dose-response functions that makes it possible to expand the health effect coverage beyond what was done previously. Recent information is available for previously unquantified health effects, and new information on previously estimated dose-response functions is also available.

Table G-1. Quantified and Unquantified Health Effects of Lead.

Population Group	Quantified Health Effect	Unquantified Health Effect
Adult Male	*For men in specified age ranges:* Hypertension Non-fatal coronary heart disease Non-fatal Strokes Mortality	Quantified health effects for men in other age ranges Other cardiovascular diseases Neurobehavioral function
Adult Female	*For women in specified age ranges:* Non-fatal coronary heart disease Non-fatal stroke Mortality	Quantified health effects for women in other age ranges Other cardiovascular diseases Reproductive effects Neurobehavioral function
Children	IQ loss effect on lifetime earnings IQ loss effects on special educational needs Neonatal mortality due to low birth weight caused by maternal exposure to lead	Fetal effects from matemal exposure (including diminished IQ) Other neurobehavioral and physiological effects Delinquent and anti-social behavior

Methods Used to Measure and Value Health Effects

The following sections present relevant dose-response relationships for three population groups: children, men, and women. These sections also discuss data sources used for the dose-response relationships, although an extensive review of the literature is not given.[1] In addition, each section includes the methods used to value the changes in health effects determined using these dose-response relationships.

Health Benefits to Children

Changes in IQ

Elevated Pb levels may induce a number of effects on the human nervous system. Generally, these neurobehavioral effects are more serious for children than for adults because of children's rapid rate of development. It is believed that neurobehavioral deficits in children may result from both pre-natal and post-natal exposure. These nervous system effects may include hyperactivity, behavioral and attentional difficulties, delayed mental development, and motor and perceptual skill deficits. Quantification of certain manifestations of these effects is possible because sufficient data exist to estimate a dose-response relationship and IQ loss. The relationship used in the analysis is discussed below.

Quantifying the Relationship Between Blood Lead Levels and IQ

A dose-response relationship for IQ decrements has been estimated by a meta-analysis of seven research studies.[2] Regression coefficients for each study were used to determine a weighted average linear regression coefficient for the relationship between lead and IQ. Each regression coefficient was weighted by the inverse of the variance of each estimate. To determine an overall coefficient, the regression coefficients for studies that used natural logarithms of lead as the exposure index were linearized. In general, the coefficient was linearized in the blood lead range of 10 to 20 µg/dL. However, in one study (Bellinger et al., 1991), 70 percent of the data were below 10 µg/dL; thus, the Bellinger data were linearized in the 5 to 15 µg/dL range. For the studies that did not transform lead concentrations, the regression coefficients were used directly. Given the typical uncertainty within individual studies, the variation in the regression coefficients among studies was not more than would be expected. The relationship determined by Schwartz (1993) suggests that for a 1 µg/dL increase in lead, a decrease of 0.25 IQ points can be expected. The p-value (< 0.0001) indicates that this relationship is highly significant.

To obtain the total change in number of IQ points for a population of children, the 0.25 points lost per µg/dL change in blood lead is multiplied by the average blood lead level for that population. The average blood lead level modeled in this analysis is a geometric mean, not an arithmetic mean. To adjust for this, we use a relationship between the expected value and the geometric mean of a lognormally distributed random variable:

$$E[x] = \exp 1n\left[(GM) + \frac{(1n\,(GSD))^2}{2}\right] \qquad (1)$$

where E(X) is the expected value (mean) of the distribution, GM is the geometric mean, and GSD is the geometric standard deviation. Taking the natural logarithm of Equation 1 and rearranging gives the ratio between the expected value and the GM:

$$1n\,(E(X)) - 1n\,(GM) = \frac{(1n\,(GSD))^2}{2} \qquad (2)$$

$$1n\left[\frac{E(X)}{GM}\right] = \frac{(1n\,(GSD))^2}{2} \qquad (3)$$

$$\frac{E(X)}{GM} = \exp\left[\frac{(1n\,(GSD))^2}{2}\right] \qquad (4)$$

For a GSD of 1.6 (the assumed GSD of children's blood lead levels[3]), the resulting ratio between E(X) and GM is 1.117. This ratio is used in equation 5.

[1] For a detailed review of this literature see U.S. Environmental Protection Agency, (1986b) *Air Quality Criteria Document for Lead*, and 1989 Addendum. Environmental Criteria and Assessment Office, Office of Research and Development, March.

[2] Schwartz, 1993.

[3] Suggested value for sub-populations provided by IEUBK guidance manual (U.S. EPA, 1994).

The total lost IQ points for each group was estimated as:

$$(TOTAL\ LOST\ IQ)_k = \Delta GM_k \times 1.117 \times 0.25 \times (Pop)_k / 7 \quad (5)$$

where $(Pop)_k$ represents the number of children (up to age six) around a given industrial source (in the case of estimating benefits from reduced industrial emissions) or the total U.S. population of children (in the case of estimating benefits from reductions in gasoline lead emissions).

As shown in equation 5, the population of children up to age six is divided by seven to avoid double counting. If we assume that children are evenly distributed by age, this division applies this equation to only children age 0-1. If we did not divide, this equation would count a child who is age zero in the first year of the analysis and count that same child 6 more times in successive years. Dividing by seven does create some undercounting because in the first year of the analysis children from age 1 to 6 are not accounted for, while presumably they are affected by the lead exposure.

The analysis assumes a permanent loss of IQ based on blood lead levels estimated for children six years and younger. Recent studies[4] provide concrete evidence of long-term effects from childhood lead exposure.

Valuing Changes in Children's Intelligence

Available economic research provides little empirical data for society's willingness to pay (WTP) to avoid a decrease in an infant's IQ. Some research, however, has addressed monetization of *a subset* of the effects of decreased IQ. These effects would represent components of society's WTP to avoid IQ decreases. Employed alone, these monetized effects should underestimate society's WTP. Nevertheless, for the purpose of this analysis, these effects are used to approximate the WTP to avoid IQ decrements.

IQ deficits incurred through lead exposure are assumed to persist throughout the exposed infant's lifetime. Two consequences of this IQ decrement are then considered: the decreased present value of expected lifetime earnings for the infant, and the increased educational resources expended for a infant who becomes mentally handicapped or is in need of compensatory education as a consequence of lead exposure. The value of foregone earnings is addressed in this section.

The reduction in IQ has a direct and indirect effect on earnings. The direct effect is straightforward: lower IQs decrease job attainment and performance. Reduced IQ also results in reduced educational attainment, which, in turn, affects earnings and labor force participation. These effects on earnings are additive since the studies used for this analysis have controlled for these effects separately.[5] If personal decisions about the total amount of education and labor force participation were based entirely on each individual maximizing the expected present value of lifetime income, the magnitude of the indirect effect on income of a small change in educational attainment would be close to zero,[6] and certainly less than the magnitude of the direct effect. However, individuals make educational decisions based on a number of considerations in addition to the effect on the present value of lifetime earnings, such as satisfaction (utility) derived from formal education, non-compensation aspects of alternative career opportunities, the ability to pay educational costs, etc. Such considerations could lead to either a positive or negative marginal return to education. Studies[7] of educational attainment and lifetime earnings have generally identified a positive marginal return to education, suggesting that the educational attainment decision may not be based simply on expected earnings.

This analysis uses two sets of estimates of the effects of IQ on earnings. The first estimate, used by Abt Associates in a previous analysis, is based on several older studies. The second estimate is based on Salkever (1995).

Older Estimate of the Effect of IQ on Earnings: The Direct Effect of IQ on Wage Rate

Henry Aaron, Zvi Griliches, and Paul Taubman have reviewed the literature examining the relation-

[4] For example, Bellinger (1992).

[5] IQ is also correlated with other socio-economic factors which have not been quantified in this analysis.

[6] This is a straightforward result of the "envelope theorem" in economics. In this context, the envelope theorem shows that if individuals select the level of education that maximizes expected income, then the marginal benefit of additional education (i.e., the partial derivative of income with respect to education) will be zero at that optimal education level.

[7] Including Chamberlain and Griliches (1977), Ashenfelter and Ham (1979), and Salkever (1995)

ship between IQ and lifetime earnings.[8] They found that the direct effect, (schooling held constant) of IQ on wage rates ranged from 0.2 percent to 0.75 percent per IQ point. Perhaps the best of these studies is Griliches (1977).[9] He reported the direct effect of IQ on wage rates to be slightly more than 0.5 percent per IQ point. Because this is roughly the median estimate of the U.S. EPA review of the literature, this estimate is used.

Older Estimate of the Effect of IQ on Earnings: The Indirect Effect of IQ on Earnings

From Needleman et al. (1990) it is possible to estimate the change in years of schooling attained per one IQ point change. The study's regression coefficients for the effect of tooth lead on achieved grade provide an estimate of current grade achieved. However, many of these children were in college at the time and are expected to achieve a higher grade level. Following Schwartz (1990), after adjusting the published results for the fact that a higher percentage of children with low tooth lead were attending college, a 0.59 year difference in expected maximum grade achieved between the high and low exposure groups was estimated. It is assumed that educational attainment relates with blood lead levels in proportion to IQ. The difference in IQ score between the high and low exposure group was 4.5 points (from Needleman et al. (1990)). Dividing 0.59/4.5 = 0.131 suggests that the increase in lead exposure which reduces IQ by one point may also reduce years of schooling by 0.131 years.

Studies that estimate the relationship between educational attainment and wage rates (while controlling for IQ and other factors) are less common. Chamberlain and Griliches (1977) estimate that a one year increase in schooling would increase wages by 6.4 percent. In a longitudinal study of 799 subjects over 8 years, Ashenfelter and Ham (1979) reported that an extra year of education increased the average wage rate over the period by 8.8 percent. We use the average of these two estimates (7.6 percent) to calculate the indirect effect of increased schooling on the present value of lifetime income. Increased wages per IQ point are calculated using: (7.6 percent wage increase/school year) x (0.131 school years/IQ) = 1.0 percent increase in earnings per IQ point.

There is one final indirect effect on earnings. Changes in IQ affect labor force participation. Failure to graduate high school, for example, correlates with participation in the labor force, principally through higher unemployment rates and earlier retirement ages. Lead is also a strong correlate with attention span deficits, which likely reduce labor force participation. The results of Needleman et al. (1990) relating lead to failure to graduate high school can be used to estimate changes in earnings due to labor force participation. Using the odds ratio from Needleman et al., it was estimated that a one IQ point deficit would also result in a 4.5 percent increase in the risk of failing to graduate. Krupnick and Cropper (1989) provide estimates of labor force participation between high school graduates and non-graduates, controlling for age, marital status, children, race, region, and other socioeconomic status factors. Based on their data, average participation in the labor force is reduced by 10.6 percent for persons failing to graduate from high school. Because labor force participation is only one component of lifetime earnings (i.e., earnings = wage rate X years of work), this indirect effect of schooling is additive to the effect on wage rates. Combining this estimate with the Needleman result of 4.5 percent increase in the risk of failing to graduate high school per IQ point, indicates that the mean impact of one IQ point loss is a (10.6 percent x 4.5 percent) = 0.477 percent decrease in expected earnings from reduced labor force participation.

Combining the direct effect of 0.5 percent with the two indirect effects (1.0 percent for less schooling and 0.477 percent for reduced labor force participation) yields a total of 1.97 percent decrease in earnings for every loss of one IQ point.

Newer Estimate of the Effect of IQ on Earnings: Salkever (1995)

One of the most recent studies of the effects of IQ on earnings is Salkever (1995). Such an analysis with more recent data is valuable because the labor market has undergone many changes over the quarter century in which earlier studies have appeared. Like the analysis of the effect of IQ on earnings presented above, Salkever (1995) estimates this as the sum of direct and indirect effects. The *direct* effect is the sum of effects of IQ test scores on employment and earn-

[8] U.S. EPA, 1984.

[9] Griliches used a structural equations model to estimate the impact of multiple variables on an outcome of interest. This method has conceptual advantages over other empirical estimates used in the literature because it successfully controls for the many confounding variables that can affect earnings.

ings for employed persons, holding years of schooling constant. The *indirect* effect works through the effect of IQ test scores on years of schooling attained, and the subsequent effect of years of schooling on the probability of employment, and on earnings for employed persons.

Salkever (1995) provides updated estimates all of the necessary parameters using the most recent available data set, the National Longitudinal Survey of Youth (NLSY). Three regression equations provide these parameters. The years of schooling regression shows the association between IQ scores and highest grade achieved, holding background variables constant. The employment regression shows the association between IQ test scores, highest grade, and background variables on the probability of receiving earned income. This regression thus provides an estimate of the effect of IQ score on employment, holding schooling constant, and the effect of years of schooling on employment, holding IQ constant. The earnings regression shows the association between IQ test scores, highest grade, and background variables on earnings, for those with earned income.

These regressions provide parameters needed to estimate the total effect of a loss of an IQ point on earnings. The direct effects of IQ on employment and earnings for employed persons, holding schooling constant, come from the employment and earnings regressions. The indirect effect of IQ on employment through schooling is the product of the effect of IQ on years of schooling, from the years of schooling regression, and the effect of highest grade on employment, from the employment regression. The indirect effects of IQ on earnings for employed persons through schooling is the product of the effect of IQ on years of schooling, from the years of schooling regression, and the effect of highest grade on earnings for employed persons, from the earnings regression.

The total estimated effect of a loss of an IQ point on earnings is larger than the previous estimate of 1.97 percent. Based on the Salkever study, the most recent estimate of the effect of an IQ point loss is now a reduction in earnings of 1.93 percent for men and 3.22 percent for women, which is a participation-weighted average of 2.39 percent.

Value of Foregone Earnings

In the next step to monetize intelligence effects, the percent earnings loss estimate must be combined with an estimate of the present value of expected lifetime earnings. Data on earnings for employed persons and employment rates as a function of educational attainment, age, and gender were reported for the U.S. population in 1992 by the Bureau of the Census.[10] Assuming this distribution of earnings for employed persons and labor force participation rates remains constant over time, and further assuming a trend rate of real wage growth (productivity effect), an annual discount factor, and year-to-year survival probabilities, the current Census data on earnings can be used to calculate the mean present value of lifetime earnings of a person born today. This analysis assumed a person received earned income from age 18 to age 64, and assumed a real wage growth rate of one percent and an annual discount rate of five percent. Men tend to earn more than women because of higher wage rates and higher labor force participation. However, for both men and women, expected lifetime earnings increase greatly with education.

While the Census data are most likely the best available basis for projecting lifetime earnings, a number of uncertainties deserve mention. Labor force participation rates of women, the elderly, and other groups will most likely continue to change over the next decades. Real earnings of women will probably continue to rise relative to real earnings of men. Unpredictable fluctuations in the economy's growth rate will probably affect labor force participation rates and real wage growth of all groups. Medical advances will probably raise survival probabilities.

One problem that was addressed was the fact that the current educational distribution for older persons today is an especially poor predictor of educational attainment for those born today, since educational attainment has risen over time. In fact, if one simply projected educational attainment for a person born today using this method, this person would lose years of schooling with age (starting between ages 40 and 50), since average years of schooling declines with age in a one-time snapshot of the current population. To address this issue, the analysis assumes education levels cannot fall as a person ages.

Note that use of earnings is an incomplete measure of an individual's value to society. Those individuals who choose not to participate in the labor force for all of their working years must be accounted for, since the lost value of their productive services may not be accurately measured by wage rates. The largest group are those who remain at home doing housework and child rearing. Also, volunteer work contrib-

[10] U.S. Department of Commerce, 1993

utes significantly to social welfare and rates of volunteerism tend to increase with educational attainment and income.[11] If the opportunity cost of non-wage compensated work is assumed to be the average wage earned by persons of the same sex, age, and education, the average lifetime earnings estimates would be significantly higher and could be approximated by recalculating the tables using full employment rates for all age/sex/education groups. To be conservative, only the value of lost wages is considered in this analysis.

The adjusted value of expected lifetime earnings obtained above is a present value for an individual entering the labor force at age 18 and working until age 64. Given a five percent discount rate, the other assumptions mentioned, and current survival probabilities,[12] the present value of lifetime earnings of a person born today would be $170,169.

Costs of Additional Education

The increase in lifetime earnings from additional education is the gross return to education. The gross return to education, however, does not reflect the cost of the additional education. The cost of the marginal education must be subtracted from the gross return in order to obtain the net increase per IQ point from additional education. There are two components of the cost of marginal education; the direct cost of the education, and the opportunity cost of lost income during the education. An estimate of the educational cost component is obtained from the U.S. Department of Education.[13] The marginal cost of education used in this analysis is assumed to be $5,500 per year. This figure is derived from the Department of Education's reported ($5,532) average per-student annual expenditure (current plus capital expenditures) in public primary and secondary schools in 1989-'90. For comparison, the reported annual cost of college education (tuition, room and board) in 4 year public institutions is $4,975, and $12,284 for private institutions.

The estimated cost of an additional 0.131 years of education per IQ point (from the older estimate of IQ effects) is $721 (i.e., 0.131 x $5,500). Because this marginal cost occurs at the end of formal education, it must be discounted to the time the exposure and damage is modeled to occur (age zero). The average level of educational attainment in the population over age 25 is 12.9 years.[14] Therefore, the marginal educational cost is assumed to occur at age 19, resulting in a discounted present value cost of $285.

The other component of the marginal cost of education is the opportunity cost of lost income while in school. Income loss is frequently cited as a major factor in the decision to terminate education, and must be subtracted from the gross returns to education. An estimate of the loss of income is derived assuming that people in school are employed part time, but people out of school are employed full time. The opportunity cost of lost income is the difference between full-time and part-time earnings. The median annual income of people ages 18-24 employed full-time is $16,501, and $5,576 for part-time employment.[15] The lost income associated with being in school an additional 0.131 years is $1,431, which has a present discounted value at age zero of $566.

Salkever found a smaller effect of IQ on educational attainment (0.1007 years per IQ point, versus 0.131 years), which results in smaller estimated costs. Using the same method and data described above, the estimated present value of educational cost per IQ point is $219, and the income opportunity cost is $435.

Final Estimate of the Effect of IQ on Earnings.

Combining the value of lifetime earnings with the two estimates of percent wage loss per IQ point yields a low estimate of $170,169 x 1.97 percent = $3,000 per lost IQ point, and a higher estimate of $4,064 based on Salkever (1995). Subtracting the education and opportunity costs reduces these values to $2,505 and $3,410 per IQ point, respectively. This analysis uses the midpoint of these two estimates, which is $2,957. Of course, changing the discount rate would change this estimate. With an assumed discount rate of seven percent, the final estimate is only $1,311. With an assumed discount rate of three percent, the final estimate rises to $6,879.

[11] U.S. Department of Commerce, 1986. Table No. 651, p. 383.

[12] Special education costs for children who do not survive to age 18 are not counted, which results in some underestimation of benefits. However, most child mortality occurs before the age of 7, when the special education begins, so this under-counting is not substantive.

[13] "Digest of Education Statistics". U.S. Dept. of Education, 1993.

[14] "Digest of Education Statistics". U.S. Dept. of Education, 1993.

[15] "Money Income of Households, Families, and Persons in the United States: 1992". U.S. Department of Commerce, 1993.

Children with IQs Less Than 70

Quantifying the Number of Children with IQs Less than 70

In addition to the total IQ point decrements that can be predicted to occur in a population of children having a specified blood lead distribution, increases are also expected to occur in the incidence of children having very low IQ scores as the mean blood lead level for that population increases. IQ scores are normalized to have a mean of 100 and a standard deviation of fifteen. An IQ score of 70, which is two standard deviations below the mean, is generally regarded as the point below which children require special compensatory education tailored to the mentally handicapped.

The relationship presented here for estimating changes in the incidence of IQ < 70 was developed to make use of the most current IQ point decrement function provided by Schwartz (1993). It is assumed that for a baseline set of conditions where a population of children has a blood lead distribution defined by some geometric mean and geometric standard deviation, that population also has a normalized IQ point distribution with a mean of 100 and a standard deviation of 15. For this baseline condition, the proportion of the population expected to have IQ < 70 is determined from the standard normal distribution function:

$$P(IQ<70) = \Phi(z) \qquad (6)$$

where:

P(IQ <70) = Probability of IQ scores less than 70

z = standard normal variate; computed for an IQ score of 70, with mean IQ score of 100 and standard deviation of 15 as:

$$z = \frac{70 - 100}{15} = -2 \qquad (7)$$

$\Phi(z)$ = Standard normal distribution function:

$$\frac{1}{\sqrt{2\pi}} \int_{-\infty}^{z} e^{-\frac{u^2}{2}} \, du \qquad (8)$$

The integral in the standard normal distribution function does not have a closed form solution. Therefore, values for $\Phi(z)$ are usually obtained readily from software with basic statistical functions or from tables typically provided in statistics texts. The solution for $\Phi(z)$ where z = -2 is 0.02275. That is, for the normalized IQ score distribution with mean of 100 and standard deviation of 15, it is expected that approximately 2.3 percent of children will have IQ scores below 70.

To estimate changes in the proportion of children with IQ scores below 70 associated with changes in mean blood lead levels for a population of children, the following two key assumptions are made:

1. The mean IQ score will change as a result of changes in the mean blood lead level as:
   $$\Delta\overline{IQ} = -0.25 \times \Delta\overline{PbB}$$
   where
   $$\Delta\overline{IQ} \text{ and } \Delta\overline{PbB}$$
   are the changes in the mean IQ score and in the mean blood lead levels, respectively, between the no-control and control scenarios. This relationship relies on Schwartz' estimate (1993) of a decrease of 0.25 IQ points for each µg/dL increase in blood lead. Note that the mean blood lead level referred to here is the arithmetic mean (or expected value) for the distribution obtained as described previously from the GM and GSD.

2. The standard deviation for the IQ distribution remains at 15.

Using these assumptions, the change in the proportion of children having IQ <70 can then be determined for a given change in mean blood lead from:

$$\Delta P(IQ<70) = \Phi(z_{No\text{-}control}) - \Phi(z_{control}) = \Phi(z_{No\text{-}control}) - 0.02275 \qquad (9)$$

where,

$$z_{No\text{-}control} = \frac{70 - (100 + 0.25 \times \Delta\overline{PbB})}{15} \qquad (10)$$

For a given change in PbB between the control and no-control scenarios a response in terms of IQ is calculated. The procedure above yields an estimate of the percent of the population with IQs less than 70. This percentile is multiplied by the exposed population of children to estimate the increased incidence of

children with low IQs. As in the IQ point loss equation, the results of this function are applied to children age 0-6 and divided by seven to avoid double counting. (See discussion under equation 5).

This procedure quantifies only the change in the number of children who pass below the IQ=70 threshold. Any other changes in children's IQ are quantified using the IQ point loss function described previously. Treating these two endpoints additively does not result in double counting, because the value associated with the IQ point loss function is the change in worker productivity while the value associated with IQs less than 70 is the increased educational costs for the individual, as discussed below.

Valuing the Reduction in Number of Children with IQs less than 70

To value the reduction in the number of children with IQs less than 70, the reduction in education costs were measured - a clear underestimate of the total benefits.[16] Kakalik et al. (1981), using data from a study prepared for the Department of Education's Office of Special Education Programs, estimated that part-time special education costs for children who remained in regular classrooms cost $3,064 extra per child per year in 1978. Adjusting for inflation and real income growth using the GNP price deflator yields an estimate of $6,318 per child in 1990 dollars. For the calculations, this incremental estimate of the cost of part-time special education was used to estimate the cost per year per child needing special education as a result of impacts of lead on mental development. Costs would be incurred from grades one through twelve. Discounting future expenses at a rate of three percent yields an expected present value cost of approximately $52,700 per infant (assuming compensatory education begins at age 7 and continues through age 18). Note that this underestimates the cost, since Kakalik et al. measured the increased cost to educate children attending regular school — not a special education program.

Changes in Neonatal Mortality

Quantifying the relationship between PbB levels and neonatal mortality

U.S. EPA (1990c) cites a number of studies linking fetal exposure to lead (via *in utero* exposure from maternal intake of lead) to several adverse health effects. These effects include decreased gestational age, reduced birth weight, late fetal death, and increases in infant mortality.[17] The Centers for Disease Control (CDC, 1991a) presents a method to estimate changes in infant mortality due to changes in maternal blood lead levels during pregnancy.[18] The analysis links two relationships. The first relationship, between maternal blood lead level and gestational age of the newborn, was estimated by Dietrich et al. (1987). CDC then estimated infant mortality as a function of gestational age, using data from the Linked Birth and Infant Death Record Project from the National Center for Health Statistics. The resulting association is a decreased risk of infant mortality of 10^{-4} (or 0.0001) for each 1 µg/dL decrease in maternal blood lead level during pregnancy. This is the relationship used in the current analysis.

Valuing changes in neonatal mortality

The central estimate of the monetary benefit associated with reducing risks of neonatal mortality is $4.8 million per avoided mortality. This analysis attempts to capture the credible range of uncertainty associated with this estimate by describing the monetary benefit as a distribution of values: a Weibull distribution with a mean value of $4.8 million and a standard deviation of $3.24 million. Appendix I documents the derivation of this distribution and the sources of uncertainty in valuing reduced mortality risks.

Health Benefits to Men

In addition to adversely affecting children's health, lead exposure has also been shown to adversely affect adults. The health effects in adults that are quantified and included in the benefits analysis are all re-

[16] The largest part of this benefit is the parents' willingness to pay to avoid having their child become mentally handicapped, above and beyond the increased educational costs.

[17] Due to unavailability of suitable data, non-fatal health impacts due to decreased gestational age or reduced birth weight have not been included in this analysis. For example, the benefits from avoided developmental disabilities such as sensory and motor dysfunction associated with decreased gestational age have not been included.

[18] The estimated change in infant mortality due to change in birth weight was not modeled because the data relating prenatal lead exposure to birth weight are not as strong as data relating lead exposure and gestational age.

lated to the effects of lead on blood pressure.[19] The estimated relationships between these health effects and lead exposure differ between men and women. The quantified health effects include increased incidence of hypertension (estimated for males only), initial coronary heart disease (CHD), strokes (initial cerebrovascular accidents and atherothrombotic brain infarctions), and premature mortality. Other health effects associated with elevated blood pressure, and other adult health effects of lead including neurobehavioral effects, are not included in this analysis. This section describes the quantified health effects for men; the next section describes the health effects for women.

Hypertension

Quantifying the relationship between PbB levels and hypertension

Elevated blood lead has been linked to elevated blood pressure (BP) in adult males, especially men aged 40-59 years.[20] Further studies have demonstrated a dose-response relationship for hypertension (defined as diastolic blood pressure above 90 mm Hg for this model) in males aged 20-74 years.[21] This relationship is:

$$\Delta Pr(HYP) = \frac{1}{1 + e^{2.744 - .793*(\ln PbB_1)}} - \frac{1}{1 + e^{2.744 - .793*(\ln PbB_2)}} \quad (11)$$

where:

ΔPr(HYP) = the change in the probability of hypertension;

PbB_1 = blood lead level in the control scenario; and

PbB_2 = blood lead level in the no-control scenario.

Valuing reductions in hypertension

The best measure of the social costs of hypertension, society's willingness to pay to avoid the condition, cannot be quantified without basic research well beyond the scope of this project. Ideally, the measure would include all the medical costs associated with treating hypertension, the individual's willingness to pay to avoid the worry that hypertension could lead to a stroke or CHD, and the individual's willingness to pay to avoid changes in behavior that may be required to reduce the probability that hypertension leads to a stroke or CHD. Medical costs of hypertension can be divided into four categories: physician charges, medication costs, hospitalization costs and lost work time.

This analysis uses recent research results to quantify two components of this benefit category. Krupnick and Cropper (1989), using data from the National Medical Care Expenditure Survey, have estimated the medical costs of hypertension. These costs include physician care, drugs and hospitalization costs. In addition, hypertensives have more bed disability days and work loss days than others of their age and sex. Krupnick and Cropper estimated the increase in work loss days at 0.8 per year, and these were valued at the mean daily wage rate. Adjusting the above costs to 1990 dollars gives an estimate of the annual cost of each case of hypertension of $681. The estimate is likely to be an underestimate of the true social benefit of avoiding a case of hypertension for several reasons. First, a measure of the value of pain, suffering and stress associated with hypertension is not included. Second, the direct costs (out-of-pocket expenses) of diet and behavior modification (e.g., salt-free diets, etc.) are not valued. These costs are likely to be significant, since modifications are typically severe. Third, the loss of satisfaction associated with the diet and behavior modifications are ignored. Finally, the medication for hypertension may produce side effects including drowsiness, nausea, vomiting, anemia, impotence, cancer, and depression. The benefits of avoiding these side effects are not included in this estimate.

Quantifying the relationship between blood lead and blood pressure

Because blood lead has been identified as a risk factor in a number of cardiovascular illnesses,[22] it is useful to quantify the effect of changes in blood lead levels on changes in blood pressure for reasons other than predicting the probability of hypertension. Based on results of a meta-analysis of several studies, Schwartz (1992a) estimated a relationship between a

[19] Citing laboratory studies with rodents, U.S. EPA (1990c) also presents evidence of the genotoxicity and/or carcinogenicity of lead compounds. While such animal toxicological evidence suggests that human cancer effects are possible, dose-response relationships are not currently available.

[20] Pirkle et al., 1985.

[21] Schwartz, 1988.

[22] Shurtleff, 1974; McGee and Gordon, 1976; Pooling Project Research Group, 1978.

change in blood pressure associated with a decrease in blood lead from 10 µg/dL to 5 µg/dL.[23] The coefficient reported by Schwartz leads to the following function relating blood pressure to blood lead for men:

$$\Delta DBP_{men} = 1.4 \times 1n\left(\frac{PbB_1}{PbB_2}\right) \qquad (12)$$

where:

ΔDBP_{men} = the change in men's diastolic blood pressure expected from a change in PbB;
PbB_1 = blood lead level in the control scenario (in µg/dL); and
PbB_2 = blood lead level in the no-control scenario (in µg/dL).

This blood lead to blood pressure relationship is used to estimate the incidence of initial coronary heart disease, strokes (atherothrombotic brain infarctions and initial cerebrovascular accidents) and premature mortality in men.

Changes In Coronary Heart Disease

Quantifying the relationship between blood pressure and coronary heart disease

Estimated blood pressure changes can be used to predict the increased probability of the initial occurrence of CHD and stroke.[24] Increased blood pressure would also increase the probability of reoccurrence of CHD and stroke, but these quantified relationships are not available. First-time coronary heart disease events in men can be predicted using an equation with different coefficients for each of three age groups. For men between 40 and 59 years old, information from a 1978 study by the Pooling Project Research Group (PPRG) is used. PPRG (1978) presents a multivariate model (controlling for smoking and serum cholesterol) that relates the probability of coronary heart disease (CHD) to blood pressure. The model used data from five different epidemiological studies. From this study, the equation for the change in 10-year probability of occurrence of CHD is:

$$\Delta Pr(CHD_{40\text{-}59}) = \frac{1}{1 + e^{4.996 - 0.030365 * DBP_1)}} - \frac{1}{1 + e^{4.996 - 0.030365 * DBP_2)}} \qquad (13)$$

where:

$\Delta Pr(CHD_{40\text{-}59})$ = change in 10-year probability of occurrence of CHD event for men between 40-59 years old,
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

The relationship between BP and first-time CHD in older men was determined from information presented in Shurtleff (1974). This study also uses data from the Framingham Study (McGee and Gordon, 1976) to estimate univariate relationships between BP and a variety of health effects by sex and for each of the following age ranges: 45-54, 55-64, and 65-74 years. Single composite analyses for ages 45-74 were also performed for each sex. For every equation, t-statistics on the variable blood pressure are significant at the 99th percent confidence interval. For men aged 60 to 64 years old, first-time CHD can be predicted from the following equation:

$$\Delta Pr(CHD_{60\text{-}64}) = \frac{1}{1 + e^{5.19676 - 0.02351 * DBP_1)}} - \frac{1}{1 + e^{5.19676 - 0.02351 * DBP_2)}} \qquad (14)$$

where:

$\Delta Pr(CHD_{60\text{-}64})$ = change in 2 year probability of occurrence of CHD event for men from 60 to 64 years old;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

For men aged 65 to 74 years old, the following equation uses data from Shurtleff (1974) to predict the probability of first-time CHD:

$$\Delta Pr(CHD_{65\text{-}74}) = \frac{1}{1 + e^{4.90723 - 0.02031 * DBP_1)}} - \frac{1}{1 + e^{4.90723 - 0.02031 * DBP_2)}} \qquad (15)$$

where:

$\Delta Pr(CHD_{65\text{-}74})$ = change in 2 year probability of occurrence of CHD event for men from 65 to 74 years old;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

[23] Schwartz, 1992a.

[24] U.S. EPA, 1987.

The probability changes calculated using the functions above are used to estimate the number of CHD events avoided in a given year due to air quality improvements attributable to the Clean Air Act. The resulting CHD incidence estimates include both fatal and non-fatal events. However, because mortality benefits are independently estimated in this analysis, it is important to capture only the non-fatal CHD events. Shurtleff (1974) reported that two-thirds of all CHD events were non-fatal. This factor was therefore applied to the estimate of avoided CHD events for each age category.

Valuing reductions in CHD events

General methodology

Because of the lack of information on WTP to avoid an initial CHD event, WTP was estimated by estimating the associated cost of illness (COI). This will underestimate WTP, as explained in Appendix I. Full COI consists of the present discounted value of all costs associated with the event, including both direct and indirect costs incurred during the hospital stay, as well as the present discounted values of the streams of medical expenditures (direct costs) and lost earnings (indirect costs) incurred once the individual leaves the hospital.

Wittels et al. (1990) estimate the total medical costs within 5 years of diagnosis of each of several types of CHD events (including acute myocardial infarction, angina pectoris, unstable angina pectoris, sudden death and nonsudden death) examined in the Framingham Study. Costs were estimated by multiplying the probability of a medical test or treatment within five years of the initial CHD event (and associated with that event) by the estimated price of the test or treatment. All prices were in 1986 dollars. (It does not appear that any discounting was used.) The probabilities of tests or treatments were based on events examined in the Framingham Study. The authors estimate a total expected cost over a five year period (in 1986 dollars) of $51,211 for acute myocardial infarction, $24,980 for angina pectoris, and $40,581 for unstable angina pectoris. Converted to 1990 dollars (using the consumer price index for medical care, U.S. Bureau of the Census, 1992), this is $68,337 for acute myocardial infarction, $33,334 for angina pectoris, and $54,152 for unstable angina pectoris. (The figures for sudden death and nonsudden death are not included because the CHD events in this analysis exclude those that resulted in death, to avoid double counting.)

Cropper and Krupnick (1990) suggest, in an unpublished study, that CHD-related lost earnings could be a significant component of total COI, although the value of earnings lost may vary substantially with the age of onset of CHD. They estimate, for example, that an individual whose first heart attack occurs between ages 55 and 65 will have an expected annual earnings loss of $12,388 (in 1990 dollars), and a present discounted value of lost earnings over a five-year period of about $53,600, using a five percent discount rate. This is almost as much as the total medical costs over 5 years estimated by Wittels et al. (1990) for unstable angina pectoris, and substantially more than the corresponding estimate of medical costs for angina pectoris. For an individual whose first heart attack occurs between ages 45 and 54, on the other hand, Cropper and Krupnick estimate annual average lost earnings of $2,143 (in 1990 dollars), and a present discounted value of lost earnings over a five-year period of about $9,300, again using a five percent discount rate. Cropper and Krupnick do not estimate medical costs for exactly the same disease categories as Wittels et al., but their research suggests that whether the five-year COI of a CHD event, including both medical costs and lost earnings, is lower or higher than the average of the three estimates reported by Wittels et al. depends on an individual's age at the onset of CHD. Combining Cropper and Krupnick's five-year lost earnings estimates with their estimates for average annual medical expenditures for ischemic heart disease summed over five years, for example, yields a total COI of about $47,000 for a 50 year old and $72,000 for a 60 year old, compared to the $52,000 average of the three estimates reported by Wittels et al.

In addition to the variability in estimates of medical costs and lost earnings arising from CHD, there is uncertainty regarding the proportion of pollution-related CHD events associated with the various classes of CHD. To characterize this uncertainty it was assumed, in the absence of further information, that all pollution-related CHD events are either acute myocardial infarctions, angina pectoris, or unstable angina pectoris. A distribution of estimates of COI for pollution-related CHD was generated by Monte Carlo methods. On each iteration, a value was randomly selected from each of three continuous uniform distributions. Each value selected was normalized by

dividing by the sum of the three values, so that the three normalized values summed to 1.0. The resulting triplet of proportions represents a possible set of proportions of pollution-related CHD events that are acute myocardial infarction, angina pectoris, and unstable angina pectoris. The corresponding dollar value for the iteration is a weighted average of the estimated dollar values for the three types of CHD event (from Wittels et al.), where the weights are the three randomly selected proportions. The central tendency estimate of the COI associated with a case of pollution-related CHD is the mean of this distribution, about $52,000.

This estimate is likely to understate full COI because it does not include lost earnings. It is likely to underestimate total WTP to an even greater extent because it does not include WTP to avoid the pain and suffering associated with the CHD event. It is, however, substantially greater than an estimate based only on the direct and indirect costs incurred during the hospital stay.

The valuation for CHD is additive with the valuation for hypertension despite the fact that the conditions often occur together, because the two values represent different costs associated with the conditions. The valuation for hypertension is based on loss of work days as a result of hypertension and some of the medical costs associated with treating hypertension. The valuation for CHD is based on the willingness to pay to avoid the pain and suffering of the CHD itself. Therefore, these two valuations can be separated and added together.

Changes in Initial Cerebrovascular Accidents and Initial Atherothrombotic Brain Infarctions

Quantifying the relationship between blood pressure and first-time stroke

Two types of health events are categorized as strokes: initial cerebrovascular accidents (CA) and initial atherothrombotic brain infarctions (BI). The risk has been quantified for the male population between 45 and 74 years old.[25] For initial cerebrovascular accidents, the logistic equation is:

$$\Delta Pr(CA_{men}) = \frac{1}{1 + e^{8.58889 - 0.04066 * DBP_1)}} - \frac{1}{1 + e^{8.58889 - 0.04066 * DBP_2)}} \quad (16)$$

where:

$\Delta Pr(CA_{men})$ = change in 2 year probability of cerebrovascular accident in men;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

For initial atherothrombotic brain infarctions, the logistic equation is:

$$\Delta Pr(BI_{men}) = \frac{1}{1 + e^{9.9516 - 0.04840 * DBP_1)}} - \frac{1}{1 + e^{9.9516 - 0.04840 * DBP_2)}} \quad (17)$$

where:

$\Delta Pr(BI_{men})$ = change in 2 year probability of brain infarction in men;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

Similar to CHD events, this analysis estimates only non-fatal strokes (to avoid double-counting with premature mortality). Shurtleff (1974) reported that 70 percent of strokes were non-fatal. This factor was applied to the estimates of both CA and BI.

Valuing reductions in strokes

Taylor et al. (1996) estimate the lifetime cost of stroke, including the present discounted value (in 1990 dollars) of the stream of medical expenditures and the present discounted value of the stream of lost earnings, using a five percent discount rate. Estimates are given for each of three separate categories of stroke, separately for males and females at ages 25, 45, 65, and 85. For all three types of stroke, the indirect costs (lost earnings) substantially exceed the direct costs at the two younger ages, and are about the same as or smaller than direct costs at the older ages.

Both types of stroke considered in this analysis fall within the third category, ischemic stroke, considered by Taylor et al. To derive a dollar value of avoiding an initial ischemic stroke for males, a dollar value for avoiding ischemic stroke among males age 55 was interpolated from the values for males ages 45 and 65; similarly, a dollar value for avoiding ischemic stroke among males age 75 was interpolated from the values for males ages 65 and 85. Of males in the United

[25] Shurtleff, 1974.

States between the ages of 45 and 74 (the age group for which lead-related stroke is predicted), 41.2 percent are ages 45-54 and the remaining 58.8 percent are ages 55-74. The value of an avoided stroke among males was calculated as the weighted average of the values for males in age group 45-54 and males in age group 55-74 , where the weights are the above percents. The value for age group 45-54 is the average of the values for ages 45 and 55; the value for age group 55-74 is the average of the values for ages 55, 65 and 75. The resulting average value of an avoided stroke among males aged 45-74 is about $200,000.

Changes in Premature Mortality

Quantifying the relationship between blood pressure and premature mortality

Information also exists to predict the increased probability of premature death from all causes as a function of elevated blood pressure. U.S. EPA (1987) used population mean values for serum cholesterol and smoking to reduce results from a 12 year follow-up of men aged 40-54 in the Framingham Study (McGee and Gordon, 1976) to an equation in one explanatory variable:

$$\Delta Pr(MORT_{40\text{-}54}) = \frac{1}{1 + e^{5.3158 - 0.03516 * DBP_1)}} - \frac{1}{1 + e^{5.3158 - 0.03516 * DBP_2)}} \quad (18)$$

where:

$\Delta Pr(MORT_{40\text{-}54})$ = the change in 12 year probability of death for men aged 40-54;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

Information from Shurtleff (1974) can be used to estimate the probability of premature death in men older than 54 years old. This study has a 2 year follow up period, so a 2 year probability is estimated. For men aged 55 to 64 years old, mortality can be predicted by the following equation:

$$\Delta Pr(MORT_{55\text{-}64}) = \frac{1}{1 + e^{4.89528 - 0.01866 * DBP_1)}} - \frac{1}{1 + e^{4.89528 - 0.01866 * DBP_2)}} \quad (19)$$

where:

$\Delta Pr(MORT_{55\text{-}64})$ = the change in 2 year probability of death in men aged 55-64;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

For men aged 65 to 74 years old, premature mortality can be predicted by the following equation:

$$\Delta Pr(MORT_{65\text{-}74}) = \frac{1}{1 + e^{3.05723 - 0.00547 * DBP_1)}} - \frac{1}{1 + e^{3.05723 - 0.00547 * DBP_2)}} \quad (20)$$

where:

$\Delta Pr(MORT_{65\text{-}74})$ = the change in 2 year probability of death in men aged 55-64;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

Valuing reductions in premature mortality

As discussed above, premature mortality is valued at $4.8 million per case (discussed further in Appendix I). Because this valuation is based on the willingness to pay to the risk of death, and the CHD valuation is based on the willingness to pay to avoid the pain and suffering of a CHD event (defined as a CHD event that does not end in death, to avoid double counting), these two endpoints are additive as well.

Health Benefits to Women

Available evidence suggests the possibility of health benefits from reducing women's exposure to lead. Recent expanded analysis of data from the second National Health and Nutrition Examination Survey[26] (NHANES II) by Schwartz (1990) indicates a significant association between blood pressure and blood lead in women. Another study, by Rabinowitz et al. (1987), found a small but demonstrable association between maternal blood lead and pregnancy hypertension and blood pressure at time of delivery.

[26] The Second National Health and Nutrition Examination Survey (NHANES II) was conducted by the U.S. Department of Health and Human Services from 1976 to 1980 and provides researchers with a comprehensive set of nutritional, demographic and health data for the U.S. population.

The effect of lead exposure on the blood pressure of women, relative to the effect on men, is examined in a review of ten published studies.[27] All of the reviewed studies included data for men, and some included data for women. A concordance procedure was used to combine data from each study to predict the decrease in diastolic BP associated with a decrease from 10 µg/dL to 5 µg/dL PbB. The results suggest that the effect on blood pressure for women of this decrease in blood lead is 60 percent of the effect of the same change observed in men. Thus, for women, Equation can be rewritten as:

$$\Delta DBP_{women} = (0.6 \times 1.4) \times 1n\left(\frac{PbB_1}{PbB_2}\right) \quad (21)$$

where:

ΔDBP_{women} = the change in women's diastolic blood pressure expected from a change in PbB;
PbB_1 = blood lead level in the control scenario; and
PbB_2 = blood lead level in the no-control scenario.

Although women are at risk of having lead-induced hypertension, there is not a dose-response function for hypertension in women available at this time. Omitting the hypertension benefits for women creates an underestimate of the total benefits, but the impact on the total benefits estimation will likely be small. Lead raises blood pressure in women less than in men, so the probability of causing hypertension is likely to be less than in men, and the total value of hypertension in men is a small portion of the overall estimated benefits.

Changes in Coronary Heart Disease

Quantifying the relationship between blood pressure and coronary heart disease

Elevated blood pressure in women results in the same effects as for men (the occurrence of CHD, two types of stroke, and premature death). However, the general relationships between BP and these health effects are not identical to the dose-response functions estimated for men. All relationships presented here have been estimated for women aged 45 to 74 years old using information from Shurtleff (1974). First-time CHD in women can be estimated from the following equation:

$$\Delta Pr(CHD_{women}) = \frac{1}{1 + e^{6.9401 - 0.03072*DBP_1)}} - \frac{1}{1 + e^{6.9401 - 0.03072*DBP_2)}} \quad (22)$$

where:

$\Delta Pr(CHD_{women})$ = change in 2 year probability of occurrence of CHD event for women aged 45-74;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

Again, non-fatal CHD events were estimated by assuming that two-thirds of the estimated events were not fatal (Shurtleff, 1974).

Valuing reductions in CHD events

Values of reducing CHD events for women are assumed to be equal to those calculated for men (above): $52,000 per CHD event.

Changes in Atherothrombotic Brain Infarctions and Initial Cerebrovascular Accidents

Quantifying the relationship between blood pressure and first-time stroke

For initial atherothrombotic brain infarctions in women, the logistic equation is:

$$\Delta Pr(BI_{women}) = \frac{1}{1 + e^{10.6716 - 0.0544*DBP_1)}} - \frac{1}{1 + e^{10.6716 - 0.0544*DBP_2)}} \quad (23)$$

where:

$\Delta Pr(BI_{women})$ = change in 2 year probability of brain infarction in women aged 45-74;
DBP_1 = mean diastolic blood pressure in the control scenario; and
DBP_2 = mean diastolic blood pressure in the no-control scenario.

The relationship between BP and initial cerebrovascular accidents can be predicted by the following logistic equation:

$$\Delta Pr(CA_{women}) = \frac{1}{1 + e^{9.07737 - 0.04287*DBP_1)}} - \frac{1}{1 + e^{9.07737 - 0.04287*DBP_2)}} \quad (24)$$

[27] Schwartz, 1992b.

where:

$\Delta Pr(CA_{women})$ = change in 2 year probability of cerebrovascular accident in women aged 45-74;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

The predicted incidences of avoided BI and CA were multiplied by 70 percent to estimate only non-fatal strokes (Shurtleff, 1974).

Valuing reductions in strokes

The value of avoiding an initial cerebrovascular accident or an initial atherothrombotic brain infarction for women was calculated in the same way as for men (see above). Of women in the United States between the ages of 45 and 74 (the age group for which lead-related stroke was predicted), 38.2 percent are ages 45-54 and the remaining 61.8 percent are ages 55-74. Using these percentages, and the gender- and age-specific values in Taylor et al. (1996) the average value among women ages 45-74 of avoiding either type of stroke was estimated to be about $150,000.

Changes in Premature Mortality

Quantifying the relationship between blood pressure and premature mortality

The risk of premature mortality in women can be estimated by the following equation:

$$\Delta Pr(MORT_{women}) = \frac{1}{1 + e^{5.40374 - 0.01511 * DBP_1)}} - \frac{1}{1 + e^{5.40374 - 0.01511 * DBP_2)}} \quad (25)$$

where:

$\Delta Pr(MORT_{women})$ = the change in 2 year probability of death for women aged 45-74;

DBP_1 = mean diastolic blood pressure in the control scenario; and

DBP_2 = mean diastolic blood pressure in the no-control scenario.

Valuing reductions in premature mortality

The value of reducing premature mortality for women is assumed to be equal to that estimated for all premature mortality, $4.8 million per incident (see Appendix I).

Quantifying Uncertainty

Characterizing Uncertainty Surrounding the Dose-Response Relationships

The dose-response functions described for each health endpoint considered above generally quantify the adverse health effects expected due to increased exposure to lead. For children, these relationships are described directly in terms of changes in blood lead. For adults, effects are estimated in terms of changes in blood pressure (which are related to changes in blood lead levels). As with any estimate, uncertainty is associated with the dose-response relationships.

Consistent with the approach outlined in Appendix D for the non-lead criteria air pollutants, this analysis attempts to capture the uncertainty associated with these relationships. This is accomplished by estimating a distribution associated with each dose-response coefficient using the information reported in the literature. This analysis assumes these distributions to be normal. For each of the coefficients used to relate adverse health effects to lead exposure, Table G-2 summarizes the means and standard deviations of the normal distributions used in this analysis.

Characterizing Uncertainty Surrounding the Valuation Estimates

The procedure for quantifying uncertainty associated with the valuation estimates is similar to that used to characterize the dose-response coefficient estimates. The valuation distributions for health effects considered in the lead analysis are documented in Appendix I.

Table G-2. Uncertainty Analysis: Distributions Associated With Dose-Response Coefficients Used to Estimate Lead Health Effects.

Health Effect	Parameters of Normal distributions describing Dose-Response Coefficients	
	Mean	Standard Deviation
Blood Lead-Blood Pressure Coefficient (Adults)	1.44	0.85
Adult Males		
Mortality (ages 40-54)	0.03516	0.16596
Mortality (ages 55-64)	0.01866	0.00533
Mortality (ages 65-74)	0.00547	0.00667
Chronic Heart Disease (ages 40-59)	0.030365	0.003586
Chronic Heart Disease (ages 60-64)	0.02351	0.028
Chronic Heart Disease (ages 65-74)	0.02031	0.00901
Cerebrovascular Accidents	0.04066	0.00711
Atherothrombotic Brain Infarctions	0.0484	0.00938
Hypertension	0.793	not available
Adult Females		
Mortality (ages 45-74)	0.01511	0.00419
Chronic Heart Disease	0.03072	0.00385
Cerebrovascular Accidents	0.04287	0.00637
Atherothrombotic Brain Infarctions	0.0544	0.00754
Children		
Infant Mortality	0.0001	not available
Lost IQ Points	0.245	0.039
IQ<70 (cases)	relies on Lost IQ Point distribution	

Industrial Processes and Boilers and Electric Utilities

This section describes the methods and data sources used to estimate changes in blood lead levels due to changes in lead emissions from industrial processes and boilers between 1970 and 1990 and from electric utilities between 1975 and 1990. The estimates of the changes in health effects resulting from changes in lead emissions due to the CAA are also presented.

Methods Used to Determine Changes in Lead Emissions from Industrial Processes from 1970 to 1990

This analysis used several sources to determine the changes in facility-specific emissions of lead from industrial processes. To summarize, the analysis extracted 1990 facility-specific lead emissions data from the Toxics Release Inventory (TRI), which provides recent emissions data for over 20,000 U.S. manufacturing facilities. This study then adjusted these data by the relative changes in lead emissions between 1970 and 1990; these relative changes were derived from several data sources described below. This method yielded facility-specific emissions for five year intervals between 1970 and 1990 for both the controlled and uncontrolled scenarios. The five-year values were interpolated to derive annual changes for each year between 1970 and 1990. Specific details on this approach are given below.

TRI Data

The Toxics Release Inventory (TRI) is mandated by the Superfund Amendment Reauthorization Act (SARA) Title III Section 313 and requires that U.S. manufacturing facilities with more than 10 employees file annual reports documenting multimedia environmental releases and off-site transfers for over 300 chemicals. Facilities report both stack and fugitive releases to air. Reported releases are generally estimates rather than precise quantifications. Emissions data can be presented as numerical point estimates, or, if releases are below 1,000 pounds, as an estimated range of emissions.

From the TRI data base, this analysis extracted data from the reporting year 1990 for all facilities reporting emissions of lead to air, as either stack or fugitive emissions. Data were reported as annual emissions (in pounds per year). Where emissions are reported as a range, this analysis used the upper bound of the range to represent the emissions.[28] TRI facilities also report their location by latitude and longitude. In order to later match facilities emitting lead with Census data on surrounding exposed populations, this analysis uses the latitudes and longitudes of lead-emitting facilities.

Derivation of Industrial Process Emissions Differentials 1970-1990

The TRI database is the Agency's single best source of consistently reported release data; however, the database does not include information for most of the years modeled in this analysis. Furthermore, this analysis required estimates of hypothetical emissions in the absence of the CAA. Therefore, estimates were created for the emissions of lead from industrial sources under the CAA, and in the absence of the CAA, for the years 1970, 1975, 1980, 1985, and 1990. The *percent changes*, or differentials, reflected by these estimates were then applied to the 1990 TRI data to obtain facility-level release estimates for the years of interest for the control and no-control scenarios.

The method for creating these differentials captured the two potential causes of the differences between emissions from industrial sources regulated by the CAA and emissions from those same sources in the absence of the CAA. The first cause of the difference in emissions is a change in overall industrial output, resulting from the macroeconomic impact of the CAA. The second element is a change in emissions per unit of output, which results from the adoption of cleaner processes and the application of emissions control technology mandated by the CAA. The methods used to project the effects of these two causes, described below, were designed to be as consistent as possible with other emissions projection methods for other segments of the CAA retrospective analysis.

Data sources

Data for the differentials estimates were taken from the following sources:

- the Jorgenson/Wilcoxen (J/W) model projections, conducted as part of the section 812 analysis. This data source addresses the first cause of changes in emissions: the macroeconomic changes that resulted from the implementation of the 1970 CAA. The J/W model calculated the change in economic output for each of thirty-five industrial sectors, roughly analogous to two-digit standard industrial classification (SIC) codes, that resulted from the CAA's implementation. The specific output used from the J/W model in this analysis was the percentage change in economic output for the various industrial sectors, rather than any absolute measure of economic activity.

- the 1991 OAQPS Trends database. This data base is an emissions projection system that was used to produce the report, "The National Air Pollutant Emission Estimates, 1940-1990." It contains information on economic activity, national level emissions and emission controls, by industrial process, from 1970 through 1990. Three different elements were extracted from the Trends database: the emissions of lead per unit economic output for various industrial processes for the years 1970-1990; annual economic output data for these industrial processes; and the emission calculation formula.

- the National Energy Accounts (NEA), compiled by the Bureau of Economic Analysis. This database records the historical levels of industrial energy consumption, disaggregated by fuel type at the approximately three-digit SIC code level.

The manner in which these data were combined to derive lead emissions estimates is described below.

Estimates of industrial process emissions in the control scenario

Emissions data for industrial processes were estimated for the years 1970, 1975, 1980, 1985, and 1990. For each of these years, this analysis extracted an emission factor and a control efficiency for each lead-

[28] Ranges are infrequently reported and are either reported as 0-500 lbs. or 500-1000 lbs. The infrequency of the incidence of a facility reporting a range and the relatively small quantities of lead released by those facilities means any overestimation of benefits that results from using the upper limit of the range is extremely minor.

emitting industrial process in the Trends database. Emissions factors are expressed as amount of lead emitted per unit of economic activity, and control efficiencies are reported as the percent that emissions are reduced through the application of pollution control technology to the process. The year-specific emission factors and control efficiencies were multiplied by the economic activity data for that year, for that process, as reported in the Trends database, using the following equation found in the Trends report:

$$Emissions = (Economic\ Activity) \times (Emission\ Factor) \times (1 - Control\ Efficiency) \quad (26)$$

This calculation yielded the estimated control scenario emissions, by industrial process. Industrial processes were then assigned to an NEA code. Finally, all processes assigned to a given NEA code were summed to give a total emissions estimate for that NEA code.

Estimates of industrial process emissions in the no-control scenario

The results from the J/W model were used to estimate process emissions in the no-control scenario. As stated above, the J/W model provides percent changes in economic outputs by industrial sector. To use these values, lead-emitting industrial processes (in the Trends database) were assigned to a J/W sector. The percent change for that sector from the J/W model was then used to adjust the economic activity data for that process from the Trends database. These adjusted economic output figures were used together with 1970 emission factors and control efficiencies to derive the estimated lead emissions for each industrial process in the no-control scenario. The 1970 emission factors and control efficiencies were used for all years in the analysis (1970, 1975, 1980, 1985 and 1990) in the no-control scenario; this assumes that emissions per unit economic output and control efficiencies would have been constant over time in the absence of the CAA. This is the same approach that was used to project the changes in emissions from industrial processes for other criteria pollutants in other portions of the CAA retrospective analysis. The process-level emissions were then aggregated to the NEA-code level, as in the controlled scenario.

Matching TRI Data to Industrial Process Emissions Differentials

The methods described in the preceding section yielded emissions estimates from industrial processes in the control and no-control scenarios, by NEA code. We used these estimates to derive percent changes in emissions between control and no-control scenarios, by NEA code, for application to the TRI emissions data. However, since TRI data are reported by SIC code, we first mapped" NEA codes to the appropriate SIC codes, and used the percent change for each NEA code to represent the percent change for all SIC codes covered by that NEA code.

It should be noted that the Trends data base covers only the most important sources of lead in air, not all sources; as a result, not all SIC codes reporting lead emissions in TRI correspond to an NEA code for which emission differentials have been estimated. However, we assume that the TRI emissions sources that have a match are the most important sources of lead air emissions. In fact, although only 48 out of 519 legitimate SIC codes reporting lead emissions in TRI have matching differentials, these SIC codes account for over 69 percent of the lead emissions reported in TRI. The remaining 31 percent of the emissions are distributed relatively evenly among the remaining 471 SIC codes, each of which contributes a small amount to total emissions.

For the 31 percent of the emissions without differentials, this analysis has no information regarding the change in the lead emissions over time or between the control and no-control scenarios; therefore, we are unable to predict benefits attributable to the CAA for these emission sources. Although excluding these sources may lead us to underestimate total benefits, we believe these sources are unlikely to contribute significantly to the difference between control and no-control scenarios. The Trends data focus on the point sources of lead emissions of greatest concern to the Project Team and of greatest regulatory activity. If a process within an SIC code does not appear in the Trends, it is unlikely to have had specific CAA controls instituted over the past 20 years. A lack of control efficiencies for smaller sources prevents them from being included.

It should also be noted that the total industrial process emissions of lead estimated in the 1990 Trends report actually exceeds the reported lead emissions in

TRI, despite the fact that TRI covers more SIC codes. This is probably attributable in part to the fact that TRI covers only a subset of the facilities contributing to economic output in an SIC code. TRI reporting rules only require facilities with greater than 10 employees and who use certain amounts of lead in their processes to submit information to TRI, while the Trends report attempted to estimate emissions from all sources contributing to the economic output for the industrial sector, regardless of size. However, the components of the Trends data base used in this analysis (i.e., emissions factors, economic output data) represent typical conditions at average facilities; they do not allow for the representation of the distribution of emissions across particular facilities. In contrast, a major strength of the TRI is its match of emissions data with geographical information. Because the distribution of emissions geographically determines the size of exposed populations, this analysis used the TRI data, rather than Trends data, to characterize lead release quantities, and used the Trends figures only to characterize relative emissions and changes over time, rather than to estimate total quantities.

Because the Trends data are intended only as an estimate of emissions using typical conditions at average facilities, and do not capture the differences in facility-level emissions, the data do not provide sufficient information to make specific quantitative adjustments to the TRI-based benefits estimates to account for the overall higher emissions estimates in Trends. However, since Trends does generally suggest that there are many more sources than are accounted for by TRI, it is possible that our benefits calculations may be underestimated.

Some additional assumptions were necessary when matching the TRI lead release data and the differentials from the Trends data. Ideally, we would like to know whether the facilities present at a given location, as reported in the 1990 TRI, were present and operating in earlier years; whether facilities operating in 1970 have ceased to operate; and whether new facilities would have been constructed in the no-control situation. Unfortunately, data do not exist in an accessible form at this level of detail for the years 1970 through 1990. Therefore, for the purposes of this exercise, we have assumed that the locations and numbers of the 1990 sources are the same as they were in 1970.

Methods Used to Determine Changes in Lead Emissions from Industrial Boilers from 1970 to 1990

Several sources were used to determine the change in lead emissions from industrial boilers. TRI locational data, Trends database national fuel consumption levels and emissions factors, and NEA and SIC codes were used to derive the emissions for the control and no-control scenarios.

TRI Data

The TRI does not appear generally to contain combustion emissions data. In general, the emissions data are from process sources. We reached this conclusion based on two pieces of information:

(1) TRI reporting requirements: TRI has three reporting requirements: (a) the facility must fall in SIC codes 20-39; (b) the facility must employ more than 10 persons; and (c) the facility must manufacture or process more than 25,000 pounds of a TRI chemical, or otherwise use more than 10,000 pounds Firms must submit reports only for the chemical that exceeds the thresholds given in item (c), but they must report all releases of that chemical, including releases from uses that would not qualify alone. If the TRI chemical is part of a blended substance and the quantity of the TRI chemical in the blend exceeds the threshold, it must be reported. For industrial boilers, if the amount of lead in the fuel were to exceed the 10,000 pounds threshold, then the firm would be required to report all emissions of lead from combustion of fuel. There is an exemption, however, for ingredients present in small proportions. If the amount of lead in the oil were less than 0.1 percent (1,000 ppm), then the firm would not be required to report the emissions.

The conclusion from the above information is that most firms burning used oil are probably not reporting lead combustion emissions to TRI because these releases fall outside the TRI reporting requirements. The concentration at which lead is typically found is used oil (100 ppm) (NRDC, 1991) is much less than the minimum concentration required for reporting (1,000 ppm).

(2) Use data from the TRI data base: The hypothesis that firms do not report lead combustion was confirmed by an analysis of the data submitted by the

firms reporting lead use to TRI. On the TRI submission forms, firms must indicate how the chemical is used. Our analysis of category codes submitted by firms reporting lead emissions showed the following four use category reports: as a formulation component; as a reactant; as an article component; and repackaging only. None of these category codes suggest that the source of the reported lead release is combustion. Therefore, we may conclude that all of the lead emissions reported in TRI are process emissions.

Based on these analyses, the Project Team could not use the TRI release data to evaluate releases of lead from industrial combustion. However, the TRI geographical information was used to locate industrial facilities by longitude and latitude in order to combine combustion data with population information. For combustion emissions, the calculations included all TRI reporting facilities, not just those who reported lead emissions. The assignment of combustion emissions to these facilities is described below.

Derivation of Industrial Combustion Emissions 1970-1990

As with industrial process emissions, estimates were created for the emissions of lead from industrial combustion under the CAA, and in the absence of the CAA, for the years 1970, 1975, 1980, 1985, and 1990. These emissions estimates were used, in combination with the TRI data base geographic information, to obtain facility-level release estimates for the years of interest for the control and no-control scenarios. The method for deriving these emissions estimates included both the macroeconomic impact of the CAA and the change in emissions per unit of output that resulted from specific pollution control mandates of the CAA. The same data sources were used to derive combustion differentials as were used to derive process differentials. The particular data elements and the methods by which these data were combined to derive lead emissions estimates from industrial combustion are described below.

Estimates of combustion emissions under the control scenario

The Trends database contains a national aggregate industrial fuel consumption estimate, by fuel type (coal, natural gas, oil). For each fuel type, the fuel consumption estimate was disaggregated by the share of that fuel used by each NEA industrial category, using the NEA data base. It should be noted that the NEA includes data only for the years 1970 through 1985. For 1990, the 1985 figures were used to disaggregate the national-level consumption figure into NEA industrial categories.

The Trends database also contains emissions factors for industrial fuel use, by fuel type, as well as control efficiencies. The lead emissions from industrial combustion for each NEA category was derived by multiplying the fuel-specific combustion estimate for each NEA category by the emission factor and control efficiency for that fuel type. The result was emissions of lead by NEA code and by fuel type. Emissions from all fuel types were then summed by NEA code. By using the NEA data to disaggregate the industrial fuel consumption figures, the analysis assumes that the industrial combustion emissions are the same among all industries covered by a given NEA code, an assumption which may bias the analysis.

Estimates of combustion emissions under the no-control scenario

As in the control scenario, the national aggregate industrial fuel consumption estimate, by fuel type (coal, natural gas, oil), was disaggregated by the share of that fuel used by each NEA industrial category. The fuel use was then adjusted by one of two factors: (1) seven of the NEA codes were specifically modeled by the Industrial Combustion Emissions (ICE) model — for these sectors, the ICE modeled percent changes were used instead of J/W percent changes; or (2) the remaining NEA codes were matched to J/W sectors — the J/W percent changes were then applied to those matched NEA codes. These fuel use estimates were then combined with the 1970 emission factors and control efficiencies for industrial combustion by fuel type from the Trends database to obtain combustion-related lead emissions from industrial boilers in the no-control scenario, by NEA code.

The process-specific data in the Trends database, and the energy use data in the NEA, are much more disaggregated than the J/W sectoral projections. For the purpose of the analysis, it was assumed that all of the specific industrial processes in the Trends database and industrial categories in the NEA data set assigned to a given J/W sector changed at the same rate as the entire J/W sector. For example, if the economic activity in the J/W Sector 20, "Primary Metals," changed by one percent between the control and no-control scenarios, then the analysis assumed that economic activity in each industrial process assigned to

the Primary Metals sector also increased by one percent. This approach assumes that the economic activities of specific industries within a sector are equally affected by the imposition of the CAA. This assumption is consistent with the projection of the change in emissions from industrial processes for the other criteria air pollutants, which were calculated using a similar process.

Matching TRI Data to Industrial Combustion Emissions Data

Because of the structure of the TRI reporting requirements, it does not appear that TRI generally contains releases from combustion sources. Although TRI may incidentally contain lead combustion emissions, TRI would contain data on such releases only if the reporting facility also used more than 10,000 pounds of lead per year for manufacturing or processing. As a result, the combustion releases, estimated using the methods described above, do not have corresponding data in the TRI data base. Therefore, we devised a different method for estimating benefits from changes in combustion releases.

The first step in the method was to divide the estimates of total releases of lead from industrial combustion, by NEA code, by an estimate of the number of facilities in each NEA code. The number of facilities in each NEA category was estimated using the 1987 Census of Manufactures. This Census, conducted by the U.S. Department of Commerce, tallies the number of facilities by four-digit SIC code; these SIC codes were matched to the NEA codes.

Dividing total lead emissions emitted by number of facilities yielded the average yearly lead emissions from industrial combustion for each SIC code. We then assigned this average value to *all reporting TRI facilities* in the SIC code. The consequence of this approach is that the modeling of combustion from industrial facilities includes substantially more sources than the modeling of industrial process emissions; combustion emissions are assigned to essentially all facilities reporting to TRI, while the process emissions are only evaluated for facilities actually reporting lead air emissions from processes.

One unavoidable drawback to this approach is that it cannot capture differences in release quantities among facilities within an SIC code. Furthermore, this approach does not capture all combustion emissions because we assign average emissions only to facilities that report to TRI. TRI facilities account for between two percent and 50 percent of all facilities listed in the Census of Manufacturers, depending on the SIC code. Because of the inability to place the remaining facilities geographically, this analysis excludes the consideration of emissions from non-TRI facilities.

Methods Used to Determine Changes in Lead Emissions from Electric Utilities from 1975 to 1990

The estimation of lead emissions from electric utilities required data from three different sources. Energy use data for the control and no-control scenarios were obtained from the national coal use estimates prepared for the section 812 analysis by ICF Incorporated. The OAQPS Trends Database provided emissions factors and control efficiencies. Individual plant latitudes, longitudes, and stack information were collected from the EPA Interim Emissions Inventory. This analysis combines these three sets of data and estimates annual lead emissions at the plant level for coal burning electric utilities in the control and no-control scenarios. This section describes the sources and the methods used to create the final data set.

Coal-Use Data

The energy use data obtained from national coal-use estimates provide plant level energy consumption information for 822 electric utilities. The data set were separated into four distinct sets for the years 1975, 1980, 1985, and 1990. Each set of data provided the state where the plants are located, the plant names, and the amount of coal consumed, for both the control and no-control scenarios. The four data sets were combined into one comprehensive set by matching the plants' names and states.

The EPA Interim Emissions Inventory

The EPA Office of Air Quality Planning and Standards Technical Support Division provided the 1991 EPA Interim Emissions Inventory. The Interim Inventory contains data for all electric utility and industrial plants in the United States including latitude, longitude, stack height, stack diameter, stack velocity, and stack temperature. The additional stack parameter data allowed the use of plant-specific parameters in the air modeling for electric utilities rather than average parameters for all facilities as was done for industrial emissions.

Matching the Coal-Use Data to the Interim Emissions Inventory

The combination of the Interim Emissions Inventory and the coal-use data required two steps. First, the Interim Emissions Inventory had to be pared down to include only electric utility data, and to narrow the information provided for each utility. Second, the two databases had to be combined. One difficulty in combining them was the lack of a common data field that would allow a quick and complete matching process.

Electric utility plants were identified in the Interim Emissions Inventory by SIC code (code 4911). The associated stack information file, which lists the size of every stack on every plant, was reduced to include only the tallest stack for each plant. This provides a reasonable estimate of the stack height at which most emissions occur. The air modeling assumes that each electric utility releases its emissions from the largest stack that exists at that plant.

Next, the procedure matched the abridged Interim Emissions Inventory file with the coal use data. Due to the lack of a common data field between the two sets, this process required several phases. Both data sets had name fields, but these fields utilized different naming conventions for the plants. Therefore the name fields were matched directly, with individual words in the names, and then with abridged words from the names. Abridged word matches were double checked by ensuring that the names were indeed similar and by verifying that the state fields matched. Finally some matches were made by hand.

Only 27 unmatched plants with positive coal use remained. There were 493 matched plants with positive coal usage and these were included in the final data set.[29] To eliminate under-counting of emissions, the emissions from the 27 unmatched plants were allocated to matched plants within the states where the unmatched plants were located. Allocations were weighted according to the emission level for each matched plant within that state in the year in which the allocation was being made.

Emissions Factors and Control Efficiencies

At this stage, the electric utilities data set contained coal consumption by plant by year in the control and no-control cases as well as air modeling parameters. Using emission factors for lead and control efficiencies for electric utilities, estimates of lead emissions per plant per year could now be calculated. As in the industrial source analysis, the emission factors and control efficiencies come from the 1991 OAQPS Trends database.

Control efficiencies are available for coal-fired electric utilities in each year between 1975 and 1990. As in the industrial source analysis, it is assumed that pollution control on coal-burning power plants without the CAA would be the same as the pollution control level in 1970. Therefore, the control efficiency from 1970 is used in the no-control analysis.

The emission factor obtained from the Trends database is expressed in terms of lead emitted per ton of coal burned (6,050 grams per 1,000 tons).[30] The combined data set, though, contains quantity of coal burned per plant per year in energy units (trillions of BTUs). To reconcile this difference, a conversion factor was obtained from a 1992 DOE report titled *Cost and Quality of Fuels for Electric Utility Plants 1991*. The conversion factor used (20.93 million BTUs per ton of coal) is the average BTU per pound of coal burned for all domestic electric utility plants in 1990. Data for a small subset of other years were also provided in the DOE report, but they did not differ significantly from the 1990 number. Therefore, the 1990 conversion factor (637.3 pounds of lead per trillion BTU) is assumed valid over the entire study period. The final equation for lead emissions looks quite similar to the equation used in the industrial source analysis.[31] The only change is that "Economic Activity" has been replaced by "Coal Consumed" for this particular analysis:

$$\textit{Emissions} = \textit{(Coal Consumed)} \times \textit{(Emission Factor)} \times (1 - \textit{Control Efficiency}) \quad (26)$$

This equation produces estimates of the emissions per plant per year in both the control and the no-control scenarios.

[29] Plants with zero coal usage were not immediately excluded from the analysis due to the possibility of analyzing lead emissions from oil combustion at these plants. However, OAQPS has suggested that oil combustion comprises under two percent of the total lead emitted from electric utilities. For this reason, the electric utility analysis focused entirely on coal.

[30] The actual figure cited is 12.1 metric pounds per 1,000 tons. A metric pound is one two-thousandth of a metric ton.

[31] U.S. EPA, 1991a

Use of Air Dispersion Modeling to Estimate Ambient Air Lead Levels

To link estimates of lead emissions to blood lead levels of populations living in the vicinity of a facility, the lead benefits model first uses air dispersion modeling to estimate air lead concentrations surrounding facilities that emit lead into the air. The air concentrations are then linked to blood lead levels.

This analysis uses the Industrial Source Complex Long Term (ISCLT) air dispersion model, a steady-state Gaussian plume model, to estimate long-term lead concentrations downwind of a source. The concentration is modeled as a function of site parameters (stack height, stack velocity).[32] The general form of the concentration equation from a point source at a distance *r* greater than one meter away is as follows:[33]

$$C_{air,r_{ijk}} = \frac{2K}{\sqrt{2\pi}\, r\Theta} \cdot \frac{Q\ f\ S\ V\ D}{u\sigma_z} \qquad (28)$$

where,

- C_{air} = concentration at distance *r* ($\mu g/m^3$),
- Q = pollutant emission rate (g/sec),
- f = frequency of occurrence of wind speed and direction,
- Θ = sector width (radians),
- S = smoothing function used to smooth discontinuities at sector boundaries,
- u = mean wind speed (m/sec),
- σ_z = standard deviation of vertical concentration distribution (m),
- V = vertical term (m),
- K = scaling coefficient for unit agreement.

For each facility modeled in the lead benefits model, a 21 by 21 kilometer grid around the source is specified. The model stores data in 1 km by 1 km cells and calculates the air lead concentrations for each of the 441 cells surrounding a given facility. Fugitive sources are modeled similarly, the only difference being a modified form of Equation 28.

For facility-specific weather data, the model used Stability Array (STAR) data. The STAR data contain information on typical wind speed and direction for thousands of weather stations in the U.S. For each facility, the model accesses the STAR data for the weather station nearest the source. Standard default parameters are used for the other parameters because facility-specific data are not available for them (except for utilities). Table G-3 lists default parameters for the ISCLT, and summarizes sources for other parameters.

Industrial process emissions were modeled as either point or fugitive sources, depending on how they were reported in TRI. All industrial combustion emissions were modeled as "fugitive" emissions. This is a more appropriate model scenario for boiler emissions than a 10 meter stack scenario. All electric utility sources were modeled as point sources.

The model tracks all lead emissions to a given grid cell. That is, if the plumes of two or more sources overlap in a given cell, the air concentration in the given cell is determined from the sum of all of the contributing sources.

Determination of Blood Lead Levels from Air Lead Concentrations

Once the air lead concentrations surrounding a given plant are estimated, the model estimates blood lead levels for children and adults living in those areas. This section describes the methods and data sources used to derive blood lead levels from estimated air lead concentrations.

Relationship Between Air Lead Concentrations and Blood Lead Levels

The rates at which lead is absorbed from air depend on the age of the exposed individual, distance from the facility, the initial concentration of blood lead, and other factors. In addition, rates determined from empirical data may differ depending on whether or not the analyses from which rates are derived have controlled for factors such as lead exposure through deposition on dust and soil (i.e., "indirect exposure"). Especially when children constitute the exposed group, the inclusion of indirect exposure results in higher air lead to blood lead slopes. In both cases, the slope re-

[32] Ideally, reported stack and fugitive air releases would be modeled using site-specific data (such as source area or stack height). However, since TRI does not contain such facility-specific information, default values are used to model TRI facilities.

[33] This equation is from U.S. EPA (1992). The equation is for a specific wind speed, direction, and category (*ijk*). Each facility has several combinations of these that must be added to arrive at a total concentration at that point. The equation for area sources is similar.

Table G-3. Air Modeling Parameters.

Parameter	Industrial Source Value	Electric Utility Value	Source/ Comment
Stack height	10 m	site-specific or 115.0 m*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Exit velocity	0.01 m/s	site-specific or 22.5 m/s*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Stack diameter	1 m	site-specific or 5.15 m*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Exit gas temperature	293° K	site-specific or 427.5*	Industrial – U.S. EPA (1992) Utilities -- U.S. EPA (1991b)
Area source size	10 m^2	10 m^2	U.S. EPA (1992)
Area source height	3 m	3 m	U.S. EPA (1992)
Lead emission rate	site-specific	site-specific	Industrial – TRIS (lbs/yr) Utilities -- SAI & OAQPS (lbs/yr)
Frequency of wind speed and direction	site-specific	site-specific	STAR data
Sector width	22.5°	22.5°	360° divided by 16 wind directions
Wind speed	site-specific	site-specific	STAR data (m/sec)
Smoothing function	calculated	calculated	
Vertical term	calculated	calculated	

* average value for electric utilities, utilized for utilities without this information

lationship is expressed as the change in blood lead (µg/dL) per change in air concentration (µg/m^3).

In performing this analysis, a choice had to be made between the use of air lead:blood lead relationships that account for inhalation exposure ("direct" slopes) and those that account for exposure to lead deposited from air onto soil and dust ("indirect" slopes). The choice of which slopes to use considered both the effects on the estimate of benefits over time (from 1970 to 1990) and the estimate of the difference in benefits between the control and no-control scenarios. The indirect slope is more comprehensive in its coverage of the types of exposures that will result from air releases, and thus captures more of the health effects predicted to occur from lead exposures, especially to children. For this reason, indirect slopes are preferred to direct slopes, especially when comparing the control and no-control scenarios: using only the direct slope would underestimate the benefits of avoiding deposition that controls confer. However, indirect slopes may capture effects from exposure to soil and dust lead deposited from both current air releases and historic air releases. Since lead's dissipation from soil is slow relative to its removal from air, the reservoir of lead in soil and dust is unlikely to change at the same rate as the reductions in air lead concentrations. Therefore, using indirect slopes to represent a change in blood lead over time due to reduced air emissions may overestimate the change in blood lead, and thus overestimate the benefits of reductions over time, to the extent that the indirect slope captures exposure to the total reservoir of soil and dust lead, rather than only recently deposited lead.

Given that the focus of this analysis is the *difference* between the control and no-control scenarios, it is important to capture both the benefits from reduced lead deposition that result from the CAA, and the direct benefits from reduced air concentrations. Therefore, this analysis modeled changes in blood lead levels using indirect slopes. It should be kept in mind

that this choice may overestimate blood lead changes over time for both the control and no-control scenarios.

The relationship between concentrations of lead in ambient air and blood lead concentrations has been evaluated by a variety of methods. These include experimental studies of adult volunteers, as well as epidemiological studies of different populations of children and adults. The discussion below describes the slopes used in this analysis for children and adults, and for individuals with blood lead values greater than 30 µg/dL.

Children

U.S. EPA (1986b) reports that slopes which include both direct (inhalation) and indirect (via soil, dust, etc.) air lead contributions vary widely, but typically range from three to five µg/dL increment in children's blood lead per µg/m^3 increment in air lead concentration (roughly double the slope due to inhaled air lead alone). Since hand dust levels can play a significant role in blood lead levels (U.S. EPA, 1986b), this higher slope may be due to mouthing behavior of children that brings them into contact with dust and soil.

Specific values for estimating contribution of air lead to blood lead, including indirect pathways, are cited in U.S. EPA (1986b); slope values (ranging from -2.63 to 31.2) and data sources for these values are presented in Table 11-36 of U.S. EPA (1986b). The median of these values is 4.0 µg/dL per µg/m^3, which matches the midpoint of the range of typical slope values. This analysis used this value to represent the relationship between air lead concentrations and blood lead concentrations for children living in the vicinity of point sources of lead emissions.

The use of this slope assumes that indirect exposure" principally measures indirect effects of lead emissions to air (through deposition to dust and soil). However, it is possible that these slopes include other exposures not related to air lead. In many cases researchers have measured other possible exposures, such as water and food, and have confirmed that the most significant contribution comes from soil and dust lead, which is assumed to result from air deposition of lead. Those studies that measured lead in tap water showed that mean levels were generally low or not significantly related to blood lead. Landrigan et al. (1975) measured lead in pottery and food; lead in pottery was found in only 2.8 percent of homes, and food and water made no more than a negligible contribution to lead uptake. Lead in paint was measured in some studies.[34] Landrigan and Baker (1981) measured lead in paint at levels greater than one percent in about one fourth to one third of the houses in each area studied. Brunekreef et al. (1981) measured high levels of paint in some houses, but excluded these data points from the analysis.

Despite the possibility of confounding factors, this analysis uses the median value determined above (4.0 µg/dL per µg/m^3) as the appropriate slope for children living within five kilometers of the point source. Five kilometers is chosen as the cut off point because the data from most of the studies cited collected the majority of their data points near lead smelters.[35] Furthermore, these slopes, although measured primarily in the vicinity of smelters, are assumed applicable to all point sources that emit lead into the air.

Adults

For adult males and females, the air lead/ blood lead slopes that include indirect effects due to soil and dust differ very little from slopes that include only direct effects. This result is expected since the higher indirect slope values estimated for children are assumed to be as a result of mouthing behavior typical of young children.

U.S. EPA (1986b) describes several population studies that estimate indirect slopes for men; these slopes range from -0.1 to 3.1 µg/dL per µg/m^3.[36] Snee (1981) determined a weighted average of these studies and one other study.[37] The average slope, weighted by the inverse of each study's variance, is 1.0 µg/dL per µg/m^3. However, the Azar study measured the direct relationship between air lead and blood lead. Excluding the Azar study from the weighted average, the average slope is 1.1 µg/m^3. Excluding the highest and lowest slopes from this group (from Goldsmith,

[34] Landrigan and Baker, 1981; Brunekreef et al., 1981.

[35] U.S. EPA, 1986b, Table 11-36.

[36] Johnson et al., 1976; Nordman, 1975; Goldsmith, 1974; Tsuchiya et al., 1975; Fugas et al., 1973.

[37] Azar et al., 1975.

1974 and Tsuchiya et al., 1975), both of which had difficulties,[38] the resulting slope is 1.4 µg/dL per µg/m³.

Slopes for females range from 0.6 to 2.4 for general atmospheric conditions.[39] Snee determined an average slope for women of 0.9 µg/dL per µg/m³, weighted by the inverse of the variances of the studies. Excluding the slope for women from Goldsmith (1974), the resulting slope for women is 1.0 µg/dL per µg/m³.

These values are adjusted by a factor of 1.3 to account for the resorption of lead from bone tissue (according to Chamberlain, 1983), thus deriving an adjusted slope estimate of 1.8 µg/dL blood lead per µg/m³ increment in air lead concentration for men and 1.3 for women. These are the slope estimates used in this analysis.

Individuals with initial blood lead levels of 30 µg/dL and greater

For individuals with high blood lead levels, the air lead to blood lead uptake slopes have been shown to be much shallower, as described by U.S. EPA (1986b). An appropriate change in blood lead per change in air lead is 0.5 µg/dL per µg/m³ for individuals that have initial blood lead levels in the range of 30 to 40 µg/dL. This value is based on cross-sectional and experimental studies.[40] For individuals with initial blood lead levels greater than 40 µg/dL, an appropriate range of slopes is 0.03 to 0.2, as determined by occupational studies listed in Table 11-37 of U.S. EPA (1986b). The median value of these studies is 0.07. These two slopes (0.5 for the population with blood lead levels between 30 and 40 µg/dL and 0.07 for blood lead levels greater than 40 µg/dL) are used for both children and adults in this analysis. These relationships are summarized in Table G-4.

Estimates of Initial Blood Lead Concentrations

The benefits model requires an initial distribution of blood lead levels in the exposed populations to model health benefits of reducing lead air emissions. The model estimates the new distribution of blood lead levels that would exist after a given change in air concentrations using the slopes described above. Finally, the model estimates the difference between the two distributions. This analysis begins with an initial 1970, no-control scenario blood lead distribution from which all subsequent changes are modeled. This approach requires an estimate of the blood lead distributions in the U.S. population in 1970. Unfortunately, there are no actual national blood lead distribution estimates for 1970. Although the first NHANES study covered 1970, blood lead data were not collected in this study.[41] Nonetheless, a 1970 distribution of blood lead was estimated using NHANES II data (from 1976-1980), combined with estimates of typical changes in blood lead levels from 1970-1976 observed in localized screening studies.

Table G-4. Estimated Indirect Intake Slopes: Increment of Blood Lead Concentration (in µg/dL) per Unit of Air Lead Concentration (µg/m³).

	Individuals with blood lead levels < 30 µg/dL	Individuals with blood lead levels 30-40 µg/dL	Individuals with blood lead levels > 40 µg/dL
Adult Males	1.8	0.5	0.07
Adult Females	1.3	0.5	0.07
Children	4.0	0.5	0.07

[38] Goldsmith (1974) refrigerated (rather than froze) the blood samples, and did not analyze the samples until 8 or 9 months after they were taken, and restricted the analysis to one determination for each blood sample. Tsuchiya et al. (1975) measured air lead concentrations after blood samples were taken; blood was drawn in August and September of 1971, whereas air samples were taken during the 13 month period from September 1971 to September 1972.

[39] Tepper and Levin, 1975; Johnson et al., 1976; Nordman, 1975; Goldsmith, 1974; Daines et al., 1972.

[40] U.S. EPA, 1986b.

[41] NCHS, 1993a.

A major drawback to this approach is the uncertainty in deriving the 1970 estimates. Another drawback to beginning with the 1970 level and modeling changes from that point is the analysis only represents changes in lead exposure from air; reductions from other sources of lead exposure are not accounted for. The purpose of this analysis is to identify changes attributable to the CAA mandates; changes from other sources of lead exposure should not be considered. However, due to nonlinear nature of the lead concentration-response functions (see above), the overall exposure context in which the air lead exposure reductions take place will influence the estimate of benefits from those reductions. Specifically, at higher blood lead levels, the slope of the concentration-response curve is shallower than at lower levels. As a result, a given change in the mean blood lead level may result in a smaller change in the health effect if the change occurs from a relatively high starting level. On the other hand, if one accounts for the fact that other sources of lead exposure are reduced at the same time that the given air reductions occur, then those air emissions reductions may result in greater changes in health risk.

This issue is of concern even though the analysis focuses on the difference between the control and no-control scenarios, since the health benefit implications of the emissions differentials between the scenarios will depend on the point on the blood lead distribution curve at which the differences are considered. That is, a difference between a mean blood lead of 25 µg/dL and one of 20 µg/dL may have different health implications than a difference between 15 µg/dL and 10 µg/dL, even though the absolute value of the difference is the same (5 µg/dL).

An alternative method is to "start" with a 1990 blood lead level and to "back-calculate" benefits by representing the differentials as increases over the 1990 levels, rather than decreases from 1970 levels. The advantage of this approach is that it accounts for reductions in lead exposure from other sources, as represented by current blood lead levels. Its disadvantage is that it holds other sources constant to (lower) 1990 levels, and thus the modeling may underestimate actual blood lead distributions in earlier years, and thereby overestimate benefits from controls during those years. This analysis presents the results of both approaches, indicated as "forward-looking" and "backward-looking".

Combination of Air Concentration Estimates with Population Data

The modeled air lead concentrations at various distances from the sources were combined with population data from the Census Bureau to arrive at an estimate of the number of cases of health effects for each of the years from 1970 to 1990 in both the control and no-control scenarios. The primary census information was accessed from the Graphical Exposure Modeling System Database (GEMS), an EPA mainframe database system. The following data were obtained from GEMS for the years 1970, 1980, and 1990: total population for each Block Group/Enumeration District (BG/ED); state and county FIPS codes associated with each BG/ED; latitude and longitude of each BG/ED; and population of males under 5 and females under 5 for each BG/ED. The intervening five year intervals (1975 and 1985) were estimated using the Intercensal County Estimates from the Census, which estimate annual populations on a county by county basis. The decennial Census data and the Intercensal County Estimates data sets were related by county FIPS codes; the population in each BG/ED was assumed to grow or shrink at the same rate as the county population as a whole.

Since the concentration-response data are particular to specific sex and adult age groups, additional population data were also required to determine the sizes of affected subpopulations. For 1990 age and sex, the U.S. Census, 1992 was used, with age groups tallied as necessary. For 1980 age and sex, the U.S. Census, 1982 was used, with age groups also tallied as necessary. The 1970 age and sex breakdowns were obtained through personal communication with the Census Bureau.[42] The age and sex percentages were interpolated for intervening years.

Pregnant women are often a subpopulation of interest for lead effects. Although pregnant women themselves may be harmed by exposure to lead, this analysis was concerned with pregnant women because of possible effects on their fetuses who will be born

[42] Personal communication, Karl Kuellmer, Abt Associates and the Bureau of Census, Population, Age and Sex telephone staff, March, 1994.

and evince effects as young children. To estimate the number of exposed fetuses who were born during the years of interest,[43] birth rates for 1970, 1980 and 1990 were obtained from the Census Bureau.[44] These birth rates were used to interpolate for years between 1970 and 1980, and for the years between 1980 and 1990.

Results

For both the control and no-control scenarios, Table G-5 shows estimated lead emissions from electric utilities, industrial processes, and industrial combustion. Tables G-6 and G-7 show the differences in health impacts between the two scenarios (for industrial processes, industrial combustion and electric utilities only) for the "forward-looking" and "backward-looking" analyses. The modeled population for each year is also presented.

Table G-5. Estimated Lead Emissions from Electric Utilities.Industrial Processes.and Industrial Combustion (in Tons).

	1970	1975	1980	1985	1990
Electric Utilities[a] Control Scenario		1,351	636	175	190
Electric Utilities[a] No-control Scenario		2,309	3,143	3,670	3,864
Industrial Processes Control Scenario	7,789	3,317	1,032	670	658
Industrial Processes No-control Scenario	7,789	7,124	6,550	5,696	5,305
Industrial Combustion Control Scenario	4,329	4,354	1,880	190	187
Industrial Combustion No-control Scenario	4,329	4,457	4,653	4,584	4,596

[a] Appropriate data on electric utilities do not exist for years prior to 1975.

[43] Note that we do not record the number of pregnancies, since the valuation only applies if the child is born and lives to exhibit the effect. Neither are we concerned with whether the births are single or multiple births, since each fetus is at risk, whether a pregnant women carries one or more fetuses.

[44] Personal communication, Karl Kuellmer, Abt Associates and the Bureau of Census, Population, Fertility/Births telephone staff.

Table G-6. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Industrial Processes, Boilers, and Electric Utilities (Holding Other Lead Sources at Constant 1970 Levels).

Health Effect	1975	1980	1985	1990
Mortality				
Men (40-54)	0.1	1.5	2.5	2.7
Men (55-64)	0.0	1.1	1.8	1.8
Men (65-74)	0.0	0.4	0.7	0.8
Women (45-74)	0	0.8	1.3	1.4
Infants	0	0.001	0.002	0.002
Total	0.1	3.9	6.3	6.7
Coronary Heart Disease				
Men (40-54)	0.1	1.8	3.0	3.3
Men (55-64)	0.0	0.7	1.2	1.2
Men (65-74)	0.0	1.0	1.6	1.7
Women (45-74)	0.1	1.3	2.1	2.1
Total	0.2	4.8	8.0	8.3
Strokes				
Cerebrovascular Accident (men 45-74)	0.1	1.1	1.8	1.8
Cerebrovascular Accident (women 45-74)	0	0.5	0.9	0.9
Brain Infarction (men 45-74)	0	0.7	1.1	1.1
Brain Infarction (women 45-74)	0	0.4	0.6	0.6
Total	0.1	2.7	4.4	4.4
Hypertension (men 20-74)	149	3,790	6,350	6,670
IQ Decrement				
Lost IQ Points	630	14,300	22,700	23,900
IQ<70 (cases)	3	60	120	125
Population Exposed (millions)	188	197	207	217

Table G-7. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Industrial Processes, Boilers, and Electric Utilities (Holding Other Lead Sources at Constant 1990 Levels).

Health Effect	1975	1980	1985	1990
Mortality				
Men (40-54)	0.3	6.9	11.5	12.5
Men (55-64)	0.2	5.1	8.3	8.2
Men (65-74)	0.1	2.0	3.5	3.9
Women (45-74)	0.2	3.9	6.4	6.4
Infants	0	0.001	0.002	0.002
Total	0.8	17.9	29.7	31.0
Coronary Heart Disease				
Men (40-54)	0.4	8.3	13.8	15.0
Men (55-64)	0.1	3.4	5.6	5.6
Men (65-74)	0.2	4.4	7.6	8.0
Women (45-74)	0.2	5.9	9.6	9.7
Total	0.9	22.1	36.6	38.3
Strokes				
Cerebrovascular Accident (men 45-74)	0.2	5.0	8.1	8.2
Cerebrovascular Accident (women 45-74)	0.1	2.6	4.1	4.2
Brain Infarction (men 45-74)	0.1	2.8	4.6	4.7
Brain Infarction (women 45-74)	0.1	1.6	2.7	2.7
Total	0.5	12.0	19.5	19.8
Hypertension (men 20-74)	422	10,800	18,100	19,000
IQ Decrement				
Lost IQ Points	630	14,300	22,700	23,900
IQ<70 (cases)	0	31	50	61
Population Exposed (millions)	188	197	207	217

Reduction in Health Effects Attributable to Gasoline Lead Reductions

Estimating Changes in Amount of Lead in Gasoline from 1970 to 1990

The relationship between the national mean blood lead level and lead in gasoline is calculated as a function of the amount of lead in gasoline consumed. Thus, to calculate the health benefits from gasoline lead reductions, necessary inputs are estimates of lead in gasoline consumed over the period 1970 to 1990 and the amount of lead in gasoline that would have been consumed in the absence of the Clean Air Act. These values are calculated using the quantity of both leaded and unleaded gasoline sold each year and the concentration of lead in leaded and unleaded gasoline for each year in the period of interest. For each year, the relationship is expressed as:

$$LEAD = \left(\frac{SOLD}{365\ days}\right) \times [FRAC_{Pb} \times PB_{leaded} + (1 - FRAC_{Pb}) \times PB_{unleaded}] \quad (29)$$

where:

- $LEAD$ = average lead per day in gasoline sold in a given year (metric tons/day),
- $SOLD$ = total quantity of gasoline sold (million gal/yr),
- $FRAC_{Pb}$ = fraction of total gasoline sales represented by leaded gasoline (dimensionless),
- Pb_{leaded} = lead content of leaded gasoline (g/gal), and
- $Pb_{unleaded}$ = lead content of unleaded gasoline (g/gal).

Gasoline Sales (SOLD): Data on annual gasoline sales were taken from a report by Argonne National Laboratories (1993) which presented gasoline sales for each state in five year intervals over the period 1970-1990. This analysis used linear interpolation to estimate the gasoline sales for years between the reported years. These data were summed to obtain national sales figures.

Fraction of Total Sales Comprised of Leaded Gasoline ($FRAC_{Pb}$): For the control scenario, this analysis used information reported by Kolb and Longo (1991) for the fraction of the gasoline sales represented by leaded gasoline for the years 1970 through 1988. For 1989 and 1990, data were taken from DOE (1990 and 1991, respectively). For the no-control scenario, all of the gasoline sold was assumed to be leaded for all years.

Lead Content of Gasoline (Pb_{leaded} and $Pb_{unleaded}$): Argonne National Laboratory in Argonne, Illinois was the source for the data on the lead content of leaded and unleaded gasoline for the period 1974-1990. Argonne compiled these data from historical sales data submitted to EPA, from Clean Air Act regulations on lead content, and from recent Motor Vehicle Manufacturers Association (MVMA) surveys. For 1970 through 1973, this analysis assumed the lead content of gasoline to be at the 1974 level. For the no-control scenario, this analysis used the 1974 lead content in leaded gasoline as the lead content in all gasoline for each year.

Estimating the Change in Blood Lead Levels from the Change in the Amount of Lead in Gasoline

Several studies have found positive correlations between gasoline lead content and blood lead levels.[45] Data from the National Health and Nutrition Examination Survey (NHANES II) have been used by other researchers who determined similar positive correlations between gasoline lead and blood lead levels.[46]

The current analysis used a direct relationship between consumption of lead in gasoline and blood lead levels to estimate changes in blood lead levels resulting from Clean Air Act regulation of the lead content of gasoline. This relationship was based on regression analyses of the reduction of leaded gasoline presented in the 1985 Regulatory Impact Analysis (RIA).[47] Several multiple regressions were performed in the RIA to relate gasoline usage with individuals' blood lead lev-

[45] U.S. EPA, 1985; Billick et al., 1979; Billick et al., 1982.

[46] Janney, 1982; Annest et al., 1983; Centers for Disease Control, 1993; National Center for Health Statistics, 1993b.

[47] U.S. EPA, 1985.

els, which were taken from NHANES II. These regressions of blood lead on gasoline usage controlled for such variables as age, sex, degree of urbanization, alcohol consumption, smoking, occupational exposure, dietary factors, region of the country, educational attainment, and income. The regressions suggested that a decrease of 100 metric tons per day (MTD) of lead used in gasoline is associated with a decrease in mean blood lead concentration of 2.14 µg/dL for whites and 2.04 µg/dL for blacks. In both of these regressions, gasoline use was found to be a highly significant predictor of blood lead ($p < 0.0001$).[48]

To determine a single gasoline usage-blood lead slope for the entire population of the U.S., this analysis used the average of the slopes for blacks and for whites, weighted by the percentage of blacks and whites in the U.S. during the time period of the analysis.[49] The resulting relationship is 2.13 µg/dL blood lead per 100 metric tons of lead in gasoline consumed per day. The same relationship was used to model changes in both children's and adults' blood lead levels. The U.S. EPA (1985) analyzed data from a study of black children in Chicago during the time period 1976 to 1980 and determined a slope of 2.08 µg/dL per 100 MTD. This slope for children is very similar to the one used in this analysis.

1970-Forward and 1990-Backward Approaches

As with the industrial processes and boilers analysis, this analysis used two different approaches to determine mean blood lead levels based on changes in lead concentrations in gasoline. In the 1970-forward approach, the calculations began with the estimated blood lead level for 1970. The change in blood lead level from one year to the next was based upon the change in the amount of lead in gasoline sold, as discussed above, for both the control and no-control scenarios. For example, to calculate the blood lead level for 1971, the calculated change in blood lead from 1970 to 1971 was added to the 1970 value. This process was repeated for each succeeding year up to 1990.

The 1990-backward approach began with a mean blood lead level in 1990 for the control scenario. For the no-control scenario, the starting blood lead was estimated from the 1990 level used in the control scenario, plus an additional blood lead increment resulting from the difference between the 1990 consumption of lead in gasoline under the two scenarios. Again, the difference in mean blood lead levels from one year to the next was based on the change in gasoline lead for the corresponding years. For example, the difference in blood lead levels between 1990 and 1989 was subtracted from the 1990 level to determine the 1989 level. The process was continued for each year back to 1970.

Relating Blood Lead Levels to Population Health Effects

The mean blood lead levels calculated using the methods described above were used in the dose-response functions for various health effects (e.g., hypertension, chronic heart disease, mortality). This information was then combined with data on the resident population of the 48 conterminous states in each year to determine the total incidence of these health effects attributable to lead in gasoline. A Department of Commerce Publication (1991) was used to obtain the total population in 1970, 1980, and 1983-1990, while a different publication was the source of the 1975 population values.[50] Linear interpolation was used to estimate the populations in years for which specific data were not available.

For certain health effects, it was necessary to know the size of various age groups within the population. Two different sources were used to estimate the proportions of the population in the age groups of interest. A U.S. Census summary (U.S. Dept. of Commerce, 1990) was used for information for 1990 for children and adults and for 1980 for adults, and Census Telephone Staff (U.S. Dept. of Commerce, 1994) provided information for 1980 for children and 1970 for children and adults. The populations for the intervening years were estimated by linear interpolation.

Changes in Leaded Gasoline Emissions and Resulting Decreased Blood Lead Levels and Health Effects

Table G-8 shows the estimated quantity of lead burned in gasoline in the five year intervals from 1970 to 1990. Tables G-9 and G-10 show the difference in

[48] U.S. EPA, 1985.

[49] U.S. Department of Commerce, 1992. Although the percentages of blacks and whites changed slightly over this time period (1970-1990), the change did not affect the value of the weighted slope.

[50] U.S. Dept. of Commerce, 1976.

health impacts between the two scenarios (for lead in gasoline only) for the "forward-looking" and "backward-looking" analyses. In general, health effect benefits resulting from gasoline lead reductions exceed those predicted from lead reductions at the point sources examined (i.e., industrial processes and boilers and electric utilities) by three orders of magnitude.

Table G-8. Lead Burned in Gasoline (in tons).

	1970	1975	1980	1985	1990
Control Scenario	176,100	179,200	86,400	22,000	2,300
No-control Scenario	176,100	202,600	206,900	214,400	222,900

Table G-9. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Lead in Gasoline only (Holding Other Lead Sources at Constant 1970 Levels).

Health Effect	1975	1980	1985	1990
Mortality				
Men (40-54)	309	1,820	3,340	4,150
Men (55-64)	220	1,340	2,380	2,700
Men (65-74)	81	520	999	1,260
Women (45-74)	155	939	1,710	2,060
Infants	456	2,340	3,930	4,940
Total	1,220	6,960	12,400	15,100
Coronary Heart Disease				
Men (40-54)	230	1,360	2,540	3,280
Men (55-64)	92	563	1,030	1,220
Men (65-74)	113	723	1,380	1,750
Women (45-74)	73	442	805	965
Total	508	3,090	5,760	7,210
Strokes				
Cerebrovascular Accident (men 45-74)	147	884	1,610	1,960
Cerebrovascular Accident (women 45-74)	73	442	805	965
Brain Infarction (men 45-74)	85	508	927	1,130
Brain Infarction (women 45-74)	47	286	521	624
Total	352	2,120	3,862	4,679
Hypertension (men 20-74)	677,000	4,200,000	7,840,000	9,740,000
IQ Decrement				
Lost IQ Points	1,030,000	5,020,000	8,580,000	10,400,000
IQ<70 (cases)	3,780	20,100	36,500	45,300
Population Exposed (millions)	214	225	237	247

Table G-10. Yearly Differences in Number of Health Effects Between the Control and No-control Scenarios: Lead in Gasoline only (Holding Other Lead Sources at Constant 1990 Levels).

Health Effect	1975	1980	1985	1990
Mortality				
Men (40-54)	476	3,040	6,140	7,950
Men (55-64)	342	2,250	4,430	5,240
Men (65-74)	128	886	1,880	2,480
Women (45-74)	242	1,590	3,210	4,030
Infants	456	2,340	3,930	4,940
Total	1,640	10,100	19,600	24,600
Coronary Heart Disease				
Men (40-54)	356	2,280	4,690	6,310
Men (55-64)	142	945	1,910	2,370
Men (65-74)	176	1,220	2,570	3,380
Women (45-74)	113	740	1,490	1,860
Total	787	5,180	10,700	13,900
Strokes				
Cerebrovascular Accident (men 45-74)	225	1,460	2,940	3,720
Cerebrovascular Accident (women 45-74)	113	740	1,490	1,860
Brain Infarction (men 45-74)	129	837	1,680	2,120
Brain Infarction (women 45-74)	73	477	955	1,190
Total	540	3,514	7,065	8,890
Hypertension (men 20-74)	984,000	6,350,000	12,300,000	15,600,000
IQ Decrement				
Lost IQ Points	1,030,000	5,030,000	8,580,000	10,400,000
IQ<70 (cases)	3,790	20,100	36,500	45,300
Population Exposed (millions)	214	225	237	247

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Appendix H: Air Toxics

Introduction

Air toxics are defined as air pollutants other than those six criteria pollutants for which EPA sets acceptable concentrations in ambient air. The SARA 313 Toxic Release Inventory (TRI), covering 328 of the approximately 3000 potentially hazardous compounds detected in air, estimated that approximately 1.2 million tons of air toxics were released to the atmosphere in 1987 from U.S. stationary sources alone. While the TRI estimate tends to understate emissions of toxics for a number of reasons, it does show that large quantities of toxics are emitted into the atmosphere annually.

Effects of air toxics emissions are divided into three categories for study and assessment: cancer; "noncancer" effects, e.g. a wide variety of serious health effects such as abnormal development, birth defects, neurological impairment, or reproductive impairment, etc.; and ecological effects. Each year, these air toxics emissions contribute to significant adverse effects on human health, human welfare, and ecosystems. In EPA's 1987 *Unfinished Business Report*[1] cancer and noncancer air toxics risk estimates were considered sufficiently high, relative to risks addressed by other EPA programs, that the air toxics program area was among the few rated "high risk".

Limited Scope of this Assessment

The effects of air toxics emissions are difficult to quantify. The adverse health effects of toxics are often irreversible, not mitigated or eliminated by reduction in ongoing exposure, and involve particularly painful and/or protracted disease. Therefore these effects are not readily studied and quantified in human clinical studies, in contrast to, for example, ambient ozone. In addition, epidemiological studies of effects in exposed populations are often confounded by simultaneous exposure of subjects to a variety of pollutants. Therefore, the effects of air toxics are often quantified by extrapolating data from animal studies to human exposure and expressed as risk per unit of exposure. Incidence of noncancer effects, for example, often are difficult to translate into monetized benefits.

Similarly, the quantification of ecological effects due to emissions of air toxics is hampered by lack of sufficient information regarding contribution of sources to exposure, associations between exposure to mixtures of toxics and various ecological endpoints, and economic valuation for ecological endpoints.

The air toxics portion of this study is, of necessity, separate and more qualitative in nature than the benefit analysis conducted for the criteria air pollutants. Limitations in the quantitative analyses of air toxics effects led the Project Team to decide to exclude the available quantitative results from the primary analysis of CAA costs and benefits. Table H-1 presents the range of potential human health and ecological effects that can occur due to air toxics exposure. As indicated, this appendix presents quantitative estimates of benefits of CAA air toxics control for the cancer mortality endpoint for only nonutility stationary source and mobile source categories. Noncancer effects and ecological effects are described qualitatively.

[1] U.S. EPA. Office of Policy Planning and Evaluation. *Unfinished Business: A Comparative Assessment of Environmental Problems*. February 1987.

Table H-1. Health and Welfare Effects of Hazardous Air Pollutants.

Effect Category	Quantified Effects	Unquantified Effects	Other Possible Effects
Human Health	Cancer Mortality - nonutility stationary source - mobile source	Cancer Mortality - utility source - area source Noncancer effects - neurological - respiratory - reproductive - hematopoetic - developmental - immunological - organ toxicity	
Human Welfare		Decreased income and recreation opportunities due to fish advisories Odors	Decreased income resulting from decreased physical performance
Ecological		Effects on wildlife Effects on plants Ecosystem effects Loss of biological diversity	Effects on global climate
Other Welfare		Visibility Materials Damage	

History of Air Toxics Standards under the Clean Air Act of 1970

The 1970 Clean Air Act required the EPA to list a chemical as a hazardous air pollutant if it met the legislative definition provided:

> *"The term 'hazardous air pollutant' means an air pollutant to which no ambient air quality standard is applicable and which in the judgment of the Administrator may cause, or contribute to, an increase in mortality or an increase in serious irreversible, or incapacitating reversible, illness."*[2]

Once a HAP was listed, the EPA Administrator was required to:

> *"establish any such standard at the level which in his judgment provides an ample margin of safety to protect the public health from such hazardous air pollutant."*[3]

In other words the EPA had to first determine that a chemical was a HAP, and then regulate the emissions of each HAP based solely on human health effects and with an ample margin of safety. This regulatory mandate proved extremely difficult for EPA to fulfill, for reasons discussed below, and the result was that only seven HAPs were regulated over a period of 20 years.

Listing chemicals became a difficult task because of debates within and outside of the EPA surrounding issues of how much data are needed and which meth-

[2] 42 U.S.C. §1857(a)(1).

[3] 42 U.S.C. §1857(b).

odologies should be used to list a chemical as a HAP. An even more difficult issue was how to define the Congressional mandate to provide an "ample margin of safety." For carcinogens, there is generally no threshold of exposure considered to be without risk. What level of risk, then, is acceptable, and how should it be calculated? The EPA struggled to provide answers to these questions, and was challenged in court. The end result was a 1987 ruling by the D.C. Circuit Court that provided the EPA with a legal framework with which to determine an "ample margin of safety." This framework was interpreted and used by the EPA in its 1989 benzene regulations.

Quantifiable Stationary Source Air Toxics Benefits

One might be tempted to presume that the few federal HAP standards set would have achieved relatively substantial reductions in quantifiable risk. While some standards set under section 112 of the Clean Air Act appear to have achieved significant reductions in cancer incidence, the coverage, quantification, and monetization of the full range of potential adverse effects remains severely limited. This fact serves to highlight the inadequacy of current methods of evaluating HAP control benefits. This limited ability to estimate the total human health and ecological benefits of HAP reductions is an important area for future research. Thus the quantifiable benefits for CAA air toxics control presented here are limited in scope.

There are three sources of information that provide a picture of potential stationary source air toxics benefits of the CAA. EPA's Cancer Risk studies attempted to broadly assess the magnitude and nature of the air toxics problem by developing quantitative estimates of cancer risks posed by selected air toxics and their sources. Secondly, risk assessments conducted in conjunction with the promulgation of National Emissions Standards for Hazardous Air Pollutants (NESHAPs) offer a snapshot of potential monetized cancer mortality benefits. Finally, the Project Team attempted to estimate historical non-utility stationary source HAP-related direct inhalation cancer incidence reductions. Results from each of these studies are presented below.

EPA Analyses of Cancer Risks from Selected Air Toxic Pollutants

The Agency conducted two efforts to broadly assess the magnitude and nature of the air toxics problem. The 1985 report entitled, "The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants"[4] otherwise known as the "Six Month Study," was intended to serve as a "scoping" study to provide a quick assessment of the air toxics problem utilizing only readily available data on compound potencies, emissions, and ambient pollutant concentrations. The Agency updated this analysis of cancer risks in the 1990 report entitled "Cancer Risk from Outdoor Exposure to Air Toxics" referred to here as the "1990 Cancer Risk study."[5]

For the pollutant and source categories examined, the 1990 Cancer Risk study estimated the total nationwide cancer incidence due to outdoor concentrations of air toxics to range from 1,700 to as many as 2,700 excess cancer cases per year, with 14 compounds accounting for approximately 95 percent of the annual cancer cases. Additionally, point sources contribute 25 percent of annual cases and area sources contribute 75 percent of annual cases. Mobile sources account for 56 percent of the nationwide total.[6]

The Six Month study indicates that the criteria air pollutant programs appear to have done more to reduce air toxics levels during the 1970 to 1990 period than have regulatory actions aimed at specific toxic compounds promulgated during the same period. Metals and polynuclear compounds usually are emitted as particulate matter and most of the volatile organic compounds are ozone precursors. As such, they are regulated under State Implementation Plan (SIP) and New Source Performance Standard (NSPS) programs and Title II motor vehicle regulations. A number of reports cited indicate significant reductions in air toxics emissions attributable to actions taken un-

[4] U.S. EPA. Office of Air Quality Planning and Standards. *The Air Toxics Problem in the United States: An Analysis of Cancer Risks for Selected Pollutants*. May 1985. EPA-450/1-85-001.

[5] U.S. EPA. Office of Air Quality Planning and Standards. *Cancer Risk from Outdoor Exposure to Air Toxics*. September 1990. EPA-450/1-90-004a.

[6] The 1990 Cancer Risk study reported approximately 500 - 900 more cancer cases per year than the Six Month Study due primarily to the inclusion of more pollutants, better accounting of emissions sources, and, in some cases, increases in unit risk estimates.

der SIP, NSPS and mobile source programs. Additionally, EPA conducted a comparison of air quality and emissions data for 1970 with the estimates of cancer incidence for 1980.[7] Methods, assumptions and pollutants included were held constant over the period. The analysis showed a significant decrease in incidence during the decade due to improvements in air quality, presumably related to general regulatory programs. For the 16 pollutants studied, estimated nationwide cancer incidence decreased from 3600 in 1970 to 1600 in 1980. The 1990 Cancer Risk Study did not attempt to update this analysis.

Although it is difficult to draw quantitative conclusions from these two studies regarding the benefits of CAA air toxics control, it is apparent that the pollutant-specific and source category-specific NESHAPs were not structured to reduce significant air toxic emissions from area and mobile sources. In fact, the 1990 Cancer Risk Study indicates that considerable cancer risk remained prior to passage of the 1990 CAA Amendments: as many as 2,700 excess cancer cases annually. Some studies indicate that the criteria air pollutant program played a critical role during the 1970 to 1990 period in achieving air toxic emission reductions and therefore decreasing cancer risk.

Cancer Risk Estimates from NESHAP Risk Assessments

In looking back at the estimated effects of the HAP standards, EPA found that the effects of the NESHAPs were not quantified completely. These estimates occurred at a time when emission estimation and risk assessment methodologies for HAPs were first being developed. One consequence is that because emissions were not fully characterized, air toxics exposures could not be completely assessed. Additionally, most assessments only focused on the specific HAP being listed under the CAA and did not assess the reduction of other pollutants, which are currently considered HAPs. For example, while the vinyl chloride standard reduces emissions of ethylene dichloride, these emission reductions were not assessed in the risk assessment. In a different context, reductions of HAP may also achieve reductions of VOC and PM. The benefits of such reductions generally were also not evaluated. In addition, EPA generally did not assess the potential exposure to high, short-term concentrations of HAP and therefore did not know whether toxic effects from acute exposures would have been predicted and possibly addressed by the HAP standards.

In addition, people living near emission sources of concern are often exposed to a mix of pollutants at once. Some pollutants have been shown to act synergistically together to create a health risk greater than the risk that would be expected by simply adding the two exposure levels together. More research is needed to understand the effects of multiple-pollutant exposures. Finally, HAP risks tend to be distributed unevenly across exposed populations, with particularly high exposures occurring closest to emission sources. It should be noted that HAP exposure to specific populations may tend to fall disproportionately among the poor and minorities, who are more likely to live in close proximity to emitting facilities.

With the above caveats in mind, Table H-2 provides information about maximum individual risk taken from the Federal Register notices for the NESHAPs promulgated before the 1990 amendments to the Clean Air Act. The benefits are calculated by multiplying the estimated annual incidence reduction by the $4.8 million valuation per statistical life (1990 dollars). These benefit estimates provide a snapshot of potential monetized benefits for the year in which each NESHAP was promulgated. Of course these estimates do not include air toxics benefits for other health and ecological benefit categories, or air toxics benefits from co-control of criteria air pollutants. All uncertainties associated with the original estimates remain.

Non-utility Stationary Source Cancer Incidence Reductions

The Project Team commissioned two studies to estimate reductions in cancer incidence due to pre-1990 NESHAPs: the PES Study and the ICF Re-analysis. The methodology used for most air pollutant evaluations involved a “back calculation” for the estimation of incidence reductions. However, the EPA has elected not to rely on the results of this analysis given critical methodological flaws. Despite the Project Team’s concerns, the methodology and results of the two studies are presented below in the interest of full disclosure and to guide efforts to develop a more valid

[7] Hunt, W.F., Faoro, R.B. and Curran, T.C., “Estimation of Cancer Incidence Cases and Rates for Selected Toxic Air Pollutants Using Ambient Air Pollution Data, 1970 vs. 1980”. U.S. EPA. April 1985.

Table H-2. Cancer Incidence Reductions and Monetized Benefits for NESHAPs.

Pollutant	Source Category	Year Promulgated	Pre-Reg Maximum Individual Risk	Post-Reg Maximum Individual Risk	Reduction in Cancer Incidence (per year)	Benefits in $million per year (1990$)
benzene		1985	$1.5x10^{-3}$	$4.5x10^{-4}$	.31	1.5
benzene	coke by-product	1984	$7x10^{-3}$	$2x10^{-4}$	1.95	9.4
benzene	storage vessels	1982	$4.5x10^{-4}$	$3x10^{-5}$	0.01 to 0.06	0.05 to 0.3
benzene	waste operations	1986	$2x10^{-3}$	$5x10^{-5}$	0.55	2.6
benzene	transfer operations	1987	$6x10^{-3}$	$4x10^{-5}$	0.98	4.7
arsenic	primary copper	1986	$1.3x10^{-3}$ to $5x10^{-6}$	$1.2x10^{-3}$ to $3x10^{-6}$	0.09	0.4
arsenic	glass manuf.	1986	$7x10^{-4}$ to $3x10^{-5}$	$1.7x10^{-4}$ to $6x10^{-6}$	0.117 to 0.0034	0.02 to 0.6
asbestos	demolition	1973			100	480
vinyl chloride	PVC production	1975			10.5	50.4

and reliable analysis of the health-related benefits of HAP reductions in the upcoming section 812 Prospective studies.

PES Study

Methodology

The first attempt to estimate, for this study, historical non-utility stationary source HAP-related direct inhalation cancer incidence reductions was conducted by Pacific Environmental Services (PES). The basic approach used in the PES study was to adjust the cancer incidence estimates developed for EPA's 1990 Cancer Risk study to reflect the changes in emissions of, and exposures to, 14 key HAPs: arsenic, asbestos, benzene, 1,3-butadiene, carbon tetrachloride, chloroform, hexavalent chromium, dioxin, ethylene dichloride, ethylene dibromide, formaldehyde, gasoline vapors, products of incomplete combustion (PICs), and vinyl chloride.

The first step was to compile baseline incidence levels, defined as cancer cases per million population, for each of the 14 pollutants. The point estimates of incidence from the 1990 Cancer Risk study were used for this purpose. For some source categories, the "best point estimate" from the 1990 Cancer Risk study was used, for others a mid-point was selected.[8] These baseline incidence levels were based on measured ambient concentrations of the pollutant, modeled concentrations, or both.

The second step involved allocating baseline incidence levels to the individual source categories known to emit the relevant pollutant. In some cases,

[8] For some of the source categories, the original NESHAP/Air Toxic Exposure and Risk Information System (NESHAP/ATERIS) estimates of incidence were not available, in which case the baseline incidence was obtained from the 1989 National Air Toxics Information Clearinghouse(NATICH) Database Report. (See PES, "Draft Summary of Methodology Used for Cancer from Stationary Sources," memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA, March 22, 1993, p. 2.)

adjustments were made to reflect differences among the vintages of source category-specific data.[9] All baseline incidence estimates were ultimately expressed relative to a 1985 base year.[10] The assumption was then made that source-category incidence rates were proportional to the level of emissions from that source category.

Next, levels of control for each source category-specific incidence rate were estimated for each of the target years of the present analysis (i.e., 1970, 1975, 1980, 1985, and 1990).[11] Source category-specific activity level indicators were then established and linked to changes in corresponding activity indicators provided by the J/W macroeconomic modeling results. Activity levels were estimated for each source category, for each of the target years, and for each of the two scenarios.

Finally, source category/pollutant combination incidence levels for both the control and no-control scenarios were developed. These incidence levels were developed based on the baseline incidence levels, the activity indicators, and the control levels for each year. Both of these latter two factors varied between the control and no-control scenarios. The activity levels differed based on the specific levels of related sector economic activity predicted by the J/W model for the control and no-control scenario. The control levels prevailing in each of the target years were used for the control scenario, and the 1970 control level was applied throughout the 1970 to 1990 period for the no-control scenario.[12] The formula used for these calculations was as follows:[13]

$$I_{ty} = I_{by} \times [\frac{A_{ty}}{A_{by}}] \times [\frac{P_{ty}}{P_{by}}] \times [\frac{(1 - C_{ty})}{(1 - C_{by})}] \qquad (1)$$

where:

- I = cancer incidence for a source category-pollutant combination
- A = activity level for a source category
- P = population
- C = control level for a source category-pollutant combination
- ty = target year (1970 ... 1990)
- by = base year

Findings

The PES analysis concluded that substantial reductions in HAP-related cancer cases were achieved during the reference period of the present study. The vast majority of these estimated reductions were attributable to reduced exposures to asbestos, particularly from manufacturing and fabricating sources.[14] In fact, roughly 75 percent of the total reduction in cancer cases averaged over the 1970 to 1990 period were attributed to asbestos control.[15] Figure H-1 summarizes the PES study overall cancer incidence reductions and the relative contribution of asbestos-related reductions over the study period.

The Project Team had several concerns about the PES results. First and foremost, the reductions in asbestos-related cancer cases appeared to be substantially higher than expected, particularly in the earlier target years. Second, the control scenario activity level indicators for several sources with which Project Team members were familiar did not appear to be even remotely consistent with actual historical activity patterns.[16] Finally, the level of documentation of the analytical methodologies, assumptions, and results was insufficient to ascertain the validity and reliability of

[9] For example, six discrete sources for vinyl chloride were identified in the Six-Month Study Update. Point estimate incidences for each of these source categories came from separate references with databases corresponding to different years. (See PES, "retrospective analysis for section 812(a) Benefits Study," September 30, 1992, p. 8.)

[10] See PES, March 22, 1993 memorandum, p. 3.

[11] Control level estimates were based on one of the following: control efficiencies for related criteria pollutants defined in the criteria pollutant analysis, reference documents such as Control Technology Guidelines (CTGs) or Background Information Documents (BIDs), preambles for related regulations, or EPA experts. (See PES, March 22, 1993 memorandum, p. 3.)

[12] More detailed descriptions of the methodology and associated uncertainties are provided in "Retrospective Analysis for section 812(a) Benefits Study," a September 30, 1992 memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA.

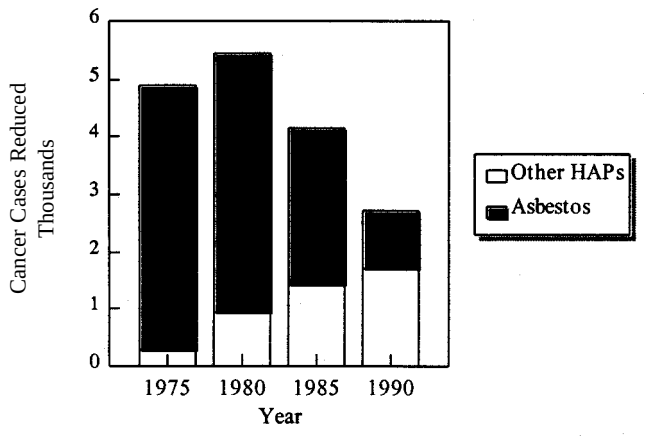
[13] See PES, March 22, 1993 memorandum, p. 4.

[14] PES, "Cancer Risk Estimates from Stationary Sources," memorandum from Ken Meardon, PES to Vasu Kilaru, US EPA, March 5, 1993.

[15] ICF, "Direct Inhalation Incidence Benefits," Draft Report, November 11, 1994, p. 10.

[16] For example, the activity indicators for Municipal Waste Combustors (MWCs) incorporated in the PES analysis decline dramatically throughout the 1975 to 1990 period. (See PES, March 5, 1993 memorandum to Vasu Kilaru, p. 10). In reality, overall MWC capacity and throughput increased significantly over this period.

Figure H-1. PES Estimated Reductions in HAP-Related Cancer Cases.



the results. Ultimately, the Project Team determined that it was necessary to conduct a formal review and re-analysis of the cancer incidence reductions associated with non-utility stationary source HAP controls. The results of the PES analysis remain a relevant part of the record of the present study, however, since they provided a substantial basis for the subsequent re-analysis by ICF Incorporated.

ICF Re-analysis

Methodology

The purposes of the ICF Re-analysis were to examine the methodology and results of the PES study, particularly to address the aforementioned concerns of the Project Team, and to develop a revised set of estimates. Due to significant constraints on the resources remaining for HAP analysis in the section 812 study, however, only a few key HAPs could be investigated in depth and many important issues could not be addressed.[17] Furthermore, the effects of two early and potentially important HAP standards –the Beryllium and Mercury NESHAPs– could not be evaluated. Nevertheless, the ICF Re-analysis clarified some potential sources of uncertainty in the PES results and provided revised cancer incidence reduction estimates for several HAPs.

A key uncertainty in the PES results was associated with the use of a "back-calculation" technique to estimate incidence reductions for some HAPs. The back-calculation technique estimates uncontrolled incidence by dividing residual incidence by the assumed control efficiency. This approach means uncontrolled incidence, and therefore incidence reductions, are highly sensitive to small changes in assumed control efficiency.[18] In some cases, the PES analysis may have used control efficiencies which were too high, resulting in overestimation of uncontrolled incidence and therefore incidence reductions attributable to the CAA.[19] The vinyl chloride incidence reduction estimates appear to be significantly influenced by the use of this back-calculation technique. Another important source of uncertainty identified by ICF involved the potential overestimation of incidence totals when source apportionment is based on measured ambient concentrations.[20] ICF was unable, however, to perform an extensive evaluation of the activity level indicators used in the PES study.[21]

The first step undertaken in the re-analysis was to conduct a screening test to identify the HAPs which accounted for the most significant estimated incidence reductions. Based on this screening analysis, ICF eliminated 1,3-butadiene, carbon tetrachloride, chloroform, gasoline vapors, chromium, formaldehyde, and PICs from the detailed re-analysis effort.

Detailed reviews were then conducted for the remaining HAPs: vinyl chloride, dioxins, ethylene dibromide (EDB), ethylene dichloride (EDC), benzene, asbestos, and arsenic. In the re-analysis of these HAPs, ICF determined whether a forward- or back-calculation technique was used for the relevant source categories of a given HAP, reviewed the regulatory

[17] For example, the Project Team sought to develop and apply a methodology for estimating a central tendency estimate for the total carcinogenic risk imposed by all the HAPs examined. The intent was to address concerns about potential overestimation of aggregate risk measures when combining upper bound risk estimates of multiple HAPs. Unfortunately, resources were insufficient to continue development of this methodology.

[18] An example of this back-calculation technique illustrating the sensitivity to the assumed control efficiency is presented on page 12 of the draft ICF report.

[19] See ICF Draft Report, p. 12.

[20] See ICF Draft Report, p. 9.

[21] See ICF Draft Report, p. 13.

history of the relevant source categories to re-evaluate the assumed control efficiencies, and reviewed the upper-bound unit risk factor for each HAP. Revised total incidence reduction estimates for each HAP and for each target year were then calculated using the same basic calculation procedure used by PES. Finally, ICF identified a number of residual deficiencies in the analysis which could only be addressed through additional research and analysis.[22]

Findings

The ICF Re-analysis largely affirmed the original results obtained by PES; primarily because the PES analysis itself served as the basis for the re-analysis and only minor adjustments were adopted for many critical variables. In particular, most Project Team concerns regarding the PES methodology could not be resolved, including uncertainties associated with activity levels, assumed control efficiencies, and the unexpectedly high estimated incidence reductions associated with asbestos. In fact, the ICF Re-analysis produced a revised upper bound estimate for vinyl chloride-related incidence reductions which were even higher than the asbestos benefits.

Several sets of results were developed by ICF and presented in either the November 1994 draft report or in briefing materials prepared for the Science Advisory Board Clean Air Act Compliance Analysis Council Physical Effects Subcommittee (SAB/ACCACAPERS) in May 1995. The first set of results is based on the assumption of 100 percent source compliance with HAP control requirements. An alternative set of results was developed assuming an 80 percent compliance rate with applicable standards. Given the linear effect of changes in compliance rates, these results were precisely 20 percent lower than the first set of estimates. At the May 1995 ACCACAPERS briefing, estimates based on the 100 percent compliance estimates were presented. For asbestos, the revised incidence reductions were presented and characterized as upper bound. The asbestos estimates were then combined with upper and lower bound estimates for vinyl chloride and for "all other compounds." Figure H-2 presents the total cancer incidence reductions derived from the ICF Re-analysis, using the asbestos estimates combined with the lower bound estimates for non-asbestos HAPs.

Figure H-2. ICF Estimated Reductions in Total HAP-Related Cancer Cases Using Upper Bound Asbestos Incidence and Lower Bound Non-Asbestos HAP Incidence.

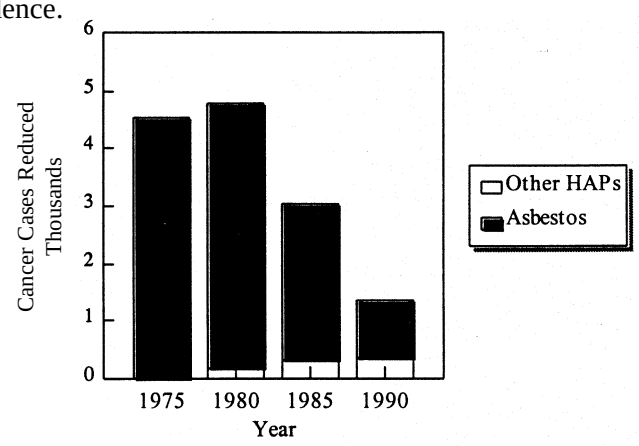


Figure H-3. ICF Estimated Reduction in Total HAP-Related Cancer Cases Using Upper Bound Incidence for All HAPs.

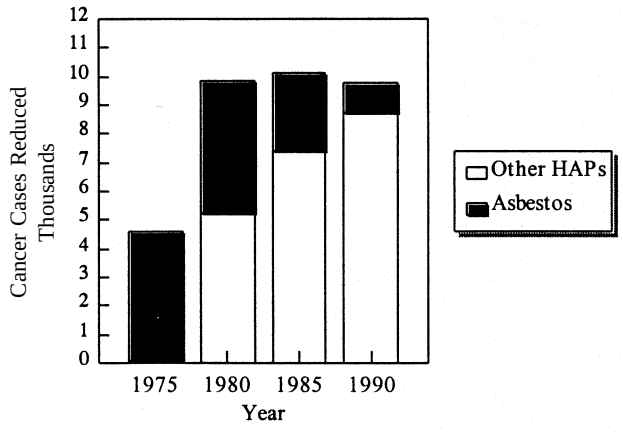


Figure H-3 presents a comparable compilation reflecting the upper bound estimates for all HAPs.

The Project Team remains concerned about these incidence reduction estimates, particularly given the doubts raised by the SAB/ACCACAPERS at the May 1995 presentation of these results. For instance, several critical assumptions are needed to make this analysis valid when applied to EPA's NESHAPs. The flaws in these assumptions are described below.

(1) The risk estimates described in the 1990 Cancer Risk study, which served as the baseline for determining risk reductions, were accepted without question. There are myriad uncertainties in these estimates

[22] Additional details of the ICF Re-analysis methodology can be found in ICF, "Direct Inhalation Incidence Benefits," Draft Report, November 11, 1994.

that must be recognized, as the study was designed only to generate rough order-of-magnitude estimates of the extent of the air toxics cancer problem.

(2) The percent control efficiency for emission reductions, which is calculated in each standard, would have to apply across every facility. Typically, the emissions reductions vary between facilities; using a single average reduction could skew the results.

(3) There is a direct correlation between the number of tons of emissions reduced and incidence reduced by a specific regulation. Given the assumption of a linear, non-threshold dose-response curve (as is typically done for cancer), this is theoretically correct.

(4) Finally, the back calculation approach assumes that there is 100 percent compliance with the regulation.

EPA staff reviewed the "back calculation" approach for one of the more controversial aspects of the vinyl chloride (VC) NESHAP. The PES study estimates benefits at 426 cases reduced in 1990. The ICF Re-analysis resulted in an even higher estimate, between 1,000 and 7,000 cases annually. An analysis by EPA staff indicated that these vinyl chloride risk estimates are highly suspect given historical cancer incidence data for hepatic angiocarcinoma, a specific cancer that has been linked to vinyl chloride (Koppikar and Fegley, 1995). The following analysis demonstrates the inadequacies of the assumptions in the 1993 study.

(1) In the actual standard, no control technology was required for emissions from oxychlorination vents at ethylene dichloride (EDC)/VC plants. Applying "back calculation" for these emissions is inappropriate.

(2) In 1985, there were an estimated 8,000 fabrication plants which processed resins produced by PVC plants, thus resulting in VC emissions, which were exempt from the VC NESHAP. They emit very small quantities of VC and back calculation is not appropriate.

(3) The 1993 study uses a baseline estimate of 18 residual cases from the NESHAP/ATERIS data base. There is no evidence that these cases resulted only from emissions from PVC and EDC/VC plants.

(4) The risk analysis performed for the October 21, 1976 final VC regulation projected an incidence reduction of 11 cases per year.

In contrast, the PES study, using the "back calculation" method derived the following annual incidence reductions:

1980 - 250 cases
1985 - 360 cases
1990 - 430 cases

The subsequent back calculation conducted in the ICF Re-analysis resulted in incidence reductions as much as an order of magnitude higher than these.

Even considering the slightly different industrial output assumptions imposed by macroeconomic modeling, such a stark contrast is difficult to explain except for a critically flawed approach. Growth in activity and population nor other factors explain the difference in these two estimates. Given that the same general methodology was used for all of the air toxic pollutant assessments as was used for the VC NESHAP evaluation, there is reason to believe that cancer incidence results for the other air toxic pollutants are also flawed.

Mobile Source HAP Exposure Reductions

EPA's Cancer Risk report estimated that approximately 60 percent of the total carcinogenic risk posed by HAPs was attributed to mobile sources, with stationary sources contributing 15 percent and area sources contributing the remaining 25 percent.[23] The relative importance of mobile sources to total HAP exposure was a significant motivation behind EPA's subsequent effort to examine exposures and risks from mobile source HAPs.[24] Although available analytical resources were severely limited, the Project Team nevertheless decided it was necessary to perform at least an initial screening analysis to estimate the differences in mobile source HAP exposures between the control and no-control scenarios configured for the present study.

[23] Cancer Risk report, Page ES-12.

[24] See US EPA/OAR/OMS, "Motor Vehicle-Related Air Toxics Study," EPA 420-R-93-005. April 1993.

Methodology

The approach used by ICF/SAI in conducting the mobile source HAP analysis closely followed the approach used in the EPA Motor Vehicle-Related Air Toxics Study (MVATS).[25] Recognizing the dearth of HAP ambient concentration and exposure data, both studies use carbon monoxide (CO) concentrations as the basis for estimating mobile source HAP concentrations and exposures. An important difference between the two studies, however, is that the ICF/SAI study adjusted the estimated change in ambient CO concentrations to take account of background[26] and non-mobile source[27] CO emissions. The HAP exposure function used in the ICF/SAI analysis is summarized by the following equation:

$$E = ((C \times A) - B) \times S \times M \times \frac{(VOC \times HAP)}{CO} \quad (2)$$

where :

- E = exposure to motor vehicle-emitted HAP
- C = annual ambient CO concentration to annual CO exposure concentration conversion factor
- A = county-level annual average ambient CO concentration
- B = background CO concentration
- S = no-control to control scenario CO concentration adjustment factor (equals 1 for the control scenario)
- M = total CO exposure to mobile source CO exposure conversion factor
- VOC = VOC emissions by year, county, and scenario
- HAP = VOC speciation factor by mobile source HAP
- CO = CO emissions by year, county, and scenario

Details of the derivation of each of the variables applied in the above equation are provided in the ICF/SAI report. However, in essence, the calculation involves the following basic steps.

First, annual average county-level CO ambient monitoring data are compiled from the EPA Aerometric Information Retrieval System (AIRS) database. After adjusting for background and non-mobile source contributions, these annual average ambient CO concentrations are converted to annual average CO exposure concentrations. As in the EPA MVATS, this conversion is made based on the Hazardous Air Pollutant Exposure Model - Mobile Sources (HAPEM-MS) population exposure model, which takes account of time spent in five indoor and outdoor microenvironments: indoors at home, other indoor, in-vehicle, outdoors near roadway, and other outdoor.[28] After adjusting for CO exposures attributable to non-mobile sources of CO, the CO exposures are converted to exposures for each of the mobile source HAPs based on available VOC speciation data and the ratio of co-located VOC and CO emissions.[29] These calculations are repeated for the no-control scenario after adjusting for differences in CO ambient concentrations for each target year and for differences in fuel composition.

Results

By 1990, CAA controls resulted in significant reductions in exposure to motor vehicle HAPs. Figure H-4 summarizes the nationwide annual average exposure levels, in micrograms per cubic meter, for each of the five HAPs analyzed under the control and no-control scenarios. Additional detailed results, including breakdown by urban versus rural environments and comparisons with the EPA MVATS estimates, are provided in the ICF/SAI report.

Analytical resources to carry forward these exposure estimates to derive estimates of the changes in motor vehicle HAP-related adverse effects attributable to historical CAA programs were not available.

[25] ICF/SAI, "Retrospective Analysis of Inhalation Exposure to Hazardous Air Pollutants from Motor Vehicles," October 1995, p. 4.

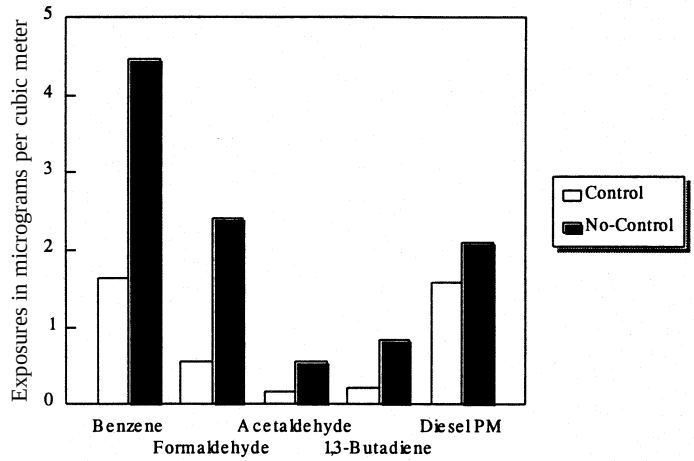
[26] Background CO is produced by the oxidation of biogenic hydrocarbons. See ICF/SAI, p. 7.

[27] The EPA MVATS attributed all measured CO to motor vehicles, resulting in an overestimation of motor-vehicle contributions to CO concentration changes. See ICF/SAI, p. 8. The MVATS assumption would also lead to a subsequent overestimation of changes in HAP exposures.

[28] See ICF/SAI, p. 3.

[29] The same HAP emission fractions used in the EPA MVATS were used herein, except for diesel PM which is not proportional to VOC emissions. Instead, diesel PM emission factors were developed using year-specific PART5 diesel PM emission factors and VMT estimates for diesel-powered vehicles.

Figure H-4. National Annual Average Motor Vehicle HAP Exposures (µg/m3).



Non-Cancer Health Effects

Broad gaps exist in the current state of knowledge about the quantifiable effects of air toxics exposure. This is particularly true for a wide range of health effects such as tumors, abnormal development, birth defects, neurological impairment, or reproductive impairment, etc. For example, the EPA's Non-Cancer Study[30] found that ambient concentrations for a substantial number of monitored and modeled HAPs exceeded one or more health benchmarks.[31] However no accepted methodology exists to quantify the effects of such exceedences. More data on health effects is needed for a broad range of chemicals.

Ecological Effects

Through the 1970s and 1980s, the adverse effects of toxic pollution on the Great Lakes became clear and undeniable. Over the same time period, scientists began collecting a convincing body of evidence that toxic chemicals released to the air can travel long distances and be deposited on land or water far from the original sources. An example of this evidence is the presence of such contaminants as PCBs, toxaphene, and other pesticides in fish in Lake Siskiwit, a lake on an island on upper Lake Superior, which has no waterborne sources of pollution. Toxaphene, a pesticide used primarily in the southeastern U.S. cotton belt, has been found as far away as the Arctic, with a decreasing air concentration gradient from the southeast toward the Great Lakes and the north Atlantic regions.

Similarly, a growing body of evidence showed that pollutants that were persistent (do not easily break down) and bioaccumulating (not significantly eliminated from the body) were magnifying up the food chain, such that top predator fish contained levels up to millions of times greater than the harmless levels in the water. As such, those who ate those large fish, such as humans, eagles, mink, and beluga whales could receive very high exposures to the pollutants. Wildlife were beginning to show adverse effects in the wild, that could be duplicated in the lab. In the Great Lakes, such chemicals as PCBs, mercury, dieldrin, hexachlorobenzene, Lindane, lead compounds, cadmium compounds, DDT/DDE, and others are of significant concern. In other places in the country, similar effects are being experienced, especially with mercury, which is transported primarily by air, but exposure to which is primarily through contaminated fish. It was this kind of information about DDT and toxaphene that led to their being banned in the U.S. under FIFRA.

While ecological and economical sciences are not yet sufficiently advanced to support the kind of comprehensive, quantitative evaluation of benefits needed for the present study, selected local and regional scale adverse ecological effects of HAPs, and their adverse consequences for human health and welfare, can and have been surveyed. In May 1994, the EPA issued its first "Report to Congress on Deposition of Air Pollutants to the Great Waters."[32] The Great Waters Report examined the pollutants contributing to adverse ecological effects, the potential significance of the contribution to pollutant loadings from deposition of airborne pollutants, and the potential adverse effects associated with these pollutant loadings. Key HAPs identified in the Great Waters Report include PCBs, mercury, dioxins, and other heavy metals and toxic organics.

[30] U.S. Environmental Protection Agency, "Toxic Air Pollutants and Noncancer Risks: Screening Studies," External Review Draft, September, 1990.

[31] Relevant benchmarks include Acceptable Daily Intake (ADI), the estimate of daily exposure at which adverse health effects are unlikely; and Lowest Observed Actual Effect Level (LOAEL), which is the lowest exposure level at which significant adverse health effects are observed.

[32] USEPA/OAR/OAQPS, "Deposition of Air Pollutants to the Great Waters, First Report to Congress," EPA-453/R-93-055, May 1994.

Of particular relevance to the present assessment, the Great Waters Report demonstrated the significance of transport and transformation of HAPs through food webs, leading to increased toxicity and biomagnification. A prime example of adverse transport and transformation is mercury. Transformation from inorganic to methylated forms significantly increases the toxic effects of mercury in ecosystems. A prime example of biomagnification is PCBs. As noted in the Great Waters Report:

> *"Pollutants of concern [such as PCBs] accumulate in body tissues and magnify up the food web, with each level accumulating the toxics from its diet and passing the burden along to the animal in the next level of the food web. Top consumers in the food web, usually consumers of large fish, may accumulate chemical concentrations many millions of times greater than the concentrations present in the water...High risk groups...include breast-feeding mothers because breast-fed babies continue to accumulate [pollutants] from their mothers after birth. For example, they can have PCB levels four times higher than their mothers after six to nine months of breast feeding."*[33]

Because of the risk of significant exposure to infants and other high-risk groups, such as "sport anglers, Native Americans, and the urban poor,"[34] a substantial number of fish consumption advisories have been issued in recent years. Current fish advisories for the Great Lakes alone include widespread advisories for PCB's, chlordane, mercury and others, cautioning that nursing mothers, pregnant women, women who anticipate bearing children, female children of any age and male children age 15 and under not eat certain high-food chain fish species. It should be noted as well that 40 states have issued mercury advisories in some freshwater bodies, and nine states have issued mercury advisories for every freshwater waterbody in the state (these states are Maine, New Hampshire, Vermont, Massachusetts, New York, New Jersey, Missouri, Michigan, and Florida).

There is little evidence indicating that the CAA had much beneficial effect on air toxic deposition to water bodies. Since the early NESHAPs were based on direct inhalation, primarily cancer effects close to a plant, they did not address the issue of cumulative effects of persistent pollutants far from the source. It was for this reason that section 112(m) was included in the 1990 CAA Amendments, with requirements to study and document the atmospheric contribution of water pollutants, the adverse human health and environmental effects resulting and the sources that should be controlled to prevent adverse effects, and additionally, to promulgate regulations to prevent adverse effects.

Conclusions — Research Needs

As has been demonstrated, there are broad gaps in the current state of knowledge about the quantifiable effects of air toxics exposure for a wide range of both human health and environmental effects. The following discussion outlines areas in which further research is needed in order to adequately quantify the benefits of air toxics control.

Health Effects

- Develop health effects data on pollutants for which limited or no data currently exists. Such studies should be focused on pollutants with a relatively high probability of exposure and/or potential adverse health effects.

- Understand mechanism of action of pollutants, for example through pharmacokinetic modeling. This will allow for a more accurate assessment of the effects of these pollutants on humans.

- Conduct research on factors that affect variations in susceptibility of human populations and determine the distribution of these factors in the U.S.

- Conduct research to better understand interactive effects of multiple pollutant exposures.

- Develop methodologies to derive alternative estimates of human cancer risk from existing upper-bound methods.

- Acquire data and develop dose-response relationships for critical noncancer effects such as developmental, neurotoxic, mutagenic, res-

[33] EPA-453/R-93-055, May 1994, p. ix.

[34] EPA-453/R-93-055, May 1994, p. x.

piratory and other effects. In particular, design methodology to quantify effects of exposures above health benchmarks.

- Acquire data and develop methods to estimate effects from acute exposure.

Exposure Assessment

- Expand data collection efforts: pre- and post-control emissions; HAP speciation; facilities location; facility parameters (stack heights, distances from stacks to fencelines, etc.).

- Develop more comprehensive exposure models which incorporate activity patterns, indirect exposures, total body burden, ratios of time spent indoors to outdoors.

- Continue to refine uncertainty analysis methods.

Ecosystem Effects

- Reliable estimates/measures of the levels of persistent bioaccumulating toxics in different media (air, water column, soils and sediments)

- Work to correlate levels of persistent bioaccumulating toxics with exposures, biota concentrations/accumulation, and adverse effects, especially subtle effects such as wasting, behavioral effects, and developmental effects.

- Criteria for effects, such as a wildlife correlate to a RfD or dose-response curve. This work should be done to complement the mass balance efforts now being completed, which will model source emissions to water column concentrations, then design research to predict effects on living resources given those predicted levels.

- Work to determine the effects of mixtures of persistent bioaccumulating toxic pollutants, and to determine cause-effect relationships of exposures over long periods of time.

- Studies to evaluate toxic effects in less well understood terrestrial systems such as: soil organisms/invertebrates, food web effects, amphibian effects, effects on endangered species and phytotoxic effects.

- Work to improve understanding of effects of toxic air pollutants on wetland species and wetland functions.

Economic Valuation

- Develop valuation estimates for endpoints for which inadequate estimates currently exist. These valuation estimates must be consistent with the kinds of damages expected.

- Initiate broad-scope economic valuation of air toxics program using survey techniques.

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