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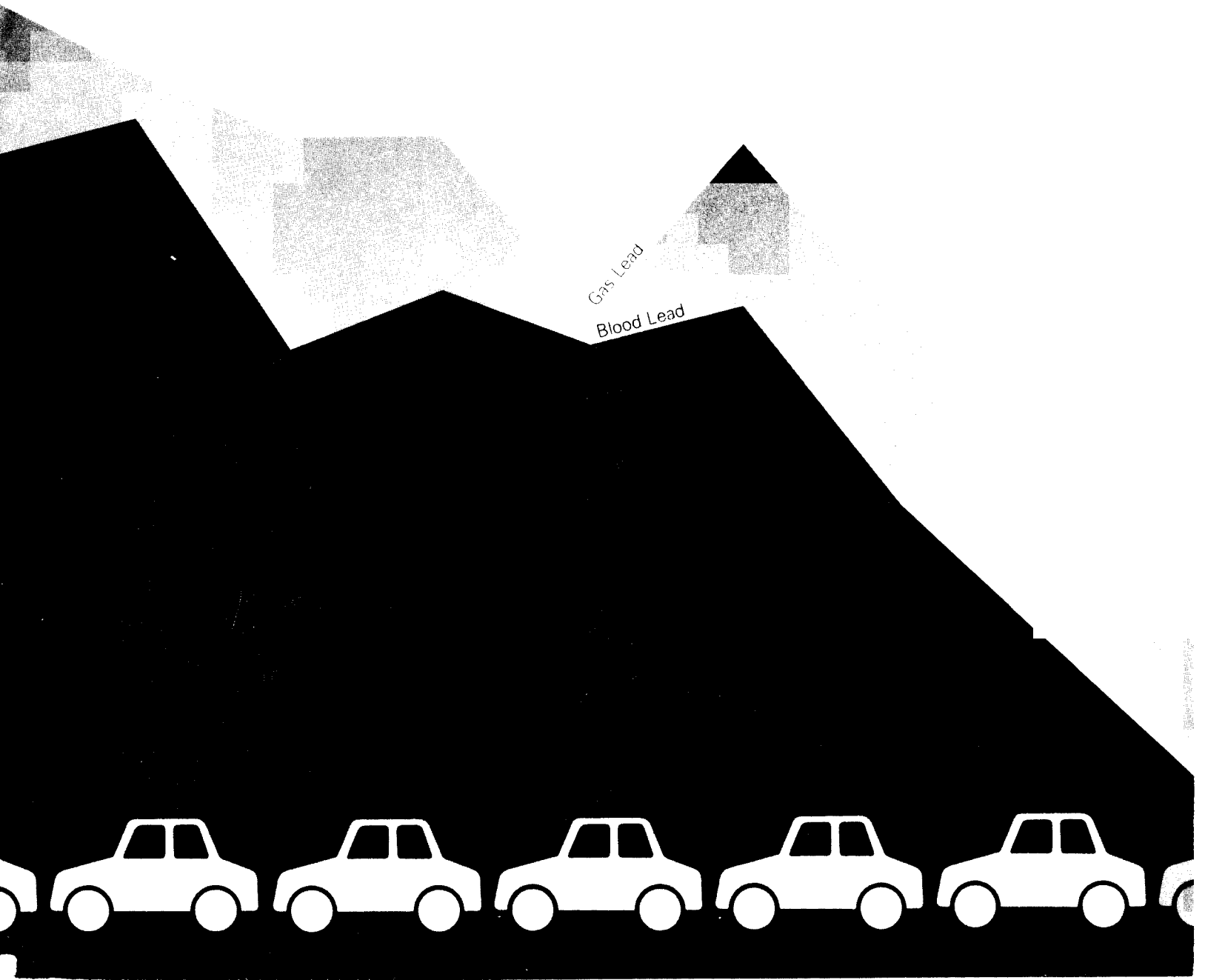
Costs and Benefits of Reducing Lead in Gasoline

Final Regulatory Impact Analysis



COSTS AND BENEFITS OF REDUCING LEAD IN GASOLINE:

FINAL REGULATORY IMPACT ANALYSIS

February 1985

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EXECUTIVE SUMMARY

The Environmental Protection Agency (EPA) is promulgating a rule to reduce the amount of lead in gasoline from its current limit of 1.10 grams per leaded gallon (gplg) to 0.50 gplg on July 1, 1985 and to 0.10 gplg effective January 1, 1986. EPA's primary objective in promulgating this rule is to minimize the adverse health and environmental effects of lead in gasoline. To increase flexibility in meeting the phasedown schedule, EPA has proposed to allow refineries that reduce lead ahead of schedule in 1985 to "bank" those extra lead rights for use in 1986 or 1987. The Agency also is considering the possibility of a complete ban on leaded gasoline, to take effect as early as 1988. This Regulatory Impact Analysis addresses only the final phasedown rule; a separate document summarizes the costs and benefits of a possible ban.

Basis for Action

Section 211(c)(1) of the Clean Air Act gives EPA's Administrator broad authority to "control or prohibit the manufacture . . . or sale of any fuel additive" if its emission products (1) cause or contribute to "air pollution which may be reasonably anticipated to endanger the public health or welfare," or (2) "will impair to a significant degree the performance of any emission control device or system . . . in general use." Reductions in the lead content of gasoline are justified under both of these criteria.

Lead in gasoline has been shown to increase blood lead levels, which in turn have been linked to a variety of serious health effects, particularly in small children. Recent studies linking lead to blood pressure in adult males also are a source of concern about the health effects of lead in gasoline; because these studies have just been published, however, EPA will not rely upon that evidence until there has been a greater opportunity for scientific review and public comment.

Lead in gasoline also impairs the effectiveness of pollution-control catalysts. A 1983 EPA survey of vehicles in use showed that about 15.5 percent of the vehicles that should use unleaded gasoline to protect the effectiveness of their pollution-control catalysts are misfueled with leaded gasoline, resulting in significant excess emissions of hydrocarbons (HC), nitrogen oxides (NO_x), and carbon monoxide (CO). In addition to these health and environmental effects, reducing lead in gasoline will reduce vehicle maintenance costs associated with the corrosive effects of lead on engines and exhaust systems, and will improve fuel economy.

EPA has considered a variety of alternatives for reducing the health and environmental problems caused by lead in gasoline, and has concluded that the most effective approach is to reduce the amount of lead in gasoline as quickly as possible. EPA is confident that the phasedown schedule mandated by the rule can be met by the refining industry with existing equipment. The Agency is not promulgating a complete ban on lead in gasoline at this time because of continuing concern about the possible effects of such a ban on certain engines that may rely on lead for protection against valve-seat recession. EPA will consider the valve-seat issue, along with the recent evidence on lead and blood pressure, in reaching a final decision on a ban.

Costs of Reducing Lead in Gasoline

Since the 1920s, petroleum refiners have added lead to gasoline as a relatively inexpensive way of boosting octane. To meet octane requirements with less lead, refiners have several options, including additional processing in reforming or isomerization units and the use of alternative additives, such as MMT or alcohols. At the margin, however, each of these alternatives is more expensive than lead for producing octane. Higher refining costs constitute virtually all of the rule's social costs.

We estimated the additional refining costs using a model of the petroleum refining industry developed for the Department of Energy (DOE). This model employs a linear programming framework to represent U.S. refining operations, and can find the minimum-cost method for producing a particular product slate subject to various constraints (including the amount of lead allowed in gasoline). The model has been used by both EPA and DOE in previous rulemakings, and several verification checks have indicated that it performs well.

To estimate the costs of alternative rules, we first ran the model specifying the existing lead limit of 1.10 gplg. We then ran the model with a tighter constraint on lead. In both types of runs, the model also was constrained to meet projected demands for gasoline and other refined products. The difference between the costs at the two lead limits is the estimated cost of the rule. This procedure limits the possibility of underestimating costs if the model assumes more flexibility than is in fact possible; any overoptimization affects both estimates, and thus has little impact on the difference between the two. In addition, we placed many constraints on the model to reflect real-world limitations in the ability of the refining industry to fully optimize production.

Our base case results suggest that the final rule will cost less than $100 million for the second half of 1985, when the 0.50 gplg limit will apply. For later years, when the 0.10 gplg limit will apply, the estimated costs range from $608 million in 1986 to $441 million in 1992. (The estimated costs fall over time because of projected declines in the demand for leaded gasoline, even in the absence of this new rule.)

We ran extensive sensitivity analyses to probe the limits of feasibility. Those analyses focused on 1986, because that is the first year in which the 0.10 gplg will apply, and refineries will not be able to undertake substantial new construction by then. Those sensitivity analyses show that the rule remains feasible (i.e., product demands can be met with existing refining capacity) under most conditions. Only when extremely unlikely combinations of multiple adverse conditions are assumed does feasibility appear to be in doubt, and then only for the peak-demand summer months. Additional sensitivity analyses show that even in those worst-worst cases, the 0.50 gplg limit in 1985 does not approach infeasibility.

We also examined the potential impact on costs of the banking rule that EPA recently proposed and which it may promulgate shortly. Under the banking rule, refineries that reduced their lead use below applicable limits in 1985 (1.10 gplg prior to July 1 and 0.50 gplg for the second half of the year) could bank those extra reductions and use them in 1986 or 1987. Banking would allow refineries to follow their own least-cost schedules of lead reduction, so long as their total usage over the three years did not exceed the amount allowed by this rule. Because banked rights would be freely transferable among refineries, they also would increase individual refineries' flexibility by allowing those refineries with relatively high costs to buy rights from refineries with lower costs. Our estimates suggest that banking would reduce the present value of the rule's cost by about $200 million over the 1985-to-1987 period. Moreover, it appears that banking would alleviate the potential unfeasibility found in the most extreme sensitivity analyses.

Benefits of the Rule

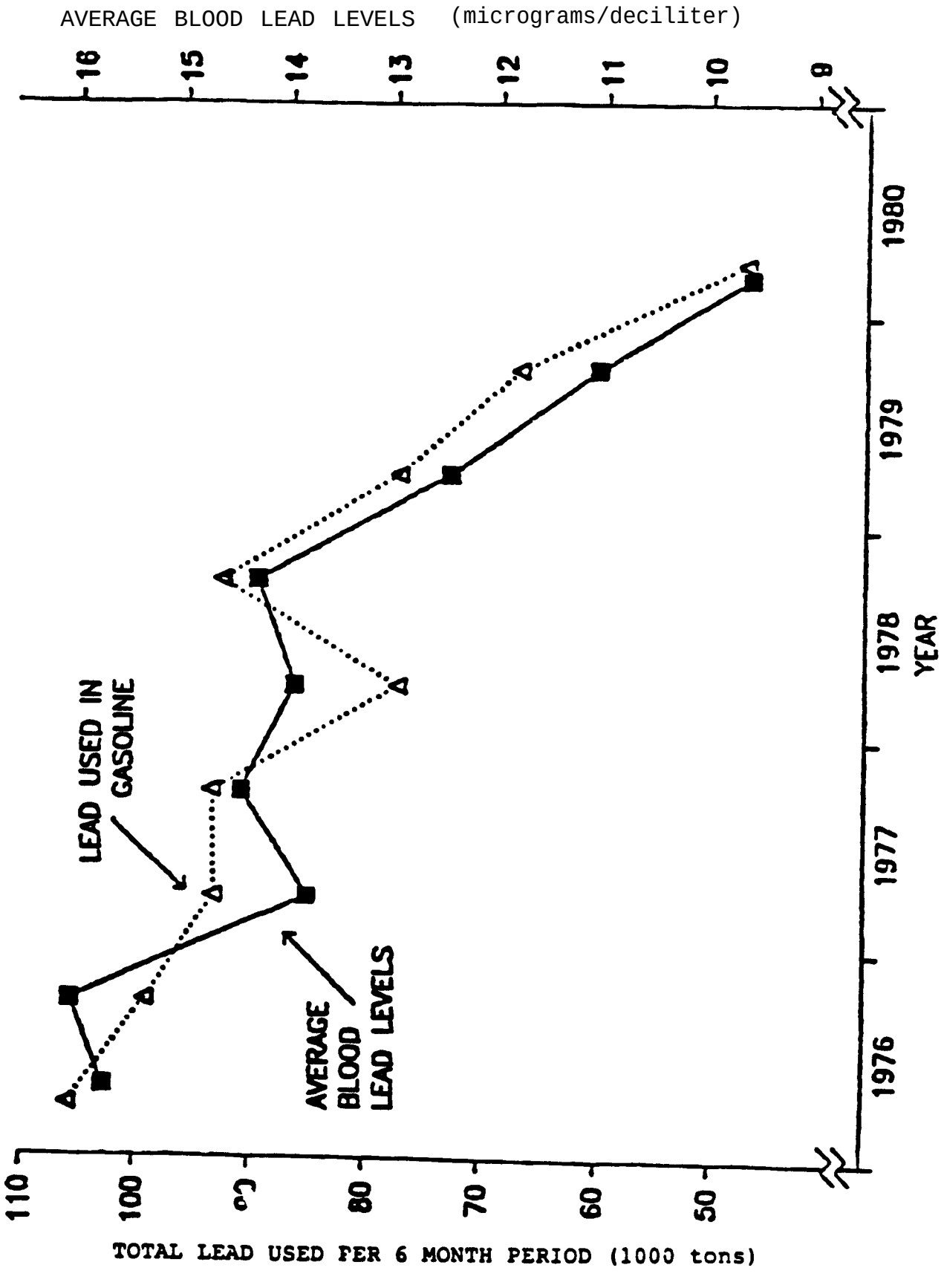
We estimated benefits in four major categories: (1) children's health and cognitive effects associated with lead; (2) blood-pressure-related effects in adult males due to lead exposure; (3) damages caused by excess emissions of HC, NO_x, and CO from misfueled vehicles; and (4) impacts on maintenance and fuel economy. In each category, our estimates do not cover all of the likely benefits because of gaps in the data or difficulties in monetizing some types of benefits. Nonetheless, the estimates are substantial, and far exceed the costs.

Human exposure to lead from gasoline. To predict the lead-related health effects of the rule, we began by estimating its impact on lead in individuals' blood. People are exposed to lead from gasoline through a variety of routes, including direct inhalation of lead particles when they are emitted from vehicles, ingestion of lead-contaminated dust or inhalation of such dust when it is stirred up, and ingestion of food that has been contaminated with lead. Although it is difficult to estimate the contributions of these individual pathways, several large data sets make it possible to estimate the total contribution of lead in gasoline to concentrations of lead in human blood.

These data sets include records of lead-screening programs from the Centers for Disease Control (CDC), records from screening programs in individual cities, and, most importantly, the Second National Health and Nutrition Examination Survey (NHANES II), which provides blood lead measurements (and other important information) on a large representative sample of the U.S. population surveyed during the late 1970s. By linking these data to data on gasoline lead use, it is possible to estimate statistically how blood lead levels respond to changes in gasoline lead.

Several studies have shown remarkably strong and consistent relationships between gasoline lead and blood lead. Figure E-1 plots those two measures over time using data from NHANES II. Note how strong the relationship is; blood lead tracks both the seasonal variations in gasoline lead (rising during the summer months, when more gasoline is used) and the long-term downward trend in gasoline lead. Multiple regression analyses show that this relationship continues to hold after controlling for other factors (such as socioeconomic status, nutritional factors, and exposure to other sources of lead). Such studies suggest that during the 1970s, gasoline was responsible on average for about half of the lead in blood (other sources of lead include lead paint, stationary sources, and lead solder in cans). Experimental studies, where the isotopes in gasoline lead have been modified so that its presence in blood can be distinguished from lead from other sources, have yielded similar conclusions.

Statistical analyses indicate that gasoline lead not only raises the average level of lead in blood, but also contributes substantially to the incidence of lead toxicity in children. Based on an analysis of NHANES II, we predict that the 0.10 gplg limit will roughly halve the number of children with blood lead levels above those recognized as harmful. Since 1978, the CDC has recommended that children with blood lead levels above 30 micrograms per deciliter (ug/dl) receive follow-up testing and possible treatment. The CDC recently reduced that recommended level to 25 ug/dl. We estimate that in 1986 alone, the rule will prevent 172,000 children from exceeding 25 ug/dl blood lead.



LEAD USED IN GASOLINE PRODUCTION AND AVERAGE NHANES II BLOOD LEAD LEVELS (FEB. 1976 – FEB. 1980)

FIGURE E-1

Children's health and cognitive effects. Elevated blood lead levels have been linked to a wide range of health effects, with particular concern focusing on young children. These effects range from relatively subtle changes in biochemical measurements at low doses (e.g., 10 ug/dl) to severe retardation and even death at very high levels (e.g., 100 ug/dl). Lead can interfere with blood-forming processes, vitamin D metabolism, kidney functioning, and neurological processes. The negative impact of lead on cognitive performance (as measured by IQ tests, performance in school, and other means) is generally accepted at moderate-to-high blood-lead levels (30 to 40 ug/dl and above). Several studies also suggest cognitive effects at lower levels. Changes in electroencephalogram readings have been found at levels as low as 10 to 15 ug/dl.

For children's health effects, we estimated benefits in two categories: medical care for children exceeding the new CDC cutoff and compensatory education for a subset of those children who may suffer cognitive effects from exposure to lead. These estimates are conservative in that they do not include many benefit categories (e.g., lasting health and cognitive damage not reversed by medical treatment and compensatory education), nor do they attribute any benefits to reducing lead levels in children whose blood lead levels would be below 25 ug/dl even in the absence of the rule.

To estimate reductions in medical care expenses, we relied on recently published recommendations for testing and treating children with blood lead levels above 25 ug/dl. Such treatment, we estimate, costs about $900 per child over 25 ug/dl. (This average reflects lower costs for most of these children, but much higher costs for the subset requiring chelation therapy.)

The estimates for compensatory education assumed three years of part-time compensatory education for roughly 20 percent of the children above 25 ug/dl; that averages about $2600 per child above that blood-lead level. Thus we estimated a total of $3500 in monetized benefits for each child brought below 25 ug/dl. Our estimates of aggregate benefits in this category ranged from about $600 million in 1986 to roughly $350 million in 1992.

Blood-pressure-related benefits. Lead has long been associated with elevated blood pressure, but until recently most of the studies have focused only on hypertension and relatively high lead levels (typically found only in those occupationally exposed to lead). Two recent studies, however, have found a continuous relationship between blood lead and blood pressure using data from a large representative sample of the U.S. population (NHANES II, the same data set used to estimate the relationship between gasoline lead and blood lead). These studies show a strong relationship that has proved robust in the face of exhaustive statistical tests involving many possible confounding factors and alternative specifications of the model.

These findings, if verified, have important implications for the benefits of the phasedown rule. EPA has not relied upon them in setting the final phasedown rule, because they are too recent to allow widespread review and comment. They will be an important element, however, in the decision on a final ban, so we present estimates of blood–pressure-related benefits in this RIA for informational purposes.

To calculate the benefits, we used logistic regression equations estimated from NHANES II to predict how the rule would affect the number of hypertensive. These estimates cover only males aged 40 to 59, because the effect of lead on blood pressure appears to be strongest for men and because estimates for that age range are not confounded by a strong covariance between age and blood lead. We estimate that the rule will reduce the number of hypertensives in that group by about 1.8 million in 1986. We valued reductions in hypertension based on estimates of the costs of medical care, medication, and lost wages; they yielded a value of $220 per year per case of hypertension avoided.

We also estimated how reductions in blood pressure would affect the incidence of various cardiovascular diseases, based on our projections of changes in blood pressure as a result of the rule and estimates of the relationships between blood pressure and heart attacks, strokes, and deaths from all causes. The latter estimates were derived from several large-scale epidemiological studies, primarily the Framingham study. Because those studies included very few nonwhites, we restricted our estimates to white males aged 40 to 59.

We valued reductions in heart attacks and strokes based on the cost of medical care and lost wages for nonfatal cases (the fatalities from heart attacks and strokes were included in the estimate of deaths from all causes). That procedure yielded benefits of $60,000 per heart attack and $44,000 per stroke avoided. It is important to note that these estimates do not account for any reductions in the quality of life for the victims of heart attacks and strokes (e.g. , the partial paralysis that afflicts many stroke victims).

Valuing reductions in the risk of death is difficult and controversial, with a wide range of estimates in the literature. EPA’s RIA guidelines, for example, suggest a range of $400,000 to $7 million per statistical life saved. For our point estimate, we used a value from the lower end of that range, $1 million per case. The benefits of reduced mortality dominate our estimates of total blood-pressure-related benefits, which range from almost $6 billion in 1986 to about $4.5 billion in 1992.

Benefits of reducing pollutants other than lead. Reducing the amount of lead in gasoline should decrease emissions of several pollutants in addition to lead. Most of these reductions

will result from less misuse of leaded gasoline in vehicles that should use unleaded to protect the effectiveness of their pollution-control catalysts. EPA expects the rule to significantly reduce misfueling because it will be more expensive to produce leaded regular gasoline (at 89 octane) with 0.10 gplg than to make unleaded regular (at 87 octane). As a result, the gap between the retail prices of unleaded and leaded regular gasolines should narrow, and may well reverse. (At present, leaded regular is slightly cheaper to make than unleaded regular, but the retail price differential is much larger than the manufacturing cost differential, apparently because of marketing strategies by retailers.)

Eliminating misfueling would substantially reduce emissions of HC, NO_x, and CO. All three of these pollutants have been associated with damages to health and welfare, and contribute to ambient air pollution problems covered by National Ambient Air Quality Standards (NAAQS). To predict the emission reductions that would be associated with eliminating misfueling, we used survey data on the extent of misfueling, tests showing the effect of misfueling on emissions per mile traveled, and estimates of the numbers of miles traveled by vehicles of different ages and types. The rule also should reduce emissions of benzene (a hydrocarbon that has been associated with leukemia) and ethylene dibromide (a suspected human carcinogen, which is added to leaded gasoline to control excess lead deposits in engines).

We valued these reduced emissions in two ways. The first involved direct estimation of some of the health and welfare effects associated with these pollutants. This method is conceptually correct, but suffers from various uncertainties and the inability to generate estimates for some potentially important categories. Virtually all of the benefits that we could quantify were associated with projected declines in ozone as a result of reductions in HC and NO_x emissions.

Our second method valued the emission reductions based on the implicit cost per ton controlled of the emission control equipment destroyed by misfueling. The final estimate, used in computing total and net benefits, was the simple average of the two different methods. Assuming that the 0.10 gplg rule will eliminate 80 percent of misfueling, those estimates range from $222 million in 1986 to $248 million in 1992. (The estimates for this category increase over time because the total amount of misfueling is projected to increase in the absence of the rule.)

Maintenance and fuel economy benefits. Reducing lead in gasoline will decrease vehicle maintenance expenses associated with the corrosive effects of lead and its scavengers on engines and exhaust systems. The rule also is likely to increase fuel economy, both because additional reforming to replace the octane now provided by lead will increase the energy content of gasoline and because leaded gasoline fouls oxygen sensors in newer misfueled vehicles.

Three categories of maintenance benefits were estimated: exhaust systems, spark plugs, and oil changes. Estimates for the first two categories were based on fleet studies of vehicles in use, which showed that exhaust systems and spark plugs last longer with unleaded than with leaded gasoline. Estimates of oil change benefits were based on studies showing that oil maintains its quality longer with unleaded than with leaded. Summing together these three categories, we estimate that reducing lead in gasoline from 1.10 gplq to 0.10 gplq will yield benefits of $0.0017 per vehicle mile, or about $17 per year for a vehicle driven 10,000 miles. Because of the large number of vehicles affected, the aggregate benefits are large, ranging from about $900 million in 1986 to roughly $750 million in 1992.

The fuel economy estimate, as noted above, has two components. To estimate the gain in fuel economy due to higher energy content, we used the change in fuel density predicted by the DOE refining model and applied it to a fuel economy formula developed by the Society of Automotive Engineers. To estimate the portion due to reduced fouling of oxygen sensors, we estimated the change in the number of misfueled sensor-equipped vehicles and used experimental data on how much extra fuel is consumed by vehicles with fouled sensors. Total estimated fuel economy benefits exceed $100 million in most years.

Costs and Benefits of Alternatives

Table E-1 summarizes several important non-monetary measures of the benefits of the 0.10 gplg standard for a single year, 1986. These estimates assume that the rule will eliminate all misfueling. We also examined a wide range of alternative standards, however, and considered various assumptions about the impacts of those rules on misfueling, ranging from the rule eliminating all misfueling to the rule having no impact on current misfueling rates. In addition, we computed net benefits with and without the preliminary estimates of blood-pressure-related benefits.

Regardless of the assumption about misfueling, and whether or not the blood-pressure-related benefits were included, we found that net benefits were maximized at the tightest of the alternative standards considered, 0.50 gplg for the second half

TABLE E-1. Nonmonetary Measures of Health and Environmental Benefits of 0.10 gplg in 1986, Assuming No Misfueling

Reductions in thousands of children above selected blood lead levels	
30 ug/dl	52
25 ug/dl	172
20 ug/dl	563
15 ug/dl	1,726
Reductions in thousands of tons of pollutants	
Hydrocarbons	305
Nitrogen oxides	94
Carbon monoxide	2,116
Reductions in adult male health effects	
Thousands of hypertensives	1,804
Myocardial infarctions	5,350
Strokes	1,115
Deaths	5,160

of 1985 and 0.10 gplg for 1986 and subsequent years. Table E-2 presents year-by-year estimates of the costs and benefits of the final rule under our “partial misfueling” case, which assumes that misfueling will continue unabated under the 0.50 gplg standard in 1985, and then will be reduced by 80 percent under the 0.10 gplg standard in 1986 and subsequent years. The net benefits without blood-pressure-related effects exceed $1 billion per year in 1986 and later years. If the blood-pressure-related benefits are included, net benefits exceed $5 billion per year. Although many of the individual components of these estimates are subject to uncertainty, the magnitude of the estimated monetized benefits relative to the costs, together with the many potentially important unmonetized benefits, indicate that rapid reductions of lead in gasoline are amply justified.

TABLE E-2. Year-by-Year Costs and Monetized Benefits of Final Rule, Assuming Partial Misfueling (millions of 1983 dollars)

	1985	1986	1987	1988	1989	1990	1991	1992
MONETIZED BENEFITS								
Children's health effects	223	600	547	502	453	414	369	358
Adult blood pressure	1,724	5,897	5,675	5,447	5,187	4,966	4,682	4,691
Conventional pollutants	0	222	222	224	226	230	239	248
Maintenance	102	914	859	818	788	767	754	749
Fuel economy	35	187	170	113	134	139	172	164
TOTAL MONETIZED BENEFITS	2,084	7,821	7,474	7,105	6,788	6,517	6,216	6,211
TOTAL REFINING COSTS	96	608	558	532	504	471	444	441
NET BENEFITS	1,988	7,213	6,916	6,573	6,284	6,045	5,772	5,770
NET BENEFITS EXCLUDING BLOOD PRESSURE	264	1,316	1,241	1,125	1,096	1,079	1,090	1,079

CHAPTER I

INTRODUCTION

The Environmental Protection Agency (EPA) is promulgating a rule to sharply reduce the lead content of gasoline from its current limit of 1.10 grams per leaded gallon (gplg) to 0.10 gplg. The phasedown will take effect in two steps: effective July 1, 1985, the limit will be 0.50 gplg; effective January 1, 1986, the limit will be 0.10 gplg. Although EPA believes that these standards are feasible for the industry as a whole, to increase individual refineries' flexibility in meeting that schedule, EPA has proposed a rule change to allow refineries to reduce lead use below allowable limits in 1985 and "bank" those credits for use in 1986 and 1987.

EPA's goal in promulgating this phasedown schedule is to minimize the adverse health and environmental impacts of lead in gasoline. To further aid in meeting that goal, the Agency is considering the elimination of all lead in gasoline.

The health and environmental consequences of lead in gasoline include both the direct health effects of exposure to lead and the effects of higher emissions of conventional pollutants (hydrocarbons, nitrogen oxides, and carbon monoxide) from vehicles whose pollution-control catalysts have been poisoned by the misuse of leaded gasoline. In addition to these health and environmental benefits, which form the basis for this rule, reducing lead in gasoline will provide benefits to vehicle owners in the form of increased fuel economy and reduced maintenance expenditures for lead-induced corrosion of engines and exhaust systems. On the

other hand, lead is a low-cost octane booster, so the proposed rule will raise the cost of producing gasoline.

EPA has determined that both the final phasedown rule and the possible ban are "major" regulatory actions under the criteria of Executive order 12291, because higher gasoline production costs would exceed $100 million per year under each action. For major rules, the Executive Order requires a Regulatory Impact Analysis (RIA); this document constitutes the final RIA for the phasedown being promulgated. Although EPA has not relied on banking in analyzing the costs or establishing the feasibility of the final phasedown, this RIA also examines the impact of the banking proposal on the costs and benefits of the phasedown rule. A separate preliminary RIA has been prepared for the ban, now under consideration for as early as 1988; it employs the same methods used in this document, and summarizes the costs and benefits of a ban. That preliminary RIA incorporates by reference large parts of this final RIA.

The remainder of this chapter provides an overview of the problem of lead in gasoline, a brief review of earlier rulemakings and the proposal made in August 1984, an analysis of the rationale for government intervention, and a discussion of alternative regulatory approaches.

I.A. Background Information

The rules being promulgated and proposed are part of a series of actions taken by EPA over the past eleven years to address the health and environmental hazards posed by lead in gasoline. This section provides some background information

for these latest actions.

I.A.1. Previous Rulemakings

EPA has regulated lead in gasoline since 1973 to meet two goals: assure the availability of unleaded gasoline for those vehicles with pollution-control devices (catalysts) that are rendered ineffective by leaded fuel, and reduce the adverse health effects associated with exposure to lead.

EPA's original rule for lead in gasoline limited the lead content per gallon averaged over all gasoline (both leaded and unleaded) sold by each refinery (38 FR 33741; December 6, 1973). EPA also temporarily set separate and less stringent interim limits for small refiners.

In 1982, EPA promulgated new rules (47 FR 49331; October 29, 1982) that: (1) switched the basis of the standard from all gasoline to leaded only, (2) set a limit of 1.10 gplg, (3) phased in uniform treatment of all refiners regardless of size, and (4) allowed "constructive averaging" or "trading" of lead use across refineries (e.g., one refinery could produce a gallon with 1.20 grams of lead if it traded with another refinery that produced a gallon with only 1.00 gram).

The purpose in switching from a standard based on the overall pool average to one based only on leaded gasoline was to reduce total lead usage as sales of leaded gasoline declined with the retirement of the older vehicles allowed to use it. (With the overall gasoline-pool standard, refiners would have been allowed to increase the amount of lead per gallon of leaded gasoline as the total demand for leaded declined.) When EPA

promulgated the 1982 rule, it projected a steady and substantial decline in total lead in gasoline over the next decade.

1.A.2. Continuing Problems

Since 1982, new studies and reanalyses have shown lead to be a greater health risk than previously thought. In addition, total lead in gasoline has not declined as projected in 1982. Figure 1-1 compares the 1982 lead projections with actual lead use in 1983 and with current projections for later years.

The major cause of higher lead levels than previously projected is misfueling, the use of leaded gasoline in vehicles designed to use unleaded. EPA's 1983 survey of vehicles in use indicated that about 15.5 percent of vehicles required to use unleaded gasoline were misfueled with leaded (U.S. EPA, 1984e). By comparison, in the 1982 survey, the overall rate was 13.5 percent, with lower average rates in areas with Inspection and Maintenance (I/M) mobile source enforcement programs (6.2 percent) than in areas without such programs (15.1 percent) (U.S. EPA, 1983a). The 1983 survey showed increased misfueling in I/M areas, reducing the gap. These estimates probably understate the true extent of misfueling because owners were not required to submit their vehicles for testing. Moreover, the surveys show higher misfueling rates in older vehicles, so misfueling rates are likely to grow in future years as the average age of catalyst-equipped vehicles increases.

Misfueling not only increases lead emissions, but by poisoning pollution-control catalysts, it increases emissions of hydrocarbons (HC), nitrogen oxides (NO_x), and carbon monoxide

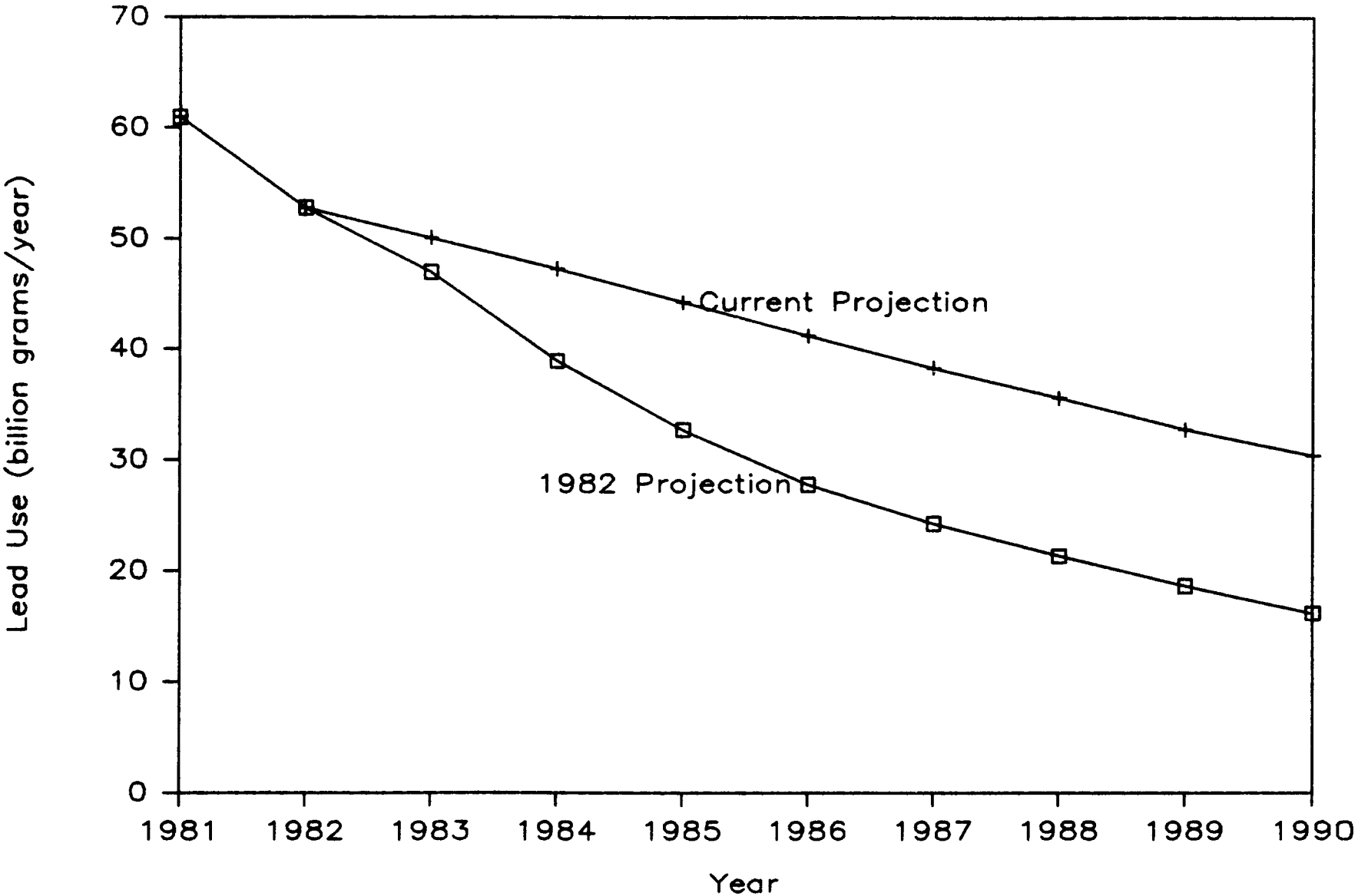


FIGURE I-1. Comparison of 1982 and Current Projections of Lead Use in Gasoline

(CO). In vehicles equipped with three-way catalysts (virtually all post-1981 cars), lead-induced poisoning of catalysts increases emissions by 500 percent for HC, 100 percent for NO_x, and 300 percent for CO (U.S. EPA, 1983b). Each of these pollutants is regulated by vehicle emission standards. Each is also covered by a National Ambient Air Quality Standard (NAAQS) or contributes to a pollution problem covered by a NAAQS (HC and NO_x form ozone, and NO_x forms NO_2).

Continued use of lead in gasoline, whether to meet the demands of misfuelers or of legal users, poses a serious threat to health. Several studies have shown a strong relationship between lead in gasoline and levels of lead in children's blood, with blood lead levels following gasoline lead closely, tracking seasonal fluctuations as well as long-term trends. Analyses using several different data sets show that this relationship remains strong and statistically significant even when other potentially confounding variables are controlled for using multiple regression and other statistical techniques. These analyses show that gasoline lead is related to blood lead levels in both adults and children.

Analyses also show that gasoline lead not only increases average blood lead levels, but also raises the number of children with dangerously high blood-lead levels. Statistical analyses indicate that lead in gasoline accounts for about half of the number of children above 30 micrograms per deciliter (ug/dl), until recently the level set by the Center for Disease Control (CDC) for follow-up testing and possible treatment. Lead paint,

emissions from stationary sources, and other non-gasoline sources of lead exposure account for the other children at high levels, although some of these additional exposures, including lead in food, partially reflect past emissions of lead from gasoline.

The adverse health consequences of high levels of lead in children are well accepted. They include damage to the kidney, the liver, the reproductive system, blood creation, basic cellular processes, and brain functions. The CDC recommends that children with blood lead levels above 25 ug/dl receive follow-up testing and possible medical treatment; upon further testing, about 70 percent of children with blood lead levels above 25 ug/dl are expected to be classified as “lead toxic” under CDC criteria.

Increasing evidence also points to health effects at blood lead levels below 25-30 ug/dl. These effects include inhibition of certain enzyme activities, changes in EEG patterns, impairment of heme synthesis, increases in levels of a neurotoxic chemical, possible interference with neurotransmission processes, impairment of vitamin D activity, and impairment of globin synthesis. Several studies also have found indications of lead affecting cognitive functions (as measured by IQ tests and other means), as well as having other neurobehavioral effects, at levels well below 30 ug/dl. Although EPA has not reached definite conclusions regarding the specific blood lead levels at which such effects occur, the Clean Air Scientific Advisory Committee (CASAC) has recommended that the Agency consider these studies in the Criteria Document for the ambient air quality standard for lead.

Concerns about the health effects of ambient exposure to lead traditionally have focused on children. Although lead has a variety of adverse effects on the health of adults, most of the known effects appear not to be of substantial concern except at blood-lead levels in excess of 30-40 ug/dl. Recent analyses, however, indicate a strong and robust relationship between blood lead levels and blood pressure, with no apparent threshold. Those findings have important implications for estimating the benefits of reducing lead in gasoline, because high blood pressure, in turn, is linked to a variety of cardiovascular diseases including hypertension, myocardial infarction and strokes.

I.A.3. The August 1984 Proposal

Concerns about the health risks posed by lead in gasoline and about the growing misuse of leaded gasoline in catalyst-equipped vehicles led EPA to consider additional restrictions on the lead content of gasoline. In August 1984, the Agency proposed to reduce the allowable lead limit to 0.10 gplg, effective January 1, 1986. EPA also indicated that it would consider alternative phasedown schedules with more than one step that would start earlier than 1986. In addition, it offered two alternatives for the final elimination of lead in gasoline: a ban to take effect in the mid-1990s, or allowing market forces to eliminate leaded gasoline as demand shrank.

The proposal and the alternatives reflected EPA's desire-to eliminate lead in gasoline quickly, tempered by two concerns. First, the Agency wanted to ensure that the phasedown schedule

could be met without excessive costs or gasoline shortages due to lack of time to build additional refining capacity. The Agency's analyses, based on the Department of Energy's (DOE) refinery yield model, indicated that refineries could meet the 0.10 gplg standard with existing equipment, but in the proposal EPA solicited comments and additional data on that issue.

Second, EPA was concerned that eliminating lead in gasoline altogether might pose a serious risk of premature valve-seat wear for certain older engines. These engines include those in older automobiles (primarily those manufactured before 1971) and some gasoline-powered trucks and off-road vehicles (including farm equipment) that do not have hardened valve seats. Dynamometer and track tests have shown that such engines can suffer premature erosion of valve seats ("valve recession") with unleaded gasoline under severe conditions (sustained high speeds and heavy loads), but studies of vehicles in normal use generally have failed to find excessive wear. However, based on the available studies, the proposal allowed 0.10 gplg to provide a margin of safety for those engines that might need lead to protect against undue valve seat recession.

I.A.4. Information Received After August 1984 Proposal

Several pieces of information received after the August 1984 proposal have strengthened EPA's determination to reduce lead in gasoline as quickly as possible. The CDC has now lowered its recommended cut-off level for follow-up testing (from 30 ug/dl to 25 ug/dl) as well as its definition of lead toxicity. The CDC cautions, however, that even this new, lower level should

not be regarded as “safe”; rather, it reflects tradeoffs between protecting children against the adverse effects of lead and the risks associated with treatment, as well as the limited ability of screening tests to reliably measure blood-lead levels below 25 ug/dl. The reduction in this cut-off level more than triples the number of children at risk under CDC criteria.

Additionally, comments and data from refiners, plus additional sensitivity analyses using the DOE refining model, have reinforced the Agency’s conviction that the refining industry as a whole can meet a 0.10 gplg standard within a year of promulgation using existing equipment. Moreover, these same sources indicate that it is possible to achieve significant reductions even sooner. Thus, the Agency has decided to add an interim standard of 0.50 gplg to take effect on July 1, 1985.

Although EPA believes that both standards are feasible for the industry as a whole, EPA’s recent proposal to extend the lead trading program to allow “banking” also will increase flexibility and help to ensure that individual refineries can meet the phasedown schedule in the most cost-effective manner. Under that proposed rule change, refineries using less lead than allowed under the applicable limits in 1985 could “bank” those extra lead rights for use in meeting the tighter limits in 1986 and 1987. Banking should reduce significantly the overall costs of meeting the phasedown limits and increase a refinery’s flexibility in meeting unexpected problems, without permitting any additional use of lead over the three-year period, 1985 through 1987.

Three additional types of information have contributed to EPA's decision to consider a ban on lead in gasoline by 1988. First, the Agency has received data indicating that valve-seat recession with unleaded gasoline may be a much less serious problem than earlier feared. The Agency has reviewed data from a large test performed by the U.S. Army in the mid-1970s that involved switching many types of vehicles -- including heavy-duty trucks, construction equipment, motorcycles, and stationary engines (such as generators), as well as light-duty vehicles -- from leaded to unleaded gasoline. The Army's study showed no detectable increase in valve-seat problems and resulted in all of the armed services switching to unleaded gasoline (where available) by 1976. In addition, the U.S. Postal Service switched its heavy-duty trucks to unleaded in 1980, and a review of their computerized maintenance records shows no evidence of abnormal rates of valve seat recession. Moreover, reanalyses of other studies suggest that lead may cause serious engine durability problems. EPA is reviewing these and other studies relevant to this issue.

Second, although EPA continues to believe that the phasedown rule will have a significant impact on misfueling, it is less confident that the problem will be eliminated so long as any lead remains in gasoline. Moreover, the 1983 EPA misfueling survey, the results of which became available after the August proposal, showed that misfueling continues to be a serious problem, and that the rates appear to be increasing.

Finally, two recently published studies (Pirkle et al., 1985; Harlan et al., 1985) indicate a strong relationship between blood

lead and blood pressure in adult males. Because of the well-established relationships between gasoline lead and blood lead, and between blood pressure and cardiovascular disease, these studies have potentially important implications for public health. Preliminary estimates based on Pirkle et al. indicate that lead in gasoline may be responsible for well over one million cases of hypertension per year and for over 5,000 deaths from heart attacks, strokes, and other diseases related to blood pressure. Moreover, these estimates cover only males aged 40 to 59 and, in the case of heart attacks, strokes, and deaths, only white males in that age group. (The Pirkle et al. study did not find a statistically significant association between lead and blood pressure in females, and available studies of the effects of blood pressure on cardiovascular risks provide the most reliable dose-response estimates for whites in that age range.) The benefits may extend to older males and to nonwhites as well.

The Pirkle et al. and Harlan et al. studies have been published in peer-reviewed journals. A summary of the Pirkle et al. study also was placed in the docket of the phasedown rulemaking (Schwartz, 1984c). Because the papers have only recently been published and have not yet received widespread review by the scientific community, EPA has not relied on estimates of health effects related to blood pressure in deciding on the phasedown rule being promulgated now. These effects will be considered, however, in reaching a decision on a ban. We have included a chapter in this RIA on the potential impacts of the phasedown on blood pressure and cardiovascular disease.

I.B. Need for Government Intervention

Lead in gasoline is a classic example of what economists call a negative externality; individual users of leaded gasoline do not bear all of the costs it imposes on society as a whole. Users of leaded fuel reap short-term savings in the form of lower fuel costs; they also bear higher maintenance costs. Individually, however, they bear only an infinitesimal fraction of the costs of the health and environmental damages caused by their vehicles' emissions of lead and, in the case of misfuelers, other pollutants from poisoned catalysts. This disparity between private and social costs generates an overuse of the good, in this case lead in gasoline, and increases the damages imposed on society.

The need for government intervention to rectify significant negative externalities is well recognized and provides the primary intellectual basis for virtually all EPA regulations. The predominant approach in the United States has been to impose standards that limit the level of the externality (in this case, the amount of lead permitted in gasoline). Market-oriented approaches -- such as pollution taxes or charges on emissions and marketable permits -- are rarely used, although they may be highly efficient means for reducing negative externalities. These alternative approaches are discussed more fully in later sections of this chapter.

Section 211(c)(1) of the Clean Air Act [42 U.S.C. §7545(c)(1)] gives the Administrator of EPA broad authority to "control or prohibit the manufacture . . . or sale of any fuel additive" if its emission products (1) cause or contribute to "air pollution

which may be reasonably anticipated to endanger the public health or welfare", or (2) "will impair to a significant degree the performance of any emission control device or system . . . in general use . . ."

EPA believes that further reductions in the lead content of gasoline are justified by both of the tests under Section 211(c)(1). Lead in gasoline has been shown to raise blood lead levels, which endangers public health, and the misuse of leaded fuel damages pollution control devices, substantially increasing emissions of HC, NO_x, and CO.

I.C. Alternatives to New Regulations

Regulation of lead in gasoline is amply supported both by statutory authority and the significant negative externalities associated with leaded gasoline. In this instance, however, EPA already has regulations in place, so the issue is not whether there should be any regulation at all, but whether the regulations should be tightened. Before proposing stricter rules, the Agency carefully considered alternative approaches that would not require new rules. Three such alternatives -- no change in policy, public education efforts, and stepped-up enforcement against misfueling -- are discussed in this section.

I.C.1. No Change in Policy

Under this approach, the Agency would not change existing regulations and policies regarding lead in gasoline. Lead use would decline over time, as existing vehicles designed to use

leaded gasoline retired and the number of vehicles designed to use unleaded increased. As discussed earlier, however, lead use would decline less rapidly than expected at the time of the previous rulemaking, and the number of misfuelers would grow. Our best estimate is that leaded fuel would not decline below 20 percent of total gasoline fuel use under this scenario.

All of the benefit-cost calculations in later chapters implicitly evaluate this alternative, because it is the baseline from which costs and benefits are measured; by definition, it has zero costs and benefits, and thus zero net benefits in each year. Policies with positive net benefits yield higher net benefits than this alternative. Conversely, policies with negative net benefits are less efficient, in economic terms, than the status quo.

I.C.2. Public Education

Efforts to educate the driving public about the problems caused by misfueling offer potentially useful supplements to current policy. As discussed more fully in Chapter VII, the use of leaded gasoline increases vehicle maintenance costs. Indeed, EPA estimates that the maintenance savings of reducing lead in gasoline would exceed the higher costs of manufacturing unleaded fuel. EPA is undertaking a variety of initiatives to inform vehicle owners of these maintenance costs, as well as the adverse environmental effects of misfueling.

Public education efforts, while useful, are unlikely to significantly reduce the use of leaded gasoline, in large part because retail price differentials between leaded and unleaded

gasoline are high, roughly three to four times higher than the manufacturing cost differentials. (See Chapter 11 for further discussion of this issue.) In addition, the health and environmental costs of using leaded gasoline are externalities that are not borne by individual users of leaded gasoline. Thus, even if the social benefits of reducing lead in gasoline exceed the social costs by a large margin, the strictly private benefits (reduced maintenance costs) to most individual purchasers may be less than their private cost (the retail price differential).

I.C.3. Stepped-Up Enforcement

Federal law makes it illegal for service stations and other gasoline dispensing outlets to put leaded fuel in vehicles requiring unleaded. Federal law does not apply to individual vehicle owners who misfuel, although many states have such laws. In theory, therefore, the misfueling problem could be mitigated by stepped-up enforcement of existing state and federal laws.

Unfortunately, however, massive enforcement efforts would be very expensive, and only partially successful in eliminating misfueling. Moreover, even if it were possible to eliminate misfueling, the serious health effects associated with legal use of leaded gasoline would continue unabated.

Misfueling presents a serious enforcement problem. Currently, almost 100 million vehicles require unleaded fuel, and there are over 100,000 retail gasoline outlets, plus even more private outlets (e.g., private tanks used to fill commercial

fleets). Inspection and Maintenance (I\M) mobile source enforcement programs test the emissions of individual cars, requiring owners to make repairs if their cars exceed emission standards. I/M programs appear to offer the most practical means of enforcement, but less than one-fifth of all vehicles are in areas with such programs.

I/M programs are used in areas to help achieve compliance with National Ambient Air Quality Standards, primarily by encouraging the improved maintenance of vehicles and their emission control equipment. They are an important part of strategies to attain air quality standards, and they produce substantial emission reductions independent of their effects on misfueling. Extending I/M programs to areas already attaining standards would be expensive; a typical program costs about $6.50 per vehicle inspection (U.S. EPA, 1981). By the mid-to-late 1980s, more than 100 million vehicles requiring unleaded gasoline will be registered in areas that do not currently have I/M programs. If we assume annual inspections, the cost of extending I/M programs to cover all unleaded vehicles would be about $650 million per year, which is higher than the estimated cost of reducing lead to 0.10 gplg in all leaded gasoline.

This estimate understates the cost of an I/M approach because it does not include the costs of required repairs, the time spent by owners to have their vehicles inspected, or the higher costs of manufacturing additional unleaded gasoline to meet the demand of existing and potential misfuelers deterred by inspections. These categories of costs are likely to be substantial.

A national I/M program also is likely to miss many misfuelers. Surveys show that while misfueling rates are lower in areas with I/M programs than in those without, they are still significant; the average misfueling rate in I/M areas appears to be about two thirds of the overall national average, but the difference has been shrinking (see Chapter VI). An I/M program aimed specifically at misfueling (e.g., including tests for lead in tailpipes) probably would be more successful, but even an extensive I/M program is unlikely to solve the misfueling problem.

More importantly, I/M programs and other aids to enforcement have no impact on lead emissions from vehicles legally permitted to use leaded gasoline. EPA estimates that in 1983, legal users accounted for about 85 percent of all leaded gasoline sales. Over time, with a decrease in the number of vehicles allowed to use leaded and a rise in the number of vehicles designed for unleaded, that proportion will fall. Even in 1988, however, EPA projects that, in the absence of new rules, roughly two-thirds of the demand for leaded gasoline will be legal. Thus, we estimate that even a fully effective enforcement program targeted on misfueling could not reduce lead emissions from vehicles by more than 30 percent for the period from 1986 to 1990.

I.D. Market-Oriented Alternatives

Market-oriented approaches to environmental protection offer the advantage of allocating control efforts on the basis of marginal control costs, yielding minimum costs for any given level of overall protection. The two major alternatives are marketable permits and emission charges.

I.D.1. Marketable Permits

EPA's current lead-in-gasoline regulations, by allowing "constructive averaging," in effect set up a system of marketable permits for lead in gasoline. Refineries with lower-than-average costs for producing octane without lead are allowed to reduce their lead content below the limit of 1.10 gplg and to sell the excess lead rights to refiners with higher-than-average costs, who may then produce leaded gasoline with lead content above 1.10 gplg. This approach offers clear advantages over a uniform standard of 1.10 gplg. Lead is reduced by the same amount, but costs are lower. For those refineries that lower the lead content of their gasoline, the sale of excess lead rights more than offsets higher manufacturing costs. Conversely, for those refineries that buy lead permits, the savings in manufacturing costs more than offset the purchase price of the permits. Consumers presumably also benefit when these lower costs translate into lower gasoline prices.

The current system is working well. Based on confidential reports filed with EPA by the industry, about 73 percent of refiners participated in lead trading during the second quarter of 1984. During that same period, about 14 percent of the total amount of lead was traded. Thus, the lead trading system initiated in 1982 appears to be a major success story for introducing market principles into environmental regulation.

Under the new rule being promulgated, refineries will be allowed to continue constructive averaging through 1985, until the 0.10 gplg standard takes effect, Once the 0.10 gplg standard

applies, however, inter-refinery averaging no longer will be allowed, because that standard is intended to protect those few engines that may need a small amount of lead (or some other additive) to protect against premature valve-seat recession. Although EPA believes that the risk is small and that minor fluctuations around this level over time will not damage engines (hence, the rule permits quarterly averaging by refiners), constant use of fuel with a lower lead content might damage a few engines. For a trading system to work, some refiners must produce gasoline with a lower lead content than permitted by the rules, but that could cause damage to some engines because many vehicle owners consistently purchase fuel produced by the same refinery. Moreover, the refinery cost savings from allowing trading at 0.10 gplg would be trivial, as the amount of lead involved will be so small.

EPA has proposed a rule change that will allow refineries greater flexibility in meeting lead-phasedown goals without any detrimental effects on health, the environment, or those engines that may need protection against valve seat recession. Under this proposal, refiners would be able to save some of the lead that they are allowed to use during 1985 for use in 1986 or 1987. Thus, for example, if a refinery produced five million gallons of leaded gasoline at 0.90 gplg during the second quarter of 1985 (when the limit will still be 1.10 gplg), and did not sell the extra lead rights to other refineries, it could “bank” one million grams of lead ([1.10 - 0.901 x [5 million] = 1 million) for use in 1986 or 1987. Similarly, a refinery that

produced five million gallons at 0.40 gplg during each of the last two quarters of 1985 (when the limit will be 0.50 gplg) also would have one million grams available for supplemental use in 1986 or 1987. These banked lead rights could be used as desired by the refinery in 1986 or 1987; it could, for example, produce 10 million gallons at 0.20 gplg (rather than the 0.10 gplg standard that will apply then), or sell the million grams to another refinery with higher marginal production costs. This change will increase refineries' flexibility (thus lowering production costs) without increasing the amount of lead released to the environment.

I.D.2. Pollution Charges

The second market-oriented alternative to mandatory lead content reductions would be to impose a charge on gasoline based on its lead content. Ideally, the charge per gram of lead should equal the marginal external damage caused by a gram of lead in gasoline, thereby internalizing the health and environmental damages caused by lead use. With such a charge, the price of leaded gasoline would rise, encouraging consumers to switch to unleaded. Refiners would have an incentive to reduce the lead content of gasoline to the point where the marginal cost of additional reductions would equal the charge. As with marketable permits, refiners would end up producing leaded gasoline with different lead levels, depending on the refiners' marginal costs.

Despite these attractive features, a charge has several drawbacks and faces some obstacles. The primary drawback is the same as identified earlier with regard to permits: leaded gaso-

line that contains much less than 0.10 gplg will not protect those few engines that may need valve lubrication. The charge might be modified in one of two ways to address this problem: it could be levied only on lead in excess of 0.10 gplg, or EPA could set a minimum lead-content rule for leaded gasoline. In either case, EPA believes that an appropriate charge would be high enough to drive virtually all leaded gasoline to the minimum level of 0).10 gplg. If that happened, the cost-minimizing advantage of this approach would be lost; the effect would not be materially different from a uniform standard of 0.10 gplg.

Even with these limitations, a charge would still offer some advantages. It would give refiners additional flexibility over the short run while they made needed adjustments in equipment and operating practices. If the charge were levied on all lead in gasoline (not just that above 0.10 gplg), it also would provide an additional disincentive for misfueling with 0.10 gplg gasoline by raising its price. In addition, it would discourage the use of leaded gasoline in other vehicles unless required to prevent valve-seat recession.

One potential obstacle to levying a charge to reduce lead in gasoline is that EPA may not have the necessary statutory authority. If new legislation, were needed, it almost certainly would be accompanied by extensive debate about both the general principle of using charges or taxes to control environmental hazards and the specific tax rate to be set for lead, which could cause significant delays in solving a serious environmental problem. EPA believes that the foregone benefits caused by such a delay would outweigh any potential efficiency gains.

I.E. Alternative Standards

EPA has considered a wide range of alternative phasedown schedules. These alternatives included both different lead levels and different effective dates. Chapters II through VII present the methods used to estimate the costs and benefits of these alternatives. In each chapter, we survey the effects in question and discuss the methods used to derive monetized estimates. Each chapter contains year-by-year estimates of the relevant effects for three sets of rules, as summarized in Table I-1: (1) the primary Proposal made in August (0.10 gplg in 1986), (2) the sample Alternative schedule discussed in the August Notice of Proposed Rulemaking, and (3) the Final Rule. (Because the 1985 standard only applies to the second half of the year, our estimates for that year are for half a year.) In all cases, the costs and benefits depend on the amount of misfueling eliminated. The sample benefit estimates presented in Chapters III-VII assume that all misfueling is eliminated. The cost estimates in Chapter II assume that the amount of misfueling declines linearly with the level of the standard, from 100 percent of current levels at 0.50 gplg or above, to zero with a ban. A range of alternative assumptions about misfueling, and their impacts on the costs and benefits, is explored in Chapter VIII.

Throughout this document, we use a 10 percent real discount rate to compute present values, and costs and benefits are expressed in 1983 dollars. The choice of a discount rate is controversial, the subject of much debate among economists. Generally, the higher the discount rate, the lower the net benefits, because costs usually are incurred sooner than benefits.

TABLE I-1. Alternative Phasedown Schedules

Phasedown Schedules	Lead Limit (gplg)			
	1985*	1986	1987	1988
Proposed	N.A.	0.10	0.10	0.10
Alternative	0.50	0.30	0.20	0.10
Final	0.50	0.10	0.10	0.10

* 1985 limit applies as of July 1.

The rate we use, 10 percent, is at the high end of the ranges typically discussed; many economists would regard a much lower rate as more appropriate. We have used 10 percent to be consistent with guidelines from the Office of Management and Budget (U.S. OMB, 1981). We also considered presenting sensitivity analyses using lower rates, but decided not to do so in the interests of simplifying the presentation; lower discount rates would increase our estimates of net benefits.

Chapter VIII summarizes the costs and benefits of the final phasedown rule and compares them to a wide range of alternative schedules. It also examines the impact of different levels of misfueling under each of the alternatives. Regardless of the assumption about misfueling, the schedule contained in the final rule appears to offer the highest net benefits of the options considered. Chapter VIII also examines the costs and benefits of the recent proposal to allow banking of lead rights.

CHAPTER II

COSTS OF REDUCING LEAD IN GASOLINE

Since the 1920s, petroleum refiners have added lead to gasoline as a relatively inexpensive way to boost octane. To meet octane demands with reduced amounts of lead, refiners have a variety of options, the most important of which is to perform additional processing of gasoline components in reforming and isomerization units. In addition, refiners also may employ additives other than lead, such as alcohols or MMT, or they may purchase additional high-octane components, such as aromatics or butane. At the margin, however, all of these alternatives are more expensive than lead for making octane, so the cost of producing gasoline will rise under the rule being promulgated.

These higher refining costs will comprise virtually all of the costs of the rule. (The one potential exception, damage to the valve seats of some engines with unleaded gasoline, applies only to the possible ban, not to the final phasedown rule; the valve-seat issue is discussed in Chapter VII.) To estimate those costs, we used a model of the refining industry originally developed for the Department of Energy (DOE).

Section A of this chapter explains why we estimated the social cost of the rule using predicted changes in manufacturing costs, rather than changes in retail prices. Section B provides an overview of the DOE model and refinery processes generally. Section C discusses the input data and assumptions used for our projections and presents year-by-year cost estimates for reducing lead in gasoline. Section D reports the results of numerous sensitivity

analyses conducted to test the robustness of the cost projections. Finally, Section E, for illustrative purposes, examines the impact on costs of the proposal to allow "banking" of lead rights during 1985 for use in 1986 and 1987.

II.A. Price versus Cost Differences

The costs we are measuring are the social costs of reducing lead in gasoline. Our estimates of these costs are based on estimates of changes in the costs of manufacturing gasoline (and other petroleum products). In the long run in a competitive market, the change in manufacturing costs is likely to be fully reflected in changes in the amounts paid by consumers. In the short run, however, the total amount paid by consumers may be less than or greater than the change in manufacturing costs, depending on supply and demand elasticities and other factors. The divergence between the change in manufacturing costs and the change in the amount paid by consumers, though, will be comprised mostly of transfers from one segment of U.S. society to another, rather than real social costs (or savings).

By real social costs, we mean the costs of real resources that are used to comply with the rule (i.e., he extra energy, capital, labor, etc. that is needed to meet the tighter lead standard). If retail prices rise less than costs in the short run, then the losses to refiners (and their shareholders) will be offset by gains to consumers. If prices rise more than costs, then the losses to consumers will be offset by gains to producers. In either case, the net cost to all citizens, including shareholders, is just the real resources used in meeting the rule. Thus,

so long as the rule does not affect the costs of distributing or selling the product, the change in manufacturing costs is a good approximation of the social cost of the rule.

The DOE model estimates that at current lead levels (1.10 gplg) the marginal manufacturing cost differential between unleaded and leaded regular grades of gasoline is less than two cents per gallon. Retail prices, however, diverge by an average of about seven cents per gallon (Weekly Petroleum Status Report, 1984, various issues). Some commenters suggested that the gap between the model's estimate of the cost differential and current retail price differentials indicates either an error in the model, or that it would be more appropriate to use the retail price difference as the measure of social cost. Both arguments have some superficial plausibility, but fail to withstand careful analysis.

The much larger retail price differential appears to reflect marketing strategies within the industry rather than errors in the model's cost estimates. Price differentials at the wholesale level are far smaller than the retail price spread, and discussions between EPA and petroleum refiners confirmed that manufacturing cost differences between unleaded and leaded gasolines were about two cents per gallon, and that inter-refinery trades occurred at a differential of two to two-and-a-half cents per gallon. Also, the differential between the spot prices of unleaded and leaded regular gasolines in barge quantities has been between one and four cents per gallon over the last several years. At the end of 1983, wholesale price differentials in the Gulf of Mexico were about

three cents per gallon (Platt's Oilgram).

The additional price gap between unleaded and leaded gasolines at the retail level does not appear to reflect incremental distribution or other costs. Both types of gasoline are distributed through the same network of pipelines, terminals, barges, and tank trucks, and similarities in sales volumes suggest that differential inventory carrying costs do not apply.

Two possible explanations of the price differential have been offered. The first is that service stations use their lowest cost gasoline as the "fighting grade," prominently advertising it at a low price to attract customers (including those purchasing another grade). If this explanation is correct, unleaded regular may become the new fighting grade, as it will be less expensive to manufacture 87 octane unleaded than 89 octane leaded with 0.10 gplg. (To produce 89 octane leaded gasoline at 0.10 gplg, the octane level of the gasoline must be boosted to over 88 before the lead is added.) In that case, the price of unleaded gasoline would be lower than leaded. Service stations may continue to use leaded gasoline as the fighting grade, however, because it is the lower-volume product.

The second possible explanation is that retailers are engaging in price discrimination, taking a larger margin on the grade (unleaded) with less elastic demand. This explanation presupposes that individual retail outlets do not face the flat demand curves of a perfectly competitive market, but rather that gasoline sales are characterized by monopolistic competition. Under this scenario,

the individual station owner is a geographic monopolist, i.e., the only retailer at that location. Thus, the demand curve facing the operator is not perfectly flat, but if the price is too high, customers will drive to a competitor.

Under such a market structure, prices deviate from marginal costs, roughly in inverse proportion to each product's demand elasticity, but firms do not reap economic profits (i.e., profits beyond a normal return on capital) because of free entry of new competitors. If leaded customers are more price sensitive (perhaps because they may have lower incomes and are more willing to search out lower-price stations, or because the leaded price is displayed more prominently), their demand elasticity (at the level of the individual service station) will be higher than the elasticity of demand for unleaded customers. As a result, price discrimination will cause the operating margin (price minus wholesale cost) to be lower on leaded than on unleaded. If this explanation is correct, the 0.10 gplg standard should greatly reduce the retail price differential between unleaded and leaded gasoline, though it would not necessarily reverse it.

Whichever of these explanations is correct, the difference in manufacturing costs, rather than in retail prices, would represent the social cost (as conventionally defined in cost-benefit analysis) of reducing lead in gasoline. This conclusion would fail to hold only if it were possible to show that reducing the lead content of gasoline would increase the real costs of distributing gasoline. No such evidence is available.

II. B. The Refinery Model

This section discusses the DOE refinery model used to estimate costs. Section II.B.1 provides a brief introduction to the model, while Section II.B.2 gives an overview of refining processes and how they are represented in the model.

II.B.1. Introduction to the DOE Model

The DOE refinery yield model uses a linear programming framework to simulate refining operations (sU.S. DOE 1984b). The model represents individual refinery units and their inter-relationships using a series of about 350 equations. Given a set of input assumptions and constraints, it finds the least-cost method of producing any specified set of final products. (The model also can be run to maximize profit given final-product prices.)

In addition to estimating the total cost for a set of product demands, inputs, and constraints, the model generates detailed and useful information on important aspects of industry operations, including the rates at which costs change (the shadow prices of the constraints) and the extent to which various types of equipment are utilized under different scenarios.

The model is based on many similar models developed and used widely by the petroleum refining industry for its own planning purposes. The refining industry was among the first to make extensive use of linear programming. The DOE model has been used by EPA for several years in its analyses of the impacts of regulations on the petroleum industry and on petroleum product purchasers. In the 1982 lead-in-gasoline rulemaking, EPA used the

model to predict the costs of the rule and to predict the value of lead rights under the trading program. Although it is impossible to verify directly the cost estimates made then, it is important to note that the actual prices of traded lead rights were slightly lower than predicted by the model, indicating that it did not underestimate the value of lead to refiners as an octane booster.

The model also has been used by DOE for several purposes, including: evaluating crude mixes for the selection of storage sites for the Strategic Petroleum Reserve, assessing the impacts of petroleum disruptions on product supplies, and evaluating the industry's capability to respond to changes in feedstock quality or product demands.

DOE subjected the model to two verification checks, and found that for 1982 it generated a product slate very close to the actual one. It also closely predicted refinery-gate price differentials. It is also important to note that these verifications covered a period before EPA initiated interrefinery averaging of lead use, indicating that the accuracy of the model's predictions is not dependent on the trading of lead rights. More recently, DOE verified the model using 1983 data (U.S. DOE, 1985).

II.B.2. Overview of Refining Processes

So the reader can better understand how the model works, we provide a brief description of refining processes. Figure II-1 is a schematic diagram of a very simple refinery, often called a topping plant, which processes low-sulfur crude oils. A complex

refinery contains distillation units and other types of processing units; Figures II-2, II-3, and II-4 present schematics of such refineries. (The model contains considerably more detail than even these exhibits indicate.) In any given refinery, these different process “units” are assembled into final structures that accomplish different but related purposes. The basic similarity of process units makes it possible to model refineries.

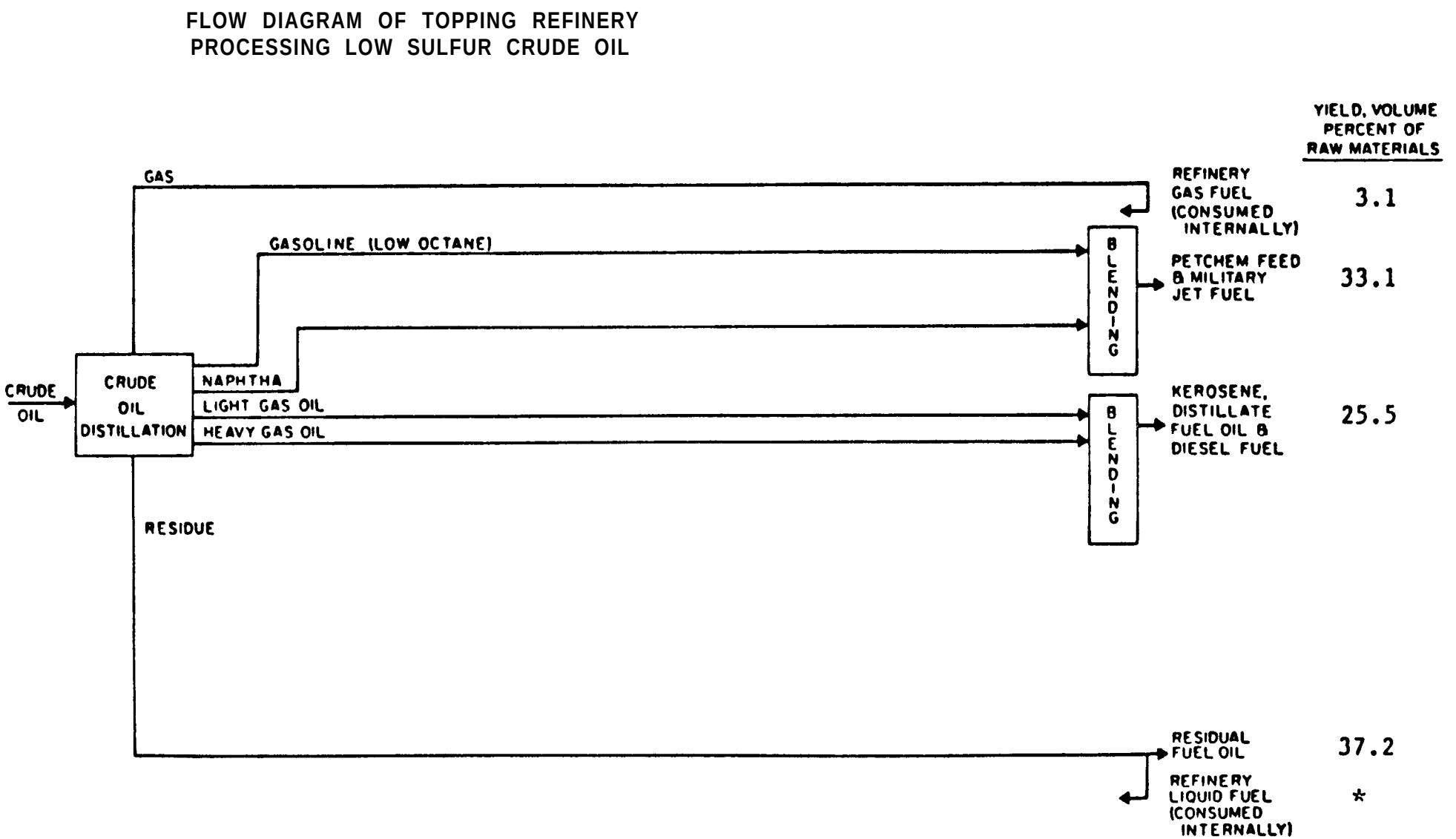
Basically, the model is a system in which the various units that make up all types of refineries are represented by the boxes in the schematics. Each unit uses inputs (crude oil or an intermediate product) to make one or more intermediate or final products. The exact types and quantities of the product(s) made are functions of the properties of the inputs of each unit and the process that each performs. Fuel and utilities (e.g., electricity and steam) are consumed, and an operating cost is incurred for each operation. A capital cost may or may not be charged, as appropriate to the particular analysis being performed. Table II-1 provides a summary of the basic types of refinery processes. Appendix A contains a more detailed description of processing operations.

Because all refineries are made up of these building blocks, the smallest structure in the model is a process unit rather than a plant. The individual functions that are modeled are the inputs and outputs from each type of unit. The model is made up of refinery units, each of which has an output (or a series of products), the quantity of which is a function of the material that the unit is "fed." Some refinery process units incur some costs that vary with how intensely they are run -- called “severity”. In the case

Figure II-1

Schematic Diagram of a Simple Oil Refinery

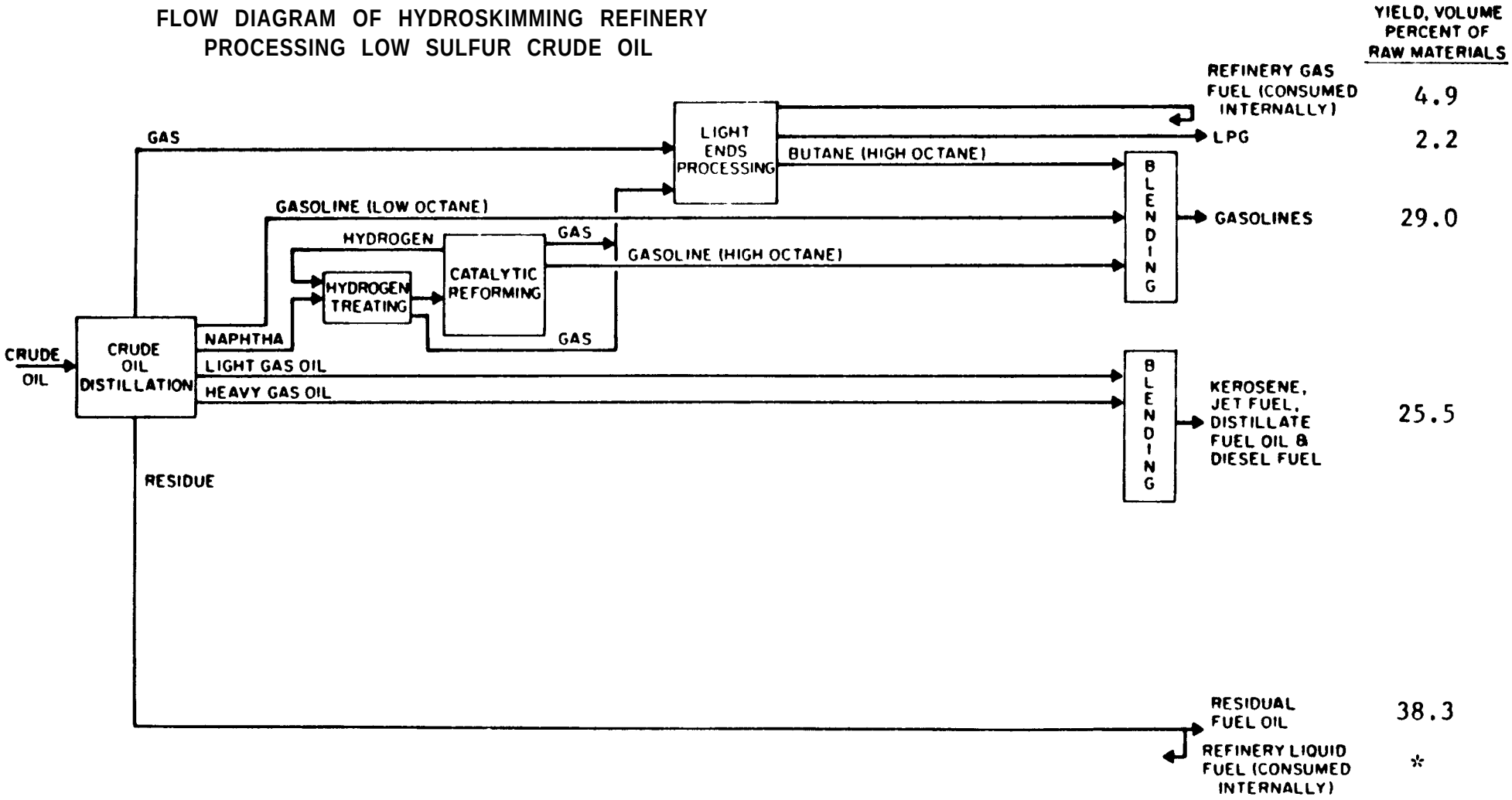
(Topping Plant)



*Included with gas fuel

Figure II-2

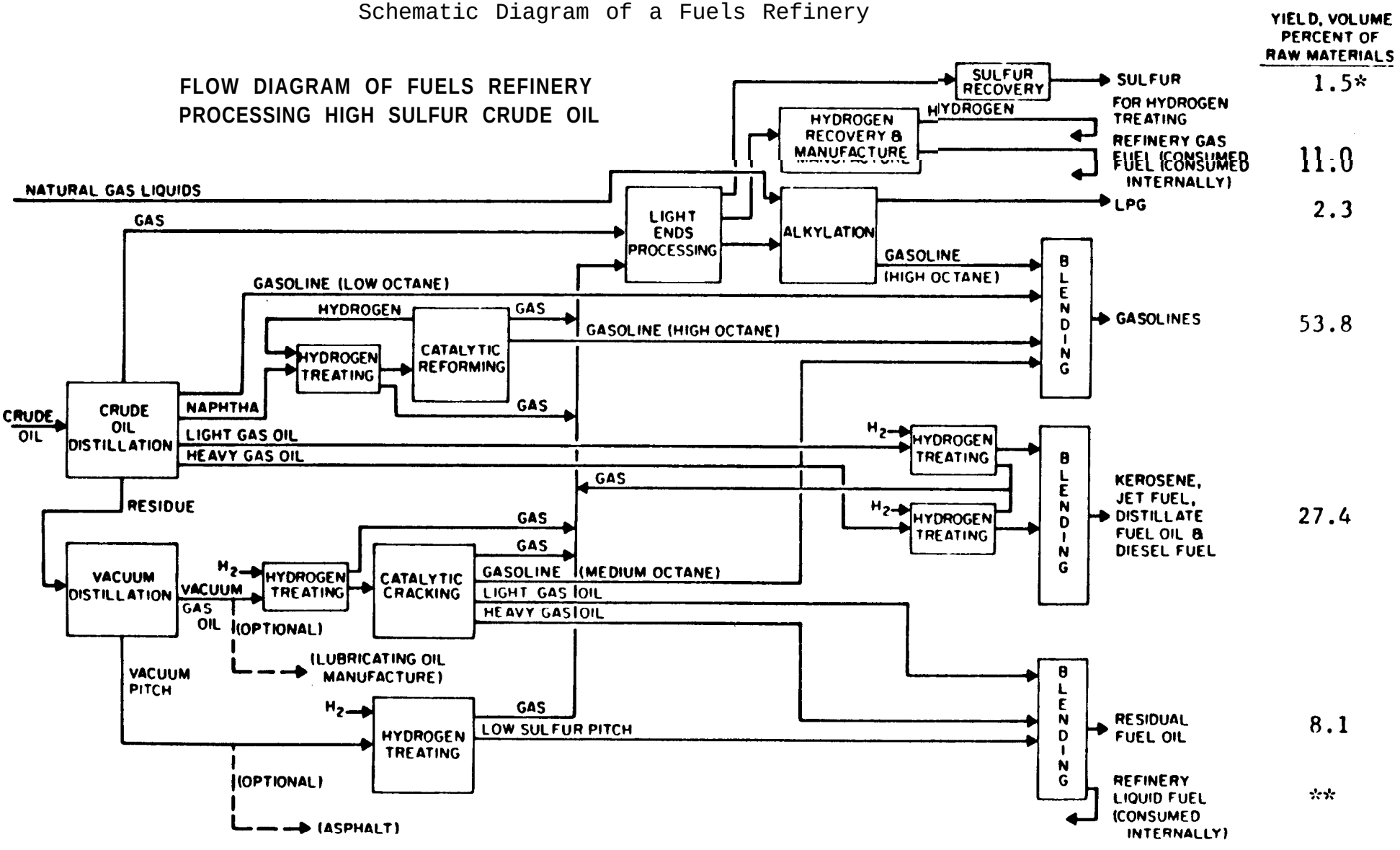
Schematic Diagram of a Hydroskimming Refinery



* Included with gas fuel

Figure II-3

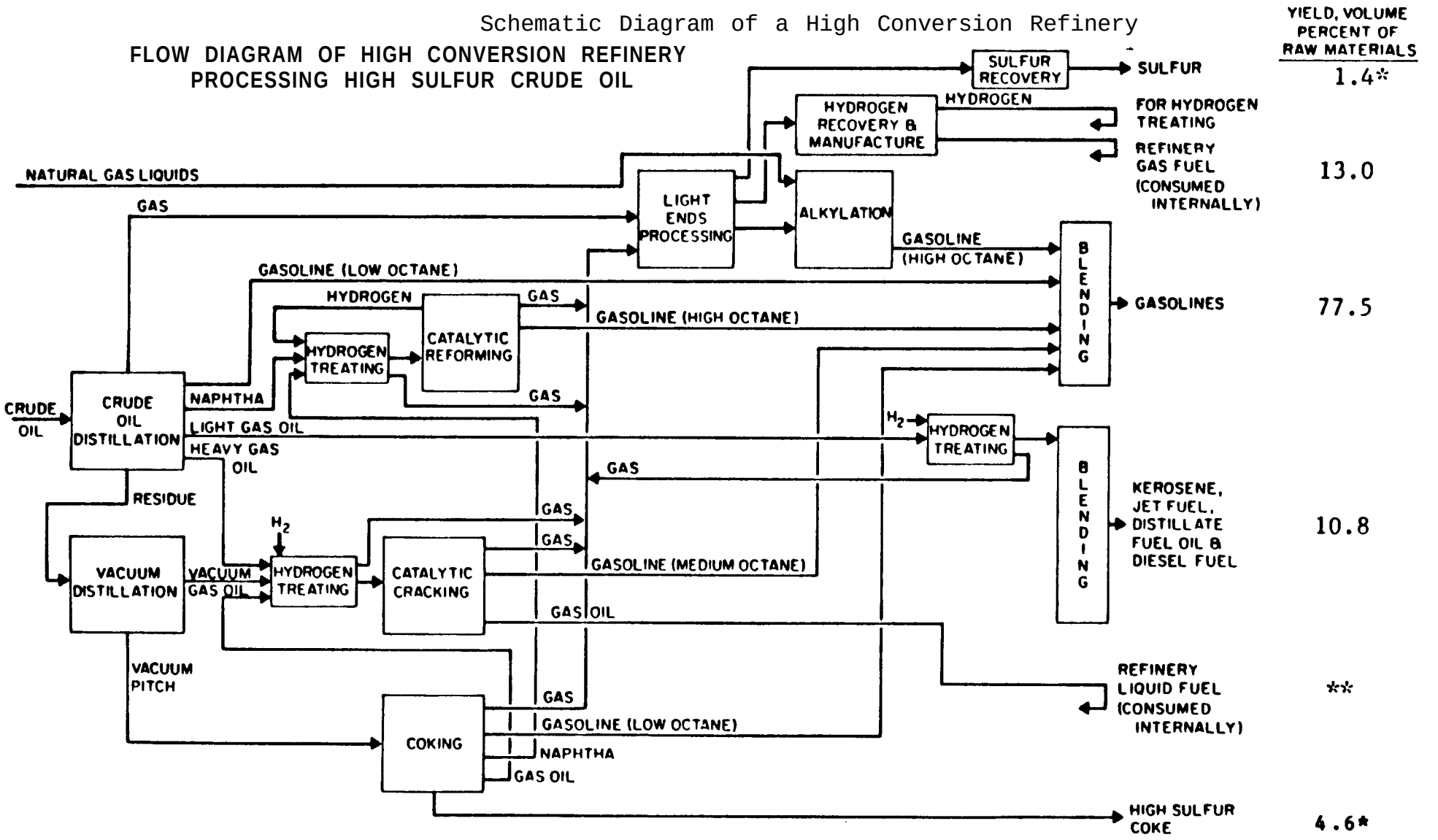
Schematic Diagram of a Fuels Refinery



* Percent by weight
** Included with gas fuel

Figure II-4

Schematic Diagram of a High Conversion Refinery



* Percent by weight

** Included with gas fuel

TABLE II-1. Functional Characterization of Refinery Processes

SEPARATION	ALTERATION (CONVERSION)
Separation on the Basis of molecular Weight	A. Conversion on the Basis of Molecular Weight
Distillation (atmospheric and vacuum fractionation of crude oil, naphtha splitting, depropanizing, stabilization)	Thermal cracking (visbreaking, coking)
	Catalytic cracking
Absorption (recovery of olefins from catalytic cracked gas, recovery of propane from natural gas or hydrocracked gas)	Hydrocracking
	Alkylation
	Polymerization
Extraction (deasphalting of feedstock for lubricating oil manufacture or for catalytic cracking)	
Separation on the Basis of molecular Structure	B. Conversion on the Basis of Molecular Structure
Extraction (recovery of benzene, toluene, and zylenes from catalytic reformate, removal of aromatics from lubricating oil feedstock)	Catalytic reforming (benzene, toluene, and xylene manufacture and octane improvement)
Crystallization (dewaxing of lubricating oils, recovery of paraxylene from mixed xylenes)	Isomerization (normal butane to iso for alkylation, normal pentane and hexane to iso for octane improvement)

TREATMENT TO REMOVE IMPURITIES
Hydrogen treatment (hydrotreating)
Caustic treatment (Merox, Bender)
Clay treatment (of lubricating oils)
Acid treatment

of some important octane-improvement processes (particularly reformers), the higher the severity, the higher the octane produced, but also the lower the "yield" (the amount of gasoline material produced per unit of input). In general, the more the process unit is used, the higher its marginal cost of processing the next increment of feed will be.

In the model, all processes consist of a series of linear relationships that describe the process output and operating cost, given specified inputs and a set of operating conditions. The relationships are stored in the model in the form of a process data table. (Table II-2 shows an example of such a table.) Each column in this process table represents the processing of a specific type of crude oil and each row represents a specific input or output stream, fuel, utility consumption etc. For example, the model specifies that as one barrel of Saudi light crude is processed, a mix of 15 intermediate streams is created. The operation consumes fuel, power, steam, and capacity, and incurs variable operating costs of 9.3 cents per barrel.

For producing high-octane components for low-lead or unleaded gasoline, four types of "downstream" units (i.e., units that process outputs from the crude distillation process) are particularly important: fluid catalytic cracking (FCC) units, isomerization units, alkylation units, and reformers. Each of these units enhances the octane of a different stream of intermediate products.

Finally, after all processing is complete, the refinery ends up with numerous process output streams that are blended together to produce final, salable refined products. This activity is

TABLE II-2. Sample Process Data Table from Refinery Model: Yields and Operatinq Cost Coefficients for Crude Distillation-Unit

	Crude Oil Type		
	Saudi Arab Light	Saudi Arab Heavy	Mexican Maya
Saudi Arab light	-1		
Saudi Arab heavy		-1	
Mexican maya			-1
Capacity factor	1.0	1.0	1.0
Yields (Fraction of Intake)			
Still gas	0.001	0.OO1	0.001
Propane	0.003	0.003	0.003
Isobutane	0.002	0.002	0.002
Normal butane	0.013	0.015	0.009
Lt. st. run (C5-175) low oct.	0.051		
Lt. st. run (C5-175) int. oct.	0.040	0.035	
Lt. naph. (175-250) parf.	0.070	0.060	0.025
Lt. naph. (175-250) intro.			0.025
Naph. (250-325) parf.	0.050	0.044	0.010
Naph. (250-325) intro.	0.020	0.011	0.050
Hvy naph. (325-375) parf.	0.020	0.020	0.005
Hvy naph. (325-375) intro.	0.020	0.014	0.030
Kero. (375-500) jet fuel quality	0.115	0.090	0.070
Kero. (375-500) other	0.015	0.005	0.040
Dist. (500-620) hi sulfur	0.130	0.090	0.100
Hy gas oil (800-BTMS) (2.0% S)	0.180	0.180	0.105
Asph., very hi sul.(4.3% S)	0.143	0.300	0.350
Operating Cost Coefficients (Per Barrel of Throughput)			
Fuel, fuel oil equivalent	-0.021	-0.022	-0.020
Power, KWH	-0.6	-0.6	-0.6
Steam, LB	-60.7	-63.4	-57.9
Other var. op. cost, $	-0.092	-0.093	-0.092
Capital charge		varies	

Note: The negative signs (-) indicate consumption of crude oil, fuel oil, power, steam, etc.

represented in the model by product blending units. The blending units contain quality data for all refinery streams and quality specifications for final products. The components are then combined by the model such that the qualities of the blended mixes meet the minimum requirements of product specifications.

The refinery model can be operated in either of two modes -- minimum cost or maximum profit. It can constrain product quantities and compute a minimum cost solution. (This is useful for analyzing the country as a whole or large refining regions in which aggregate demands can be forecast.) Alternatively, the simulation can vary product quantities to maximize profits at preselected prices.

The principal reason to use computer models to simulate petroleum operations is to measure differences between alternative scenarios, and thereby estimate the changes in petroleum activities when some conditions change.

It is important to note that the procedure used does not simply find the optimal way to make up the difference between the current lead level and the lower level. Rather, subject to constraints discussed later, we first run the model at the current lead standard and find the costs of meeting that standard; then we repeat the process, finding the cost of making gasoline at the O.10 gplg standard. The <u>difference</u> between the cost of producing gasoline under the current standard and the new standard is the cost estimate of the rule change.

This approach minimizes the risk of error in the cost estimate, because if the model mistakenly assumes some flexibility that the

industry does not have, it would reduce the estimated cost of meeting both the current and the new standard. The difference between the costs is only affected if the overoptimistic assumption generates greater savings in one case than in the other case, and even then the size of the error is only the difference in overoptimization, not the full impact of the overoptimistic assumption. EPA believes that this approach to using the model adds considerable confidence to the results.

The refinery linear programming model assumes (when unconstrained) that the least expensive refinery equipment is used to meet both the current and the proposed gasoline lead standard. In a competitive market, this is a reasonable assumption, because if one refiner's marginal costs are lower than competitors', that refiner would be expected to increase market share. As its market share increased, its utilization of refinery equipment would increase, particularly of octane-making equipment, and therefore the refiner's marginal cost of octane would increase to the level of its competitors. A new equilibrium would be reached with some market share having shifted, but not necessarily a large share. The fact that a linear program gives the same results as a competitive industry had long been known and used in economics (see, for example, Baumol, Economic Theories and Operations Analysis, Prentice Hall, 1961).

The gasoline marketing system, which already moves millions of barrels of gasoline per day in pipelines around the country, helps assure that the more efficient producers can get their product to market. In addition, the industry has a long tradition

of trading components and products, which also results in a more efficient allocation of feedstock-to-equipment. A competitive market and an increasing marginal cost of octane for all refiners means that the model simulates the industry reasonably accurately. The verification tests for 1982 showed the model accurately predicting refinery products and prices.

II.C. Base-Case Assumptions and Cost Estimates

Refineries are complex operations with multiple inputs and outputs. The costs of producing any product depend in part on the quantities of other products produced and their prices. For example, the cost of producing gasoline depends partially on how much residual oil is sold and its selling price. The more residual oil is sold and the higher its price, the lower the cost of producing gasoline. As a result of such joint costs, cost estimates for a change in any one product must consider the full range of refinery operations.

To estimate the costs of reducing lead in each year, we first ran the DOE model in its cost-minimization mode with the constraint that lead use not exceed 1.10 gplg (the current limit), and calculated the costs of the resulting solution. We then reran the model at lower lead limits (varying from 0.50 gplg to zero, depending on the year) and recalculated the costs; our cost estimates are the differences between the two runs. At both 1.10 gplg and the lower levels, the model had to meet specified product demands.

In using the refining model, it is necessary to supply it with the values of many parameters. Section II.C.1 describes the

key input assumptions used in making our base-case estimates. Section II.C.2 presents the resulting year-by-year cost estimates based on those inputs. Section II.D describes the results of multiple sensitivity analyses conducted to test the robustness of our conclusions.

II.C.1. Base-Case Parameter Values

The key assumptions input to the model can be divided into two general categories: gasoline demand and quality constraints, and refinery operations. Each category is discussed below.

II.C.l.a. Gasoline Demand

Demand for gasoline in 1983 was about 6.6 million barrels per day, or a little over 100 billion gallons per year. To project gasoline demand in future years, we examined a 1983 study by Data Resources Incorporated (DRI) and projections from DOE's Energy Information Administration (1983), and adjusted them upwards to reflect smaller projected gains in fuel economy.* We project that gasoline demand will fall to about 6.5 million barrels per day in 1988, because of expected improvements in the average fuel efficiency of vehicles in use, but then will level off in 1990 as increases in vehicle miles travelled balance improvements in miles per gallon. Since we made our original projections, the DOE projections have been revised upward to about our level (U.S. DOE, 1984 Annual Report to Congress).

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* Compliance with the rule is harder and more costly with higher gasoline demand.

We projected the split in demand between leaded and unleaded gasoline in two ways. First, we fit linear and logistic regressions to the monthly leaded-unleaded split documented in the Monthly Energy Review (from the Department of Energy), using time as the explanatory variable. We also regressed the thirteen-month moving average, to remove seasonal and random variation. These all suggested an unleaded share of 67.5 percent (plus or minus 0.6 percent at a 95 percent confidence level) of the gasoline market in 1988. Our vehicle fleet model (described in Appendix B), using historic scrappage rates from DRI, predicted essentially the same unleaded share.

We also had to estimate what portion of leaded demand is due to "misfueling", as we expect the rule to have a major impact on the misuse of leaded gasoline in vehicles equipped with catalysts. Deliberate misfueling (i.e., the use of leaded gasoline in vehicles designed for unleaded) appears to occur because leaded gasoline is both cheaper and higher in octane than unleaded gasoline. Several EPA and private studies have showed that widespread misfueling has slowed the decline in lead emissions significantly, and challenged the assumption that leaded gasoline would soon be eliminated because of lack of demand. According to a 1983 survey by EPA (U.S. EPA, 1984e), the current misfueling rate of light-duty vehicles designed to use unleaded gasoline is about 15.5 percent. (This figure was obtained after making certain adjustments; see Chapter VI for more details.) The 1982 survey found a 13.5 percent misfueling rate (U.S. EPA, 1983a). A panel study (Energy and Environmental Analysis, 1984) in which consumers kept diaries of

their gasoline purchases indicates that misfueling is even more widespread, though many people misfuel only occasionally.

We used the 1983 survey data to estimate misfueling rates by age of vehicle (the older the vehicle, the higher the misfueling rate) and by whether or not the vehicle was in an area with an Inspection and Maintenance (I/M) program (vehicles in I/M areas misfuel less). We also adjusted for the fact that older vehicles travel fewer miles per year, on average. These adjusted rates were then applied to our fleet model (described in Appendix B) to estimate the demand for leaded gasoline by misfuelers. Our estimates of misfueling did not influence our estimate of total demand for leaded gasoline (which was based on a statistical analysis of historical trends, as discussed earlier), only the split in leaded demand between misfuelers and legitimate users.

The model requires further disaggregation of unleaded demand into the demand for premium and regular. In our base case we assumed that the demand for premium unleaded would comprise 25 percent of the total demand for unleaded gasoline. This is up slightly from the current level of 24 percent.

Table II-3 presents our year-by-year estimates of gasoline demand, both with and without misfueling. Note that we project that total demand for leaded gasoline will decrease, but misfuelers' demand (in the absence of further regulation) will increase in both absolute and proportional terms.

Refining costs depend on the octane level of gasoline, as well as the split between leaded and unleaded. For each of the three

grades of gasoline (leaded, unleaded regular, and unleaded premium), we assumed that octane levels would remain at their current averages, which are slightly higher than 87 octane for unleaded regular, slightly higher than 91 octane for unleaded premium, and a little above 89 octane for leaded gasoline. These octane ratings reflect the average of two measures, "motor octane" and "research octane." We constrained the model to meet or exceed all three octane measures in current gasoline.

For 1986 and subsequent years, we assumed that the regulations would reduce misfueling. In these runs we assumed that half of the misfuelers do so strictly for price, but that the others also want the higher octane provided by leaded regular. That is, we assumed that of the potential misfuelers deterred by the price change resulting from the rule, 50 percent would switch to regular unleaded and the other 50 percent would require unleaded gasoline averaging 89 octane. (The latter could be either an intermediate grade of unleaded, or a mixture of regular and premium unleaded.) Thus, we assumed that deterred misfuelers would demand 88 octane unleaded, on average. Because 0.10 grams of lead only adds about one octane number to a gallon of gasoline, 89 octane leaded gasoline must start with an unleaded gasoline with a "clear-pool" octane rating rating slightly above 88. Therefore, our cost estimates at 0.10 gplg are not very sensitive to assumptions about the amount of misfueling eliminated; indeed, the costs rise slightly with the misfueling rate.

We also had to specify the split between domestic and imported gasoline supply. Over 4 percent of gasoline is now imported

TABLE II-3. Year-by-Year Estimates of Gasoline Demand (billions of gallons per year)

Rule	1985	1986	1987	1988	1989	1990	1991	1992
With Misfueling								
Leaded	40.2	37.5	34.9	32.4	29.8	27.6	25.2	24.3
Unleaded	60.3	62.8	65.1	67.3	69.5	71.4	73.8	74.7
Without Misfueling								
Leaded	32.1	28.8	25.6	22.4	19.2	16.4	14.9	13.4
Unleaded	68.5	71.5	74.4	77.2	80.1	82.6	84.1	85.6
Total	100.6	100.3	100.0	99.6	99.3	99.0	99.0	99.0

and over 60 percent of imports are unleaded, but our base case constrained the model to meet all demand for gasoline using domestic refineries. (For modeling purposes, “domestic refineries” include those in Puerto Rico and the Virgin Islands.)

We also constrained the model to meet other important characteristics of current gasoline, including its Reid Vapor Pressure (RVP). Because certain high-octane blending components (such as butane) and additives (such as alcohols) have high RVPs, this constraint raised the estimated cost of meeting the rule.

In addition to gasoline demand, we also had to specify demands for other products. These were based on a study done for DOE’S Energy Information Administration (Decision Analysis Corporation, 1983). In most cases, we constrained the model to produce these quantities domestically. In a few cases, where there are well established and large international markets (e.g., for residual oil), we allowed the model to slightly increase or decrease imports at fixed prices if that was the lowest-cost solution.

II.C.l.b. Assumptions About Refinery Operations

The DOE model requires that many individual parameters be specified for refinery operations. Here we discuss only the most important of those assumptions.

We based our estimates of available capacity on published tabulations of existing equipment and announced projects that will be completed in the relevant years. Table II-4 presents the estimates of available “stream-day” capacity used in the model. For such capacity, we did not include a capital charge for its use

in meeting tightened lead rules because the cost of that equipment is already "sunk"; it does not vary with the standard. In our estimates for 1985, 1986, and 1987, we constrained the model to use this existing capacity because it takes two to three years for refiners to plan, obtain permits for, and complete major construction projects. (There are "debottlenecking" and upgrading actions that refiners can take to improve their ability to make octane in as little as six months, other steps that might take a year, etc. We have ignored such changes and, therefore, somewhat underestimated industry capacity.) For the later years, however, we allowed the model to determine whether construction of additional capital equipment was economical. In those later years, the model predicted that costs would be slightly lower if new equipment (primarily isomerization units) were added. This new equipment, added in response to the rule, was included in the cost estimates using a real capital charge rate of 15 percent (which corresponds to a discount rate of 10 percent and an equipment lifetime of just under 10 years).

Because of maintenance and breakdowns, equipment cannot be used to its full daily capacity, called "stream-day" capacity in the refining industry. To account for this fact, we limited capacity utilization to 90 percent. This figure reflects tight, but feasible, operating conditions, based on industry practice and the judgment of EPA's expert consultants on the refining industry (Sobotka and Company). This judgment was also reinforced in a letter to the National Petroleum Refiners Association from one of its consultants, who suggested that reformers (a key piece of

TABLE II-4. Estimated U.S. Refinery Processing Unit Capacities for 1988 (thousands of barrels per day)

Processing Unit	Capacity
Crude distillation	15,900
Vacuum distillation	6,880
Coking	1,340
Visbreaking	170
Low pressure reforming (150 psig)	940
Medium pressure reforming (250 psig)	2,280
High pressure reforming (450 psig)	750
Catalytic cracking	5,340
Hydrocracking	980
Alkylation	960
Polymerization	78
Butane isomerization	56
Pen./Hex. Isomerization	206

downstream equipment in meeting low-lead requirements) could be operated at 92 percent of capacity (Soloman and Associates, 1984, in Docket EN-84-05). Although no data are publicly available on the utilization of individual pieces of equipment, it is important to note that during the last four years of the 1960s, the refining industry used more than 90 percent of its crude capacity; in 1966, the utilization rate was 91.8 percent.

In addition to specifying maximum utilization rates for all equipment, we also specified minimum utilization rates for certain relatively inefficient reformers. Earlier versions of the DOE model included two types of reformers. In response to criticisms of that assumption, we modified the model to distinguish among three types of reformers: low-pressure (the most modern and efficient), medium-pressure, and high-pressure (the oldest and least efficient). When unconstrained, the model uses the available capacity of the low-pressure reformers first, then the medium-pressure reformers, and then, only if more reforming is needed, does it use any high-pressure reformers.

Some refineries, however, do not have any medium- or low-pressure reformers. To account for this fact, we determined how many refineries have only high-pressure reformers and required that the model use 75 percent of their capacity. This is a very strong assumption, as it is tantamount to saying that those refiners neither lose market share nor purchase such components from other refiners. Together with that requirement, we also assumed that refineries with both high-pressure and better-quality reformers would use the less-efficient reformers to some limited

extent, and constrained the model to direct at least 270,000 barrels per day to high-pressure reformers; that works out to a minimum capacity utilization rate for such reformers of 36 percent. Because the model is more tightly constrained by the availability of reformer feed than by reformer capacity, this minimum utilization constraint increases the costs and makes the low lead levels more difficult to achieve. In most runs, for both 1.10 gplg and lower levels, the model used the maximum amount of low-pressure capacity, the minimum amount of high-pressure capacity, and all of the swing took place in the utilization of medium-pressure reformers.

Several more detailed input assumptions also are worthy of note. In the Preliminary RIA (U.S. EPA, 1984f) issued at proposal, we assumed that refiners would continue to use the current mix of catalyst grades in their FCC units. During the comment period, EPA received information from commenters about some new, more efficient catalysts now in use at some refineries. After checking with some catalyst manufacturers (W.R. Grace, 1984, and Union Carbide, personal communication) to assure that capacity is available to produce sufficient quantities of these new catalysts, we assumed that 25 percent of FCC capacity could use the new catalysts by the second half of 1985, and that 50 percent could use them by 1986; to be conservative, we doubled the selling price to account for the cost of replacing existing catalysts early.

The DOE model distinguishes among three types of naphtha produced by the crude distillation process; different types of crude produce different proportions of these naphthas. In the

Preliminary RIA, we allowed the model to allocate those naphthas optimally for further processing in isomerization units or for direct blending into gasoline. In practice, such optimal allocation is likely to be difficult, although it can be approached by segregating the storage of different naphthas, and through careful purchases of different types of crude by refineries with different isomerization units. To limit this optimization, our base case now forces the model to process a preselected mix of these naphthas that achieves only 20 percent of the gain possible with optimal segregation. In response to several comments, we also reduced the reformer yields with parafinic naphthas at higher severity levels.

In some runs, the model found that the most economical method of meeting demand with less lead included altering the mix of crude inputs, switching from heavy crudes (such as those from Saudi Arabia) to lighter, high-quality crudes (such as those from Nigeria), despite the fact that we include a price differential between such crudes. To account for possible rigidities in long-term contracts and the like, we constrained the model not to reduce its use of heavy crude in going from 1.10 gplg to a lower level.

The model also found it more economical to increase the use of alcohols in gasoline in some cases, despite the fact that we forced the model to increase gasoline production when alcohol was used (alcohol has a lower BTU content than other gasoline components and, therefore, delivers fewer miles per gallon). Gasoline containing alcohol, however, faces uncertain consumer acceptance, and many pipelines will not accept it because of possible moisture

problems. Thus, we constrained the model to use no more alcohol at low lead levels than it used at 1.10 gplg. Because of scheduled expansion in ethanol capacity we allowed the model to use somewhat more ethanol at either level than is currently used, but oxinol levels were kept at current usage, and no MTBE use was allowed, despite current usage of about 13,000 barrels per day for octane enhancement of unleaded gasoline.

In addition to limiting the model's use of alcohol to boost octane at low lead levels, we did not allow it to use any MMT, another octane enhancer. During the mid-1970s, refiners used MMT to increase octane in unleaded gasoline. After tests showed that MMT harmed catalysts, such use was banned. MMT is still legal in leaded gasoline, however, and some unknown amount currently is used. Nonetheless, to be conservative, our base case did not include any possible use of MMT to increase the octane of low-lead gasoline.

One additional change has been made in our cost estimates. When EPA began analyzing potential changes in its lead regulation in late 1983, it assumed that oil prices would remain constant in 1983 dollars. That is, they were assumed to increase with inflation. These price assumptions were kept in the March 1984 Cost-Benefit Study, Prelimary Regulatory Impact Analysis, and subsequent docket submissions. In fact, however, oil prices, far from increasing by a few dollars per barrel since 1983, have fallen by several dollars per barrel, even in nominal terms. Table II-5 shows the original price estimates for Nigerian light and Saudi heavy crude used in our model runs, the prices at the end of

TABLE II-5. Prices of Crude Oil and Petroleum Products in 1983 and 1985 (dollars per barrel)

Product	1983 Price	1984 Price	
		Current Dollars	1983 Dollars
Crude Oil			
Nigerian Crude	31.50	29.18	27.40
Saudi Heavy	28.00	28.60	26.88
Residual Oil			
0.3% Sulfur	28.00	28.00	26.29
0.5% Sulfur	27.00	27.43	25.76
1% Sulfur	26.00	26.76	25.13
2% Sulfur	24.75	26.18	24.58
Other Products			
Isobutane	26.50	24.50	23.00
Normal Butane	25.70	23.75	22.30
LPG	18.20	17.78	16.70

1984, and what those prices are equivalent to in constant 1983 dollars. It also shows those same prices for residual oil, butanes, and propane.

Product prices are based on about a six-month period in 1984 to avoid seasonal distortions. The differences between the first and last columns show that this assumption has seriously distorted our cost estimates.

Rather than rerun all of the refinery analyses to find the costs under these lower prices, we have adopted the following approach. We have assumed that oil prices stopped falling in December 1984 and will henceforth increase with inflation. We have repriced the cost estimates of our model runs using the new prices, assuming (to be conservative) that the costs of crude fell by the same percentage drop as the heavier crudes, not the lighter crudes. To test the reliability of this approach, we reran the model for 1986 and 1988 with the new prices in, and compared it to the results of our repricing. These costs should be somewhat lower, because, in response to the change in the relative prices of crudes and products, refiners may find it less expensive to change some of their operating procedures. As Table II-6 shows, the costs using our procedure are quite close, but slightly higher than the costs given by completely rerunning the model. We also checked the marginal cost estimates for some of the more highly constrained sensitivity analyses, and achieved close agreement as well.

Because the British coal strike may have been keeping up the price of high sulfur residual oil on the world market, we also

TABLE II-6. Cost of 0.10 gplg Standard with New Oil Prices: New Model Run versus Repricing, Assuming No Misfueling (millions of dollars)

Year	New Model Run	Repricing
1986	573	608
1988	502	531

recalculated the costs assuming that high sulfur residual oil was selling for $1 per barrel less than current prices. This only increased the cost of our 1986 case by $14 million, so these estimates are not very sensitive to that potential factor.

II.C.2. Base-Case Results

Table II-7 presents information on the model's estimates at 1.10 gplg and at lower lead levels in 1985, 1986, 1987, and 1988. (The figures for 1985 apply only to the second half of the year.) These estimates assume that 0.50 gplg in 1985 does not eliminate any misfueling and that 0.10 gplg in the later years reduces misfueling by 80 percent. (See Chapter VIII for a discussion of alternative assumptions about misfueling and the costs associated with those assumptions.) In addition to the cost estimates, the table reports several other important pieces of information: the capacity utilization rates for various types of reformers (low-, medium-, and high-pressure, plus the aggregate figure) and the marginal cost of producing an octane-barrel of gasoline. (An octane-barrel is defined as raising the octane of a barrel of gasoline by 1 point.) Note that the 0.50 gplg standard for the second half of 1985 has relatively little impact. The cost is only $96 million; the overall utilization of reformers rises from 50 percent at 1.10 gplg to 59 percent at 0.50 gplg. The marginal cost of producing an octane-barrel rises from 15.8 cents at 1.10 gplg to 20.4 cents at 0.50 gplg. Thus, refiners should be able to meet the 0.50 gplg standard with relative ease and at a relatively moderate marginal cost by using excess capacity.

With regard to these estimates of marginal costs, it is interesting to note the comment of one refiner (Amoco submission to Public Docket EN-84-05) critical of EPA's cost estimates: "Current wholesale spot market price differentials between leaded and unleaded imply a marginal cost of about 20 cents/BON [barrel of octane number]. The trading value of lead rights reflects a similar cost of octane. .." As shown in Table II-7, the model with EPA's base-case assumptions projects a marginal cost of 15.8 cents per octane-barrel at 1.10 gplg in 1983 dollars, or 16.7 cents in 1984 dollars, very similar to the Amoco estimate for current (late 1984) conditions.

The base-case results for 0.10 gplg in 1986 and 1987 show somewhat tighter operating conditions, but no Feasibility problems. In both years, overall reformer utilization rises to 66 or 67 percent, and the marginal cost of an octane-barrel rises to a little more than 29 cents.

The results for 1988 are of interest because that is the first year in which we assumed that additional capacity could be added. The model found it most economical to add about 98,000 barrels per stream-day of isomerization capacity. That allows slightly lower use of reforming capacity than in 1986 or 1987, and reduces the marginal cost of octane to 28.4 cents per barrel at 0.10 gplg. The cost of that extra isomerization capacity ($322 million), annualized at 15 percent, is included in the $535 million cost estimate for 1988, and also in the cost estimates for subsequent years.

TABLE II-7. Base-Case Results for 1985-1988, with Partial Misfueling

Year Lead limit	Reformer utilization (%) by reformer type Low	Med.	High	Agg.	Marginal Cost of Octane (¢/barrel)	Total Cost (million $)
1985						
1.10 gplg	90	40	36	50	15.8	N.A.
0.50 gplg	90	53	36	59	20.4	96*
1986						
1.10 gplg	90	40	36	50	16.3	N.A.
0.10 gplg	90	67	36	67	29.2	608
1987						
1.10 gplg	90	41	36	52	16.4	N.A.
0.10 gplg	90	66	36	66	30.0	558
1987						
1.10 gplg	90	42	36	52	17.2	N.A.
0.10 gplg	90	62	36	64	28.4	532

*Cost for quarters III and IV.

The 1988 results also are interesting because they reflect the long-run marginal cost of producing octane (including capital charges). The reason that the marginal costs for 1.10 gplg are lower is that the refining industry has excess octane-producing equipment, and we have not included the costs of that sunk capital in estimating either total or marginal costs. Note also that the marginal cost of 0.50 gplg in 1985 (20.4 cents per octane-barrel) is far below the long-run marginal cost, which suggests that refiners would not find it cost-effective to build new capacity to meet that standard even if it were possible; the minimum-cost solution for 1985 would not include any new equipment. In 1986 and 1987, however, at 0.10 gplg, the marginal costs slightly exceed the long-run figure, indicating that it would be cheaper to build new capacity, if it were possible (although it is still possible to meet the 0.10 gplg standard without new capacity).

Table II-8 presents the year-by-year cost estimates under the base-case assumptions. Estimates are shown for three different rules: the original proposal (0.10 starting 1/1/86), the illustrative alternative discussed in the Notice of Proposed Rulemaking (0.50 gplg on 7/1/85, 0.30 on 1/1/86, 0.20 on 1/1/87, and 0.10 on 1/1/88), and the Final Rule (0.50 gplg on 7/1/85 and 0.10 on 1/1/86). Again, these estimates assume full misfueling in 1985 and 80 percent reductions in misfueling in subsequent years, when the 0.10 gplg standard applies. The costs fall over time because of projected declines in the demand for leaded gasoline.

TABLE II-8. Year-by-Year Estimates of Costs of Meeting Alternative Rules, with Partial Misfueling (millions of 1983 dollars)

Rule	1985	1986	1987	1988	1989	1990	1991	1992
Proposed	0	608	558	532	504	471	444	441
Alternative	96	364	448	532	504	471	444	441
Final	96	608	558	532	504	471	444	441

II. D. Sensitivity Analyses

The base-case results shown in Table II-8 represent EPA's "best estimates" of the cost of complying with the phasedown rule; i.e., they are based on what the Agency believes are the most realistic assumptions about refinery capabilities and gasoline demands. The Agency also conducted numerous sensitivity analyses to test the robustness of the results. Our efforts focused on 1986, because that is the first year in which the 0.10 gplg standard will apply. As shown in Table II-7, the 0.50 gplg standard in 1985 is much easier to meet, as measured either by reformer utilization or by the marginal cost of an octane-barrel. The 1987 results are similar to those for 1986, but in practice the 0.10 gplg standard should he easier to achieve in that year because refiners will have two years to adjust their operations.

Most of the sensitivity runs focused on changes unfavorable to the rule; i.e., on alternative parameter values that would increase the estimated costs. We did so not because we believe that such values are more likely to occur than those that would reduce the cost of the rule, but rather to probe the hypothetical conditions under which the rule would become extremely costly or, possibly, infeasible with existing refinery equipment. Many of the sensitivity analyses, particularly those varying several parameters simultaneously, responded to comments received on the August 1984 proposal.

We performed two types of sensitivity analyses. The first dealt with the issue of aggregation: to what extent does a national model underestimate costs by failing to account for regional

differences? We concluded that the base-case cost estimates are not artificially low because we used a national model. The disaggregate runs also indicated, however, that the rule would cause tighter operating conditions in Petroleum Allocation for Defense Districts (PADDs) I-IV and VI than in PADD V (the West Coast, Alaska, and Hawaii), so our second set of sensitivity analyses, designed to probe the limits of feasibility, focused on PADDs I-IV/VI (the rest of the country).

II.D.1. Level of Aggregation

Several comments received before and after the August 1984 proposal criticized EPA's cost analysis for relying on a national model. To examine that issue, we conducted two sets of sensitivity analyses.

The first compared EPA's estimates to those based on an analysis performed by Turner-Mason Associates (TMA) (1984) for the Lead Industries Association. TMA disaggregate the refining industry into six groups, based on geographic location, size and type of refinery, and other factors. It estimated the cost of meeting the 0.10 gplg in 1988 to be $995 million, compared to EPA's estimate at the time of proposal of $503 million. In addition to disaggregating, however, TMA changed several other assumptions. To see to what extent those changes, rather than the level of aggregation, explained the difference in results, we incrementally made each of those changes in the inputs to the national DOE model. The end result was that our national model with all of the TMA assumptions predicted slightly higher costs

than the TMA analysis, $1,016 million vs. $995 million (September 19, "Supplemental Analysis of Refining Costs," submission to Public Docket EN-84-O5). This result indicated that different levels of aggregation were not the source of discrepancy between EPA's and TMA's estimates, and that EPA's model accurately reproduced the results obtained with TMA's substantially finer level of disaggregation.

Note that the base-case estimates in this Final RIA incorporate several of the more pessimistic assumptions used by TMA, in particular a reduction in reformer yields at high severities. Other assumptions made in the TMA analysis, however, do not appear reasonable, in particular the capital charge rate (27 percent real) and a reduction in butane use of 50 percent from its current level.

The second approach to the aggregation issue divided the refining industry into two units, PADD V (the West Coast) and PADDs I-IV (the rest of the continental U.S.) plus PADD VI (the Virgin Islands and Puerto Rico). The rationale for this analysis was that, while PADDs I-IV/VI are tightly interconnected by pipelines and water shipping, PADD V is relatively isolated; only a few small pipelines connect PADD V to the rest of the country, and transport by sea between the Gulf and the West Coast is relatively expensive, though by no means impossible.

Table II-9 compares the national base-case estimates with the separate estimates for the two parts of the country for 0.10 gplg in 1986. Note that the sum of the two regional estimates is insignificantly different than the national estimate, $611

million vs. $608 million, which indicates that using the national model did not bias downward our base-case estimates. Table II-9 also reveals, however, that the rule generates somewhat higher operating rates and marginal costs for producing octane in refineries in PADDs I-IV/VI, which suggests that any problems from tighter operating constraints would show up in that part of the country before they occurred in PADD V.

It is also important to note that very fine levels of dis-aggregation can lend a strong upward bias to the cost estimates by failing to account for the flexibility in the refining industry to trade products and shift production in the face of changes in market conditions. Suppose, for example, that each refinery were modeled separately, with the constraint that it produce exactly the same slate of products that it produces now. That approach would overestimate total costs, because it would not allow for adjustments in product mixes across refineries. Such adjustments may be quite substantial, as refineries with better octane-making equipment would increase their total production of gasoline or increase the proportion of production that is premium gasoline, while other refineries would reduce their total gasoline output (or premium gasoline share), possibly shifting to the production of other petroleum products that place less burden on octane enhancement.

II.D.2. Other Parameters

We conducted extensive sensitivity analyses of other key parameter values in PADDs I-IV/VI. Table II-10 summarizes the

TABLE II-9. Costs of Meeting the 0.10 gplg Standard: Comparison of National and Regional Results for 1986

Model	Reformer Utilization (%) by Reformer Type				Marginal Cost of Octane	Total Cost
	Low	Med.	High	Agg.	(¢/barrel)	(million $)
National Model						
Base case	90	67	36	67	29.2	608
Regional Models						
PADDs I-IV/VI	90	71	36	69	31.6	531
PADD V	90	60	36	60	21.0	80
Total						611

base-case and alternative assumptions explored. As noted earlier, we focused on adverse changes; i.e., those that would make it more difficult to meet the standard. We also ran some sensitivity analyses of changes that would make it easier to meet the rule than our base case predicts.

II.D.2.a. Assumptions Varied

The first item listed in Table II-10 is the demand for unleaded premium gasoline. In our base case, we assumed that the demand for unleaded regular would be 25 percent of the total demand for unleaded gasoline, up slightly from its current level of 24 percent. As shown in Table II-10, we also ran sensitivity analyses that assumed that the fraction of premium would grow. Case A1 assumes that the demand for premium would grow by about 1.5 percentage points per year, reaching 27 percent of total unleaded demand in 1986 and 30 percent of the total in 1988. Case A2 assumes that premium demand grows at an even higher rate, about 5 percentage points a year, reaching 34 percent of total unleaded demand in 1986. This high rate of growth is out of line with historical trends and appears implausible, particularly if the marginal cost of producing octane rises, as it will with the phasedown.

Case B tightens the constraint on capacity utilization of all downstream processing units (including, most importantly, FCC and reforming units) from 90 percent of stream-day capacity to 85 percent. Several commenters suggested that EPA's assumption of 90 percent was too optimistic. Although the Agency continues to

TABLE II-10. Parameters Examined in Cost Sensitivity Analyses

Case	Parameter/Assumption	Base-Case Value	Alternative Value
A1	Premium share of unleaded demand	25%	27%
A2	Premium share of unleaded demand	25%	34%
B	Maximum capacity utilization of downstream processing units	90%	85%
C	Share of FCC units using new catalyst	50%	0%
D	Maximum ethanol use,'000 barrels/day	60	30
E1	Summer RVP limit	No	Yes
E2	Summer RVP limit and 5% higher demand	No	Yes
E3	Summer RVP, summer gasoline demand, and summer distillate demand	No	Yes
F	Substitution of light for heavy crudes permitted	No	Yes
G	MMT use allowed in leaded gasoline	No	Yes
H	Premium share of unleaded demand	25%	23%
I	Percent of naphtha segregated for gasoline processing	20%	100%

believe that 90 percent utilization can be achieved, this sensitivity analysis examined the impact of a lower utilization rate.

Case C assumes that all FCC units continue to use the same catalysts that they do now; the base case, as discussed earlier, assumes that by 1986 half of the FCC capacity would have switched to one of the newer, more efficient catalysts now available.

Case D examines the impact of reduced alcohol use. In the base case, we constrained the model to use no more alcohol (ethanol) at 0.10 gplg than it used at 1.10 gplg, which was 60,000 barrels per day. For this sensitivity analysis, we limited ethanol use to 30,000 barrels per day.

Case E analyzes only the summer quarter, when vapor pressure constraints are tighter. This case makes one extreme assumption about the summer quarter: that the refining industry's normal reliance on seasonal storage of gasoline components to meet summer demand does not occur, and that all summer demand must be met by current production. This seems unlikely, however, because the industry has 300 million barrels of gasoline storage capacity, in part because it normally produces extra gasoline in the winter and spring and stores it to meet summer demand peaks (Schwartz, memo in Docket EN-84-05, September 11, 1984e). Case El looks at the RVP change in isolation, while case E2 combines it with 5 percent higher daily demand for gasoline, which is typical of the summer quarter. In both cases, distillate production is kept at the annual average (whereas it normally is reduced in the summer to allow for greater gasoline production), and no account is taken of imports. Case E3 combines case E2 with a 50,000 bpd reduction

in distillate. In all three cases, the cost estimates presented later are only for one quarter, and are not comparable to cost estimates for the other (annual-cost) runs. The other measures of "tightness," however, can be compared.

Case F allowed the model more flexibility in choosing the crude slate. The base case constrained the solution to use as much heavy crude at 0.10 gplg as it did at 1.10 gplg. This sensitivity analysis allowed the solution to include some substitution of high-quality light crudes for heavy crudes if that was the most economical approach; but even in this sensitivity analysis, the swing was not allowed to exceed 250,000 barrels per day.

Case G allowed the model to use an alternative octane booster, MMT, at up to 0.05 grams per gallon, but only in leaded gasoline. That compares to the average of 0.0625 grams per gallon of MMT used in unleaded gasoline until its use in unleaded was banned because of its adverse effects on tailpipe hydrocarbon emissions. The use of MMT is still permitted in leaded gasoline, although data on current levels of use are not publicly available. The base case makes the pessimistic assumption that no MMT will be used.

Case H assumed lower octane demand, with the premium share of demand for unleaded gasoline falling to 23 percent. This could reflect several possibilities. First, if the price of leaded rises above that of unleaded, some current legal users of leaded gasoline (as well as misfuelers) may switch to unleaded regular; as discussed in Chapter VII, the experiences of the U.S. Armed Services and the U.S. Postal Service suggest that

many vehicles designed to operate on leaded regular (89 octane) will perform satisfactorily on 87 octane unleaded. During the public hearings, several refiners also mentioned that regular leaded gasoline in California has been reduced to 88 octane. Second, this drop in octane could occur if sharp increases in the marginal cost of producing octane led some refiners to slightly reduce the octane levels of their products.

Case I altered the assumption about the ability of refineries to segregate different types of naphthas for further processing. This sensitivity analysis allows for greater segregation of naphthas, as was assumed in the estimates made for the Preliminary RIA.

II.D.2.b. Results of Sensitivity Analyses

Table 11-11 presents the results of varying the parameter values individually. In all cases, the estimates are for 0.10 gplg in 1986. Note that these results cover only PADDs I-IV/VI, the part of the country where difficulties, if they occur, are likely to show up first. The first line shows the results for PADDs I-IV/VI under the base case assumptions.

As the other lines in the table show, the impacts of varying the individual assumptions are modest. The maximum cost increase is less than 19 percent, and that occurs only under the higher of the two high-octane scenarios. That scenario assumes that the premium share of unleaded demand increases at 5 percentage points each year (from a base of 24 percent in 1984), which seems highly unlikely. Indeed, that rate of growth in the premium share is so

TABLE II-11. Effects of Varying Individual Parameters/Assumptions: PADDs I-IV/VI

Case	Changes	Reformer utilization (%) by reformer type				Marginal Cost of Octane (¢/barrel)	Total Cost ($ million)
		Low	Med.	High	Agg.		
Base	None	90	71	36	69	31.6	531
A1	27% premium	90	72	36	69	32.8	541
A2	35% premium	90	82	36	74	33.0	629
B	85% utilization	85	84	36	74	29.2	486
C	Old catalysts	90	87	36	77	32.5	617
D	Reduced alcohol	90	77	36	72	33.0	554
E1	Summer RVP	90	77	36	72	33.2	135*
E2	El, summer demand	90	90	36	78	34.4	136*
E3	E2, summer distillate	90	90	36	78	31.6	127*
F	Crude flexibility	90	70	36	68	31.6	513
G	Use of MMT	90	69	36	68	29.0	489
H	23% premium	90	70	36	68	30.3	496
I	Naphtha segregation	90	66	36	66	25.2	441

*Cost for summer quarter only

high that it implies that the absolute amount of unleaded regular gasoline would fall 6 percent from 1984 to 1986, a highly improbable occurrence.

Looking at the other measures of tightness, the most severe impacts occur under case E2, which assumes summer RVP and 5 percent higher demand with no distillate adjustment, seasonal storage, or imports. Even in that case, however, the marginal cost of producing octane rises less than 9 percent (to 34 cents per octane-barrel), and utilization of high-pressure reformers does not rise above the minimum level that we forced the model to use. Overall reformer utilization was only 78 percent of capacity, up from 68 percent in the base case. These increases, of course, would be balanced by lower-than-average operating costs, marginal costs, and reformer severities in the other three quarters when RVP standards are higher than the annual average. Case E3 shows that adding even small distillate flexibility brings the marginal cost of octane back down to the base case value.

Case B, lowering maximum downstream utilization from 90 percent to 85 percent, yielded counter-intuitive results; the cost is slightly lower than in the base case, as is the marginal cost of octane. The utilization of the medium-pressure reformers increased in this case, from 71 percent of stream-day capacity to 84 percent. Closer examination of the output of the model revealed that 85 percent utilization raised the cost of both the 1.10 gplg and the 0.10 gplg cases, as expected, but reduced the difference between the two. Tightening that constraint appears to have raised the total cost of producing octane, but lowered its incremental

cost over the relevant range.

On the positive side of the ledger, the most important parameter change was case T, which allowed more optimal segregation of naphthas, as in the runs made for the Preliminary RIA; that reduced the costs in PADDs I-IV/VI from the base case by 17 percent, to $441 million. It also reduced overall reformer utilization from 68 to 66 percent of capacity, and had a more dramatic impact on the marginal cost of producing octane, reducing it by 20 percent, to 25.2 cents/barrel. Allowing the use of MMT in leaded gasoline, case G, also had a significant impact on costs.

In addition, we ran sensitivity analyses that varied several parameter values simultaneously to probe the limits of feasibility; the results are shown in Table II-12. They should be interpreted carefully, for while each parameter change may be plausible alone, increasing the number of simultaneous negative changes generates increasingly implausible circumstances. Furthermore, market forces of supply and demand create feedback loops that make some of the extreme cases exceedingly unlikely. For example, if the marginal cost of producing octane rises (due, say, to lower utilization of downstream capacity), the price of high-octane unleaded premium is likely to rise, reducing the demand for that product (or at least forestalling the increases assumed in cases Al and A2) and the demand for high-octane alcohol is likely to increase, not decrease.

Lowering the utilization rate for downstream equipment and simultaneously reducing the allowable amount of alcohol, run Ml

TABLE II-12. Effects of Varying Multiple Parameters/Assumptions: PADDs I-IV/VI

Run	Changes**	Reformer Utilization (%) by Reformer Type				Marginal Cost of Octane	Total Cost
		Low	Med.	High	Agg.	(¢/barrel)	($ million)
Base	None	90	71	36	69	31.6	531
M1	Utilization (B), less alcohol (D)	85	85	38	75	33.4	524
M2	(B), (D), plus old catalysts (C)	85	85	78	83	59.4	775
M2a	(B),(D),(C), plus lower premium (I)	85	85	70	82	52.0	754
M3	(B),(C),(D), plus higher premium (A1)	85	85	78	83	61.0	830
M3a	(B), (C), (D), (A1), plus MMT (G)	85	85	68	81	49.4	717
M3b	(B), (C), (D), (A1), plus MTBE and oxinol	85	85	61	80	48.3	773
M4	Old catalysts (C), higher premium (A1), less alcohol (D)	90	90	40	79	49.4	763
M4a	(C), (A1), (D), plus MMT (G)	90	90	36	78	41.4	655
M5	Old catalysts (C), less alcohol (D), summer RVP (E1)	90	90	58	83	54.2	217*
M5a	(C), (D), (E1), plus lower premium (I)	90	90	58	83	51.3	202*
M6	(C), (D), (E1), plus higher premium (A1)	90	90	62	84	85.4	231*
M6 a	(C), (D), (E1), (A1), plus MMT (H)	90	90	64	83	48.3	197*

(Table II-12 continues on next page)

TABLE II-12. (Continued)

Run	Changes**	Reformer Utilization (%) by Reformer Type				Marginal Cost of Octane (¢/barrel)	Total Cost ($ million)
		Low	Med.	High	Agg.		
M7	(C), (D), (A1), plus summer RVP and demand (E2)	90	90	89	90	87.0	267*
M7a	(C), (D), (A1), (E2), plus MMT (G)	90	90	86	89	62.8	220*
M7b	(C), (D), (A1), (E2), plus storage	90	90	52	82	54.5	215*
M7c	(C), (D), (A1),(E2), (G), plus imports	90	90	55	83	48.2	193*
M7d	(C), (D), (A1), (E2), MTBE , oxinol, and summer distillate	90	90	76	87	48.2	218*

*Cost for summer quarter only

**Letters refer to parameters listed in Table II-10

in Table II-12, has little impact on any of the measures. Also eliminating the use of any new FCC catalysts, as in run M2, makes for substantially tighter, though still feasible, operating conditions, raising the total cost to $775 million and the marginal cost to 59.4 cents/octane-barrel. Run M2a made the same negative changes as run M2, but lowered the demand for premium unleaded to 23 percent (2 percentage points below the base case, but only 1 percentage point below current demand); compared to run M2, that cut the cost about 3 percent, and reduced the marginal cost of octane by about 12 percent.

Run M3 added higher demand for premium to the changes made in run M2. That further tightened the predicted operating conditions, but did not make the attainment of the 0.10 gplg standard infeasible. Run M3a made those same changes, but allowed the use of MMT, which cut total and marginal costs significantly. Run M3b also duplicated M3, but allowed additional purchases of oxinol (10,000 bpd) and MTBE (27,000 bpd) at prices that reflected both their cost and their adverse impact on fuel economy. It also cut total and marginal costs substantially. Both of these increases are within existing capacity for those products.

Run M4 probed a slightly different combination of sensitivities, combining low ethanol use, high octane demand, and old catalysts (as well as no imports, no MTBE, etc.). Again, costs increased but the industry would still he able to comply. Once more, adding some MMT (run M4a) substantially reduced costs.

Runs M5-M7 were summer sensitivity runs. Again, to probe the

limits of feasibility, we began by assuming no seasonal storage was available. Run M5 assumed that no new catalysts would be used, that alcohol use could not exceed 30,000 barrels per day, and that all gasoline would have to meet summertime RVP specifications with no distillate flexibility, imports, or MTBE use. This is a very demanding, and unlikely, set of events, but 0.10 gplg remained feasible. Lowering the demand for premium (run M5a) brought the total and marginal costs down significantly; storage of high octane components produced during the first half of the year (as is done by most refiners now) would be one way to reduce the amount of premium gasoline that had to be produced in the summer. The cost estimates for these two runs (and the subsequent ones in the table) are only for the summer quarter. Again, the higher-than-average costs in the summer (when RVP standards are tighter than average) are balanced by lower-than-average costs in the winter (when RVP standards are looser than average).

Run M6, which added higher demand for unleaded premium to run M5, generated a model solution that must be characterized as infeasible. The marginal cost of producing octane was extraordinarily high, and the model ran so much extra crude oil that some of the products of crude distillation had no outlet; they were simply dumped. The conditions assumed in that run, however, are collectively highly implausible. If the marginal cost of octane were to rise to the level shown in run M6, the price of high-octane unleaded premium would rise sharply, making the surge in unleaded premium demand and lack of use of existing MTBE and oxinol capacity

contemplated in run M6 impossible. Moreover, because run M6 only applies to summer conditions, normal use of existing storage capacity could alleviate the summertime crunch, since the same case (M4) with annual average RVP levels was feasible. It is also interesting to note that modest use of MMT, as allowed in run M6a, restored feasibility and brought the estimated marginal cost down to a much more comfortable level.

Run M7 added higher overall gasoline demand to the changes in run M6; again, the result was unfeasibility, though for an even more implausible set of conditions. Again, storage of gasoline or high-octane gasoline components produced in the winter or spring could alleviate this summertime problem since M4 is the same run for the full year. Relaxing some of our least-likely conservative assumptions in other areas also restored feasibility. For example, run M7a shows that MMT restored feasibility, bringing the marginal cost of an octane-barrel back down to 62.8 cents. Run M7b shows that 400,000 barrels per day of seasonal storage also restored feasibility, reducing the marginal cost of octane to 54.5 cents, and run M7c shows that taking account of imports and MMT (but not storage) reduced costs even further. Run M7d shows the results of allowing the normal seasonal distillate flexibility, use of existing MTBE capacity, and use of 10,000 bpd more of existing oxinol capacity, but neither imports nor seasonal storage. Again, feasibility is restored.

To summarize the summer sensitivity runs, it takes an extremely unlikely combination of high-cost sensitivities (e.g.,

higher premium demand, no use of the newer catalysts, lower use of ethanol, summer RVP standards, high total demand, no imports, no MTBE, no MMT, no increase in oxinol, no seasonal storage, no distillate flexibility, and no trading with PADD V) to produce an infeasible result. Relaxing the assumption about seasonal storage, within the limits of existing storage capacity, always restores feasibility (case M4), as does relaxing many of the other assumptions singly (e.g., the assumption on MMT, on distillate flexibility, or on using existing MTBE capacity). Since the industry currently manipulates to its advantage the use of storage capacity, MMT, MTBE, swings in distillate production, and imports simultaneously, EPA believes that concern about summer unfeasibility problems is unwarranted.

We also ran the two most extreme sensitivity analyses (runs M3 and M7) on PADD V to check our assumption that problems, if they occurred at all, would show up first in PADDs I-IV/VI; Table II-13 reports the results. As a comparison with the corresponding runs in Table II-12 shows, conditions in PADD V are projected to be looser (as measured by reformer utilization and the marginal cost of producing an octane barrel) than in PADDs I-IV/VI.

To assure ourselves that the 0.50 gplg standard for the second half of 1985 would be feasible, even under worst-case conditions, we ran the two most extreme sensitivity analyses, shown in Table II-14. Even in the worst case examined (M7), overall reformer utilization did not exceed 75 percent and the marginal cost of producing an octane-barrel did not rise above 24 cents. The results show that 0.50 gplg in 1985 remains quite

TABLE II-13. Sensitivity Analyses for 1986: PADD V

Run	Changes**	Reformer Utilization (%) by Reformer Type				Marginal Cost of Octane	Total Cost
		Low	Med.	High	Agg.	(¢/barrel)	($ million)
Base	None	90	58	36	59	21.5	80
M3	Utilization (B), less alcohol (D), catalysts (C), higher premium (A1)	85	70	34	68	29.8	107
M7	Old catalysts (C), less alcohol (D), higher premium (A1), summer RVP and demand (E2)	90	77	36	76	32.2	31*

*Cost for summer quarter only

**Letters refer to parameters listed in Table II-10.

TABLE II-14. Sensitivity Analyses for 1985: PADDs I-IV/VI

Run	Changes**	Reformer Utilization (%) by Reformer Type				Marginal Cost of Octane	Total Cost
		Low	Med.	High	Agg.	(¢/barrel)	($ million)
Base	None	90	55	36	60	21	79
M3	Utilization (B), higher premium (A1) less alcohol (D) old catalysts (C)	85	78	36	71	22.3	89
M7	Old catalysts (C), less alcohol (D), higher premium (A1), summer RVP and demand (E2)	90	84	36	75	23.4	48*

*Cost for summer quarter only

**Letters refer to parameters listed in Table II-10.

feasible and not excessively costly under all circumstances.

These sensitivity analyses clearly indicate that the phase-down schedule contained in the rule is feasible with existing equipment under expected conditions. Even if several conditions are more adverse, simultaneously, than in our base case, the 0.10 gplg standard remains feasible, although it may require very efficient utilization of refineries and careful attention to the full array of methods available for meeting octane demands with reduced lead. Only in the very worst cases, which combine many adverse conditions simultaneously, does it appear that the refining industry would experience great difficulty in complying with the rule and then only for three months. Such multiple worst-case scenarios are useful for probing the limits of feasibility, but they are too implausible to deserve much weight, and certainly too implausible to affect the outcome of this rule-making. Moreover, as discussed in the next section, the proposed rule to allow banking would render even those circumstances feasible.

II.E. Impact of Banking on Costs

EPA has proposed to allow the “banking” of lead in 1985 for use in 1986 and 1987 (50 FR 718; January 4, 1985), when the 0.10 gplg standard will apply. Under this proposal, refiners will have the option of reducing lead use before it is required, and then applying those early reductions to increase the amounts of lead they are allowed to use in the two succeeding years. The purpose of this provision is to give individual refiners lower

costs and extra flexibility in reducing lead (even though the standards are both feasible and reasonable without it) without increasing the total amount of lead used in gasoline between 1985 and 1987. Although refiners would be under no obligation to avail themselves of the right to bank, EPA expects that the majority would, for at least three reasons: (1) lead provides a greater octane boost at low levels, so it is more valuable in 1986 and 1987 at 0.10 gplg than at the higher levels permitted in 1985; (2) in the short run, the marginal cost of producing octane rises with the amount of octane produced, again making lead more valuable in 1986 and 1987 than in 1985; and (3) banking lead rights in 1985 will give refiners extra flexibility to deal with unexpected problems (such as equipment breakdowns) in the later years.

II.E.1. Base-Case Banking Results

It is difficult to predict precisely how banking will be used. Some refiners are likely to cut their lead use very quickly so as to bank a large amount, either for later use at that refinery or for sale to other refineries less well-equipped to produce octane without lead, while others will bank relatively little. Whatever the specific pattern followed, however, the experience with interrefinery averaging (“trading”) over the last few years suggests that the refining industry will make effective use of this mechanism to reduce costs by reallocating lead use to those refineries (and, with banking, to those times) where lead has the highest marginal value in reducing production costs. (As

discussed in Chapter I, refiners representing about three-fourths of all refining capacity now trade in any given quarter.)

To explore the potential impacts of banking, we examined two possible phasedown patterns. For this analysis, we assumed (as above) that misfueling would continue unabated during 1985, when the standard will be 1.10 gplg during the first half of the year and 0.50 gplg during the second half of the year. For 1986 and 1987, when the limit will be 0.10 gplg, we assumed that misfueling would fall to 20 percent of its current level. We also assumed that misfueling would follow this same pattern with banking. If refiners use less than 1.10 gplg in the first half of 1985, or less than 0.50 gplg in the second half, the marginal cost of producing leaded gasoline will increase. Each gallon of leaded gasoline produced below the limit will produce banked rights that have value, so the net marginal cost of producing a gallon of leaded gasoline will be little affected. Similarly, in 1986 and 1987, refiners who use banked rights to produce leaded gasoline with more than 0.10 gplg will face lower marginal costs of production than they would have otherwise, but they also will consume rights that have a market value, so the net marginal cost will be higher, roughly the same as it would have been at 0.10 gplg. As a result, it seems reasonable to make the same misfueling assumptions with and without banking.

Table II-15 compares the schedule without banking to two possible alternatives with banking. Alternative 1 assumes that refiners do not start banking until the second quarter of 1985, at which point they use an average of 0.60 gplg, thus banking

TABLE II-15. Alternative Phasedown Patterns with Banking (gplg)

Alternative	1985 (by quarter) I	II	III-IV	1986	1987
Without Banking	1.10	1.10	0.50	0.10	0.10
With Banking					
Alternative 1	1.10	0.60	0.40	0.25	0.19
Alternative 2	0.80	0.60	0.45	0.30	0.21

0.50 grams, on average, for each gallon of leaded produced. (Note that this industry average could reflect wide variations across refiners; some might not bank at all, while others saved large amounts.) Under alternative 1, we also assume some banking in the last half of the year, with leaded gasoline averaging 0.40 gplg, slightly below the limit of 0.50 gplg. A total of 7.0 billion grams of lead (about 22 percent of the total allowed in 1985) would be banked during 1985, allowing refiners to average 0.25 gplg in 1986 and 0.19 gplg in 1987. Shaving 0.10 gplg from the annual average in 1985 translates into a larger per-gallon increase in 1986 or 1987, because the amount of leaded gasoline produced in the later years is smaller; the total amount of lead use over the three years, however, is the same as without banking.

Alternative 2 assumes that some refiners are able to reduce lead more quickly, so that banking begins in the first quarter of 1985. Those extra banked rights from the first quarter are then used to reduce banking slightly in the last half of 1985 and to achieve slightly higher lead levels in 1986 and 1987. The amount banked is 9.1 billion grams. Again, the total amount of lead used is the same as without banking.

Table II-16 compares the year-by-year costs and the present values of the costs with banking to those without. The estimated savings are substantial; $173 million for alternative 1 and $226 million for alternative 2. These estimates probably understate the actual savings that will be realized with banking, because they do not account for the extra flexibility it allows in meeting unexpected problems (e.g., equipment breakdowns or a sudden

TABLE II-16. Refining Costs Under Alternative Phasedown Patterns, with Partial Misfueling (millions of 1983 dollars)

Alternative	1985	1986	1987	Present Value
Without Banking	96	608	558	1,105
With Banking				
Alternative 1	176	420	463	932
Alternative 2	170	378	452	879

surge in summertime demand) and in reallocating lead use to those refiners with higher marginal costs of producing octane.

Table II-17 reports the marginal costs of an octane-barrel for 1985 (by quarter), 1986, and 1987 under the alternative banking scenarios. Under either alternative, the marginal cost remains under 22 cents per octane-barrel in 1985 and rises to only 26.7 cents in 1986. These estimates should be compared to the marginal costs without banking, shown in Table II-7. It is particularly interesting to note that with banking, the marginal cost of octane never rises above the level shown for 1988, when new equipment will first be available. This result has several important implications. First, it indicates that, with banking, the phasedown schedule is no more expensive at the margin than it would be if the Agency delayed the phasedown until 1988, when refiners can build new equipment. Second, it suggests that refiners would not find it cost-effective to build new capacity before 1988, even if it were possible. (It would be cheaper to buy banked lead rights.) Finally, it suggests that the 1985-to-1987 phasedown (again, with banking) should not cause financial difficulties for refiners who could profitably operate at 0.10 gplg once they had time to add equipment. Indeed, to the extent that some refiners have trouble obtaining loans for capital equipment, these results suggest that they will have an easier time from 1985 to 1987 than they will in later years, because during the earlier years they can buy lead rights, the cost of which is an operating expense, not a large capital outlay requiring loan or equity financing.

TABLE II-17. Impact of Banking on Marginal Costs of Octane (cents per barrel)

Year Quarter	Without Banking	With Banking	
		Alt. 1	Alt. 2
1985			
I	N.A.	N.A.	17.6
II	N.A.	21.4	21.4
III-IV	20.4	21.1	21.1
1986			
I-IV	29.2	26.7	22.5
1987			
I-IV	30.0	25.0	25.0

II. E.2. Sensitivity Analyses with Banking

We also reran several of the sensitivity analyses for 1986 with banking, with the results shown in Tables II-18 (for alternative 1) and II-19 (for alternative 2). As a comparison of those tables with Table II-12 shows, banking greatly reduces the difficulty of meeting the rule, even in the unlikely scenarios that make several adverse changes simultaneously. For example, without banking, as shown in Table II-12, cases M6 and M7 both drive the marginal cost of an octane-barrel in PADDs I-IV/VI in 1986 over 85 cents and are essentially infeasible. If refiners follow alternative 2 with banking, however, they can use 0.30 gplg in 1986. As Table II-19 shows, reformer utilization can then fall and the marginal cost of octane decreases by more than 50 percent, to about 40 cents per octane-barrel. This greater comfort in 1986, of course, is partly offset by higher costs in 1985, but the reductions needed in 1985 should not strain the capacity of the refining industry. Moreover, this analysis of PADDs I-IV/VI understates the benefits of banking because rights banked in PADD V could be sold to refineries in PADDs I-IV/VI. (Based on a separate analysis of PADD V, we expect such transfers to occur, as the model shows a lower marginal value of lead in PADD V than in PADDs I-IV/VI, at any given level of lead use.)

These analyses suggest that while banking is not necessary to meet the phasedown schedule, it does yield significant cost savings and, perhaps more importantly, provides an extra margin of safety against unexpectedly adverse conditions (e.g., higher octane demands or lower-than-expected ability to utilize down-

TABLE II-18. Sensitivity Analyses for 1986 with Banking: Alternative 1, PADDs I-IV/VI

Run	Changes**	Reformer Utilization (%) by Reformer Type				Marginal Cost of Octane (¢/barrel)	Total Cost ($ million)
			Med.	High	Agg.		
Base	None (no banking)	90	71	36	69	31.6	531
Base	None (with banking)	90	60	36	62	22.7	420
M1	Utilization (B), less alcohol (D)	85	81	36	72	23.7	365
M2	(B), (E), plus old catalysts (C)	85	85	49	77	40.3	489
M3	(B),(C),(E), plus higher premium (A1)	85	85	55	79	40.5	531
M4	Old catalysts (C), higher premium (A1), less alcohol (D)	90	87	36	77	32.1	514
M5	Old catalysts (C), less alcohol (D), summer RVP (E1)	90	90	40	79	39.7	140*
M6	(C), (D), (E1), plus higher premium (A1)	90	90	46	81	46.6	149*
M7	(C), (D), (A1), plus summer RVP and demand (E2)	90	90	72	86	51.5	169*

*Cost for summer quarter only

**Letters refer to parameters listed in Table II-10.

TABLE II-19. Sensitivity Analyses for 1986 with Banking: Alternative 2, PADDs I-IV/VI

Run	Changes**	Reformer Utilization (%) by Reformer Type				Marginal Cost of Octane	Total Cost
		Low	Med.	High	Agg.	(¢/barrel)	($ million)
Base	None (no banking)	90	71	36	69	31.6	531
Bank	None (with banking)	90	58	36	61	22.7	378
M1	Utilization (B), less alcohol (D)	85	78	36	71	23.3	324
M2	(B),(E), plus old catalysts (C)	85	85	43	76	34.2	411
M3	(B),(C),(E), plus higher premium (A1)	85	85	46	77	36.2	440
M4	Old catalysts (C), higher premium (A1), less alcohol (D)	90	84	36	75	29.8	453
M5	Old catalysts (C), less alcohol (D), summer RVP (E1)	90	89	36	78	30.5	121*
M6	(C), (D), (E1), plus higher premium (A1)	90	90	36	78	40.0	127*
M7	(C), (D), (A1), plus summer RVP and demand (E2)	90	90	61	84	42.6	143*

*Cost for summer quarter only

**Letters refer to parameters listed in Table II-10.

stream refinery units, such as FCC units and reformers). Moreover, these gains are achieved without any increase in the amount of lead allowed.

CHAPTER III

HUMAN EXPOSURE TO LEAD FROM GASOLINE

Estimating the health benefits of an environmental regulation requires predicting how the regulation will affect human exposure levels. In most cases, exposure estimates require extensive modeling of emissions, dispersion patterns, population distributions, and the amounts of inhaled or ingested material that are absorbed by the human body. Such modeling requires that many parameters be estimated, often on the basis of very limited information.

In the case of lead in gasoline, however, exposure can be assessed directly using several large data sets that make it possible to relate lead in gasoline directly to lead in individuals' blood, without taking the intermediate steps of dispersion modeling, etc. Analyses of these different data sets have shown a strong and consistent relationship between the amount of lead in gasoline and the amount of lead in blood, a relationship confirmed by experimental data as well.

This chapter presents the methods used to estimate the impact of reducing lead in gasoline upon levels of lead in the blood of children and adults. These projections are used in Chapters IV and V to estimate health benefits for children and adults, respectively. Section A of this chapter addresses the basic issues, including the data and the statistical methods used. Section B discusses the question of causality. Section C presents estimates of the numbers of children whose blood lead levels would be reduced below various levels as a result of reducing lead in gasoline.

III. A. The Relationship Between Lead in Gasoline and Lead in Blood

Individuals are exposed to lead from gasoline through many pathways. When leaded gasoline is burned in an engine, small amounts are deposited in the engine and exhaust system, but most of it is emitted from the tailpipe to the air, where it remains suspended for a time before settling to the ground. Some exposure occurs through direct inhalation of the emitted lead. Additional exposure occurs from ingestion of lead-contaminated dust, or inhalation of such dust that has been stirred up. Lead from gasoline also deposits on food crops and is then ingested. These multiple routes make it very difficult to model individual exposure pathways. It is possible, however, to estimate the total amount of lead exposure from gasoline using statistical methods, as discussed below.

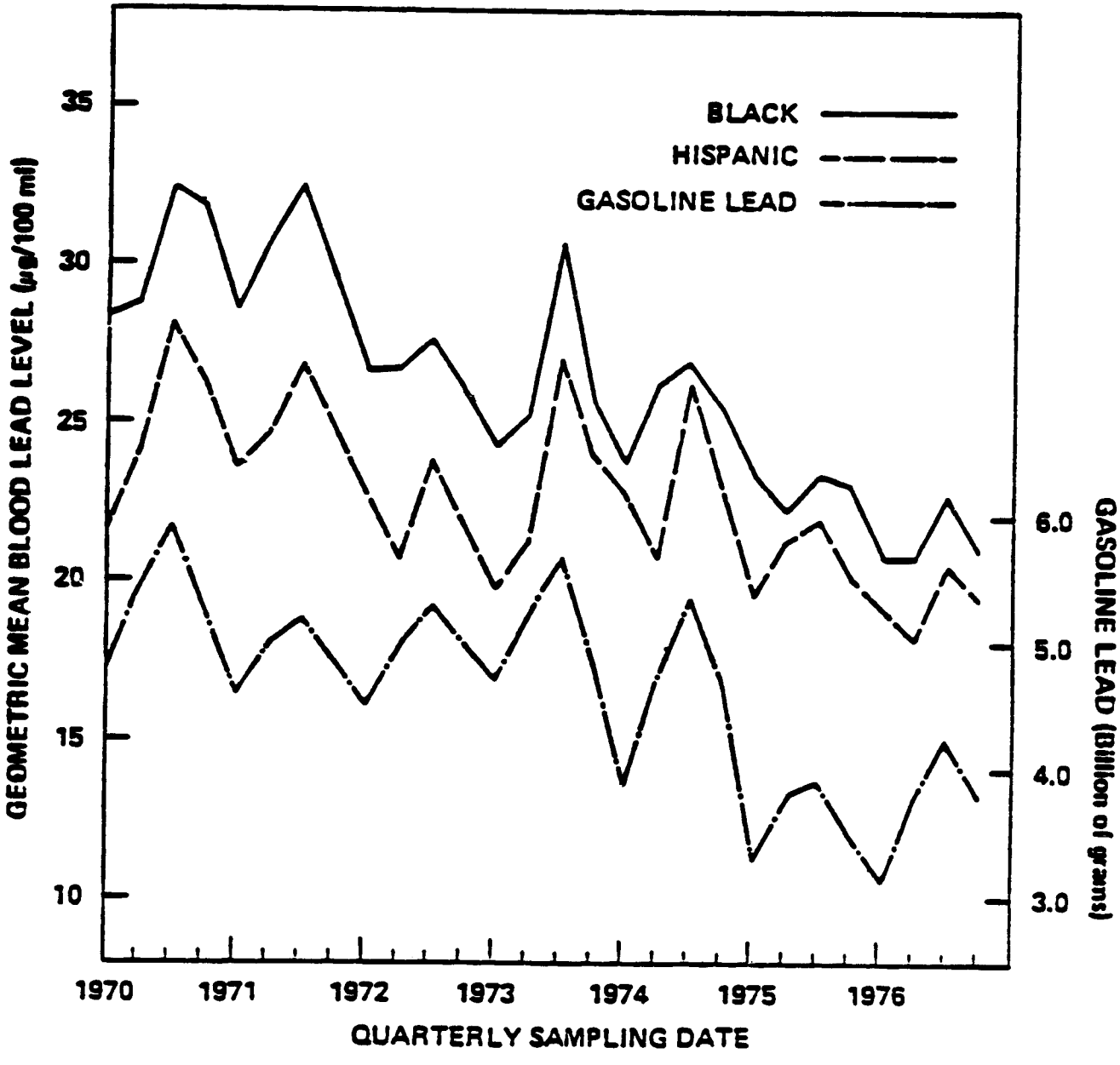
III.A.1. Recent Studies

Several recent articles have shown that blood lead levels for all age groups will fall as gasoline lead content falls. The first important statistical studies were done by Billick et al. (1979), who showed a strong relationship between the blood lead levels of several hundred thousand children screened in New York City’s lead screening program and local gasoline lead use. Figure III-1 on the next page shows this relationship graphically.

In 1982, Billick et al. presented additional regression analyses on data from New York City’s lead screening program (with data on several additional years); a Chicago screening program (800,000 children over more than ten years); and a Louisville, Kentucky program, all of which confirmed the earlier results.

FIGURE III-1

Relationship Between Gasoline
Lead and Blood Lead in New York City



An EPA report (Janney, 1982) found a strong association between gasoline lead and blood lead in the data from the second National Health and Nutrition Examination Survey (NHANES II), after controlling for age, race, sex, degree of urbanization, and income. Analyses of the NHANES data by the Centers for Disease Control (CDC) and the National Center for Health Statistics performed concurrently showed similar results.

Annest et al. (1983) published a paper analyzing the nationwide downward trend of blood lead levels from 1976 to 1980 that was demonstrated in the NHANES II data. Blind quality-control data were used to ensure that there was no drift in laboratory measurements. This downward trend was present after controlling for age, race, sex, region of the country, season, income, and degree of urbanization, and was present in each age-sex-race subgroup. Gasoline lead was a significant predictor ($p < 0.001$) of blood lead after controlling for age, race, sex, degree of urbanization, income, season, and region of the country, both in all groups and separately for blacks, whites, white males, white females, white children, white teenagers, and white adults.

A recent paper by Schwartz et al. (1984b) of EPA's Office of Policy Analysis presented the results of a study of the relationship between blood lead levels and gasoline lead. Several data sets were employed for this analysis, including the NHANES II and the CDC lead poisoning screening program. The statistical results indicated a highly significant regression coefficient for gasoline lead levels, which was consistent across all of the data sets. Estimates of environmental lead from sources

other that gasoline indicated that paint and other dietary lead were not the primary sources of the observed decline in blood lead levels during the 1970s.

Gasoline lead was strongly associated with both the level of lead in human blood and with the prevalence of elevated blood lead levels. The association appeared to be causal because other factors, such as changes in dietary lead and paint lead, did not account for the changes in blood lead levels that have been associated with gasoline lead; the results suggested that more than one-half of the lead in the average American's blood in the second half of the 1970s was due to gasoline. Since gasoline lead usage in that period was restricted by regulation to about 60 percent of what otherwise would have occurred, those regulations appeared to have reduced substantially the average blood lead level in the U.S. and the number of children with lead toxicity.

III.A.2. Available Data Sets

Because gasoline sales and the use of various gasoline additives are regulated by federal law, information on them is available from the Department of Energy and the Environmental Protection Agency. Several data sets contain information on individual blood lead measurements. Most of these data sets target children in high-risk groups who have been screened for lead poisoning. One data set, however, NHANES II, is a large representative sample of both adults and children in the U.S. This section describes the data sets used in Section III.A.3. to estimate the relationship between gasoline lead levels and blood lead levels.

III.A.2.a. Gasoline-Use Data

We combined monthly data on national leaded gasoline sales (from the U.S. Department of Energy) with quarterly average lead concentrations in grams per gallon (reported to EPA by refiners) to compute national monthly gasoline lead usage. For the Chicago, New York, and Louisville areas, we used gasoline sales data from the Ethyl Corporation's monthly survey of area gasoline sales, combined with national grams per gallon of lead, to obtain metropolitan gasoline-lead usage.

III.A.2.b. The NHANES II

The data base for the regressions used to estimate the coefficients in our prediction models was the health and demographic information collected in the NHANES II survey. The U.S. Bureau of the Census selected the NHANES II sample according to rigorous specifications from the National Center for Health Statistics so that the probability of selection for each person in the sample could be determined. The survey used subjects chosen through a random multi-stage sampling scheme, designed to utilize the variance minimization features of a stratified random sample. A total of 27,801 persons from 64 sampling areas was chosen as representative of the U.S. non-institutionalized civilian population, aged six months through 74 years. Of those 27,801 persons, 16,563 were asked to provide blood samples, including all children six months through six years and half of those aged seven through 74 years. The nonrespondent rate for blood samples was 39 percent and did not correlate with race,

sex, annual family income, or degree of urbanization.* A study of the potential nonresponse bias indicated that this was not a significant problem (Forthofer, 1983).

Lead concentrations in the blood of sampled persons and control groups were determined by atomic absorption spectrophotometry using a modified Delves Cup micro-method. Specimens were analyzed in duplicate, with the average of the two measurements being used for the statistical analysis. Bench quality control samples were inserted and measured two to four times in each analytical run to calibrate the system. In addition, at least one blind quality-control sample was incorporated with each 20 NHANES II blood samples. No temporal trend was evident in the blind quality-control measurements (National Center for Health Statistics, 1981).

The NHANES II data did, however, display a marked relationship between blood lead and gasoline lead. Figure III-2 plots gasoline lead and blood lead over time. Note how closely the changes in blood lead track the changes in gasoline lead, following both seasonal variations and the long-term downward trend. Figure III-3 plots blood lead as a function of gasoline lead after controlling for age, race, sex, income, degree of urbanization, region of the country, educational level, smoking, alcohol consumption, occupational exposure, dietary factors, and interactions among those factors; again, note the strong relationship.

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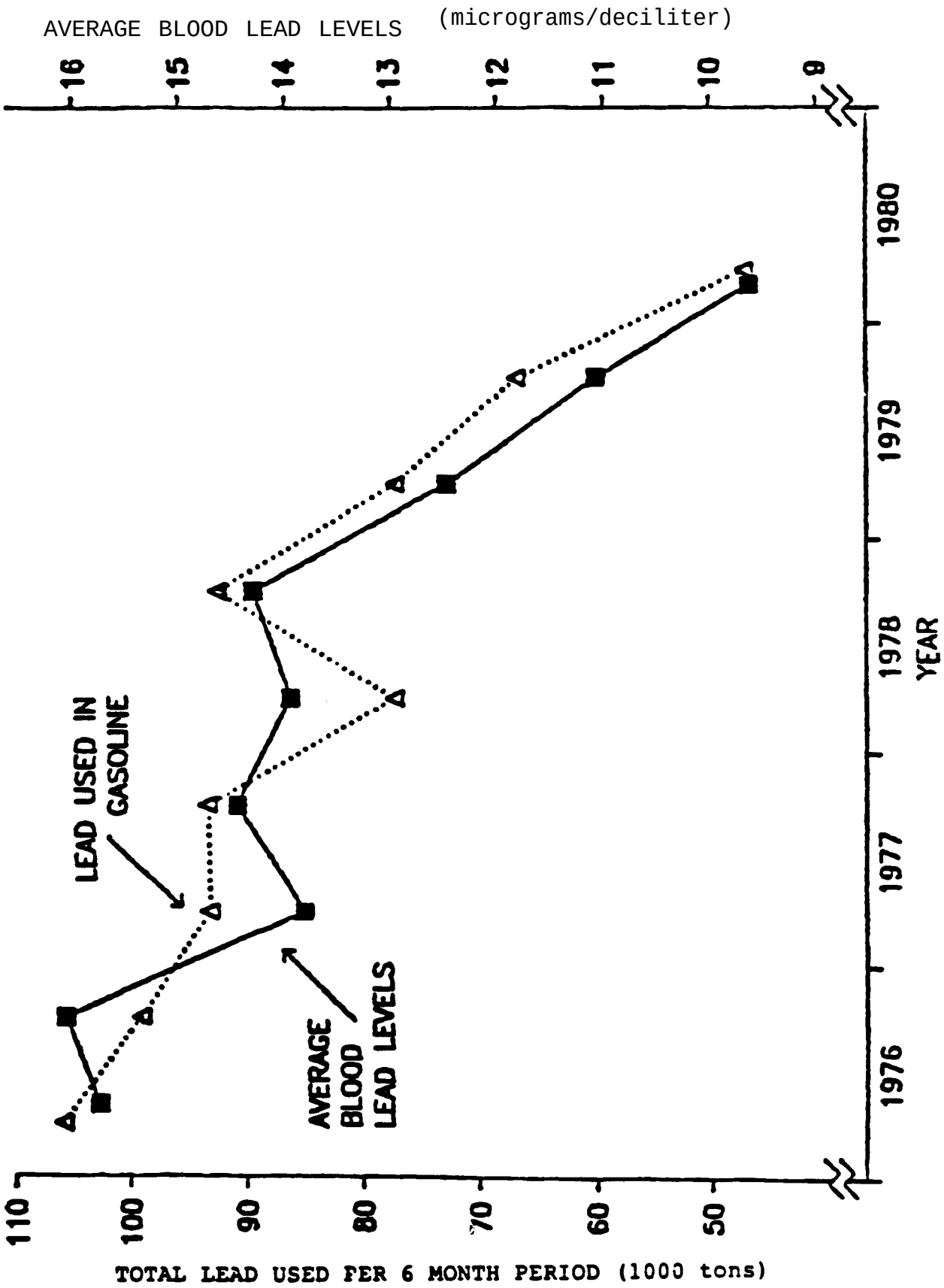
* Because children were less likely to respond (51 percent of the children did not provide blood for lead determinations in the NHANES II data set), they were double-sampled. The weights used to adjust the data to the national population accounted for both the oversampling and under-response of the children.

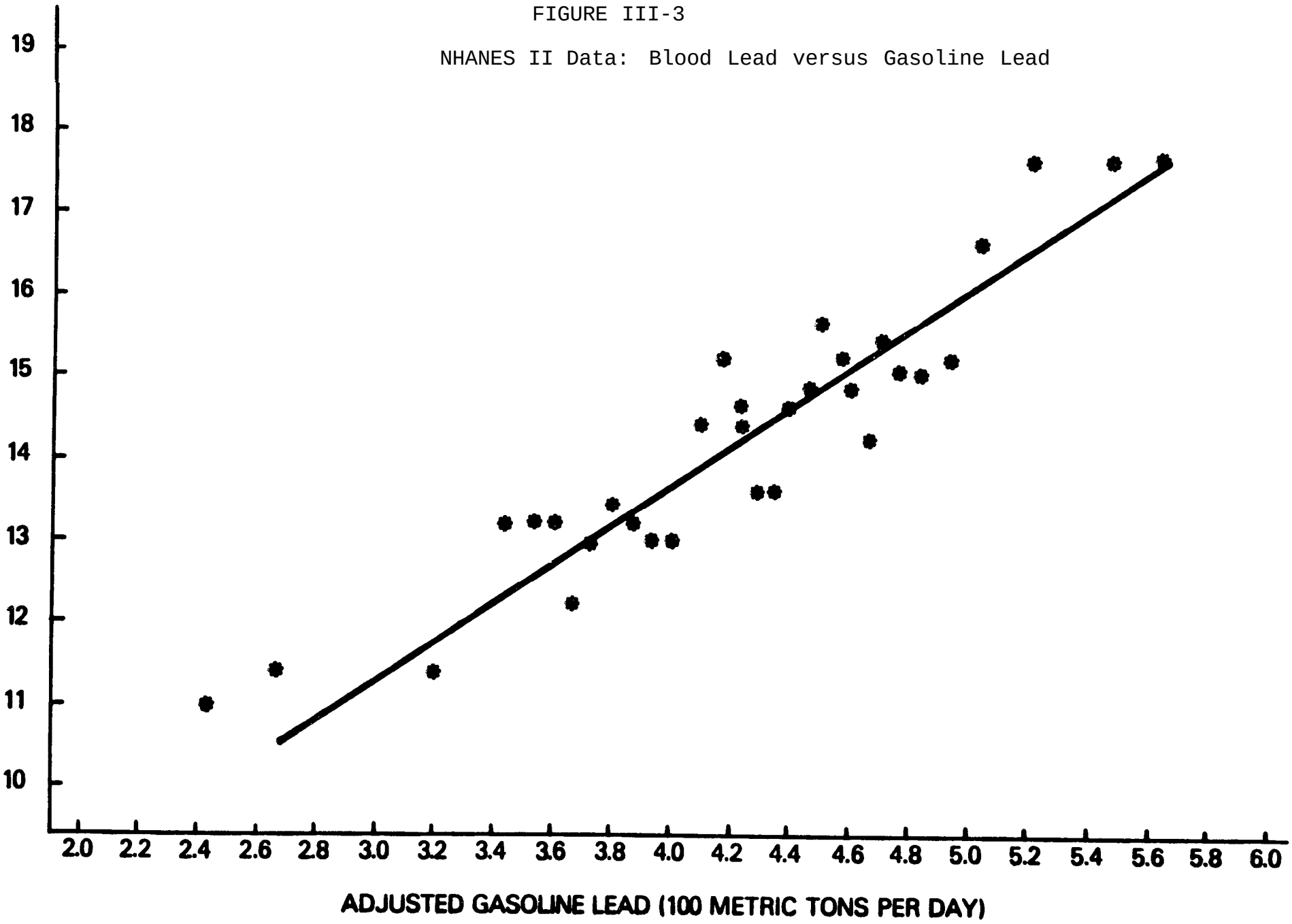


LEAD USED IN GASOLINE PRODUCTION AND AVERAGE NHANES II BLOOD LEAD LEVELS (FEB. 1976 – FEB. 1980)

FIGURE III-2

FIGURE III-3

NHANES II Data: Blood Lead versus Gasoline Lead



Note: Each point represents the average of approximately 310 observations. However the regression line shown is the true regression line for all 9987 individuals.

III.A.2.c. The CDC Screening Program for Lead Poisoning

During the 1970s, CDC provided funds for community-wide screening programs in many cities. Approximately 125,000 children were screened each quarter of the year. Results reported back to CDC included the number of children screened, the number with lead toxicity (defined as free erythrocyte protoporphyrin levels above 50 ug/dl and blood lead levels over 30 ug/dl), and the number with severe lead toxicity. Figure III-4 shows a plot of gasoline lead versus the percent of children with lead toxicity.

III.A.2.d. Chicago, New York, and Louisville, Kentucky Data

Billick analyzed detailed data from the screening programs in New York, Chicago, and Louisville, including the average blood lead levels as well as the percent of children with blood lead levels over 30 ug/dl. We have analyzed these data further, focusing particularly on the Chicago data, which include a 1-in-30 sample of the over 200,000 blood lead screening tests performed in Chicago between 1976 and 1980. Figures III-5 and III-6 show the relationship between gasoline lead and blood lead in Chicago and New York, respectively.

III.A.3. Statistical Analyses of Exposure

We used multiple regression analysis to examine both the relationship between gasoline lead and individual blood lead, and the relationship between gasoline lead and the probability of undue lead exposure (above 30 ug/dl) or lead toxicity (in the CDC data). For data from the NHANES II survey, we performed the analyses on individual blood lead measurements, with the explanatory

FIGURE III-4

CDC Data: Gasoline Lead versus Percent of Children with Lead Toxicity

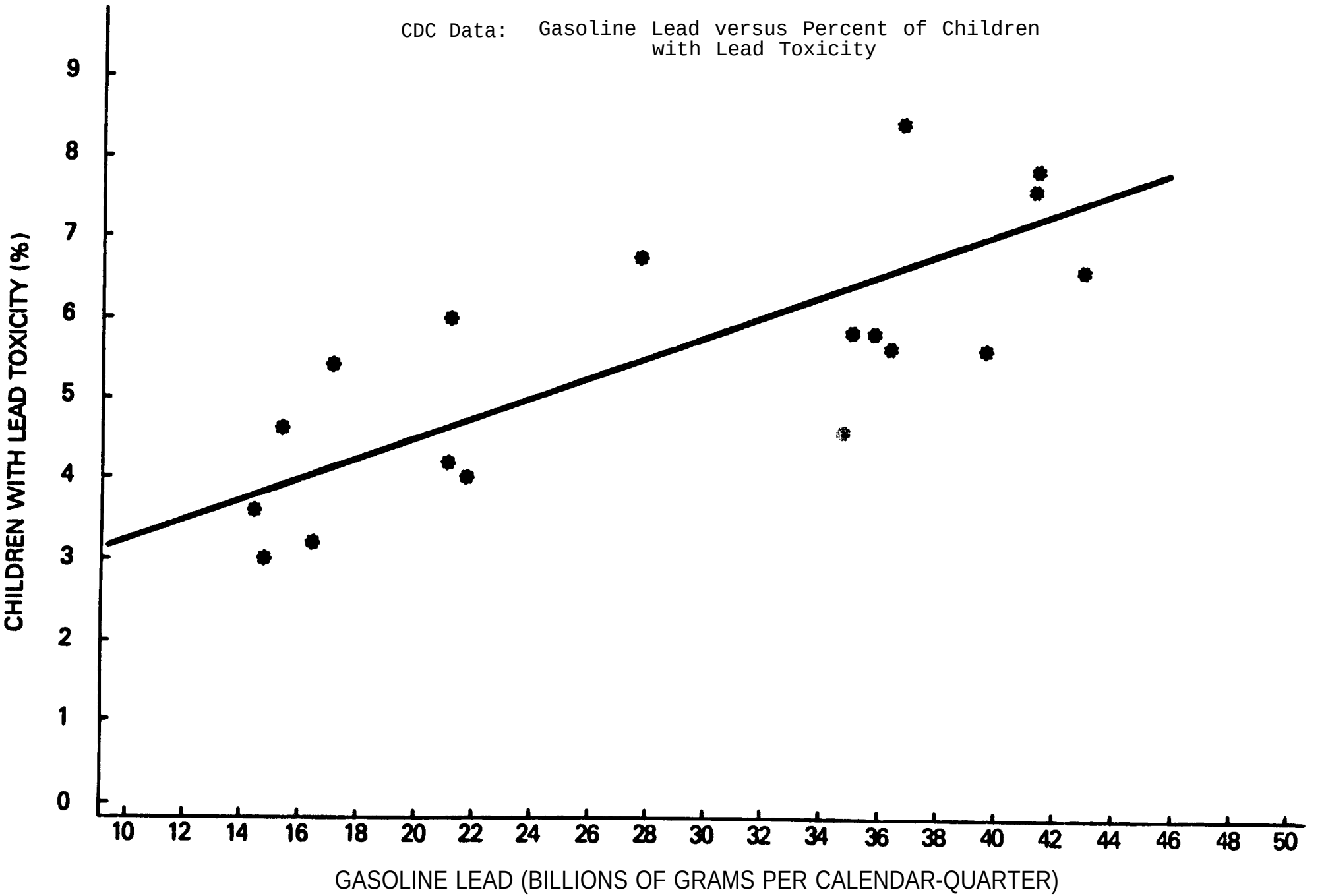


FIGURE III-5

Chicago Data: Gasoline Lead versus Blood Lead

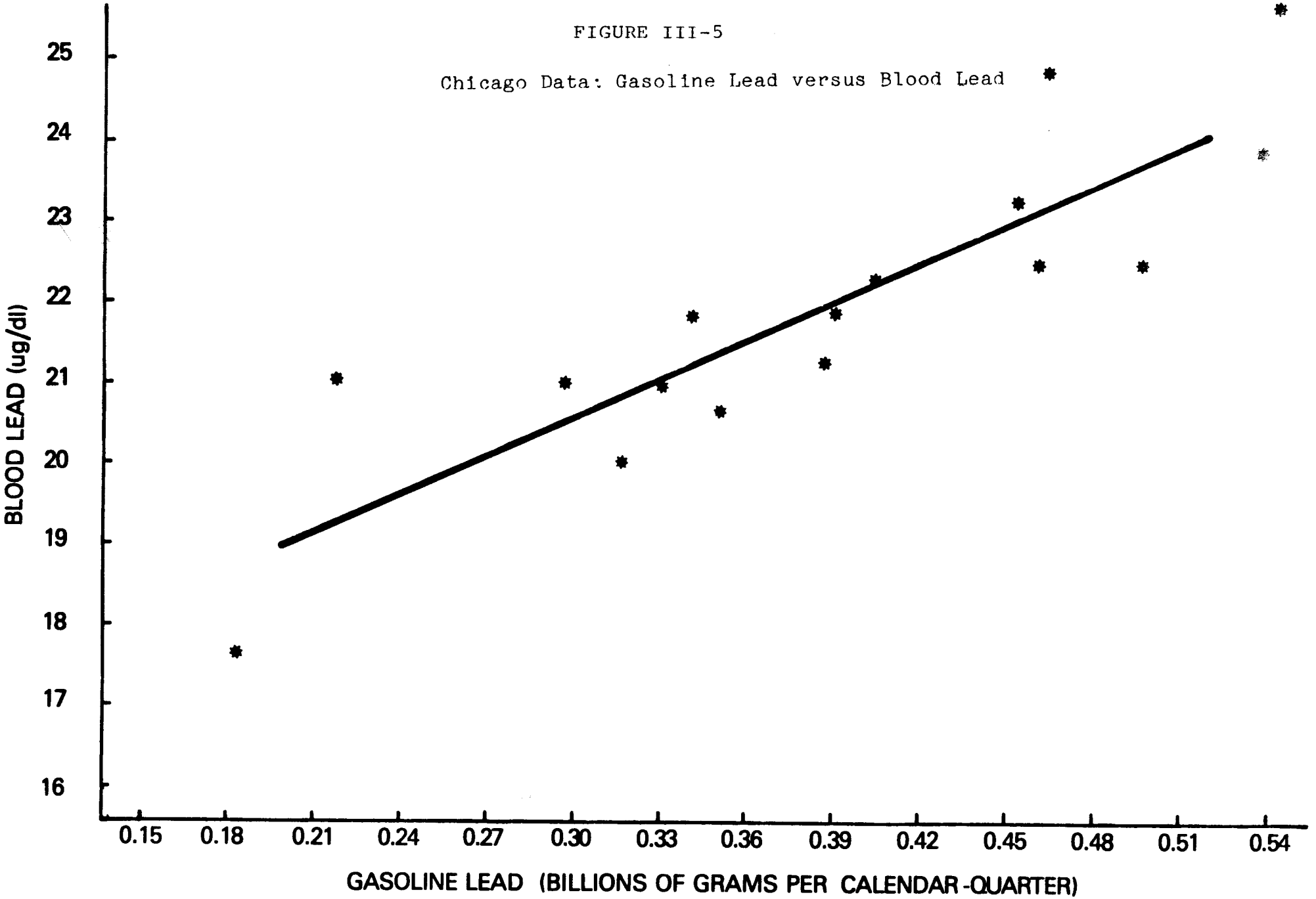
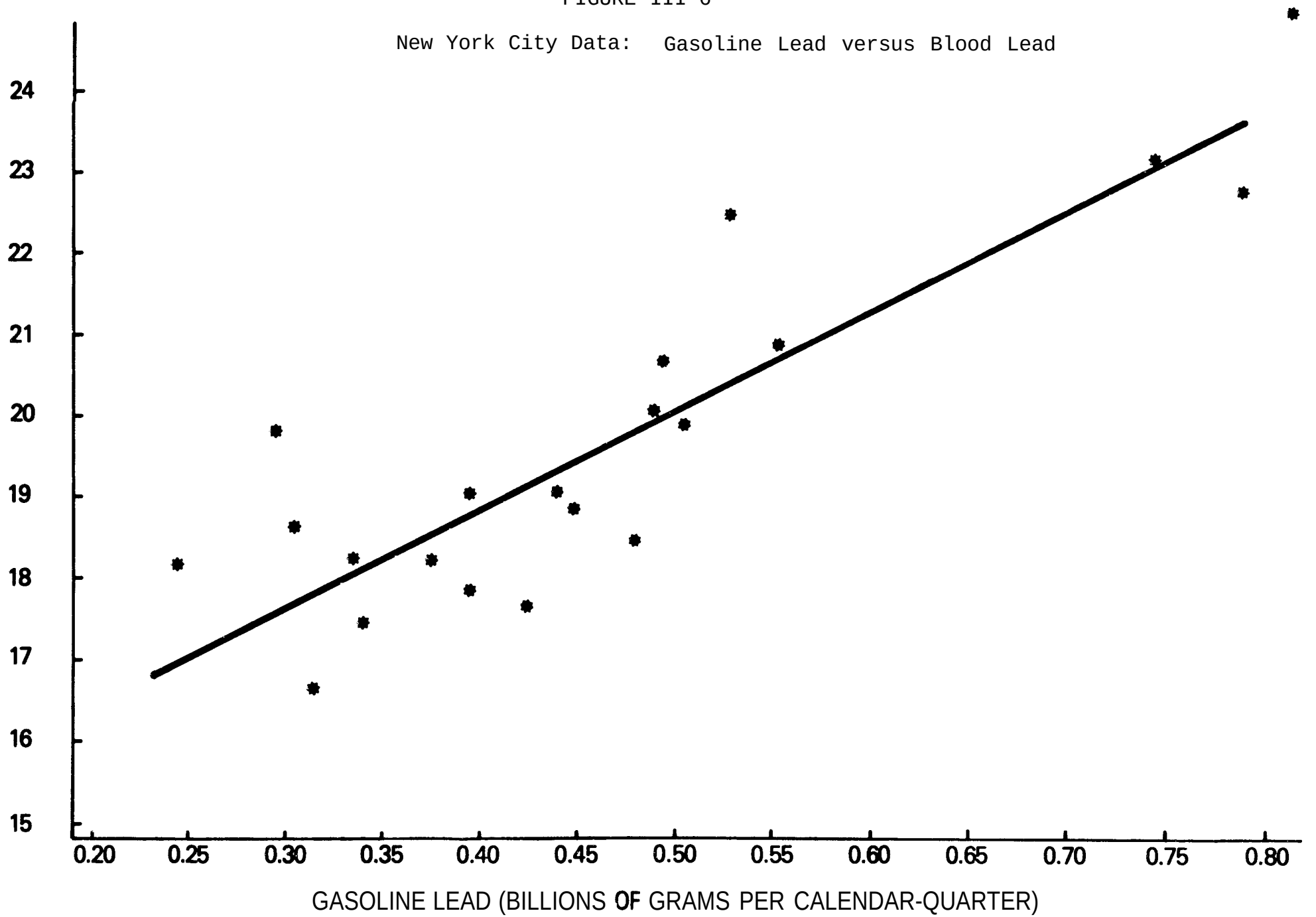


FIGURE III-6

New York City Data: Gasoline Lead versus Blood Lead



variables being gasoline lead, age, race, sex, degree of urbanization, alcohol consumption, smoking, occupational exposure, dietary factors, lead in canned food, region of the country, educational attainment, income, season of the year, and interactions among these variables. We also ran separate regressions for various age, race, and geographic groups. We weighted the results by the the inverse of the probability of selection to produce nationally representative estimates. The regressions were run using a program (SURREGR) designed to account for the stratified, clustered sampling procedure in the NHANES II survey, and many different specifications were considered.

Table III-1 presents the results for the linear regression of gasoline lead and other explanatory variables on blood lead levels. (See Appendix C.1 for definitions of the other variables.) Table III-2 presents the results for the logistic regression on the probability of blood lead level over 30 ug/dl for children aged six months to 7 years. Table III-1 shows that gasoline lead was a highly statistically significant predictor of blood lead levels ($p < 0.0001$; t-statistic > 60) even after accounting for potential confounding variables. The average value of gasoline lead usage was 426 metric tons per day, suggesting that an average of about 9 ug/dl of blood lead was due to gasoline lead during that period. Additional regression results on the NHANES II data, including results for specific subgroups used in later analyses, are in Appendix C.

For the CDC data, only quarterly data on the percent of children screened with and without lead toxicity were available.

TABLE III-1. NHANES II: Regression Results for Whites*

Effect	Coefficient	Standard Error	P-Value
Intercept	6.15		
Gasoline Lead	2.14	0.037	0.0000
Low Income	0.79	0.059	0.0025
Moderate Income	0.32	0.034	0.0897
Child (under 8)	3.47	0.125	0.0000
Number of Cigarettes	0.08	0.000	0.0000
Occupationally Exposed	1.74	0.063	0.0000
Vitamin C	-0.04	0.000	0.0010
Teenager	-0.30	0.05	0.1841
Male	0.50	0.19	0.2538
Male Teenager	1.67	0.26	0.0026
Male Adult	3.40	0.26	0.0000
Small City	-0.91	0.085	0.0039
Rural	-1.29	0.10	0.0003
Phosphorus	-0.001	0.00	0.0009
Drinker	0.67	0.03	0.0007
Heavy Drinker	1.53	0.10	0.0000
Northeast	-1.09	0.11	0.0028
South	-1.44	0.14	0.0005
Midwest	-1.35	0.25	0.0115
Educational Level	-0.60	0.02	0.0000
Riboflavin	-.188	0.005	0.0186
Vitamin A	0.018	0.000	0.0355

* The coefficients of the dummy variables show how much blood lead (in ug/dl) is, on average, attributable to a specific effect. The coefficient of gasoline lead shows the number of ug/dl of blood lead attributable to each 100 metric tons per day of gasoline lead use.

TABLE III-2. Logistic Regression on Probability of Blood Lead > 30 ug/dl for Children 6 months to 7 years

Logistic Regression Results*

Black children = under 8 years old, 479 observations

Dependent variable: 1 if blood lead is over 30 ug/dl; 0 otherwise

Model Chi square = 39.63 with 5 D.F.

Variable	Beta	Std. Error	Chi square	P
Intercept	-6.9468	1.2656	30.13	0.0000
Gaslead	0.8633	0.2452	12.40	0.0004
Poor	0.9815	0.2803	12.26	0.0005
Age 1	1.1404	0.6246	3.33	0.0679
Age 2	1.1938	0.5696	4.39	0.0361
Age 3	0.5428	0.5728	0.90	0.3433

Fraction of concordant pairs of predicted probabilities and responses = 0.718

White children = under 8 years old, 2225 observations

Dependent variable: 1 if blood lead is over 30 ug\dl; O otherwise

Model Chi square = 33.58 with 5 D.F.

Variable	Beta	Std. Error	Chi square	P
Intercept	-8.1667	1.2322	43.93	0.0000
Gaslead	0.6331	0.2160	8.59	0.0034
Poor	1.2174	0.2935	17.21	0.0000
Age 1	1.4332	0.7978	3.23	0.0724
Age 2	1.7168	0.7415	5.36	0.0206
Age 3	1.1405	0.7503	2.31	0.1285

Fraction of concordant pairs of predicted probabilities and responses = 0.637

*A1l logistic regression results were run using PROC LOGISTIC within the Statistical Analysis System (SAS). This procedure uses individual data where the dependent variable is one if the individual is above the threshold, and zero otherwise.

We performed both weighted linear and logistic regressions on the probability of lead toxicity as a function of gasoline lead.

Tables III-3a and III-3b show the results of regressing the percent of children with lead toxicity against gasoline lead for the 20 quarters between 1977 and 1981. "Dummy" represents the period before CDC modified its definition of lead toxicity in 1978. The correlation coefficients between gasoline lead and the percent of children with lead toxicity (0.8027) and between the change in gasoline lead and the change in the percent with lead toxicity (0.817) were both very large, and the regression coefficient was highly significant ($p < 0.0001$). (The magnitude of the coefficient is not directly comparable to the NHANES II both because of the difference in outcome variable -- percent toxic versus blood lead level -- and the difference in the units of gasoline lead.) The regressions predicted that if there had been no lead in gasoline at all, there would have been approximately 80 percent fewer cases of lead toxicity. This does not mean that 80 percent of the cases were due solely to gasoline lead, but rather that in 80 percent of the cases both gasoline lead and other exposures were required to bring children above the CDC definition of lead toxicity.

For the Chicago, New York, and Louisville data, we performed logistic and linear regressions of the probability of blood lead levels being over 30 ug/dl for each race separately, with explanatory variables being age, season, and gasoline lead. Gasoline lead was always significant, and explained the seasonal

TABLE III-3a. Regression of CDC Screening Data: Percent of Children with Lead Toxicity on Gasoline Lead

Variable	Coefficient	T-Statistic	Incremental R^2	P-Value
Constant	1.363	1.722	0	0.05
Gas Lead*	0.1601	5.322	0.5215	<0.001
Dummy	-1.036	-1.524	0.0427	0.07

$R^2 = 0.687$

Durbin Watson statistic = 1.786

Simple correlation coefficients:

	Gas Lead	Dummy
Percent Toxic	0.8027	0.4068
Gas Lead	—	0.6927

* Gasoline lead is in billions of grams per quarter. In the last quarter of 1978, gasoline lead use was 40 billion grams. By the second quarter of 1980, regulatoy action had reduced this to 20 billion grams.

TABLE III-3b. Regression of CDC Screening Data: Change in Lead Toxicity on Change in Gasoline Lead

Variable	Coefficient	T-Statistic	P-Value
Constant	0.3675	1.43	0.09
Delta Gas Lead	0.3938	5.84	<0.0001

$R^2 = .6670$

Durbin Watson statistic = 1.887

Simple correlation coefficient: 0.8167

variation in elevated blood lead levels. We also regressed quarterly average blood lead levels in Chicago and New York against gasoline lead, race, age, and season. Table III-4 shows the results of regressing the Chicago data for black children for the 18 quarters from 1976 until mid-1980. (Our results differ from Billick's in that he only had lead concentration values for two quarters of the year and had to interpolate the others. We had lead concentrations for all four quarters and, not surprisingly, using this better gasoline-lead data gave a stronger relationship.) Again, gasoline lead was an excellent predictor of children's blood lead levels ($p < 0.0001$). Autocorrelation corrections were run, and showed no significant correlation, nor did they produce a noticeable change in gasoline lead coefficients. Since the gasoline lead coefficient in Table III-4 was in billions of grams per quarter for the Chicago area, we had to adjust from local to national units to compare it to the NHANES II results. Scaling by the ratio of Chicago's gasoline use to the nation's, and converting to the units used in the NHANES II regressions (100 metric tons per day), the Chicago coefficient would correspond to a national coefficient of 2.08, which is essentially identical to the NHANES II results (a coefficient of 2.14, as shown in Table III-l).

Regressions on all of the data sets showed that gasoline lead was an extremely significant explanatory variable both for individual blood lead levels and for the percent of children with undue lead exposure or lead toxicity. Gasoline lead appeared to have accounted for 60 percent of the lead in Americans in the

TABLE III-4. Black Children in Chicago: Regression of Average Blood-Lead Levels on Gasoline Lead Levels

Variable	Coefficient	T-Statistic	Incremental R^2	P-Value
Constant	15.48	22.33	0	
Gas Lead*	17.02	11.27	0.5757	<0.0001
Age 1	0.85	1.53	0.0106	0.07
Age 2	1.68	3.03	0.0418	0.005
Age 3	1.01	1.84	0.0153	0.05
Age 4	0.66	1.20	0.0065	>0.10

$R^2 = 0.6194$

Durbin Watson statistic = 2.01

Simple correlation coefficients:

	Gas Lead	Age 1	Age 2	Age 3	Age 4
Average Blood Lead	0.7587	0.0009	0.01615	0.0337	0.0342
Gas Lead	—	0	0	0	0

*Gas Lead is lagged one month behind quarterly gasoline lead in billions of grams. The average for this period in Chicago was 0.379 billion grams.

second half of the 1970s, and to have explained both the seasonal increases in blood lead levels from winter to summer and the long-term drop in blood lead levels during the late 1970s. EPA refinery reports show that the rate of decline in gasoline lead accelerated after late 1978, and this was paralleled by an accelerated decline in blood lead levels.

Gasoline lead had the same coefficient in rural areas, in urban areas, and in urban areas with populations over one million. In the NHANES II regressions, a dummy variable for residence in central cities versus suburban areas was not statistically significant. The gasoline lead variable that best correlated with blood lead was gasoline sales for the preceding month.

III.B. The Question of Causality

While not crucial to a rule designed to be precautionary in nature, we did examine whether there is a causal relationship between gasoline lead and blood lead. In epidemiology there are several general criteria for determining whether association represents causality (Kleinbaum et al., 1982; Lilienfeld and Lilienfeld, 1980). The most useful criteria are:

Is there experimental evidence to support the findings?

Do several studies replicate the results?

Does a dose-response relationship exist?

Are there consistent effects in different types of studies?

Does cause precede effect?

Is the model biologically plausible?

Is it unlikely that other factors not included in the analysis would change the results?

We will address these issues in turn, with particular emphasis on the last one, as no study can ever measure all possible confounding factors.

III.B.1. Experimental Evidence

Facchetti and Geiss (1982) investigated the contribution of gasoline lead to blood lead in Turin, Italy during the late 1970s by changing the isotopic composition of the lead added to gasoline, and monitoring the isotopic composition of blood lead. This isotopic lead experiment indicated that changes in the isotopic composition of air lead followed closely and rapidly changes in the isotopic composition of gasoline lead. Changes in the isotopic composition of blood lead also paralleled changes in gasoline lead. Based on modeling of the results, Facchetti and Geiss estimated that at least 25 percent of the (high) blood lead levels in Turin were due to gasoline lead; this was at least 6 ug/dl. Since blood lead isotopic ratios were still changing when the gasoline isotopes were switched back, the actual impact of gasoline lead is probably higher.

Manton (1977) analyzed isotopic changes in blood lead in the United States and found the contribution of airborne lead (predominantly gasoline) was between 5 and 10 ug/dl in most of his subjects.

Tera et al. (1985) recently analyzed the isotopic ratios of blood lead in children in Washington, D.C. as the isotopic ratio of air lead changed. Their data showed that, as late as 1983, at least 38 percent of the lead in children's blood still came from gasoline lead, despite the 50 percent reduction in gasoline lead since 1978.

Our analysis of the impact of gasoline lead indicated that in the late 1970s, about 9 ug/dl of blood lead resulted from the lead in gasoline. This magnitude of effect is similar to that found in the isotope studies.

III.B.2. Does Cause Precede Effect?

One way to determine whether the trend in blood lead was caused by gasoline lead or was due to another variable is to examine the lag structure in light of our biomedical knowledge. The half-life of lead levels in the blood is about 30 days (Rabinowitz et al., 1976). Because the average blood test occurred on the 15th of the month, the current month's gasoline lead would have had only 15 days to affect blood lead levels, and so, though significant, should have a lesser impact. The previous month's gasoline lead, on the other hand, represents emissions on average 15 to 45 days prior to examination, and since direct inhalation and even dust exposure shows Up rapidly in the blood, we would expect this one-month lagged gas lead to be more significant, with a noticeably higher coefficient. Similarly, we expected gasoline sold two months previously to be less significant and of lesser magnitude. If gasoline sales were merely a proxy for time, however, all three months should be equally good predictors, since t, t-1, and t-2 (where t is the month since commencing the survey) equally represent the passage of time.

We regressed the individual blood lead levels in the NHANES II survey against current, one-month lagged, and two-month lagged gasoline lead simultaneously, and found one-month lagged gasoline lead was most significant and two-month lagged gasoline lead was

least significant. This suggested that the causal model was correct. The coefficient of two-month lagged gasoline lead was one-half that of one-month lagged gasoline lead, which matches the one month half-life of lead in the blood.

III.B.3. Replicability and Consistency

We replicated our analysis of the national NHANES II data with our analysis of the site-specific Chicago and New York screening data and with Billick's analysis of the screening data from Chicago, New York, and Louisville. In addition, Rabinowitz and Needleman (1983) have examined umbilical cord blood from over 11,000 consecutive births at Boston Women's Hospital between 1979 and 1981. They found a strong association ($p < 0.001$) with gasoline lead used in the Boston area, and also that one-month lagged gasoline lead had the highest correlation. No significant monthly variation was noted in the mothers' education levels, smoking, or drinking, and water lead levels increased somewhat over the period, while blood lead and gasoline lead levels fell. Thus, both local and national data from different studies, collected by different investigators, show the same pattern of gasoline lead being significantly related to blood lead.

The results of studies of both individual blood lead levels and average blood lead levels were consistent with analyses of elevated blood lead levels. Billick examined the probability of blood lead levels above 30 ug/dl in Chicago, New York, and Louisville, and found a strong relationship to gasoline lead. We repeated that analysis using logistic regressions, with the same results. Our investigation of the national CDC screening

program data, using both linear and logistic regressions, found the probability of lead toxicity was strongly dependent on the amount of lead in gasoline. Finally, we performed logistic regressions on the NHANES II data of the probability of both black and white children (under 8 years) and preteenagers (ages 8-14) being over 30 ug/dl of blood lead, and again found a strong relationship to gasoline lead levels.

III.B.4. Does a Dose-Response Relationship Exist?

To assure ourselves that the linear relationship we found was the true form of the dose-response relationship, we divided the NHANES sample in half. We repeated the regression for whites in the second half of the survey period, when the average gasoline lead levels were roughly 50 percent of those in the first half. The gasoline lead coefficient was essentially unchanged at 1.94 (compared to 2.14 for the full sample). This indicated a stable linear relationship.

For blacks, the sample size was too small to divide the sample, so we used an alternate procedure. We regressed log (blood lead) against demographic variables and log(gasoline lead), for a range of zero gasoline intercepts. This model chose the best exponent for the relationship blood lead = (gasoline lead)B. For the intercept (8 ug/dl) with the highest R^2, the exponent was 0.98, indicating a linear dose-response relationship. A square-root regression was also tried, and gave an inferior fit to the linear regression. The Chicago data, as noted before, gave the same magnitude of gasoline lead's effect on blood lead,

and visual examination of the regression plots in Figures III-3 to III-6 confirm the linearity in the relationship.

III.B.5. Biological Plausibility

Gasoline lead produced 90 percent of the emissions of lead into the air in the 1970s, and it was the major source of lead contamination of the environment. Lead is emitted as predominantly respirable particulate (less than 1 micron) from auto exhausts. Respirable particulate reach into the lung and show a high absorption rate. Gasoline lead is a major source of lead in street and household dust and in soil contamination and, therefore, in addition to direct inhalation, results in secondary exposure through the inhalation and absorption of dust. Reels (1976) has shown a high correlation between blood lead and lead on the hands, presumably from air and dust contamination. Lead is known to be absorbed from both the lung and gut. Thus, the fact that gasoline lead is related to blood lead is biologically plausible.

III.B.6. Control for Confounding Factors

Billick's analysis of children controlled for age and race, and our reanalysis controlled for season as well. The Annest et al. analysis of the NHANES II data controlled for age, race, sex, income, degree of urbanization, region of the country, and season.

We used several approaches to control for confounding factors. Where we had sufficient information, we included potential confounding factors, or surrogates for them, in our regressions. In addition, we examined external data to check for changes in

confounding factors that might bias our results, and performed several internal statistical checks to examine the likelihood of misspecification error.

III.B.6.a. External Validation

A great deal is known about the sources of lead exposure. The major general sources of body lead are food, water, and paint. While we have limited data on the specific sources of exposure of the individuals in our data sets, if all other general exposures were, on average, constant during the period, the effects of these will be part of the constant term in our regressions. Bias would occur only if these sources changed over the period.

The Food and Drug Administration's estimates of lead in the diet (based on market basket surveys) during the NHANES II period are shown on Table III-5. As there was no downward trend in dietary lead intake, this was unlikely to have been a potential source of bias in the model. Lead in canned food did change during the period, and we included a variable for this in our regression model. (See Appendix C.) Dietary variables were used to represent differences in food consumption between individuals, which were also accounted for by age, sex, race, income, education, and regional variables.

TABLE III-5. Lead in the Diet (micrograms/day)

Fiscal Year	Infants	Toddlers	Males aged 15-20
1976	21	30	71.1
1977	22	28	79.3
1978	25	35	95.1
1979	36	46	81.7
1980	--	--	82.9

The exposure to lead from water is predominantly a function of pH levels. The pH of drinking water supplies appeared to have been constant over the four to five-year period of our data.

Change in paint lead exposure was also an unlikely source of bias for several reasons. First, adult blood lead levels decreased by almost as much (37 percent vs. 42 percent) as children's blood lead, and adults generally do not eat paint. Second, because ingestion of paint lead usually results in the absorption of enough lead to produce large increases in blood lead, we would expect a drop in paint lead exposure to reduce blood lead levels only in people whose levels are above the mean. However, the drop in blood lead recorded in the NHANES II data shifted the entire distribution dramatically. Indeed, even low blood-lead groups showed major declines in blood lead. This would not have occurred if ingestion of paint lead were the determinant.

Third, the drop occurred across geographical boundaries, in suburbs as well as central cities. (Suburbs have a lower fraction of pre-1950 housing stock, and, therefore, inherently less expo-

sure to lead paint, yet they showed the same drop and the same gas lead coefficient.*) Finally, lead paint removal programs during this period reached only 50,000 of the 30 million housing units with lead paint (less than 0.2 percent), so exposure was unlikely to have changed in this period (Morbidity and Mortality Weekly Reports, 1976-1980).

To lay these issues to rest, however, we did an additional analysis of the Chicago data. For each year of the screening program, the CDC reported the percent of lead toxic children with a lead paint hazard in their home or the home of a close relative. In 1978, however, the homes of all screened children in Chicago (no matter what their blood lead level) were checked for lead paint hazard. This survey of over 80,000 housing units established the general prevalence of lead paint exposure in the screened population. With the probability of paint lead in the house given lead toxicity, and the probability of paint lead in the house in general, we used Bayes Theorem to compute the probability of lead toxicity given paint lead in the house, and the probability of lead toxicity when lead paint was not in the house. We then regressed these quarterly probabilities on gasoline lead. The results are shown in Table III-6.

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* Shier and Hall (1977) analyzed over 2,500 housing units in Pittsburgh and found that the fraction that had lead paint concentrations above 2 mg/cm2 decreased from over 70 percent in pre-1940 housing to about 43 percent in 1940-59 housing, and to 13 percent in post-1960 housing.

TABLE III-6. Chicago Probability of Blood Lead > 30 ug/dl With and Without Paint Lead Hazard in the Home

	Variable	Coefficient	t-Statistic	P-Value
Without Lead Paint Hazard				
	Constant	-2.087	-0.6583	0.26
	Gas Lead	35.398	4.423	0.0005
With Paint Lead Hazard				
	Constant	12.80	0.579	0.30
	Gas Lead	80.16	1.436	0.07

As we expected, gasoline lead was a more significant explanatory variable for children not exposed to lead paint than for the exposed group. Over 80 percent of the children were not exposed to lead paint at home, and among them gasoline lead was highly significant and explained most of the elevated blood lead levels. It was also striking that even among children with an identifiable lead paint hazard in their homes, gasoline lead was still strongly related to the probability of lead toxicity.

III.B.6.b. Seasonality

U.S. blood lead levels were strongly seasonal, with summer levels substantially higher than winter levels. However, when the NHANES II data were tested with seasonal variables, none of them was statistically significant, or even close to significant, when gasoline was included; the highest F-statistic for any season was 0.82. This indicated that the same gasoline lead

coefficient successfully explained both the short-term increases in blood lead levels from winter to summer and the long-term decrease in blood lead levels over the four-year period. The six regressions on the Chicago, New York, and Louisville data, as mentioned before, also indicated that seasonality was insignificant once gasoline lead was in the model. This suggested that other long-term trends in lead exposure cannot have biased the gasoline coefficient, as the short-term and long-term gasoline coefficients were the same.

III.B.6.c. Other Time Trends

To test the hypothesis that there was another unknown lead factor that was decreasing over the period, and whose effects might be attributed to gasoline in our regressions, we repeated our analysis with time as a variable; time was entered as the number of days after February 1, 1976 that a blood sample was taken.

The results indicated that time was not significant when gasoline lead was in the regression. Moreover, the effect of gasoline lead on blood lead was reduced by only 23 percent if we kept the insignificant time variable in. In addition, the Chicago data for average blood lead were analyzed with time as a variable. Here, again, gasoline lead was significant ($p < 0.02$) while time was insignificant ($p > 0.30$). We also tested time-squared on the CDC data, and it was insignificant.

III.B.6.d. Geographic Sampling Pattern

To be certain that the pattern of geographic sampling over time in the NHANES II period did not produce changes in blood lead

that the regression falsely attributed to gasoline, we included dummy variables for four regions of the country in our analysis. To check further, we inserted dummy variables for all 48 locations identified by the National Center for Health Statistics (NCHS). NCHS did not release locational data for 16 counties with populations below 100,000; these were all represented by one additional dummy variable. Rerunning the regressions with these identifiers changed the gasoline lead coefficient by only about 5 percent, which was insignificant.

This result was extraordinary because the NHANES II sampled different cities at different times, and the regression allowed the differences in blood lead levels over time to be attributed to changes in city location rather than gasoline lead. This meant that the coefficient of gasoline lead was heavily determined by the month-to-month changes in blood lead and gasoline lead within each city during the two months or so spent at each site. Yet the results did not change.

In addition, we performed a regression that had an interaction term between gasoline lead and the city identifiers. This procedure allowed a different slope in the gasoline lead/blood lead relationship for each county. The regression still yielded a coefficient for national gasoline lead of 1.83, with $p < 0.0001$.

Finally, the fact that analyses of many individual cities across the country (Boston, Chicago, New York, and Louisville) yielded similar results suggested geographic location was not an important source of bias.

III.B.6.e. Subgroup Analysis

To verify the robustness and stability of the relationship between gasoline lead and blood lead, we ran regressions of the NHANES II data for several demographic groups separately. This was done both to verify that we properly controlled for these variables, and to investigate whether the observed drop in blood lead levels was, in fact, due to a universal exposure such as gasoline lead, or could have been due to a source primarily affecting certain subgroups. The stability of the gasoline lead coefficient across these subpopulations reduced the likelihood of a specification bias.

This was because the bias introduced into the gasoline coefficient due to an omitted variable would be proportional to the regression coefficient that variable would have had if included. If the gasoline lead coefficient was insignificantly different among the regressions run for different subgroups, the omitted variable either must not have significantly biased the gasoline lead coefficient, or must have been coincidentally constant among all the demographic groups. The only known such variables were gasoline lead and food lead.

We performed separate regressions for males, females, adults, and children, for cities over one million, and for smaller urban areas. The maximum pair-wise difference in the gasoline lead coefficient among the six subgroups was less than 10 percent. In addition, we changed the definitions of large city (from over 1,000,000 to over 250,000), of rural (to include rural areas or cities under 10,000), and of the age categories. The coefficient

of gasoline lead changed by only 2 percent when we performed this regression. The stability of our findings, given the many additional tests we conducted, testifies to the robustness of the relationship between blood lead and gasoline lead. In addition, a study by Annest et al. (1983) of the U.S. Public Health Service, also using data from the NHANES II, found that the only reasonable explanation for the decline in blood lead levels was the decline in the amount of lead in gasoline. The Centers for Disease Control, the epidemiological branch of the Public Health Service, has also endorsed this relationship in its comments on this proposed rule (Public Docket EN 84-05) and the 1982 rule.

III.C. Impact of Rule on Numbers of Children Above Various Blood-Lead Levels

In estimating the benefits of reducing lead in gasoline, we relied on the regression coefficients estimated from the NHANES II data set because it is the largest and most representative of those available. Estimates of adult health benefits use the continuous functional forms because the relationship between blood lead and blood pressure is a continuous function of blood lead levels; their application is discussed in Chapter V. The benefit estimates for children, however, are functions of the numbers of children brought below various blood lead levels. Section III.C.1 describes how we used the results of the NHANES II logistic regressions to estimate changes in the numbers of children above these various levels. Section III.C.2 discusses the interpretation of these results with respect to prevalence and incidence.

III.C.1. Estimation Procedure

To estimate the numbers of children above different blood lead levels, we relied on logistic regressions estimated from the NHANES II data, of the type reported in Table III-2. These regressions were estimated separately for blacks and whites, and for each two-year age group from 6 months through 13 years. In each regression, the dependent variable was the natural logarithm of the odds of being above the level, while the independent variables were various demographic factors and gasoline lead.

To predict how the number of children above each level would change as the amount of lead in gasoline was reduced, a mechanism was needed to forecast the distribution of blood lead levels as a function of gasoline lead. In this analysis, we assumed that the distribution of blood lead would remain log-normal as gasoline lead levels declined. Then, estimates of the mean and variance of the associated (transformed) normal distribution could be used to determine the percentage of the population above any specific blood lead level. The estimates of the mean and standard deviation of the underlying normal distribution were derived from logistic regression estimates of the percentage of children with blood lead levels above 30 ug/dl and SURREGR estimates of the mean of the log-normal distribution using the Statistical Analysis System (SAS) procedure, SURREGR.

If the distribution ‘X’ is normal with mean ‘u’ and standard deviation ‘s’ (X:N (u,s)), then y = exp (X) is log-normal with a mean of ‘a’ and a standard deviation of ‘b’, where

(III-1) $a = \exp (u + 1/2\ s^2)$ and

(III-2) $b = \exp (2u + s^2) (\exp (s^2) - 1)$

Further, if e_g and v_g are the same percentiles of the log-normal and its corresponding normal distribution, respectively, we have

(III-3) $e_g = \exp (u + v_g\ s)$.

We used the logistic regressions to estimate e_g in equation (III-3) and the SURREGR regressions to estimate a in equation (III-l), which yielded

(III-4) $a = \exp (u + 1/2\ s^2)$

(III-5) $e_g = \exp (u + v_g\ s)$

Solving these equations for u and s produced a quadratic equation:

(III-6) $0 = (\ln (e_g) - \ln (a)) - v_g\ s + .5s^2$

which had the solutions $= v_g \pm [v_g^2 - 2 (\ln (e_g) - \ln (a))]^{0.5}$.

Only the smaller root yielded sensible values for u and s. The n $u = \ln(a) - 1/2\ s^2$. Using the estimated values for u and s, we determined percentages of the distribution above 10, 15, 20, and 30 ug/dl by looking up the results of (ln (10) - u)/s, etc., in the normal table.

We used a logistic regression equation to estimate the percentage of children over 30 ug/dl to control for problems of multiple sources of exposure. If we had simply used the regressions explaining the mean and assumed a constant standard devia-

tion, we would have predicted that removing lead from gasoline would have resulted in there being no children above 30 ug/dl. This seemed unreasonable because paint and food are known alternate sources of lead, and also are associated with high blood lead levels. The logistic regressions confirmed that the geometric standard deviation changes as the mean falls. Because of the sensitivity of the blood lead distribution to age, we estimated separate distributions for each two-year age interval. The tabulated changes in the numbers of children above various levels represented the sum of distributions for each age category. The regression results are shown in Appendix C.

For children from six months to seven years old, we used logistic regressions for the percent above 30 ug/dl of blood lead. For children aged eight to thirteen, we used logistic regressions for the percent above 20 ug/dl blood lead because there were too few observations above 30 ug/dl for the logistic procedure to yield accurate estimates.

Table III-7 presents the estimated reductions in the numbers of children over various blood lead levels from 15 ug/dl to 30 ug/dl. The estimates are presented for three phasedown schedules: the original proposal (0.10 gplg starting 1/1/86); the alternative discussed in the NPRM (0.50 gplg on 7/1/85, 0.30 on 1/1/86, 0.20 on 1/l/87, and 0.10 on 1/1/88); and the final rule (0.50 gplg on 7/1/85 and 0.10 on 1/1/86). All of the estimates assume that misfueling is totally eliminated. (The impacts of alternative assumptions about misfueling are explored in Chapter VIII.)

TABLE III-7. Estimated Reductions in Numbers of Children Over Various Blood Lead Levels, Assuming No Misfueling (thousands of children)

Blood Lead Level Rule	1985	1986	1987	1988	1989	1990	1991	1992
30 ug/dl								
Proposed	0	52	47	43	39	36	32	31
Alternative	22	46	45	43	39	36	32	31
Final	22	52	47	43	39	36	32	31
25 ug/dl								
Proposed	0	172	157	144	130	119	106	103
Alternative	72	154	149	144	130	119	106	103
Final	72	172	157	144	130	119	106	103
20 ug/dl								
Proposed	0	563	518	476	434	400	357	348
Alternative	232	501	491	476	434	400	357	348
Final	232	563	518	476	434	400	357	348
15 ug/dl								
Proposed	0	1,726	1,597	1,476	1,353	1,252	1,125	1,098
Alternative	696	1,524	1,508	1,476	1,353	1,252	1,125	1,098
Final	696	1,726	1,597	1,476	1,353	1,252	1,125	1,098
10 ug/dl								
Proposed	0	4,949	4,595	4,261	3,918	3,637	3,283	3,215
Alternative	1,972	4,354	4,333	4,261	3,918	3,637	3,283	3,215
Final	1,972	4,949	4,595	4,261	3,918	3,637	3,283	3,215

III.C.2. Incidence Versus Prevalence

Our predicted decrease in the number of children above a given threshold is for a specific point in time; our cost estimates are for an entire year. If children remain above 30 ug/dl for less than a year, there will be more children above 30 ug/dl in a year than we estimated and our benefits will be understated. Conversely, if children remain above 30 ug/dl for more than a year, these cases may be counted twice and we will overstate benefits.

This raises the difficult epidemiological issue of prevalence versus incidence. Prevalence is the percent of people who have the condition of interest at a particular time (e.g., the percent of people with the flu on February 14). Incidence is the percent of people who develop new cases of the flu in a given time period (e.g., the month of February). Prevalence is incidence times average duration.

This issue is important because the NHANES II survey, upon which we based our regressions, measured the prevalence of cases above 30 ug/dl blood lead or other thresholds, rather than the incidence. Yet the benefits we want to estimate would, in fact, be reduced numbers of cases in a time period, i.e., incidence.

Clearly an excursion of a child's blood lead level above 30 ug/dl for a day or two will produce less damage than a prolonged elevation. However, data indicate that such occurrences are not very likely. Odenbro et al. (1983) in Chicago found fairly stable blood lead levels in individual children with high

levels. For these children, levels remained high for more than a few days, usually for months or years. However, if the average duration of elevated blood lead was six months, the actual number of children affected in a year would be twice the average prevalence for the year. This obviously would affect our benefit estimates.

Because, as discussed in Chapter IV, we only valued cognitive losses for children in CDC categories III and IV,* and because data from Odenbro et al. suggested that such children's blood lead levels remain elevated for a long time unless treated, we believe our prevalence estimate is reasonable for estimating cognitive effects. Medical management costs, on the other hand, seem more reasonably associated with incidence.

In any case, it was necessary to determine the duration of elevated lead levels. To do this we evaluated several available pieces of information. They all suggested that the average duration was less than one year, so that our estimate of prevalence (based on the NHANES data) understated annual incidence.

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* CDC classifies children as "lead toxic" if they have blood lead levels above 30 ug/dl and free erythrocyte protoporphyrin (FEP) levels above 50 ug/dl. (FEP is a measure of the derangement of the heme synthesis process caused by lead.) Children with blood lead levels between 30-49 ug/dl but with FEP under 50 are in CDC category lb. Children between 30-49 ug/dl blood lead and 50-109 ug/dl FEP are category II. Category III children have blood lead levels between 30 and 49 ug/dl and FEP levels between 110 and 249 ug/dl or blood lead levels between 50 and 69 ug/dl and FEP levels between 50 and 249 ug/dl. Category IV includes all children with blood lead greater than 70 ug/dl, and children with blood lead between 60 and 69 ug/dl and FEP levels above 250 ug/dl.

Our first source was the CDC screening program data. This program screened about 100,000 to 125,000 children per quarter of the year to detect lead toxicity. Approximately 6,000 to 7,000 cases were found each quarter; this established the general prevalence of lead toxicity in the screening population. However, this prevalence rate showed strong within-year variation, with levels much higher in the third quarter, summer, when gasoline consumption was also highest. This intra-year variation suggested that the average duration was not so long that the effects of quarterly changes in exposure were swamped by cases that originated in earlier quarters.

We also have used the CDC lead screening data in another way. CDC reported, quarterly, the number of children under pediatric management, which included all the new cases discovered during that quarter plus the children remaining under pediatric management who had been discovered with lead toxicity in the previous quarters. We compared that number to the sum of the cases detected in the same quarter plus the previous two quarters and found the results were quite close. This suggested that children remained under pediatric management for an average of three quarters. However, children generally had several medical visits after their blood levels returned to normal to ensure that the decline was real. This implied that the average duration of blood lead levels above 30 ug/dl was even shorter, closer to two quarters. If this is true, then it is possible that we have underestimated the annual incidence of cases of children above 30 ug/dl by as much as a factor of two.

The amount of time it takes for lead toxicity percentages to respond to fluctuations in gasoline lead levels also may help to determine the duration of lead toxicity. If this time is relatively short (e.g., a few months or less), it is unlikely that duration would extend beyond a year. For lead toxicity to last a year or more, one would expect lead toxicity levels to be relatively insensitive to intra-annual variation in gasoline lead. The CDC data, which show such variation, suggest that the average duration is substantially less than one year.

Two other data sets supported the conclusion of a short lag between gasoline lead and blood lead levels. First, in the NHANES II data, we examined both the lag structure of blood lead's relationship to gasoline lead, and whether any seasonal dummy variables were significant in explaining the large observed seasonal variations in blood lead. Schwartz et al. (1983) found that the lag structure of average blood-lead levels' dependence on gasoline lead could extend to three months.

In addition, Billick (1982) examined the results of the screening programs for lead toxicity in Chicago (800,000 children screened) and in New York City (450,000 children screened) over a 10-year period and found a strong seasonal pattern in the number of children with lead toxicity. This pattern followed the seasonal variation of gasoline use. When Schwartz et al. (1984b) analyzed these data in a logistic regression, gasoline explained the cyclical variation in blood lead levels, with no seasonal variable obtaining a p-value of better than 0.38.

All of this suggested that the average time a child spent above 30 ug/dl was short enough so that quarterly prevalence rates corresponded well to quarterly exposure incidence. Therefore, our estimate of the annual incidence of children above 30 ug/dl is likely to be low, as is our estimate of avoided medical expenses.

CHAPTER IV

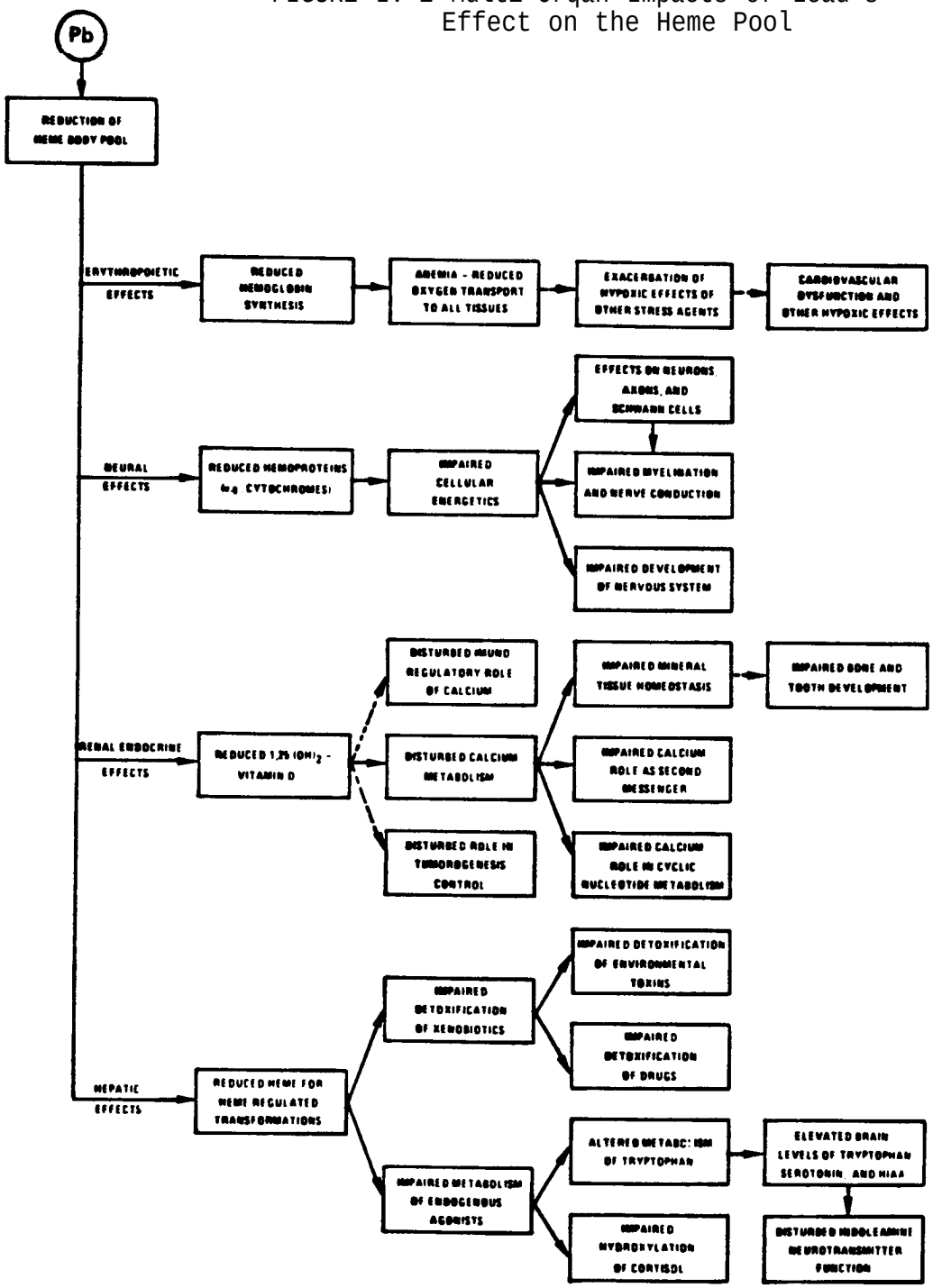
BENEFITS OF REDUCING CHILDREN'S EXPOSURE TO LEAD

The scientific literature presents evidence of a wide range of physiological effects associated with exposure to lead. These range from relatively subtle changes in various biochemical measurements at very low levels of exposure, with uncertain implications for health, to severe retardation and even death at very high levels of exposure. Although such effects are found in individuals of all ages, particular concern has focused on children, because they appear to be at greater risk.

Because the body is a complex structure of interdependent systems and processes, effects upon one component will have cascading implications throughout the body. This interdependence is well illustrated by multi-organ impacts resulting from the inhibition of heme by lead, and consequent reduction in the body heme pool. These effects are depicted graphically in Figure IV-1, taken from EPA's most recent Draft Lead Criteria Document (p. 13-31).

This chapter summarizes the available evidence of the effects of lead on children, and develops rough estimates of the benefits of reducing exposure to lead by reducing lead in gasoline. Section A deals with the pathophysiological effects of lead, while Section B addresses the evidence on neuropsychological effects (primarily reduced cognitive ability). The final section discusses the methods used to monetize the benefits of reducing children's exposure to lead. Although these estimates in no way cover the complete range of potential benefits, they total several hundred million dollars per year for the rule being promulgated.

FIGURE IV-1 Multi-Orqan Impacts of Lead's Effect on the Heme Pool



Multi-organ impact of reductions of heme body pool by lead. Impairment of heme synthesis by lead (see Section 12.3) results in disruption of a wide variety of important physiological processes in many organs and tissues. Particularly well documented are erythropoietic, neural, renal-endocrine, end hepatic effects indicated above by solid arrows (⟶). Plausible further consequences of heme synthesis interference by lead which remain to be more conclusively established are indicated by dashed arrows (⇢).

IV. A. Pathophysiological Effects

Elevated blood lead levels have long been associated with neurotoxicological effects and many other pathological phenomena: an article on lead's neurotoxicity was published as early as 1839, on anemia in the early 1930s, on kidney damage in 1862, and on impaired reproductive function in 1860. From an historical perspective, lead exposure levels considered acceptable for either occupationally-exposed persons or the general population have been revised downward steadily as more sophisticated biomedical techniques have shown formerly unrecognized biological effects, and as concern has increased regarding the medical and social significance of such effects. In the most recent downward revision of maximum safe levels, the Centers for Disease Control (CDC) lowered its definition of lead toxicity from 30 ug/dl blood lead and 50 ug/dl of free erythrocyte protoporphyrin (FEP) to 25 ug/dl blood lead and 35 ug/dl FEP. The present literature shows biological effects as low as 10 ug/dl (for heme biosynthesis) or 15 ug/dl (for renal system effects and neurological alterations).

There is no convincing evidence that lead has any beneficial biological effect in humans (Expert Committee on Trace Metal Essentiality, 1983).

The finding of biological effects at blood lead levels as low as 10 ug/dl potentially has important implications for public health, as such levels are common in the U.S. population. As Table IV-1 shows, between 1976 and 1980 over three-quarters of children under the age of 18 had blood lead levels in excess of 10 ug/dl, and 15 percent exceeded 20 ug/dl. The rates among blacks

TABLE IV-1. Blood Lead Levels Of Children in the United States 1976-80 (percent in each cell; rows sum to 100 percent)

	<10 ug/dl	10-19 ug/dl	20-29 ug/dl	30-39 ug/dl	40-69 ug/dl
All Races					
all ages	22.1	62.9	13.0	1.6	0.3
6 months-5 years	12.2	63.3	20.5	3.5	0.4
6-17 years	27.6	64.8	7.1	0.5	0.0
White					
all ages	23.3	62.8	12.2	1.5	0.3
6 months-5 years	14.5	67.5	16.1	1.8	0.2
6-17 years	30.4	63.4	5.8	0.4	0.0
Black					
all ages	4.0	59.6	31.0	4.1	1.3
6 months-5 years	2.7	48.8	35.1	11.1	2.4
6-17 years	8.0	69.9	21.1	1.0	0.0

Source : Table 1, Advance Data #79, May 12, 1982, from Vital and Health Statistics, National Center for Health Statistics (Supplemental Exhibit 4.) NOTE : These results were produced after adjusting the data for age, race, sex, income, degree of urbanization, probability of selection, and non-response to the NHANES survey.

and among preschool children were even higher.

Lead's diverse biological effects on humans and animals are seen at the subcellular level of organellar structures and processes, and at the overall level of general functioning that encompasses all of the bodily systems operating in a coordinated, interdependent way. The biological basis of lead toxicity is its ability, as a metallic cation, to bind to bio-molecular substances crucial to normal physiological functions, thereby interfering with these functions. Some specific biochemical mechanisms involve lead's competition with essential metals for binding for sites, inhibition of enzyme activity, and inhibition or alteration of essential ion transport. The effects of lead on certain subcellular organelles, which result in biochemical derangements common to and affecting many tissues and organ systems (e.g., the disruption of heme synthesis processes), are the origin of many of the diverse types of lead-based functional disruptions of organ systems.

Lead is associated with a continuum of pathophysiological effects across a broad range of exposures. In addition to the high level effects mentioned above, there is evidence that low blood-lead levels result in:

1. Inhibition of pyrimidine-5'-nucleotidase (Py-5-N) and delta-aminolevulinic acid dehydrase (ALA-D) activity, which appears to begin at 10 ug/dl of blood lead or below (Angle et al., 1982). Hernberg and Nikkanen (1970) found 50 percent of ALA-D inhibited at about 16 ug/dl.

2. Elevated levels of zinc protoporphyrin (ZPP or FEP) in erythrocytes (red blood cells] at about 15 ug/dl. This probably indicates a general interference in heme synthesis throughout the body, including inter-

ference in the functioning of mitochondria (Piomelli et al., 1977).

3. Changes in the electrophysiological functioning of the nervous system. This includes changes in slow-wave electroencephalogram (EEG) patterns and increased latencies in brainstem and auditory evoked potentials (Otto et al., 1981, 1982, 1984) which begin to occur at about 15 ug/dl. The changes in slow-wave EEG patterns appear to persist over a two-year period. Also, the relative amplitude of synchronized EEG between left and right lobe shows effects starting at about 15 ug/dl (Benignus et al., 1981). Finally, there is a significant negative correlation between blood lead and nerve conduction velocity in children whose blood lead levels range from 15 ug/dl to about 90 ug/dl (Landrigan et al., 1976).

4. Inhibition of globin synthesis, which begins to appear at approximately 20 ug/dl (White and Harvey, 1972; Dresner et al., 1982).

5. Increased levels of aminolevulinic acid (ALA) in blood and soft tissue, which appear to occur at about 15 ug/dl and may occur at lower levels (Meredith et al., 1978). Several studies indicated that increases of ALA in the brain interfered with the gamma-aminobutyric acid (GABA) neurotransmitter system in several ways (Draft Criteria Document, p. 12-128 ff).

6. Inhibition of vitamin D pathways, which has been detected as low as 10 to 15 ug/dl (Rosen et al., 1980a, 1980b; Mahaffey et al., 1982). Further, as blood lead levels increased, the inhibition became increasingly severe.

These data cite the lowest observed effect levels to date, and do not necessarily represent affirmative findings of thresholds below which exposures can be considered safe.

The types of specific effects listed above as occurring at blood lead levels below 25 ug/dl indicate (a) a generalized lead impact on erythrocytic pyrimidine metabolism, (b) a generalized lead-induced inhibition of heme synthesis, (c) lead-induced interference with vitamin D metabolism, and (d) lead-induced perturbations in central and possibly peripheral nervous system

functioning. The medical significance of all these effects is not yet fully understood. However, current knowledge regarding the deleterious and vital nature of the affected physiological functions both individually and in the aggregate suffices to warrant both public health concern and efforts to minimize their occurrence due to lead exposure.

As lead exposure increases, the effect on heme synthesis becomes more pronounced and effects broaden to additional biochemical and physiological mechanisms in various tissues, causing more severe disruptions of the normal functioning of many organ systems. At very high lead exposures, the neurotoxicity and other pathophysiological changes can result in death or, in some cases of non-fatal lead poisoning, long-lasting sequelae such as mental retardation and severe kidney disease.

This chapter discusses the known pathophysiological effects of lead that occur in children, particularly the hematological and subcellular neurotoxic effects, and the expected change in the number of children at potential risk of those effects under EPA's regulation. EPA is considering these effects in its current National Ambient Air Quality Standard process.

IV.A.1. Effects of Lead on Pyrimidine Metabolism

The best-known effect of lead on erythrocytic pyrimidine metabolism is the pronounced inhibition of Py-5-N activity. This enzyme plays a role in the maturation of erythrocytes, as well as erythrocyte function and survival; it controls the degradation and removal of nucleic acid from the maturing cell (reticulocyte).

Interference with this process can increase red cell membrane fragility. As noted earlier, the disruption of this function by lead has been noted at exposure levels beginning from 10 ug/dl. At blood lead levels of 30-40 ug/dl, this disturbance is sufficient to materially contribute to red blood cell destruction and, possibly, decreased hemoglobin production contributing to anemia (World Health organization, 1977; National Academy of Sciences, 1972; Lin-Fu, 1973; Betts et al., 1973). The significance of this interference with pyrimidine metabolism transcends the red cell; the mechanism of this inhibition suggests a wide-spread impact on all organs and tissues.

IV.A.2. Effects on Heme Synthesis and Related Hematological Processes

IV.A.2.a. Mitochondrial Effects

The mitochondrion is the critical target organelle for lead toxicity in a variety of cell and tissue types, followed probably by cellular and intracellular membranes. The scientific literature shows evidence of both structural injury to the mitochondrion (e.g., Goyer and Rhyne, 1973; Fowler, 1978; Fowler et al., 1980; Bull, 1980) and impairment of basic cellular energetic and other mitochondrial functions (e.g., Bull et al., 1975; Bull, 1977, 1980; Holtzman et al., 1981; Silbergeld et al., 1980).

IV.A.2.b. Heme Synthesis Effects

The best-documented effects of lead are upon heme biosynthesis. Heme, in addition to being a constituent of hemoglobin, is an obligatory constituent for diverse hemoproteins in all tissues, both neural and non-neural. Hemoproteins play

important roles in generalized functions, such as cellular energetics, as well as in more specific functions such as oxygen transport and detoxification of toxic foreign substances (e.g., the mixed-function oxidase system in the liver). Available data on elevated aminolevulinic acid (ALA) and free erythrocyte protoporphyrin (FEP) levels, inhibited ALA-D, and the like show inhibition in the heme biosynthetic pathway at low blood-lead levels, with statistically significant effects detectable at 10-15 ug/dl (Meredith et al., 1978; Piomelli et al., 1982; Angle et al., 1982). This heme biosynthetic disturbance may result in the impairment of many normal physiological processes in a host of organ systems and/or the reduced reserve capacity of many cells or organs to deal with other types of stress (e.g., infectious diseases).

The interference of lead with heme synthesis in liver mitochondria appears to result in the reduced capacity of the liver to break down tryptophan, which, in turn, appears to increase levels of tryptophan and serotonin in the brain (Litman and Correia, 1983). Such elevation of neurotransmitter levels may be responsible for some of the neurotoxic effects of lead, since elevated tryptophan levels have been associated with encephalopathy, and elevated serotonin levels produce necrologic symptoms similar to acute porphyria attacks.

The elevation of ALA levels is another indication of lead's interference in heme synthesis and mitochondrial functioning. Such elevations can have serious neurotoxic implications. In vitro studies have shown that ALA can interfere with several physiological processes involved in the GABA-ergic neurotrans-

mitter system, including a possible role as a GABA-agonist (Brennan and Cantrill, 1979). There appears to be no threshold concentration for ALA at the neuronal synapse below which presynaptic inhibition of GABA release ceases.

Since ALA passes the blood brain barrier and is taken up by brain tissue, it seems likely that elevated ALA levels in the blood correspond to elevated ALA levels in the brain (Moore and Meredith, 1976). Furthermore, lead in the brain is likely to enhance brain ALA concentrations because neurons are rich in mitochondria, the subcellular site of ALA production. As mentioned earlier, blood ALA elevations begin to be detectable at 18 ug/dl of blood lead (Meredith et al., 1978).

IV.A.2.c. Impact of Lead on Red Blood Cell Abnormalities

High levels of blood lead (> 40 ug/dl) are known to produce anemia. To examine the association between lead and red cell abnormalities in individuals below 30 ug/dl, we performed two analyses. First, we examined the relationship between blood lead levels and various red cell indices. Second, because FEP is a more stable indicator of a person's lead exposure over several months than a single blood lead measurement, as well as their sensitivity to lead, we also analyzed the relationship between elevated FEP levels and anemia. We found that blood lead levels below 30 ug/dl were associated with increased risks of microcytosis and hypochromia, and that FEP levels were associated with increased risks of anemia in children, even below 35 ug/dl of FEP.

For our analysis we used data from the NHANES II survey. Among the hematological information collected were mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), serum iron, hematocrit, FEP, and percent transferrin saturation. We used regression analysis of these data for 1,967 children under the age of eight to determine whether there was a relationship between blood lead levels and the presence of hematological abnormalities.

IV.A.2.c.l. Effects of Lead Exposure on Blood Cell Volume and Hemoglobin Content

We found that blood lead was inversely related to both mean cell volume (MCV) and mean cell hemoglobin (MCH), even for blood lead levels below the current CDC 25 ug/dl guideline for determining lead toxicity. Linear regressions were performed of MCV and MCH on blood lead levels in children, controlling for race, age, family-income, iron status (i.e., the level of iron in their blood), degree of urbanization, and other nutritional factors. (As previous work led us to expect, percent transferrin saturation was a superior control for iron status compared to serum iron and was used throughout our analysis.)

Table IV-2 lists the variables considered in the regressions. Income was found not to be a significant confounding variable once we controlled for iron status, and was dropped from the analysis. Race also had no bearing on MCV once iron status was controlled for, although it was a significant explanatory variable for MCH. This suggested that there may be additional dietary or biochemical factors predisposing black children to lower erythrocyte hemoglobin levels.

TABLE IV-2. Variables Considered in Regressions of FEP, MCV, MCH, and Anemia

Age under 2	Serum Copper
Age 2-4	Dietary Carbohydrates
Age 4-6	Dietary Fat
Race	Serum Iron
Sex	Blood Lead
Degree of Urbanization	Dietary Phosphorus
Family Income	Dietary Protein
Serum Albumin	Transferrin Saturation
Dietary Calcium	Dietary Vitamin C
Dietary Calories	Serum Zinc

The regressions for both MCV and MCH found blood lead to be a significant explanatory variable ($p < 0.0001$ and 0.0033, respectively) for the decreases in each. "Hockey stick" regressions on the MCV relationship indicated a threshold at 10 ug\dl, the same level at which heme synthesis disturbance by lead has been reported to begin. Figures IV-2 and IV-3 show these relationships for MCV and MCH respectively.

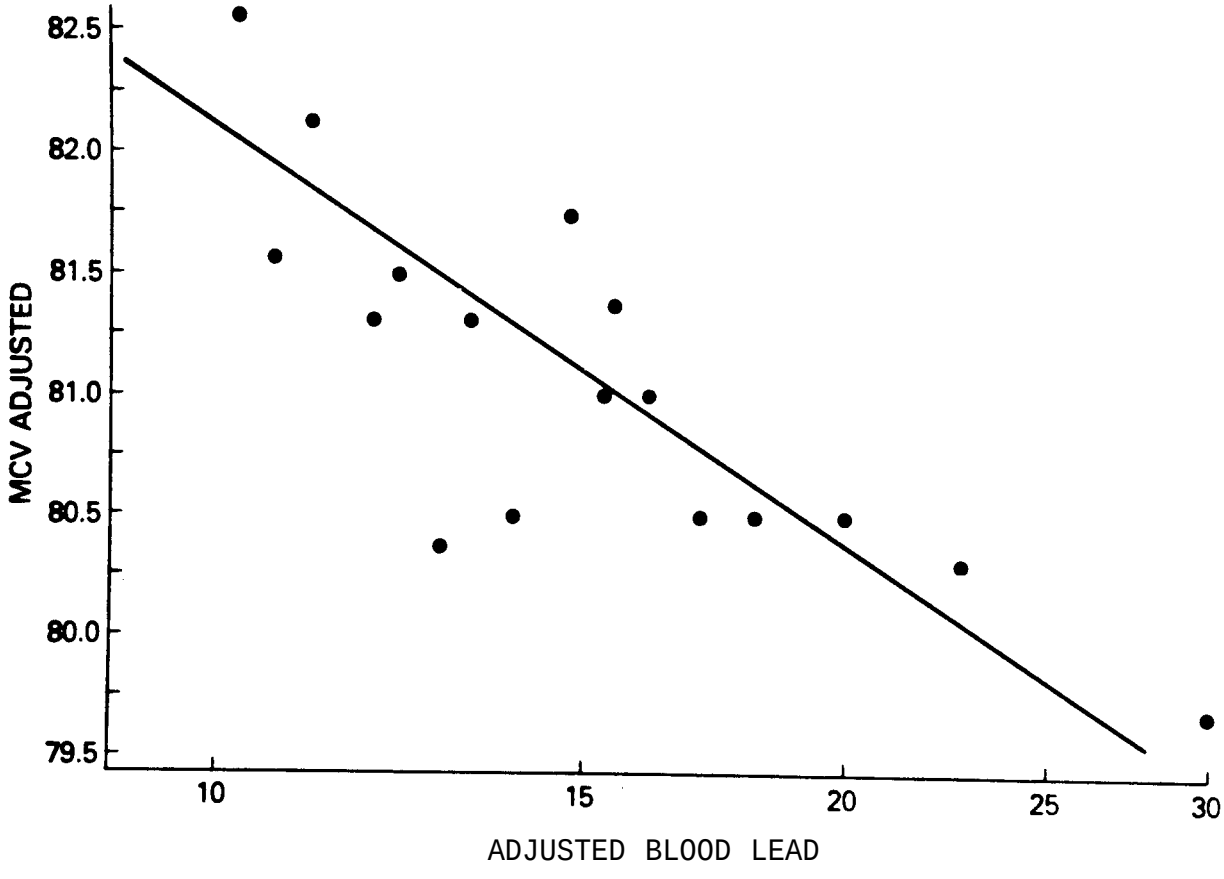
We also analyzed the probability of children having abnormally low MCV and MCH levels as a function of blood lead, since this is a clearer sign of physiological derangement. For this analysis, we used logistic regressions. Once again, blood lead was a significant explanatory factor ($p < 0.0001$), both in mean cell volume being low (MCV < 80 femptoliters [fl]) and in mean cell volume being seriously low (MCV < 74 fl). Blood lead levels were also significantly associated ($p < 0.023$) with the percent of children having MCH less than 25 picograms (pg), but only for children under six.

To test the hypothesis that the relationship with MCV held at low blood-lead levels as well as high blood-lead levels, we repeated the regression for MCV < 74 fl using only those children whose blood lead levels were less than 25 ug/dl. The regression coefficient for blood lead was unchanged and significant ($p < 0.014$). Thus, blood lead levels under 25 ug/dl were also associated with increased risks of microcytosis.

Because blacks have a higher incidence of thalessemia, we repeated the MCV regressions for white children only. Lead was

FIGURE IV-2

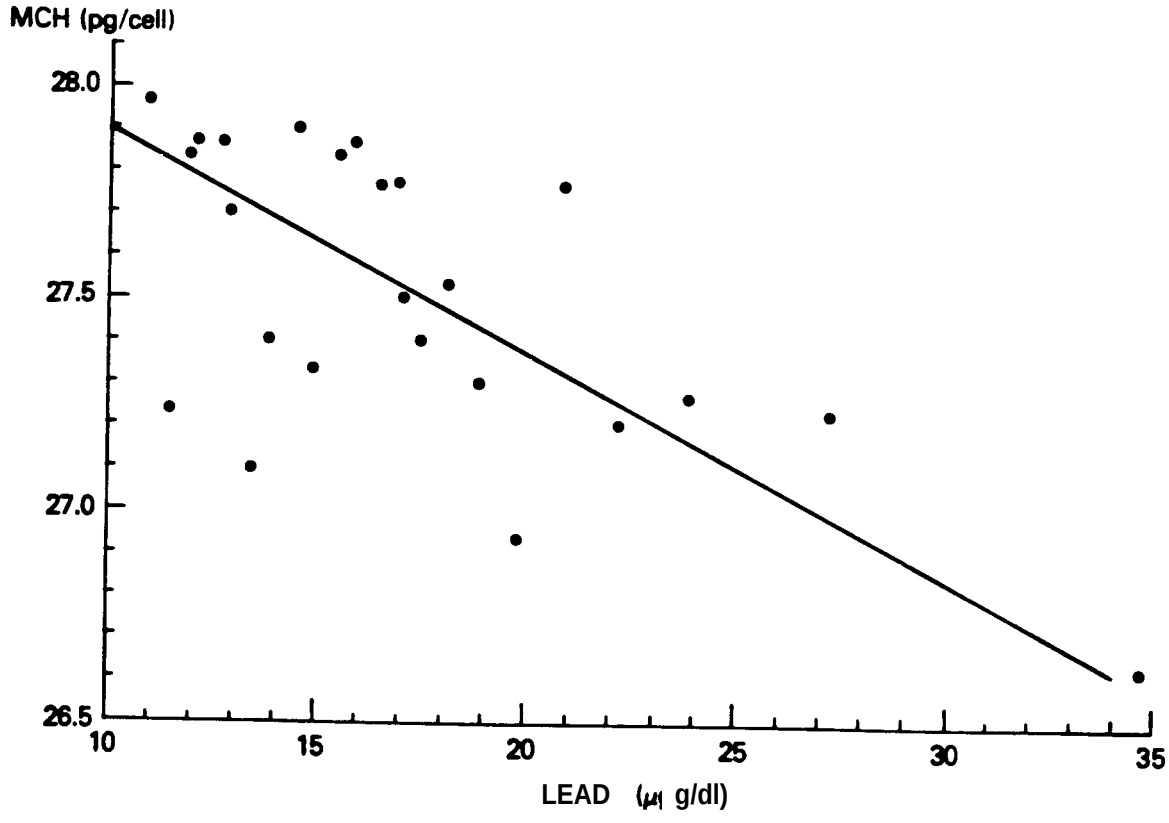
The Relationship Between MCV and Blood Lead After Adjusting for all Other Significant Variables



The regression line is the true regression for all 1,967 children. For ease of display, each point represents the average of about 100 children with consecutive blood lead levels.

FIGURE IV-3

The Relationship between MCH and Blood Lead After Adjusting for All other Significant Variables



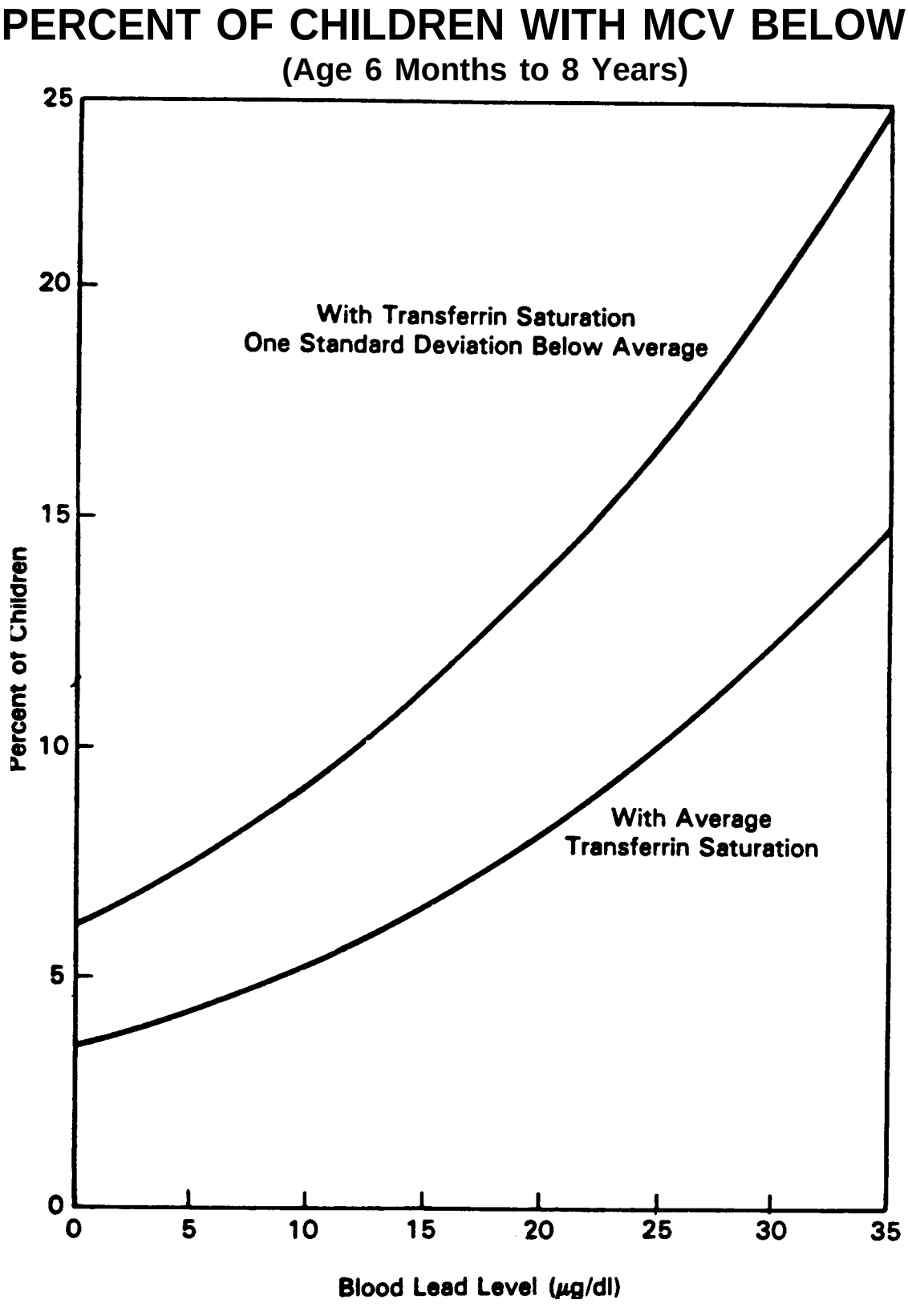
The regression line is the true regression line for all 1,967 children. For ease of display, the points shown represent the average of about 100 children with consecutive blood lead levels.

still a significant predictor of MCV ($p < 0.0001$), after controlling for all other significant variables. We also used Mentzer's index, which indicates that persons with thalessemia generally have an MCV/red cell count of < 11.5. Even after we eliminated all such people, lead was still a significant predictor of MCV, with no change in the coefficient.

To further investigate the relationship between lead and abnormal hematological variables, we used our regression to predict the percentage of children with MCV < 74 fl as a function of blood lead for two cases: children with average transferrin saturation levels (22.4 percent saturated for children in the NHANES II survey) and children with transferrin saturation levels one standard deviation below average (13.6 percent). The results are shown in Figure IV-4. Note that at 25 ug/dl of blood lead, almost 10 percent of the children with average iron levels and 17 percent of the children with below average iron levels had MCVs of less than 74 fl.

The relative risk of children having MCV levels less than 74 fl when their blood lead levels were 25 ug/dl compared to 10 ug\dl was 1.98 (the 95 percent confidence interval was 1.44-2.71). Using the same 10 ug/dl reference point, the relative risk at 20 ug/dl was 1.53 (1.27-1.95 at 95 percent). Since logistic regressions gave the same results when we used only children with blood lead levels under 25 ug/dl, and since the 95 percent confidence limits on the relative risk did not include 1.0, these results showed increased risks of hematological abnormalities in children at blood lead levels of 20 ug/dl and below.

FIGURE IV-4



Prediction of Percent of Children with MCV Below 74 fl as a Function of Blood Lead Levels

IV.A.2.c.2. The Relationship Between Blood Lead and FEP

The increased interference of lead in the formation of hemoglobin, and consequent accretion of protoporphyrins in red blood cells, has been well documented by Piomelli et al. (1982). Our analysis of the NHANES II data confirmed that study's results. Annest and Mahaffey (1985) have recently analyzed the relationship between FEP levels and blood lead in the NHANES II data and found a strong relationship after controlling for iron status. We also analyzed the NHANES II data and found that, even after controlling for iron status using transferrin saturation, the relationship was very strong.

A considerable body of literature exists suggesting that FEP levels are exponentially related to blood lead levels (Piomelli et al., 1973; Kammholz et al., 1972; Sassa et al., 1973; Lamola et al., 1975a, b; Reels et al., 1976). To test this relationship in the NHANES II population, we used several alternative specifications. We considered a linear model, a model where FEP was proportional to both exp(blood lead) and exp(percent transferrin saturation), a model where FEP was proportional to exp(blood lead) and $(\text{transferrin saturation})^{B}$, and a model where FEP was proportional to $(\text{blood lead})^{B1}$ and $(\text{transferrin saturation})^{B2}$. The model that that fit best was exp(blood lead) times $(\text{transferrin saturation})^{B}$. We examined the possibility of different additive intercepts in this model and found the highest correlation coefficient and F-statistic for a zero additive constant. This model suggested an exponential relationship to blood lead and a power law dependence on transferrin saturation. "Hockey stick" analysis

of the relationship between FEP and blood lead gave a threshold of 18 ug/dl. Figure IV-5 shows the FEP-blood lead relationship.

We also investigated the relationship between the probability of elevated FEP levels and blood lead, and verified previous findings. Again using NHANES II data, we performed logistic regressions on the probability of FEP levels being above 50 ug/dl* as a function of blood lead, using both blood lead and log(blood lead) as the independent variable; we obtained a better fit with blood lead than with log(blood lead).

Again, we checked to see whether the relationship between the risk of elevated FEP and blood lead held at lower blood-lead levels, repeating the regression only for children with blood lead levels under 30 ug/dl. Using multiple logistic regressions, blood lead was again extremely significant ($p < 0.0001$). The coefficient of blood lead for the low group was 0.178 ± 0.04 compared to 0.175 ± 0.018 for the regression with all blood-lead levels, a trivial difference between the two cases. This indicated that the risk of seriously elevated FEP levels was strongly related to blood lead, even at blood lead levels below the previously defined "safety level."

Piomelli and coworkers' studies have suggested a threshold for lead-induced increases in FEP levels of about 15 ug/dl blood lead. Taking 17.5 ug/dl of blood lead as our reference level,

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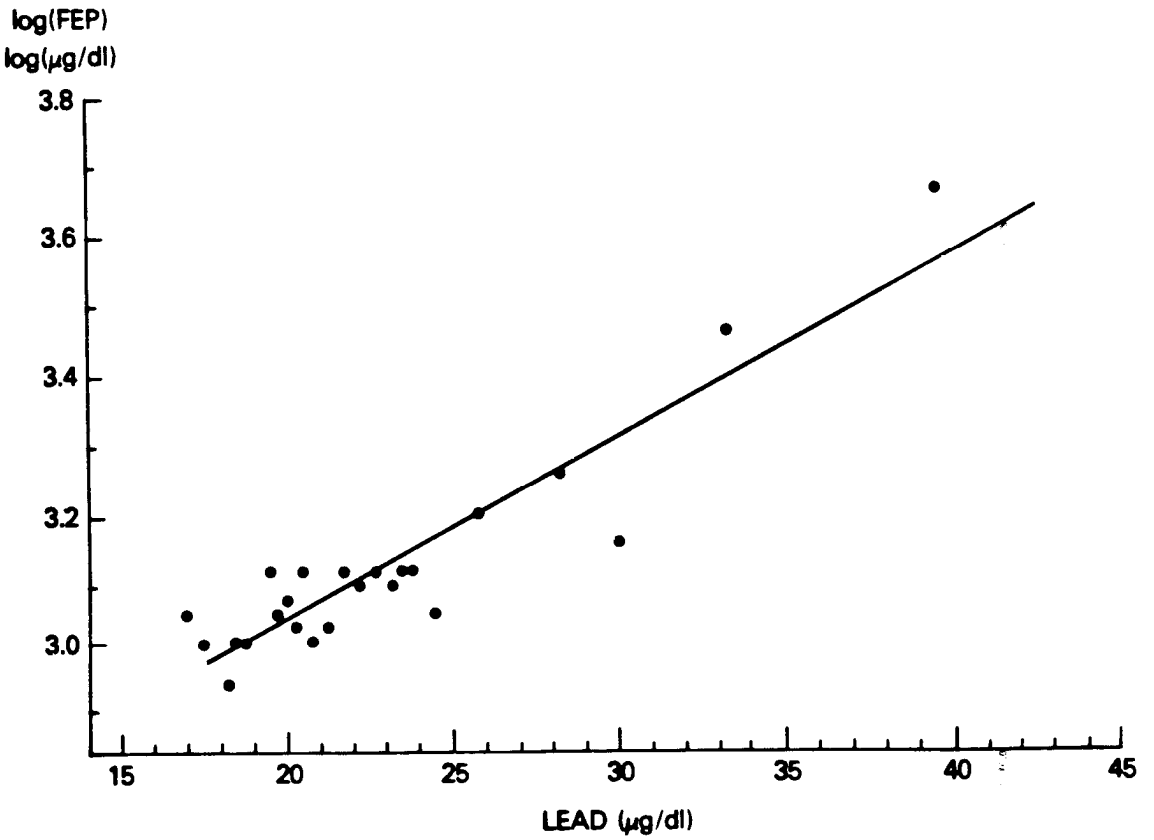
* The 50 ug/dl FEP level is considered by CDC to indicate severe enough interference with heme processes that medical attention is usually required, even when not coupled with elevated blood-lead levels.

Figure IV-5

The Relationship Between FEP Level and Blood Lead
After Adjusting for All Other Significant Variables



The regression line shows the true regression line for all 1,967 individual observations. For ease of display, each point represents the mean of about 100 children with consecutive blood lead levels.

our regression predicted that the relative risk of FEP levels over 50 ug/dl was 1.55 (1.42-1.70 at 95 percent confidence levels) at 20 ug/dl of blood lead, and was 3.73 (2.55-4.89 at 95 percent confidence) at 25 ug/dl of blood lead. This was true across all transferrin saturation levels.

IV.A.2.c.3. The Relationship Between FEP Levels and Anemia

To assess the implications of elevated FEP levels, we analyzed the relationship between log(FEP) and hematocrit, hemoglobin, and MCV. We performed linear regressions on all three outcomes as a function of log(FEP), controlling for race, age, and transferrin saturation. These analyses showed that log(FEP) was strongly inversely related ($p < 0.0001$ in all cases) to hematocrit levels, hemoglobin levels, and MCV. We then performed logistic regressions on the probability of abnormal levels of hematocrit, hemoglobin, and MCV as a function of log(FEP), with the same controls. They also showed that FEP was an excellent predictor ($p < 0.0001$) of the probability of abnormally low levels of all three indicators. Again, we repeated our regressions using only children with FEP values below 33 ug/dl, and FEP was still very significant ($p < 0.0001$). (FEP levels of less than 33 ug/dl are generally associated with blood lead levels under 30 ug/dl.) The coefficients differed by less than one standard deviation from those for the full sample. Thus, the relationship appeared to hold for low FEP levels as well as high ones.

Figure IV-6 shows the regression's prediction of the percent of children with anemia as a function of FEP levels at normal transferrin saturation levels for children. The data used in the

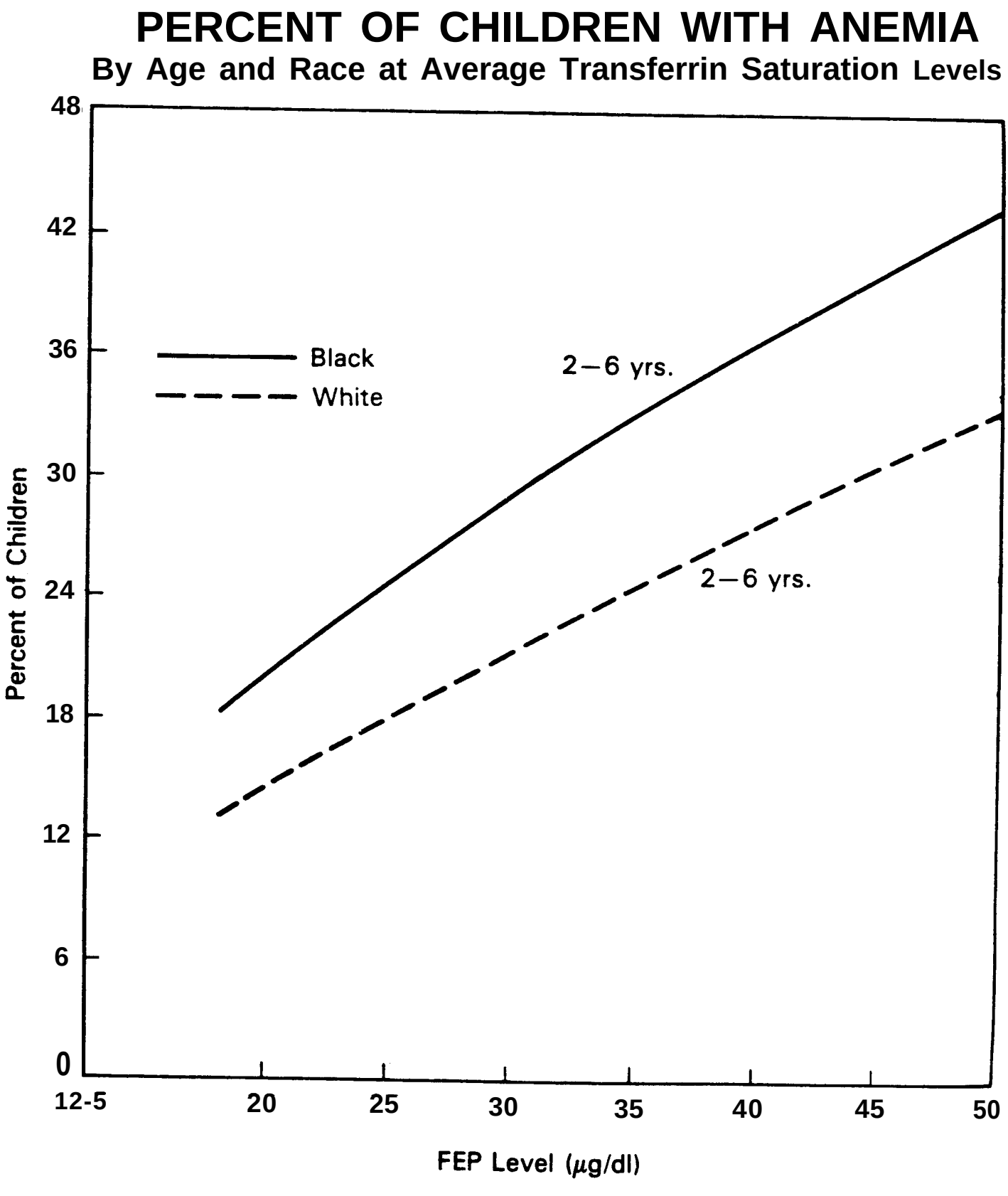
regression contained FEP levels as low as 9.6 ug/dl, but we have shown the projections only for 18 ug/dl and above. For our definition of anemia, we have used the hemoglobin and hematocrit levels representing the minimum normal range levels recommended by the Journal of Pediatrics (1977). These definitions are supported by the work of Yip et al. (1981), and the use of hematocrit and hemoglobin levels to define anemia is standard (Harrison, Principles of Internal Medicine, 9th Edition).

Figure IV-6 shows that as FEP levels increase from 20 ug/dl to 50 ug/dl, an additional 20 percent of children aged 2-6 years would develop anemia at normal iron levels. Our earlier regressions of blood lead levels on FEP suggested that blood lead levels of less than 15 ug/dl were necessary to keep average FEP levels below 20 ug/dl. Since elevated FEP is a symptom of an underlying interference in heme synthesis, it cannot be viewed as the cause of these abnormal hematocrits. The causal association must be with whatever produced the excess FEP. As the data portrayed in Figure IV-3 were for normal iron levels, the anemia appeared to be the outcome of the lead exposure, for which FEP served as a surrogate .

In summary, blood lead levels below 30 ug/dl seem to be associated with increased microcytosis and hypochromia in children and increased interference with heme synthesis producing elevated levels of free erythrocyte protoporphyrin. Elevated FEP levels, even below 33 ug/dl, are themselves associated with an increased risk of anemia. In addition, the reduced mean cell volumes and the lower hematocrit values again indicate that lead’s effect on

FIGURE IV-6

Prediction of Percent of Children with Anemia as a Function of FEP Level at Normal Transferrin Saturation Levels



heme synthesis continues at levels below 30 ug/dl of blood lead. This further strengthens the case for considering elevated FEP levels themselves, which mark lead's interference with normal body activity, as a pathophysiological effect.

IV.A.3. Lead's Interference with Vitamin D Metabolism and Associated Physiological Processes

Another potentially serious consequence of lead exposure is the impairment of the biosynthesis of the active vitamin D metabolite, 1,25-$(OH)_2$ vitamin D, which is detectable at blood lead levels of 10-15 ug/dl. Further, an inverse dose-response relationship has been reported between blood lead and 1,25-$(OH)_2$ vitamin D throughout the range of measured blood lead values up to 120 ug/dl (Criteria Document, p. 12-49; Rosen et al., 1980a, 1980b; Mahaffey et al., 1982). Interference with vitamin D production disrupts calcium, zinc, and phosphorous homeostasis, partially resulting in the reduced absorption of these elements from the gastro-intestinal tract. This alters the availability of these elements for physiological processes crucial to the normal functioning of many tissues, cell membranes, and organ systems.

The reduced uptake and utilization of calcium has two compounding consequences. First, it interferes with calcium-dependent processes that are essential to the functioning of nerve cells, endocrine cells, muscle cells (including those in the heart and other components of the cardiovascular system), bone cells, and most other types of cells. The second concern is possible increased lead absorption resulting from decreased

calcium availability. The latter can be expected to create a feedback response, further exacerbating the inhibition of vitamin D metabolism and reduced calcium availability, leading to even greater lead absorption and greater vulnerability to increasingly more severe lead-induced health effects (Rosen et al., 1980b; Barton et al., 1978). These effects are especially dangerous for young (preschool age) children who are developing rapidly. These children, even in the absence of lead, generally are susceptible to calcium deficiencies because of the large amount of calcium used for the formation of the skeletal system, as well as several other calcium-dependent physiological processes important in young children.

Even moderate levels of lead exposure in children are associated with vitamin D disturbances that parallel certain metabolic disorders and other disease states, as well as severe kidney dysfunction (Criteria Document, p. 12-37). At blood lead levels of 33-55 ug\dl, 1,25-$(0H)_2$ vitamin D is reduced to levels comparable to those observed in children who have severe renal insufficiency with the loss of ahout two-thirds of their normal kidney function (Rosen et al., 1980a; Rosen and Chesney, 1983; Chesney et al., 1983). Analogous vitamin D hormone depressions are found in vitamin D-dependent rickets (type I), oxalosis, hormone-deficient hypoparathyroidism, and aluminum intoxication in children undergoing total parenteral nutrition.

Lead-induced interference of 1,25-$(OH)_2$ D biosynthesis affects a wide range of physiological processes. The vitamin D-endocrine system is responsible in large part for the maintenance

of extra- and intra-cellular calcium homeostasis (Rasmussen and Waisman, 1983: Wong, 1983; Shlossman et al., 1982; Rosen and Chesney, 1983). Thus, modulation in cellular calcium metabolism induced by lead at relatively low concentrations may potentially disturb multiple functions of different tissues that depend upon calcium as a second messenger (Criteria Document, p. 12-40). It also appears that 1,25-$(OH)_2$ D participates directly in bone turnover by orchestrating the population of cells within the bone (Criteria Document, p. 12-41). An immunoregulatory role for the vitamin D hormone is evident through the widespread existence of 1,25-$(OH)_2$ D_3 receptor sites on immunoregulatory cells, such as monocytes and activated lymphocytes (Provvedini et al., 1983; Bhalla et al., 1983).

The negative correlation between blood lead and serum 1,25-$(OH)_2$ D, the active form of vitamin D, appears to be another example of lead's disruption of mitochondrial activity at low concentrations. While serum levels of 1,25-$(OH)_2$ vitamin D decreased continuously as blood lead levels increased from an apparent threshold of 10 to 15 ug/dl, this was not true for its precursor, 25-(OH) vitamin D. In fact, in lead-intoxicated children after chelation therapy, vitamin D levels were restored, but the precursor levels remained unchanged (Rosen et al., 1980a, 1980b; Mahaffey et al., 1982). This indicates that lead inhibits renal l-hydroxylase, the kidney enzyme that converts the precursor to the active form of vitamin D. Renal l-hydroxylase is a mitochondrial enzyme system, which is mediated by the hemoprotein, cytochrome P-450. This suggests that the damage to the mitochon-

drial systems detected at 15 ug/dl has uncompensated consequences.

If cytochrome P-450 is being inhibited at the low levels of blood lead that the reduced renal l-hydroxylase activity suggests, we must consider the possibility that other physiological functions related to cytochrome P-450 may also be disrupted. For example, reduced P-450 content has been correlated with impaired activity of the liver detoxifying enzymes, aniline hydroxylase and aminopyrine demethylase, which help to detoxify medications, hormones, and other chemicals (Goldberg et al., 1978).

While cytochrome P-450 inhibition has been found in animals, and in humans at higher lead levels, this has not yet been examined in children at low blood-lead levels (i.e., 10 to 15 ug/dl). But the disruption of vitamin D biosynthetic pathways at these levels is suggestive of an effect.

The reduction in heme caused by lead exposure probably underlies the effects seen in vitamin D metabolism. This would explain the similarity in apparent “thresholds” for the effect of lead on both erythrocyte protoporphyrin accumulation and decreases in levelS of serum 1,25-$(OH)_2$D. It would also indicate a cascade of biological effects among many organ and physiological systems of the body (depicted graphically in Figure IV-l). Together, the interrelationships of calcium and lead metabolism, lead’s effects on 1,25-$(OH)_2$D, and the apparent disruption of the cytochrome P-450 enzyme system provide a single molecular and mechanistic basis for Aub et al.'s observation in 1926 that “lead follows the calcium stream.”

IV. B. Neurotoxic Effects of Lead Exposure

Lead has been known to be a neurotoxicant since the early 1800s, and neurotoxicity is among the more severe consequences of lead exposure. At very high-blood lead levels, encephalopathy and severe neurotoxic effects are well documented; the neurotoxic effects at lower blood lead levels, however, are less clearly defined. Recent research has investigated the occurrence of overt signs and symptoms of neurotoxicity and the manifestation of more subtle indications of altered neurological functions in individuals who do not show obvious signs of lead poisoning.

IV.B.1. Neurotoxicity at Elevated Blood-Lead Levels

Very high blood-lead levels (i.e., above 80 ug/dl in children) are associated with massive neurotoxic effects that can include severe , irreversible brain damage, ataxia (i.e., the inability to coordinate voluntary muscular movements), persistent vomiting, lethargy, stupor, convulsions, coma, and sometimes death. Once encephalopathy occurs, the risk of death for children is significant (Ennis and Harrison, 1950; Agerty, 1952; Lewis et al., 1955), regardless of the quality of the medical treatment they receive.

In cases of severe or prolonged nonfatal episodes of lead encephalopathy, neurological damage occurs that is qualitatively similar to that often seen following traumatic or infectious cerebral injury, with permanent and irreversible damage being more common in children than adults (Mellins and Jenkins, 1955; Chisolm, 1956, 1968). The most severe effects are cortical atrophy, hydrocephalus (an abnormal increase in cranial fluid), convulsive seizures, and severe mental retardation. Permanent

central nervous system damage almost always occurs in children who survive acute lead encephalopathy and are re-exposed to lead (Chisolm and Harrison, 1956). Even if their blood lead levels are kept fairly low, 25-50 percent show severe permanent sequelae including seizures, nervous disorders, blindness, and hemiparesis (paralysis of half of the body) (Chisolm and Barltrop, 1979).

Even children without obvious signs of acute lead encephalopathy have exhibited persisting neurological damage. As early as 1943, Byers and Lord's study of 20 previously lead-poisoned children indicated that 19 later performed unsatisfactorily in school , "presumably due to sensorimotor deficits, short attention span, and behavioral disorders." Effects such as mental retardation, seizures, cerebral palsy, optic atrophy, sensorimotor deficits, visual-perceptual problems, and behavior disorders have been documented extensively in children following overt lead intoxication or even just known high exposures to lead (e.g., Chisolm and Harrison, 1956: Cohen and Ahrens, 1959; Perlstein and Attala, 1966).

The extent of the later manifestations seems to relate to the severity of the earlier observed symptoms. In Perlstein and Attala, 9 percent of the children studied, none of whom appeared to have severe symptoms when diagnosed for overt lead poisoning, were later observed to be minimally mentally retarded and 37 percent showed some lasting neurological sequelae.

At somewhat lower blood-lead levels (i.e., 30-70 ug/dl), substantial data confirm that a variety of neural dysfunctions

occur in apparently asymptomatic children. Several studies indicate that blood lead levels of 50-70 ug/dl are associated with IQ decrements of 5 points. Adverse electrophysiological effects, including markedly abnormal EEG patterns, slow-wave voltages, etc., are also well documented at levels of 30-70 ug/dl.

De la Burde and Choate (1972, 1975) showed persisting neurobehavioral deficits in children exposed to moderate-to-high levels of lead; most of the children appear to have had blood lead levels above 40 ug/dl. Compared to low-lead control children -- matched for age, sex, race, parents' socioeconomic status, housing density, mother's IQ, number of children in the family below age 6, presence of father in the home, and mother working -- the higher lead children averaged about five points lower in IQ and were seven times more likely to have repeated grades in school or to have been referred to school psychologists. Moreover, follow-up studies showed that these effects persisted for at least three years.

While chelation therapy may mitigate some of these persisting effects, permanent neurological and cognitive damage seems to result from very high lead levels, with or without encephalopathy. In addition, these children also appear more likely to experience neurological and behavioral impairments later in their childhood.

IV.B.2. Neurotoxicity at Lower Blood-Lead Levels

The adverse effects of lead on neurological functioning, both on the microscopic (i.e., cellular and enzymatic) level and the macroscopic (i.e., learning behavior) level, are well docu-

mented. On the micro-level, data from experimental animal studies suggest several possible mechanisms for the induction of neural effects, including: (1) increased accumulation of ALA in the brain as a consequence of lead-induced impaired heme synthesis, (2) altered ionic balances and movement of ions across axonal membranes and at nerve terminals during the initiation or conduction of nerve impulses due to lead-induced effects on the metabolism or synaptic utilization of calcium, and (3) lead-induced effects on the metabolism or synaptic utilization of various neurotransmitters.

In addition, lead-induced heme synthesis impairment, resulting in reduced cytochrome C levels in brain cells during crucial developmental periods, has been clearly associated with the delayed development of certain neuronal components and systems in the brains of experimental animals (Holtzman and Shen Hsu, 1976). (Cytochrome C is a link in the mitochondrial electron transport chain that produces energy, in the form of adenosine triphosphate (ATP), for the entire cell.) Given the high energy demands of neurons, selective damage to the nervous system seems plausible.

In addition to the effects of lead on the brain and central nervous system, there is evidence that peripheral nerves are affected as well. Silbergeld and Adler (1978) have noted lead-induced blockage of neurotransmitter (acetylcholine) release in peripheral nerves, a result of lead's disruption of the transport of calcium across cellular membranes. This disruption of cellular calcium transport may also contribute to the effects of lead on

peripheral nerve conduction velocity. Landrigan et al. (1976) have noted a significant correlation between blood lead and decreasing nerve conduction velocity in children in a smelter community. This effect may indicate advancing peripheral neuropathy.

Paralleling these cellular or biochemical effects are electrophysiological changes indicating the perturbation of peripheral and central nervous system functioning observed in children with blood lead levels of approximately 15 ug/dl. These included slowed nerve conduction velocities (Landrigan et al., 1976), reaction-time and reaction-behavior deficits (Winneke et al. 1984; Yule, 1984), as well as persistent abnormal EEG patterns including altered brain stem and auditory evoked potentials down to 15 ug/dl (Benignus et al., 1981; Otto et al., 1981, 1982, 1984). The results indicating neurological effects of lead at such low levels are particularly important because two- and five-year follow-up studies (Otto et al., 1982, 1984) indicated some persistent effects.

Aberrant learning behavior has been noted in rats with blood lead levels below 30 ug/dl. This behavior evidenced both reduced performance on complex learning problems and signs of hyperactivity and excessive response to negative reinforcement (Winneke, 1977, 1982a).

Finally, the cognitive effects of lead in children show signs of a dose-response relationship. For high level lead poisoning, adverse cognitive effects in children are indisputable and mental retardation is a common outcome. For children with

somewhat lower blood-lead levels, de la Burde and Choate (1972, 1975) found lesser but still significant cognitive effects, including lower mean IQs and reduced attention spans. Several studies discussed in more detail in Section IV.B.2.a. have found smaller effects at lower blood-lead levels. The precise biological mechanisms connected with these effects are not yet clearly defined, although hypotheses have been put forward.

While some of these effects have only been observed at higher blood-lead levels, in animals, or in vitro, they show a consistent dose-dependent interference with normal neurological functioning. Furthermore, some of these effects have been documented to occur at low blood-lead levels in children, with no clear threshold having been demonstrated.

This general pattern of lead's interference in neurological functioning on the cellular level, including effects below 30 ug/dl, form the background against which we examined the studies that investigated changes in cognitive processes in children at low blood-lead levels. Because of the intrinsic difficulties in performing such studies, and because most investigators have not employed sample sizes that would permit unambiguous detection of small effects, it is important to integrate these behavioral studies with what has been discovered on the molecular and cellular levels.

IV.B.2.a. Cognitive Effects of Moderate Blood-Lead Levels

The literature on cognitive effects at low to moderate body-lead levels is extensive. However, most of the studies have some methodological flaws, and few display indisputable results

concerning the relationship between IQ effects and changes in low body-lead levels. The Draft Lead Criteria Document (p. 12-65) divided the studies into four groups: clinical studies of high lead children, general population studies, lead smelter area studies, and studies of children who are mentally or behaviorally abnormal.

One of the larger and better designed studies of lower-level cognitive effects was the Needleman et al. study in 1979, which found a significant inverse correlation between tooth lead levels and IQ after controlling for age, parent's IQ, and socioeconomic factors. In 1983 an EPA peer-review panel asked for a reanalysis of Needleman et al.'s data, using different model specifications. EPA's Office of Policy Analysis also requested a reanalysis using a continuous lead exposure variable. Needleman submitted a reanalysis to EPA, and presented the reanalysis to the Clean Air Science Advisory Committee (CASAC) in 1984. The CASAC stated that the reanalysis adequately addressed the concerns of the peer review panel, and recommended that EPA include the study in its criteria document process. These latter results confirmed the association between lead and IQ found in the original study, and showed that the results were quite stable in response to the inclusion or exclusion of different confounding factors.

The summary table in Chapter 12 of the Criteria Document (pp. 12-65 to 12-70) indicated that virtually all of the general population studies showed high lead groups performing more poorly on a variety of tests used to assess cognitive function. For more than half of these tests, however, the probability of

falsely finding an effect due to chance was more than 5 percent; i.e., less than half of them had a p-value of less than 0.05. (Significance levels in the studies were reported as probabilities if they were below 0.05 and as “not significant” otherwise.)

For use in public policy making, rejecting the results of these studies because so many fail to attain significance at the 5 percent level may be inappropriate for two reasons. First, policy makers need to be concerned about both type I and type II errors. Significance tests guard only against the first type (falsely rejecting the null hypothesis of no effect); they help ensure that a regulation is not imposed when there is no adverse effect. Type II errors (failing to reject the null hypothesis when it is false) also can be costly, however, because they can result in the underregulation of a real hazard. With small sample sizes and subtle effects, the probability of a type II error can be large; in the case of the Smith et al. (1983) study, we calculated it to be 62 percent if the true decrease in IQ was 2 points.* The probability of a type II error in the other studies would be even higher, because of their smaller sample sizes.

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* We computed the false positive from the Smith et al. data as follows. Using Pocock and Ashby (1985), we derived the standard deviation for the difference of the high and low lead groups of 1.499. At a 5 percent chance of rejecting the null hypothesis when it is true, the normal one-tailed test statistic is 1.65. Therefore, we would reject the null hypothesis only for differences greater than (1.499) (1.65) = 2.473. If the difference in the groups was two IQ points, the probability of the difference being below 2.473 is given by p (z < [2.473-2]/ 1.499) = 0.62.

The second reason for caution in rejecting the results of these studies is that while several fail to attain statistical significance individually, they do show a consistent pattern: in nearly all of them, the children in the higher lead groups showed lower mean IQs. Figure IV-7 plots the estimated effects, along with the 90 percent confidence limits.* The higher end of the 90 percent confidence limit corresponds to the critical value for a one-tailed test at the 0.05 significance level; i.e., studies in the figure whose upper confidence limits exceed 0 are not statistically significant at the p = 0.05 level.

The consistent pattern in all of the studies suggested that combining evidence from all studies would provide a better test for a significant effect than separate evaluations of the statistical significance of the individual studies. In applying one of the available joint tests for the existence of a specific effect, we began with the six general population studies found in Table 12.1 of the draft Criteria Document. As the result of personal communication with Harvey we have not included this study, due to the younger age of the children (2.5) and the continuous nature of their study design. We did not consider clinic studies because of their higher lead (typically > 70 ug/dl) or studies of children exhibiting abnormal behavior. To the five remaining general population studies we added the smelter study by Winneke et al. (1982b), as the blood lead levels in that study

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* Some of the studies did not report p-values or standard errors, in which cases, we used the data in the study to compute these values. Where we had insufficient information, we did not include the study.

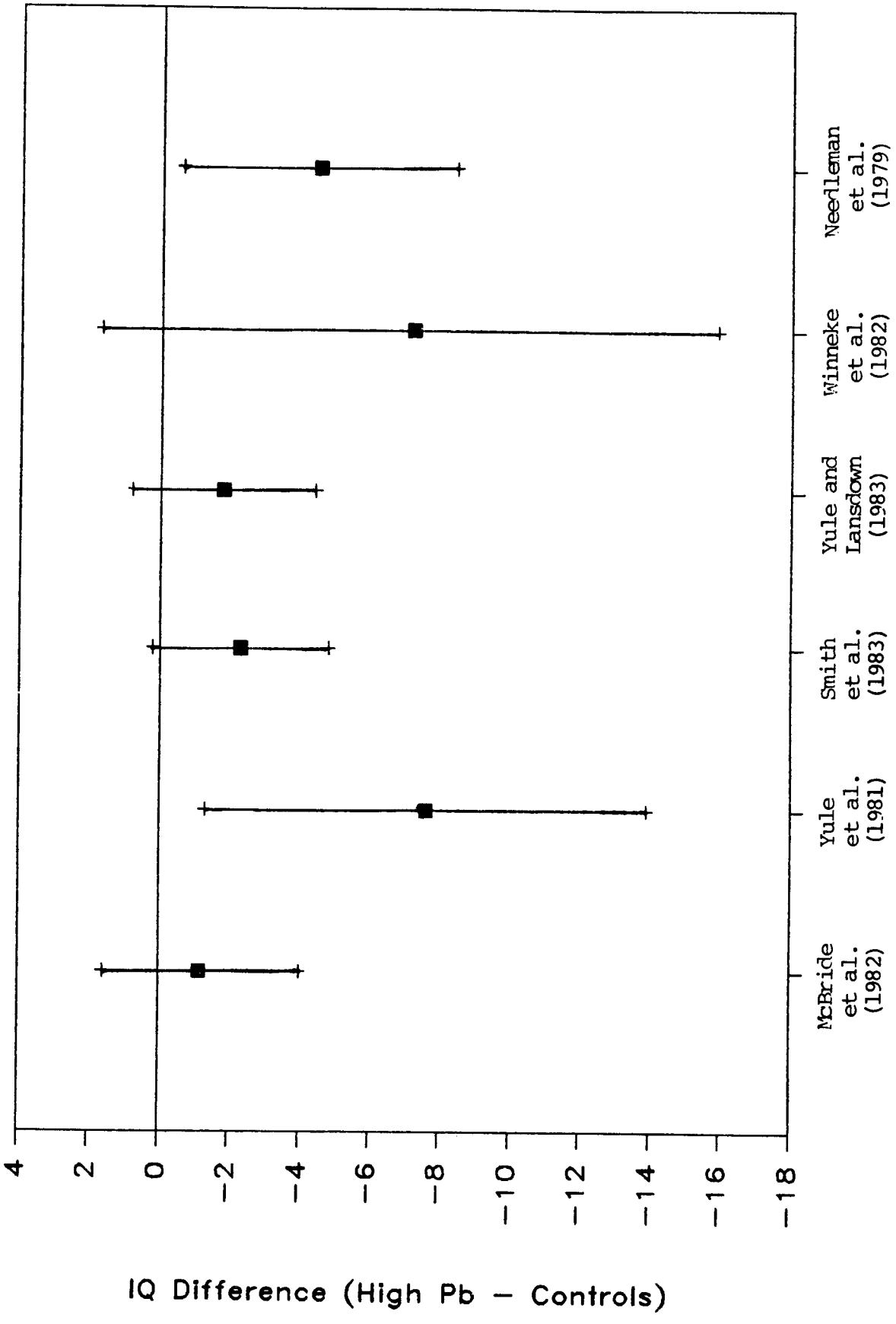

FIGURE IV-7. Mean IQ Difference Between High Lead Groups and Controls, Adjusted for Socioeconomic Factors (90% Confidence Intervals)

were in the same range as the general population studies. For three other studies, Winneke (1983), Winneke (1984), and Yule and Lansdown's study in Leeds, sufficient data were not available to include their results. These three studies generally found small or statistically insignificant effects. We also looked only at Full Scale IQ measures. While not all studies used the same IQ test, the Full Scale IQ measures employed were close enough to allow us to compare differences between groups and across studies. Table IV-3 summarizes the relevant information.

In evaluating the reported results of the combined significance of these studies, it is important to remember that because each study was performed using different protocols, study populations, levels of exposure and investigations, the resulting combined p-value should be interpreted cautiously, and viewed more for its qualitative implications than for its precise numerical result. The specific numerical result (joint p-value) also is sensitive to the studies included. Inclusion of the studies by Winneke and others discussed above, which were omitted because of insufficient data, would change the p-value. The direction of the change is difficult to predict, however, because while these studies generally found insignificant results, the inclusion of additional studies will raise the joint p-value unless the p-values in the individual studies are quite large (e.g., $p > 0.25$).

We used the Fisher aggregation procedure (Fisher, 1970, p. 99) to estimate the combined significance of the observed

TABLE IV-3. Computation of Joint P-Value from Epidemiological Studies of Cognitive Effects from Low Level Lead Exposure in Children

Study	Sample Sizes		Internal Lead Levels: Blood (ug/dl)		Internal Lead Levels: Teeth(ppm)		IQ Difference	P-Value	-2 1n p
	Control	Exposed	Control	Exposed	Control	Exposed			
McBride et al. (1982)	86	86	< 9	19-30	-----	-----	1.2 [a]	0.25	2.77
Yule et al. (1981)	20	21	7-10	17-32	-----	-----	7.6 [b]	0.027	7.22
Smith et al. (1983)	145	155	---—--	-----	< 2.5	> 8	2.3 [b]	0.067 [c]	5.41
Yule and Lansdown (1983)	80	82	7-12	13-24	-----	-----	1.8 [b]	0.13	4.08
Winneke et al. (1982a)	26	26	-----	-----	2.4	7	5 [b]	0.10	4.82
Needleman et al. (1979)	100	58			< 10	> 20	4.5 [b]	0.03	7.01
									31.31

Joint p-value for studies: $P(X^2_{12} > 31.31) < 0.005$

a Peabody Picture Vocabulary IQ Test
b Welchsler Intelligence Scale for Children-Revised
c Smith does not report a p-value but Pocock and Ashby report a 95% confidence interval of .4 to -5.5 for the Smith result which implies a standard error of 1.5 and a t-statistic of 1.53.

effects, and to derive a joint p-value for all of the studies. To do this, we needed the p-values for all of the individual studies.

For each study where p-values were not reported, we used the standard deviation of the IQ measure to compute the p-value for the difference in the mean IQs across groups. We could not use this method for the 1983 study by Smith et al. In that study, the full scale IQ effects were reported as "not significant" and no standard deviation was given. However, when we computed the p-value using the standard deviation derived from Pocock and Ashby, we found that the p-value was 0.067 when comparing high and low lead groups for the Full Scale IQ.*

The results of our application of the Fisher procedure for computing a joint probability for the observed results are presented in Table IV-3. The resulting probability of less than 0.005 indicates that it was extremely unlikely that we could get the observed pattern of results if there were really no effect. The overwhelming preponderance of the data (virtually all studies show high lead groups with lower cognitive ability) was highly unlikely to have been due to chance.

Only if the studies were consistently biased towards finding an effect would the robustness of our result be questionable. In

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* Pocock and Ashby reported that in the Smith et al. study the 95% confidence interval for the full scale IQ effect was -5.5 to +0.4. The t-statistic for 298 degrees of freedom is 1.9679, yielding a standard deviation of 1.499. Our calculation used the difference of 2.3 reported by Smith et al., rather than the difference of 2.55 implied by the recent work by Pocock and Ashby.

at least one case (Smith), a procedure was used that biased against finding an effect, and biased upward the p-values. These authors used a two-stage analysis of variance or covariance where the effects of all covariates (except lead) on IQ were controlled for in the first stage, and the only residual IQ effects were regressed on lead in the next step. Many of these covariates (e.g., parental care, income, and IQ) negatively correlate with lead exposure, and this procedure attributed all of the joint variation to the non-lead variable.

These facts, together with the very small p-value calculated in the joint test, suggest that the combined evidence of cognitive effects at moderate levels of lead exposure should be treated as statistically significant. We conclude that the combined results of available studies of cognitive effect at moderate lead levels should be taken as evidence of cognitive decrements due to lead.

IV.B.3. The Magnitude of Lead's Impact on IQ

The evidence described above indicates that exposure to lead can lower children's IQs and reduce their ability to perform well in school. In Section IV.D, we monetize the cognitive benefits of reducing these effects using the costs of compensatory education. Here, we briefly describe a more direct, but also more speculative, approach based on the improvements in IQ that might be expected with reduced exposure to lead in gasoline.

The latest draft of the Criteria Document characterizes the evidence as suggesting that, on average, blood lead levels of 30 to 50 ug/dl result in a four-point decrement in IQ, and that lead

levels of 50 to 70 ug/dl reduce IQ by roughly five points (de la Burde and Choate, 1972, 1975; Rummo et al., 1979). If we assume that preventing a blood-lead level over 30 ug/dl avoids, on average, the loss of four IQ points per child, the gain in person-IQ points from limiting lead in gasoline is substantial. In 1986, for example, as shown in Table III-7, we estimate that 52,000 fewer children will experience blood lead levels over 30 ug/dl as a result of the 0.10 gplg limit (assuming no misfueling). If we assume that each child over 30 ug/dl suffers roughly a four-point IQ loss, that implies that the final rule will yield a gain of about 200,000 person-IQ points in 1986. Table IV-4 presents year-by-year estimates for the alternative rules.

This approach suffers from two faults, which cut in opposite directions. It does not account for the fact that some children who are prevented by the regulation from going over 30 ug/dl will do so by a narrow margin (e.g., their blood lead level will be 29 ug/dl when it would have been 31 ug/dl in the absence of the rule); such children are unlikely to receive the full four point gain in IQ. On the other hand, this approach attributes no benefit to children whose blood lead levels are reduced from very high levels, but not brought below 30 ug/dl, or to those whose levels would have been under 30 ug/dl without the rule, but whose levels decrease further by the reduction in lead in gasoline.

IV.C. Fetal Effects

Because lead passes the placental barrier, a growing concern in the public health community is that the most sensitive

TABLE IV-4. Year-by-Year Estimates of Gain in Person-IQ Points Under Alternative Rules, Assuming No Misfueling (thousands of person-IQ points)

Rule	1985	1886	1987	1988	1989	1990	1991	1992
Proposed	0	208	188	172	156	144	128	124
Alternative	88	184	180	172	156	144	128	124
Final	88	208	188	172	156	144	128	124

population for lead exposure is not children, but fetuses and newborn infants. This concern is supported by both animal studies and, recently, human data in the published peer-reviewed literature.

Crofton et al. (1980) found that the development of exploratory behavior by rat pups exposed to lead in utero lagged behind that of control rats. Average blood lead levels on the 21st postnatal day were 14.5 ug/dl for the exposed pups and 4.8 ug/dl for the controls.

Gross-Selbeck and Gross-Selbeck (1981) found alterations in the operant behavior of adult rats after prenatal exposure to lead via mothers whose blood lead levels averaged 20.5 ug/dl. At the time of testing (3 to 4 months, postnatal), the lead-exposed subjects' blood lead levels averaged 4.55 ug/dl compared to 3.68 ug/dl in the controls. This suggested that changes in central nervous system function may persist for months after the cessation of exposure to relatively low blood-lead levels.

Several other papers (McCauley and Bull, 1978; Bull et al., 1979) have shown that the prenatal exposure of rats to 0.2 percent lead chloride in the mother's drinking water markedly reduced the cytochrome C content in the cerebral cortex, thereby possibly producing an uncoupling of the electron transport chain in the cortex. This reduction in cytochrome C content occurred at blood lead levels as low as 36 ug/dl, with delays in the development of central nervous system energy metabolism being seen as late as 50 days after birth (Bull et al., 1983).

Needleman et al. (1984) analyzed data from over 4,000 live births at Boston Women's Hospital and reported an association between mild congenital anomalies and umbilical-cord blood-lead malformation and lead, but only between all minor malformations and lead. There also were no significant associations between lead and any major malformations, although given the rate of such malformations in the general population, a sample this size has little power to detect such an effect. Holding other covariates constant, the relative risk of a child demonstrating a minor malformation at birth increased by 50 percent as lead levels increased from 0.7 ug/dl to 6.3 ug/dl (the mean cord-lead level). This risk increased an additional 50 percent at 24 ug/dl. (Umbilical-cord blood-lead levels are somewhat lower than, but correspond to, maternal. blood-lead levels; Lauwerys et al., 1978.)

A recent analysis by Bellinger and coworkers (1984) also found an association between increasing cord-lead levels and deficits in the child's subsequent performance on the Bayley development scales, after controlling for covariates. Again, the mean cord-lead levels in this study were very low (under 10 ug/dl).

Finally, Erickson et al. (1983) found lung- and bone-lead levels in children who died from Sudden Infant Death Syndrome were significantly higher ($p < 0.05$) than in children who died of other causes, after controlling for age. While this study suggests a potential relationship between lead and Sudden Infant Death Syndrome, this issue remains to be more fully evaluated.

In addition, lead has been implicated in complications of pregnancy, including early and still births. Fahim et al. (1976) found that women who had normal full-term pregnancies had average blood-lead levels of 14.3 ug/dl, whereas women with early membrane rupture had average blood-lead levels of 25.6 ug/dl, and women with premature delivery had average blood-lead levels of 29.1 ug/dl. Wibberly et al. (1977) found that higher lead levels in placental tissues were associated with various negative pregnancy outcomes, including prematurity, birth malformation, and neonatal death.

Bryce-Smith et al. (1977) found bone lead concentrations in still births of 0.4-24.2 ppm in the rib (average: 5.7) versus typical infant hone lead levels of 0.2-0.6 ppm.

To assess the effect of EPA's current rulemaking on fetal exposure, we performed logistic regressions of the probability of adults (over 15 years) from the NHANES II survey having blood lead levels above 30 ug/dl. The regression variables were selected by a stepwise logistic regression procedure that chose all variables that were significant at the $p = 0.05$ level. We then used these regressions to predict the percent of women who would be above 25 ug/dl in 1986-1992, under the final rule. We multiplied the change in the percent of women of child-bearing age by the expected total number of live births each year to estimate the change in the number of fetuses born to mothers exposed to more than 25 ug/dl of blood lead; this is shown on Table IV-5. (The regression coefficients are included in Appendix C.)

TABLE IV-5. Estimated Decrease in the Number of Fetuses Exposed in Utero to > 25 ug/dl of Blood Lead

1985	1986	1987	1988	1989	1990	1991	1992
1,275	3,800	3,200	2,800	2,550	2,200	2,000	1,900

IV.D. Monetized Estimates of Children's Health Benefits

The health benefits of reducing children's exposure to lead are diverse and difficult to estimate quantitatively or to value in monetary terms. To monetize the benefits, we focused on two admittedly incomplete measures: savings in expenditures for medical testing and treatment, and savings in compensatory education. These measures of benefit exclude many important factors, such as reduced pain and suffering, or higher earnings in later life.

In fact, many children with elevated blood-lead levels are neither detected nor treated. However, our estimation procedure assumes that children who go undetected and untreated hear a burden at least as great as the cost of testing, treating, and providing compensatory education for those who are detected. So, all children with high blood-lead levels are assumed to incur "costs", whether medical expenditure costs or personal costs in the form of poor health., inadequate learning, etc.

IV.D.1. Reduced Medical Costs

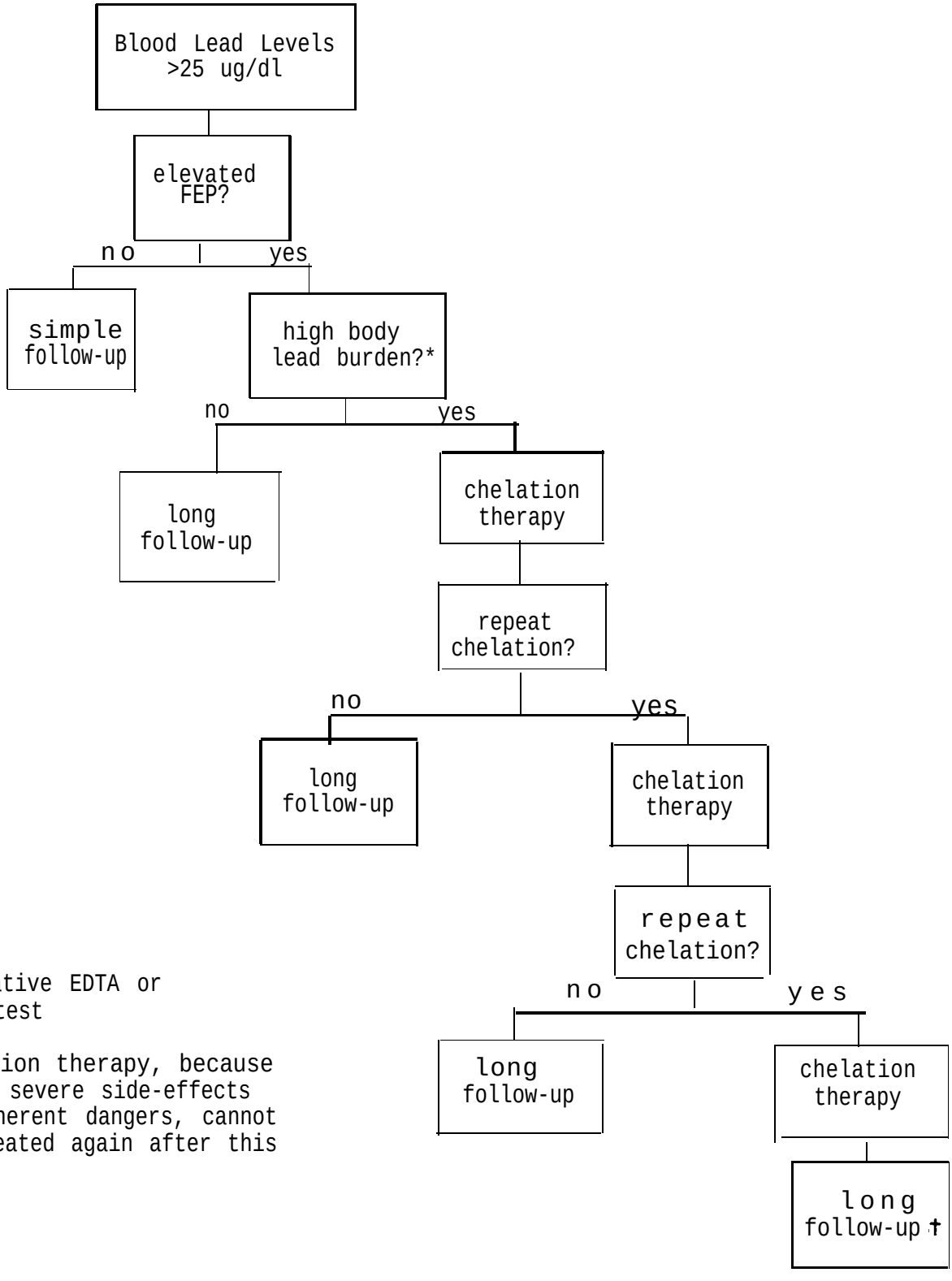
To estimate the benefits of reduced medical care expenses, we assumed that children with elevated blood lead levels would receive the treatment recommended by Drs. Piomelli, Rosen, Chisolm,

and Graef in a recent article in the Journal of Pediatrics (1984). Those four leading experts in the clinical treatment of lead toxicity combined their data and clinical experience to develop optimal diagnosis, treatment, and follow-up protocols. They also estimated the percentages of children at different blood-lead levels who would require various types of treatment. Figure IV-7 summarizes the treatment options that we considered, based on the recommendations of Piomelli et al.

We assumed that administrative expenditures and follow-up tests would cost $100 for every child found to be over 25 ug/dl at screening. Of those children over 25 ug/dl blood lead, we estimated, based on Piomelli et al. (1982) and Mahaffey et al. (1982), that 70 percent would be over 35 ug/dl FEP. Piomelli et al. (1984) recommend provocative ethyleneamentetraacetic acid (EDTA) testing for such children. EDTA testing typically requires a day in the hospital and a physician's visit; we assumed a cost of $500 per test. We also assumed that all children receiving EDTA testing would receive a series of follow-up tests and physicians' visits, at a combined cost of $300.

The purpose of EDTA testing is to see if children have a dangerously high body-lead burden (a lead excretion ratio over 0.60, per Piomelli et al.). Table IV-6 presents Piomelli et al.'s estimates of the percentages of children at various blood lead levels who will require chelation therapy; it ranges from a low of zero for those under 30 ug/dl to a high of 100 percent for those over 59 ug/dl.

FIGURE IV-8 Flow Diagram for Children with Blood Lead Levels above 25 ug/dl



NOTES:

*Provocative EDTA or other test

†Chelation therapy, because of its severe side-effects and inherent dangers, cannot be repeated again after this point

TABLE IV-6. Percent of Children Requiring Chelation Therapy

Blood Lead Levels	Percent
25-30 ug/dl	0
30-39 ug/dl	
age three and over	9.6
age under three	11.5
40-49 ug/dl	
age three and over	26.0
age under three	37.9
50-59 ug/dl	
age three and over	36.0
age under three	49.0
above 59 ug/dl	100.0

Based on our analysis of NHANES II, we estimated that, of those children over 25 ug/dl blood lead, about 20 percent are between 30 and 40 ug/dl and 10 percent are over 40 ug/dl. Based on those estimates and the percentages in Table IV-6, we assumed that 5 percent of the children above 25 ug/dl would require chelation therapy. In addition, we assumed that half of those children chelated would require a second chelation due to a rebound in their blood lead level, and that half of those children would require a third chelation treatment. Thus, we assumed a total of 0.0875 chelations would be required for every child over 25 ug/dl blood lead at screening. We assumed that chelation would require five days in the hospital, several physicians' visits, laboratory work, and a neuropsychological evaluation, for a total cost of about $2,500 per chelation.

Multiplying each of these costs by its associated probability and then summing them yields the estimated cost per child found over 25 ug/dl at screening: 1.0(100) + 0.7(500) + 0.7(300) + 0.0875(2500) = $878.75, which we round to $900. This is lower than the amount cited in a memo to the docket describing this new methodology (August 16, 1984). The reason for this difference is that, in the text of their article, Piomelli et al. recommend chelation for all children over 50 ug/dl; our earlier memo assumed that treatment in estimating costs. However, their data actually indicate that some children in that range may not require chelation, and, in this document, we have made the more conservative assumption that not all children over 50 ug/dl will receive it.

Because we have not included welfare losses (such as work time lost by parents), the adverse health effects of chelation therapy itself (such as the removal of necessary minerals and potential severe kidney damage), or such non-quantifiables as the pain from the treatment, we believe our estimate of the benefits is conservative. As mentioned previously, these medical costs are a measure of avoidable damage for all the incremental cases of lead toxicity, whether detected or not.

IV.D.2. Reduced Costs of Compensatory Education

As discussed earlier, several studies show that moderate-to-high exposures to lead reduce cognitive ability, as measured by IQ tests. The studies by de la Burde and Choate (1972, 1975) also indicate that these cognitive effects, together with lead-induced behavioral problems, translate into poorer performance in school; they found that children in their high lead group were seven times more likely than similar children with lower lead levels to repeat a grade or be referred for psychological counseling. Supplementary educational programs may compensate for some of these effects, though certainly not all of them.

To estimate roughly the cost of such compensatory education, we relied on data in a study prepared for the Department of Education's Office of Special Education Program. Kakalik et al. (1981) estimate that part-time special education for children who remained in regular classrooms cost $3,064 extra per child per year in 1978; adjusting for changes in the GNP price deflator yields an estimate of $4,290 in 1983 dollars. This figure is quite

close to Provenzano's (1980) estimate of the special education costs for non-retarded, lead-exposed children.

Based on de la Burde and Choate's finding that cognitive effects persist for at least three years, we assumed that each child needing compensatory education would require it for three years. De la Burde and Choate's high-lead group consisted mostly of children over 40 ug/dl blood lead, who make up about 10 percent of all children over 25 ug/dl. A few, however, were in the range of 30-40 ug/dl, and other studies (as discussed above in Section IV.B) have found cognitive effects at levels well below 40 ug/dl. Thus, we assumed that 20 percent of all children over 25 ug/dl are affected severely enough that compensatory education would be appropriate. Thus, our estimated average cost per child over 25 ug/dl is (0.20)(3)(4,290) = $2,574, which we round to $2,600.

IV.D.3. Summary of Estimated Benefits

Adding our estimates of compensatory education and medical costs yields a combined benefit estimate of $3,500 per case avoided of a child's blood-lead level exceeding 25 ug/dl. Although for convenience we have computed the average benefit per child over 25 ug/dl, it is important to note that most of the monetized benefits are attributable to reducing lead in children who would be at much higher levels (multiplied by the fraction of children over 25 ug/dl who are at those higher levels). It is also critical to reiterate that our estimates are incomplete, omitting many important categories, and thus are likely to be significant underestimates of the benefits of reducing lead in gasoline.

Table IV-7 presents year-by-year estimates of the monetized children's health benefits of the alternative rules. They are simply the estimated reductions in the number of children above 25 ug/dl (from Table III-6) multiplied by $3,500 per case. As before, they assume that all misfueling is eliminated in each year. The sensitivities of the results to alternative assumptions about misfueling are explored in Chapter VIII.

TABLE IV-7. Year-by-Year Monetized Benefits of Reducing Children's Exposure to Lead Under Alternative Rules, Assuming No Misfueling (millions of 1983 dollars)

Category / Rule	1985	1986	1987	1988	1989	1990	1991	1992
Medical Care								
Proposed	0	155	141	130	117	107	95	93
Alternative	65	139	134	130	117	107	95	93
Final	65	155	141	130	117	107	95	93
Compensatory Education								
Proposed	0	447	408	374	338	309	276	268
Alternative	187	400	387	374	338	309	276	268
Final	187	447	408	374	338	309	276	268
Total								
Proposed	0	602	550	504	455	417	371	361
Alternative	252	539	522	504	455	417	371	361

1987 1988 1989 1990 1991 1992

CHAPTER V

HEALTH BENEFITS OF REDUCING LEAD: ADULT ILLNESSES RELATED TO BLOOD PRESSURE

Concerns about the health effects of ambient exposure to lead traditionally have focused on children. Although lead has a variety of adverse effects on the health of adults, most of them appear not to be of substantial concern except at very high blood-lead levels. Recently, however, two new and extensive analyses of the NHANES II data set have shown a strong and robust relationship between blood lead levels and blood pressure. That finding has important implications for the benefits of reducing lead in gasoline, because high blood pressure, in turn, is linked to a variety of cardiovascular diseases.

This chapter analyzes the health benefits for adults of reducing lead in gasoline, but is limited in several ways. First, we evaluated only illnesses related to blood pressure, although lead has other adverse effects on adults. Second, the analysis is restricted to males aged 40 to 59, because lead appears to affect blood pressure only in men, not women, and because the best data are available for that age range. Finally, most of the estimates cover only whites, because the existing studies of disease associated with blood pressure have had insufficiently large samples of nonwhites. For these reasons, the estimates contained in this chapter are likely to understate significantly the adult health benefits of reducing lead in gasoline. The most important omissions are older males and black males of all ages.

The estimates presented in this chapter should be treated as preliminary. They rely heavily on a recent paper by Pirkle et al. (1985) that has been published in a peer-reviewed journal (The American Journal of Epidemiology), but has not yet been widely reviewed. A summary of that paper and the calculations underlying the estimates in this chapter was placed in the docket for this rulemaking (Schwartz, "Blood Lead and Blood Pressure", September 7, 1984). Another recently published paper (Harlan et al., 1985) also reports a statistically significant relationship between blood pressure in the NHANES II data set. Until the broader scientific community has had a chance to review these papers and comment on their findings, EPA will not rely on blood-pressure-related benefits for this lead-in-gasoline rulemaking. These health effects will be considered in the Agency's ongoing deliberations on a ban, however, and are addressed here for information purposes.

V.A. The Relationship Between Blood Lead and Blood Pressure

This section analyzes the statistical relationship between blood lead and blood pressure. The first part provides a brief overview of earlier studies on the subject, while the second part provides a detailed discussion of a recently completed statistical analysis of the NHANES II data.

V.A.l. Earlier Studies

Lead has long been associated with effects on blood pressure and the cardiovascular system, including a paper in the British

Medical Journal by Lorimer in 1886 that found that higher blood-lead levels increased the risk of hypertension. Most of the studies have focused only on hypertension and relatively high lead-exposure levels, and have not looked for a continuous effect of lead on blood pressure. Investigators reporting such an effect include Beevers et al. (1980), Morgan (1976), Richet et al. (1966), and Dingwall-Fordyce and Lane (1963). Others have failed to find effects of lead on hypertension that were significant at the 95 percent confidence level, although most of them did find a positive association. These include Ramirez-Cervantes et al. (1978) and Fouts and Page (1942).

More recently, Batuman et al. (1983) found an association between chelatable body-lead levels and hypertension in veterans, and several recent general population studies and lower lead-exposure studies (Beevers et al., 1976; Kromhout and Coulande, 1984) have found a significant association with blood lead. Moreau et al. (1982) also found a significant relationship ($p < 0.001$) between blood lead levels and a continuous measure of blood pressure in 431 French policemen, after controlling for age, body mass index, smoking, and drinking.

An even more recent British study (Pocock et al., in press) found blood lead significantly related to blood pressure at the 99 percent confidence level, but the authors felt that the small size of their correlation coefficient suggested no noticeable effect. However, that conclusion appears to reflect a misunderstanding of statistics. It is the regression coefficient that indicates the size of an effect. A correlation coefficient

confounds that measurement with the variances of the dependent and independent variables. While their full data set was not available to us, Pocock et al. presented their grouped data, and we were able to perform a regression of blood pressure versus the log of blood lead on their group averages, both before and after adjustment for confounders. The regressions were weighted by the inverse of the variance of each group, and confirmed their finding that blood lead was a significant predictor of blood pressure in their data, both before and after adjusting for covariates. Moreover, the regression coefficient indicated that the size of the effect was significant, suggesting a change of 3 mm Hg (millimeters of mercury, the standard measure of blood pressure) as blood lead goes from 5 to 15 ug/dl.

Weeden (1975) found lead associated with the vascular renal changes linked to essential hypertension, indicating a possible causal pathway. Cooper and Gaffey (1974) analyzed mortality data from 1,267 death certificates for 7,032 lead workers employed between 1900 and 1969, and found a significant excess of deaths from hypertension disease and renal disease. A later analysis of similar data from 1971 to 1975 also found an increase in cardiovascular and renal disease, but it was no longer significant at the 95 percent confidence level (Cooper, 1981).

Animal data also link lead to hypertension. Victery (1982) found lead associated with a significant elevation of blood pressure in rats with blood lead levels of 41 ug/dl. Importantly,

this study confirms Beevers et al.'s finding of a sex differential, with male but not female rats becoming hypertensive. Webb (1981) examined the vascular responsiveness of tail arteries in rats exposed to blood lead levels in the 40 ug/dl range that had suffered increases in systolic blood pressure, and found that the arteries in exposed rats had increased contraction in response to stimulation by neurotransmitters.

Iannaccone et al. (1981) also reported increased blood pressure ($p < 0.001$) in rats at blood lead levels of 38.4 ug/dl, as well as significant increases in the blood pressure response to noradrenalin. Perry and Erlanger (1979) found that low level exposure of rats to lead produced increases of 15-20 mm Hg in systolic blood pressure. Kopp (1980) repeated those findings and found electrocardiogram changes, indicating an effect on the heart itself. Subsequent tissue analysis of the heart showed reduced levels of ATP in the heart muscle, indicating that heme synthesis inhibition by lead was affecting energy availability in the heart itself.

The direct cardiological effects of lead are also indicated by electrocardiogram changes in lead-poisoned children, which are reversed by chelation therapy (Freeman, 1965; Silver and Rodrigues-Torres, 1968). Williams (1977, 1978, 1979) has shown persistant increased susceptibility to norepinephrine-induced arrhythmias in rats exposed to lead in the first three weeks of life.

V.A.2. Analysis of NHANES II Data

In light of these indications of potential effects of lead

on the cardiovascular system, the relationship between blood lead levels and blood pressure has recently been explored (Harlan et al., 1985; Pirkle et al., 1985) using the NHANES II data. The NHANES II is an excellent data base for this analysis because of the care given to accurate measurements, the great range of information on possible confounding factors, and because it is a representative sample of the U.S. population. As such it avoids the problems of selection bias, healthy-worker effect, other occupational exposures, and the choice of controls that confound many occupational studies. Harlan et al. found blood lead related to blood pressure for males aged 12 to 74 after controlling for the traditional variables associated with blood pressure (age, age-squared, body mass index, race) as well as alcohol consumption, socio-economic factors, and all nutritional variables suspected of affecting blood pressure. Moreover, this relationship held in each year of the NHANES II sample, when analyzed separately, and this relationship held for both blacks and whites.

Pirkle et al. found that blood lead levels were a statistically significant predictor of blood pressure in adult males. This relationship held not only when blood lead was evaluated in a regression with all known factors that have previously been established as correlated with blood pressure, but also when that relationship subsequently was tested against 87 additional variables representing linear and nonlinear functions of every dietary and serologic variable in the NHANES II survey.

Although final judgments about the casual relationship between blood lead and blood pressure must await further review

and study, EPA believes that the Pirkle et al. study provides a reasonable basis for estimating the potential blood-pressure-related benefits of reducing lead in gasoline. Our analysis builds on the Pirkle et al. study, and on additional work by one of its authors (J. Schwartz).

V.A.2.a. Blood Pressure Measurements

Three blood pressure measurements were taken during NHANES II. A seated measurement was taken as soon as the examinee entered. Later, a recumbent measurement was taken. A second seated measurement was taken just before the end of the examination. It is standard medical practice to prefer the second seated measurement, because nervousness on just entering a medical examination center makes the first seated measurement less stable. All of the results presented are for the second seated measurement. However, almost all of the regressions and robustness tests described were performed on all three measurements, and on the average of the first and third seated measurements; all of the conclusions concerning lead's significance held for all eight regressions (four diastolic, four systolic).

V.A.2.b. Initial Analysis

After replicating the Harlan et al. results for all adult males, the first goal was to determine if blood lead levels were related significantly to blood pressure in white males, 40 to 59 years old. This subgroup was chosen because at lower ages both blood pressure and blood lead vary with age. This collinearity could artificially mask or enhance the correlation between blood

lead and blood pressure. Between 40 and 59 years of age, however, blood pressure is essentially independent of age. Choosing this subgroup avoids any collinearity problems. We focused on whites because data relating cardiovascular disease to blood pressure are less extensive for nonwhites. The established correlates of blood pressure are age, sex, race, and one of the indices of relative height-to-weight. Body mass index (BMI = weight/height^2) was used in this analysis. By limiting attention to 40 to 59 year old white males, there was no need to control for race, sex, or, to a large degree, age. Although age was only occasionally significant in the stepwise analysis, both age and age-squared were forced into each multiple regression model to be certain any effect of lead was independent of age.

The natural log of blood lead was more normally distributed, was more statistically significant, and gave a higher R^2 than untransformed blood lead, blood-lead-squared, blood lead plus blood-lead-squared, the square root of blood lead, or blood lead to other fractional powers (0.15, 0.2, 0.3, 0.4). All of the results reported here are for the natural log of blood lead, but regressions using lead on the untransformed scale gave very similar results.

The initial regressions analyzed systolic and diastolic blood pressures for white males, 40 to 59 years old, with a model consisting of age, age-squared, BMI, and blood lead. These regressions were done to determine whether blood lead levels were significantly associated with systolic and diastolic blood pressures after controlling for age, sex, race, and BMI, which are

well-documented correlates of blood pressure. Lead was statistically significant ($p < 0.01$) for both systolic and diastolic blood pressures in all the regressions (unweighed, weighted, and weighted with design effects). The regressions also tested whether this relationship held up when other potentially confounding variables were considered.

V.A.3. Tests of Robustness

The regression models were expanded to incorporate additional variables, with particular attention directed to the stability and significance of the lead coefficient in the presence of nutritional factors and blood biochemistries.

A large set of nutritional and biochemical variables from NHANES II was included in the stepwise regressions. Additional regression analyses considered potential problems of interaction terms. To ensure the robustness of the relationships, further analyses were done to address marginally insignificant variables and nonnutrition variables. Although our analysis focused on males aged 40 to 59, additional regressions were also performed considering all males over age 20.

V.A.3.a. Nutritional and Biochemical Variables

To provide an unusually rigorous test of the independent significance of blood lead, almost all of the nutritional and biochemical variables in the NHANES II were included in stepwise regressions. In addition, to account for possible curvilinear relationships, squared and natural logarithmic transformations

of almost all of these variables were also included. The variables are listed in Table V-1. The objective was not to evaluate the possible association of nutritional or biochemical measurements with blood pressure, but rather to conservatively estimate the strength and independence of the relationship between blood pressure and blood lead.

Including these additional 87 variables increases the probability of variables being found statistically significant due to chance alone. This complicates the interpretation of nutritional and biochemical factors, but not the interpretation of the lead variable; it only makes it more difficult for lead to maintain its significance.

The general procedure for variable selection was as follows. First, weighted stepwise multiple linear regression was used to determine which variables were significantly related ($p < 0.05$) to blood pressure (using the Stepwise and MAXR options of the SAS procedure, STEPWISE). The MAXR procedure was the principle one used; it determines for any given model size (i.e., number of variables) the variables that explain the greatest amount of the variance (i.e., maximize R^2). We chose the largest model with all variables significantly related to blood pressure ($p < 0.05$). The Stepwise option, which uses forward selection with backwards elimination, chose very similar models, and also always chose blood lead. From the 87 nutritional and biochemical variables, the weighted stepwise regression selected five additional variables for diastolic pressure and six additional variables for systolic pressure using a 5 percent significance test. These were used

TABLE V-1. Variables Included in the Stepwise Regression Analyses

age *
age-squared *

body mass index

dietary sodium †

salt shaker sodium

dietary sodium X salt shaker sodium

dietary potassium †

dietary sodium - potassium ratio

dietary calcium †

dietary phosphorus †

dietary protein †

dietary fat †

dietary carbohydrate †

dietary cholesterol †

dietary saturated fatty acids †

dietary oleic acid †

dietary linoleic acid †

dietary iron †
dietary vitamin A †

dietary vitamin C †

dietary thiamine †

dietary riboflavin †

dietary niacin †

serum cholesterol †

serum vitamin C †

serum iron †

serum transferrin saturation

serum zinc †

serum copper †

serum albumin †

hemoglobin †

red blood cell count

ethanol consumption / week †

cigarettes smoked / day

total dietary grams †

total dietary calories †

cigar or pipe smoking

---

* forced into each regression to remove any possible age effects on blood pressure.

† the natural log and squared transformation of these variables were also included in the stepwise regression.

as the starting model for the SAS procedure SURREGR, which additionally incorporated the survey design effects. For both systolic and diastolic blood pressures, one variable from the weighted stepwise regression failed to maintain significance at the 5 percent level after the design effects were incorporated. The final regression results for systolic and diastolic pressures, after accounting for the weighting and design effects, are given in Table V-2.

The multiple logistic regressions (of the probability of hypertension) were also performed using programs from SAS. The procedure LOGIST was used for the stepwise unweighted multiple logistic regression, and the procedure NLIN (nonlinear regression) was used for the weighted logistic regression calculations. The selection process again chose the largest significant model that explained the greatest amount of the variance. Calculations of threshold levels for effects were made using the procedure NLIN on segmented regression models, which finds the threshold point that minimizes the sum of the squares of the error terms. The results of the logistic regression on hypertension are shown in Table V-3. Note that the logistic regressions included blacks as well as whites, because these regressions were used only to predict the effect of lead on the probability of having hypertension and were not used to estimate the number of cardiovascular diseases and deaths. As noted earlier, blacks were not included in the linear regressions because the best available coefficient for predicting cardiovascular risks included insufficient numbers of blacks.

TABLE V-2. Regression of Diastolic and Systolic Blood Pressures in White Males Aged 40 to 59

Variable	Coefficient	t-Statistic	Probability
Diastolic			
Age	0.2768	0.17	0.8636
Age^2	-0.0014	0.10	0.9321
Body Mass Index	1.131	8.55	0.0001
Log(blood lead)	3.954	2.85	0.0080
Dietary Potassium	-0.0018	4.92	0.0001
Hemoglobin	1.548	3.90	0.0005
Albumin	3.587	2.50	0.0179
Log(dietary vitamin C)	1.838	4.65	0.0001
Systolic			
Age	1.311	0.57	0.5720
Age^2	-0.0068	0.30	0.7706
Body Mass Index	1.736	9.42	0.0001
Log(blood lead)	8.436	3.24	0.0028
Albumin	7.088	2.50	0.0178
Log(dietary Vitamin C)	2.411	3.84	0.0005
Log (dietary riboflavin)	-5.509	3.07	0.0044
Log(dietary oleic acid)	3.992	2.49	0.0183
Log(serum vitamin C)	-3.472	2.47	0.0184

TABLE V-3. Weighted Logistic Regression on Probability of Diastolic Blood Pressure Greater Than or Equal to 90 mm Hg in Men Aged 40 to 59

Variable	Coefficient	t-statistic	D-Value
Constant	-16.41	10.13	0.0000
Log(Blood Lead)	0.693	3.96	0.0000
Albumin	0.0873	3.70	0.0001
Body Mass Index	1.700	9.34	0.0000
Hemoglobin	0.0329	5.25	0.0000
Log(Vitamin C)	0.3585	5.98	0.0000
Dietary Potassium	-0.00058	7.47	0.0000
Total Carbohydrates	0.00246	3.09	0.0010

After including the nutritional variables, the blood analytes, and their curvilinear transformations, lead remained significantly associated ($p < 0.01$) with both systolic and diastolic blood pressures. The magnitude of this relationship, adjusted for the other significant variables, is shown graphically in Figures V-1 and V-2. Furthermore, segmented regression analyses indicated there was no threshold blood lead level in the data.

These segmented “hockey stick” regressions fit two regression lines to the data. One, below the putative blood lead threshold T, depends on all the variables except lead. The other, for blood lead levels above T, includes lead. An iterative technique is used to find the value of T that minimizes the sum of the squares of the error terms over the full range of both regression lines. In this case, the error in the regression was minimized at a threshold of zero; that is, lead was significantly related to blood pressure at all levels down to zero.

V.A.3.b. Interaction Terms

In multiple regression analysis, another consideration is the possibility of significant interaction terms. To evaluate this possibility, an additional weighted stepwise regression analysis was done for systolic and diastolic blood pressures. The variables consisted of the linear interaction terms between the final variables in the model (shown in Table V-2) and the linear form of all the other variables originally selected for the initial stepwise regression, including their log and square transforms (Table V-l). This meant running a stepwise regression with 162 interaction terms added to the final regression models

FIGURE V-1

Adjusted Systolic Blood Pressure versus Blood Lead

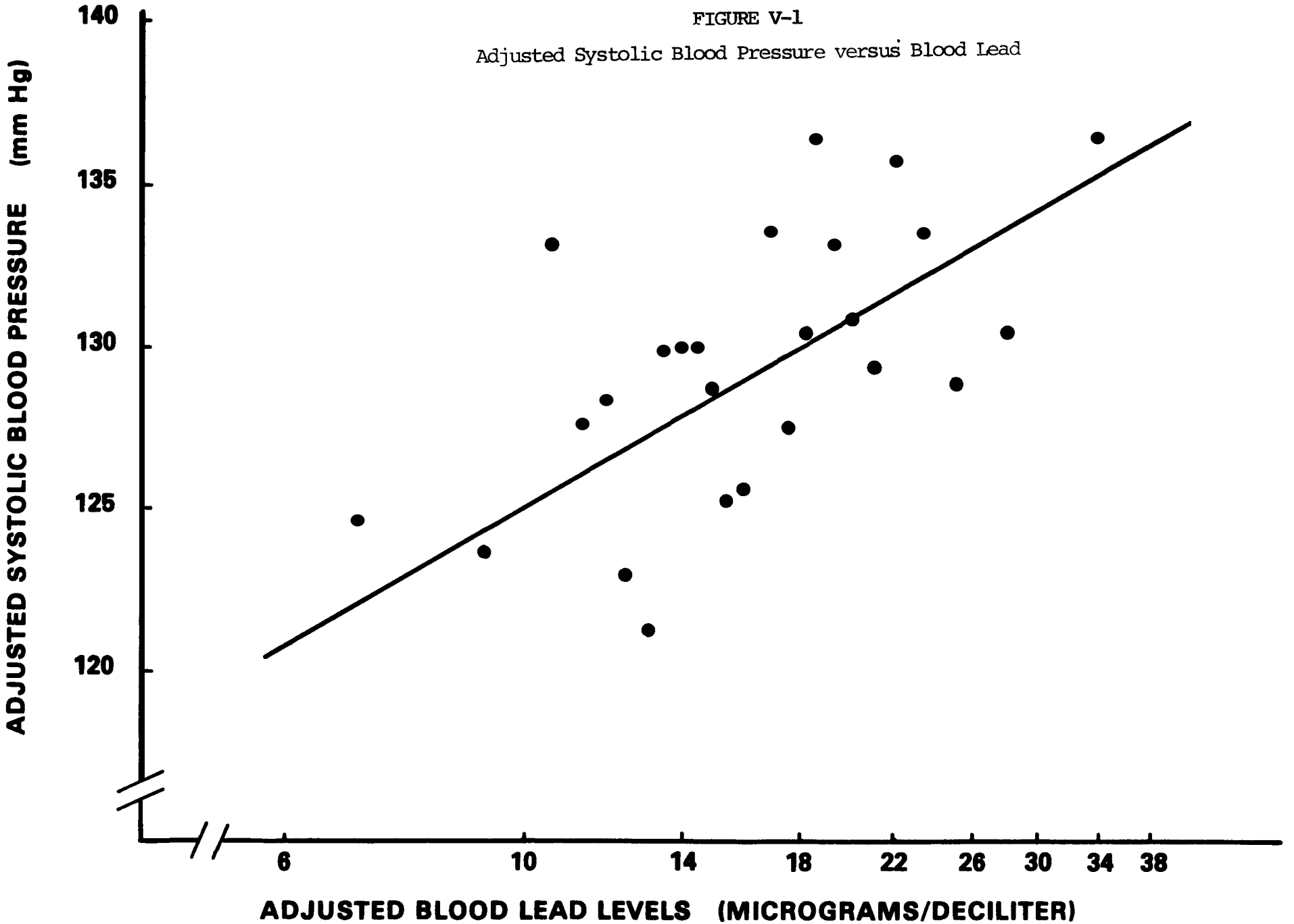
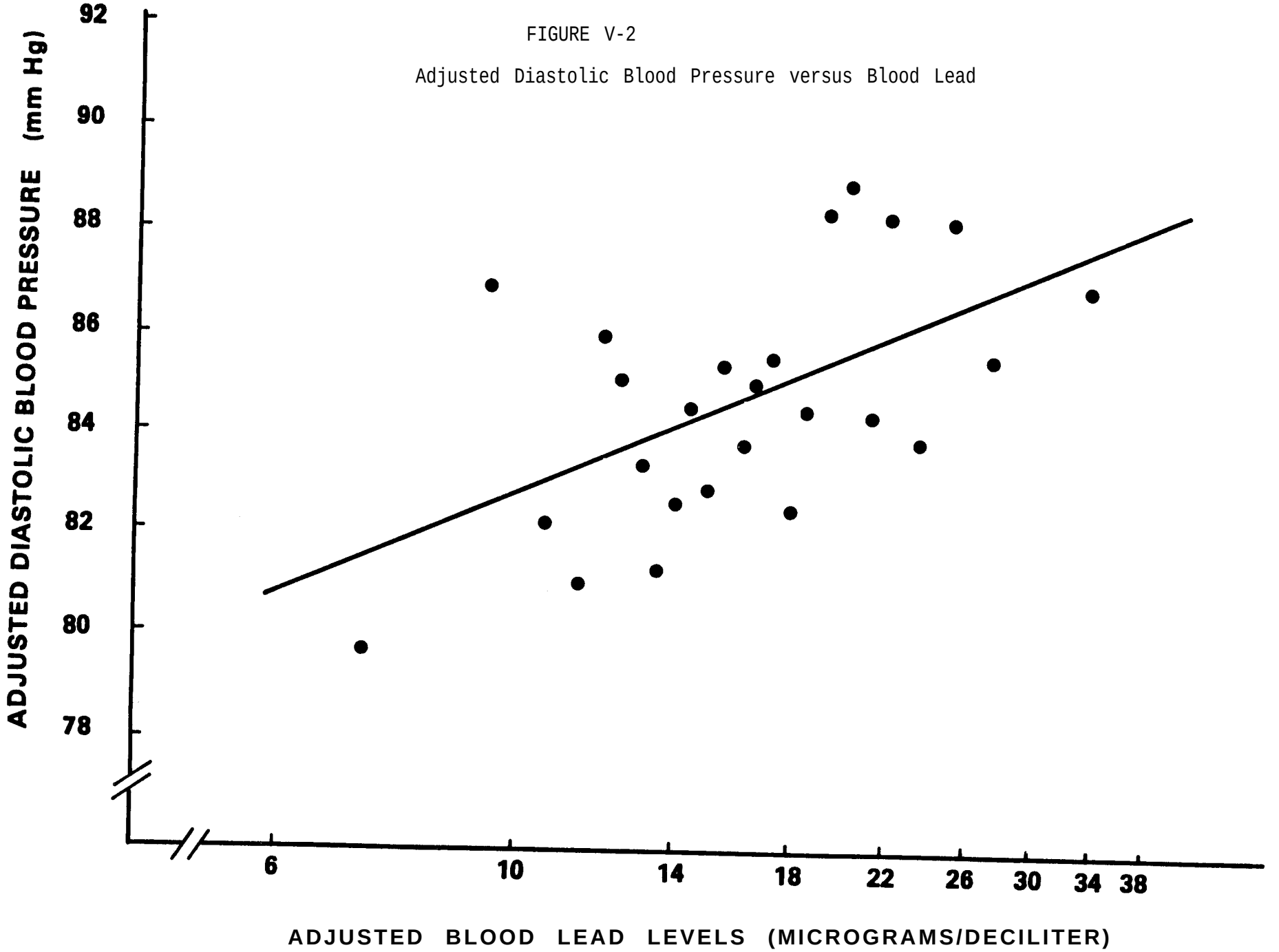


FIGURE V-2

Adjusted Diastolic Blood Pressure versus Blood Lead



for systolic and diastolic pressures. Using such a large set of variables gave a high probability that some variables would enter at the 5 percent level by chance. However, the purpose was not to determine if those variables were independently significant, but, rather, to further test the significance and independence of the relationship between blood pressure and blood lead. As expected, several interaction variables entered the systolic and diastolic regressions, but in each regression the lead coefficient varied less than 10 percent and remained significant ($p < 0.015$).

V.A.3.c. Marginally Insignificant Variables

Three other analyses were done to ensure that this relationship was robust. First, the original weighted stepwise regression was extended to include variables significant through the 15 percent level to see if marginally insignificant variables influenced the significance of lead. For both systolic and diastolic pressures, lead remained significant and there was little change in the magnitude of the coefficient.

Second, for diastolic blood pressure, all the variables were included that were significant between the $p = 0.05$ and the 0.15 levels, and every possible combination of those variables was considered. All 255 combinations were added to the variables that were statistically significant, and a regression was performed on each one. The coefficient of the log of blood lead varied by only plus or minus 10 percent from the value we obtained when we included only significant variables, and the highest p-value for lead was still less than 0.01.

The last analysis was the most demanding test of the independence of the relationship between blood pressure and blood lead. Models for diastolic and systolic blood pressures were fit by weighted stepwise regression to the original model variables (Table V-l), excluding lead. This gave all of the other variables and their curvilinear transformations the maximum opportunity to explain variation that could also be explained by lead. After obtaining this new final model without lead, a single regression was run adding the lead variable to the variables of this new final model. For both systolic and diastolic pressures, lead was still statistically significant ($p < 0.016$) and the magnitude of the lead coefficient changed less than 10 percent from those obtained in the original analysis. The results of all these analyses indicated that the strength and independence of the relationship between blood pressure and blood lead were remarkably stable.

Because some people have found small amounts of ethanol associated with reduced blood pressure, ethanol was also modeled as a quadratic function of consumption, and with two dummy variables for light and heavy drinking. The stepwise regression was repeated, with no change.

V.A.3.d. Nonnutrition Variables

Pirkle et al. then considered nonnutrition variables that might be associated with blood pressure. In additional runs completed since then, we have added several other variables. The complete set is shown in Table V-4. Socio-economic and demographic factors as well as additional medical history variables were included.

TABLE V-4. Nonnutrition Variables Tested in the Stepwise Regression

Demographic Variables	Other Personal-History Variables
Family Income	Tricep Skinfold
Poverty Index	Subscapular Skinfold
Region of the Country	Recreational Exercise
Season of the Year	Work-Related Exercise
Degree of Urbanization	Recent Weight Loss
Residence Inside Central City	Family History of Hypertension
Educational Level	Kidney Disease
	Serum Creatinine

Hypertension Variables
Hypertensive Medication
Low Salt Diet

Hypertension medication and low salt diet were tested -- not for inclusion in a final model, as they are essentially indicators of high blood pressure, but rather to see if the response to lead differed in those groups. The coefficient of lead did not change appreciably, and lead interaction terms with the two variables were insignificant.

The other variables in Table V-4 were tested in two ways. First, the stepwise regression procedure was repeated with them using all nutritional and serum measurements that were significant at the $p = 0.15$ level. The nutritional factors were limited to those significant at the 0.15 level to give the nonnutritional factors a greater chance to enter the model. Again, lead was selected ($p < 0.005$) and its coefficient changed by less than 10 percent from the original model that included only age, age^2, and body mass index.

The variables in Table V-4 were then added to <u>all</u> of those on Table V-1 (including their nonlinear transforms) and the stepwise process was repeated -- with the same results. Finally, the stepwise procedure was rerun using all of the variables in Tables V-1 and V-4 <u>except</u> lead; lead was then inserted into the model resulting from this procedure. It was still significant ($p < 0.006$), with less than a 10 percent change in its coefficient. Because the presence of two terms to describe the curvilinear dependence of blood pressure on age might reduce the chances of variable correlated with age achieving significance, age was modeled as a single curvilinear function (sine of age), and the stepwise

regression repeated; the results were the same. In addition, smoking and drinking were forced into the regression, and lead was still significant ($p < 0.01$), with only a 3 percent change in its coefficient.

Our previous studies have shown that about half of the lead in people in the NHANES II sample came from gasoline. Tetraethyl lead has very little cadmium in it, so confounding with cadmium (which is also suspected of affecting blood pressure) is unlikely. However, we repeated the regression excluding occupationally exposed workers, who may also have cadmium exposure. Lead remained significant ($p < 0.01$), and its coefficient increased somewhat. We also regressed gasoline lead directly on blood pressure, and it was significant.

Although all of these analyses make it clear that collinearity is not a problem in these regressions, variance inflation factors were computed; no significant variable had a variance inflation factor above 1.4. (Variance inflation factors below 4 are considered acceptable in multiple regression analyses.)

To ensure that the significance of lead in the regression was not due to the presence of a few influential observations, influence diagnostic procedures were run. Studentized residuals were plotted for all the observations, and the largest residuals were clustered near the middle of the data, where their influence is slight. Cook's D statistics also were computed for each observation. The highest Cook's D was 0.029, and the second highest was 0.023, both of which are very small. Moreover, of the 10 observations with the largest Cook's D statistics, six had positive residuals and four had negative residuals, indicating that

the most influential observations split almost evenly on which way they would influence the lead regression coefficient.

V.A.3.e. Other Age Groups

The 40 to 59 year old age group represents about one-third of adult males, and is the only one where the confounding of age and blood lead can be eliminated unambiguously. Additional regressions were performed, however, to confirm the Harlan et al. finding of an effect in all adult males. Tables V-1 and V-4 contain several variables that Harlan et al. did not consider in their analysis. Therefore, the stepwise regression analysis was repeated using all of the variables in both tables, and their square and natural log transforms as indicated. All males over the age of 20 were considered. Lead was selected by the regression, with a p-value less than 0.01. To check whether the relationship might be substantially different for different age groups, dummy variables for each 10-year age group (between 20 and 70 years), and interaction terms between lead and those dummy variables, were inserted in the stepwise regression. Such interaction terms check for differences in the lead/blood pressure relationship without having to subdivide the sample. None of the interaction terms was significant at even the $p = 0.15$ level.

V.A.4. Summary of Blood Lead - Blood Pressure Results

The final models for blood pressure, including all statistically significant variables, are shown in Table V-5. The final logistic model for the probability of hypertension is shown in Table V-6.

TABLE V-5. Regression of Diastolic and Systolic Blood Pressures in White Males Aged 40 to 59

Variable	Coefficient	F-Statistic	Probability
Diastolic			
Age	-0.210	0.02	0.8960
Age-squared	0.003	0.04	0.8373
Body Mass Index	1.082	67.88	0.0000
Blood lead †	4.609	12.19	0.0014
Potassium	-0.002	25.30	0.0000
Hemoglobin	0.151	16.81	0.0003
Albumin	0.354	7.42	0.0104
Dietary Vitamin C †	1.886	23.67	0.0000
Family history of hypertension	2.085	4.37	0.0446
Recreational exercise	-1.851	9.48	0.0042
Systolic			
Age	1.142	0.25	0.6226
Age-squared	-0.005	0.05	0.8208
Body Mass Index	1.710	85.90	0.0000
Blood lead †	8.510	10.54	0.0027
Albumin	0.695	6.09	0.0192
Dietary Vitamin C †	2.458	13.78	0.0008
Dietary Riboflavin †	-5.101	8.14	0.0075
Dietary Oleic Acid †	3.650	5.34	0.0275
Serum Vitamin C †	3.365	5.81	0.0218
Family history of hypertension	3.683	4.59	0.0399

† log transform

TABLE V-6. Logistic Regression on Probability of Blood Pressure Greater Than or Equal to 90 mm Hg in Men Aged 40 to 59

Variable	Coefficient	t-Statistic	p-Value
Constant	-15.40	7.0	0.0000
Log(Blood Lead)	0.793	3.20	0.0014
Albumin	0.650	2.06	0.0399
Body Mass Index	0.1571	6.57	0.0000
Hemoglobin	0.0265	3.19	0.0015
Log(Vitamin C)	0.3593	4.22	0.0000
Dietary Potassium	-0.00053	5.33	0.0000
Total Carbohydrates	0.00286	2.86	0.0080
Recreational Exercise	0.3864	0.128	0.0026

It is noteworthy that the logarithmic form of the dose-response relationship suggests a large initial effect, leveling off at higher blood-lead levels. This may explain why only about 60 percent of the occupational studies (i.e., high lead-exposure studies) have found an effect that was significant at the 95 percent confidence level, while almost all of the studies of lower lead levels have found the relationship to be significant.

The other low-exposure studies, the animal data, and the robustness of these results suggest that the relationship is causal. Moreover, specific analyses to determine whether there is a lower threshold below which lead has no effect on blood pressure showed that the data were fit best with a threshold of zero.

V.B. Benefits of Reduced Cardiovascular Disease

Reducing lead in gasoline will reduce blood lead levels, which in turn will reduce blood pressure and the number of individuals with hypertension. The reduction in hypertension will have some direct benefits from reduced medical treatment expenditures. More important, however, will be the indirect benefits in the form of reduced cardiovascular disease associated with elevated levels of blood pressure.

This section describes the methods used to estimate the benefits associated with lowering blood pressure. The first part deals with estimating the reductions in morbidity and mortality, while the second discusses the methods used to value those benefits.

V.B.1. Reductions in Hypertension and Related Morbidity and Mortality

Estimating the reduction in hypertension and cardiovascular disease requires several steps. The first is to estimate the impact of the reduction in gas lead on levels of lead in adults' blood. For that step, we used the regression analyses of the NHANES II data reported in Chapter III. Those regression coefficients were applied to the NHANES II data base to simulate the effects of gasoline lead reductions on blood lead levels. In each case, the blood lead levels in the NHANES II data were first adjusted to reflect reductions that have occurred since the time of the survey. The subsequent steps vary with the condition involved, and are described below.

V.B.1.a. Hypertension

Estimating the change in the number of cases of hypertension was straightforward; the logistic regression coefficients from Table V-6 were applied to the individual NHANES II data to predict the numbers of hypertensives at alternative levels of gasoline lead. The change due to this regulation was calculated by subtracting the number at the new lead level from the number at the original lead level (1.10 gplg). Table V-7 reports the year-by-year estimates for three cases: the Final Rule (0.50 gplg on 7/1/85 and 0.10 gplg on 1/1/86), the Proposed Rule (0.10 gplg on 1/1/86), and the Alternative discussed in the Notice of Proposed Rulemaking (0.50 gplg on 7/1/85, 0.30 on 1/1/86, 0.20 on 1/1/87, and 0.10 on 1/1/88). In all three cases, Table V-7 assumes that the rules eliminate all misfueling; alternative assumptions

TABLE V-7. Reductions in Cases of Hypertension in Males Aged 40 to 59, Assuming No Misfueling (thousands of cases)

Rule	1985	1986	1987	1988	1989	1990	1991	1992
Proposed	0	1,804	1,727	1,649	1,562	1,489	1,396	1,399
Alternative	639	1,527	1,600	1,649	1,562	1,489	1,396	1,399
Final	639	1,804	1,727	1,649	1,562	1,489	1,396	1,399

about misfueling are examined in Chapter VIII. These estimates cover only males aged 40 to 59, but include nonwhites as well as whites.

V.B.1.b. Myocardial Infarctions, Strokes, and Deaths

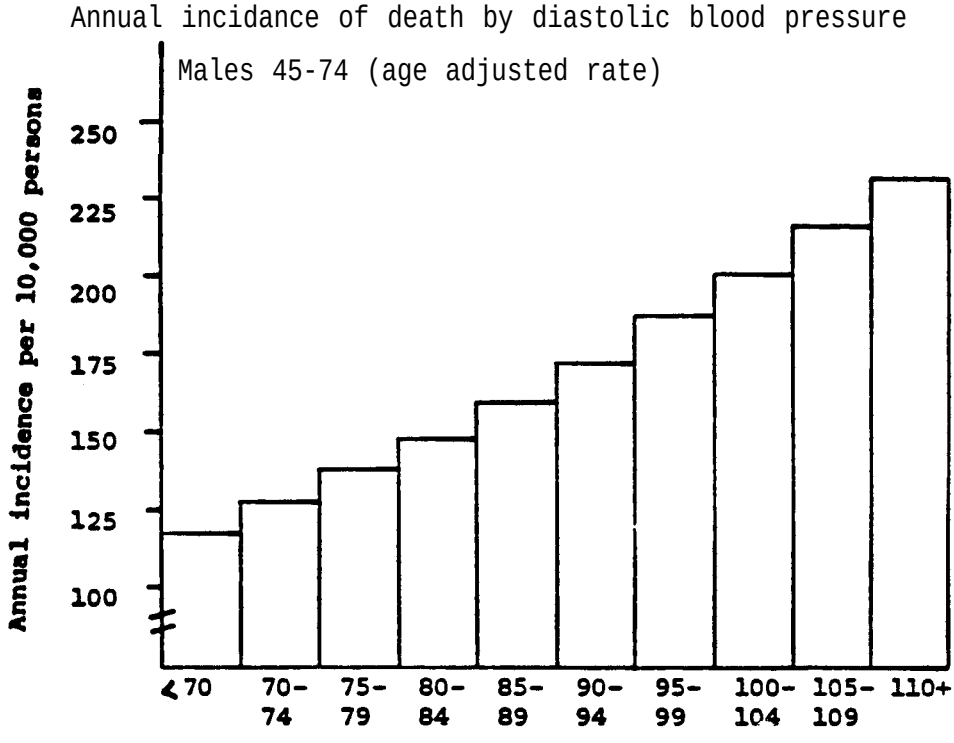
Estimating the impact of reduced blood pressure on morbidity and mortality required several additional steps. Using the NHANES II data and the regression coefficients in Table V-5, we simulated the changes in individual blood pressure levels due to reductions in gasoline lead. Coefficients from two large studies of cardiovascular disease were then used to estimate changes in the numbers of first-time myocardial infarctions, first-time strokes, and deaths from all causes.

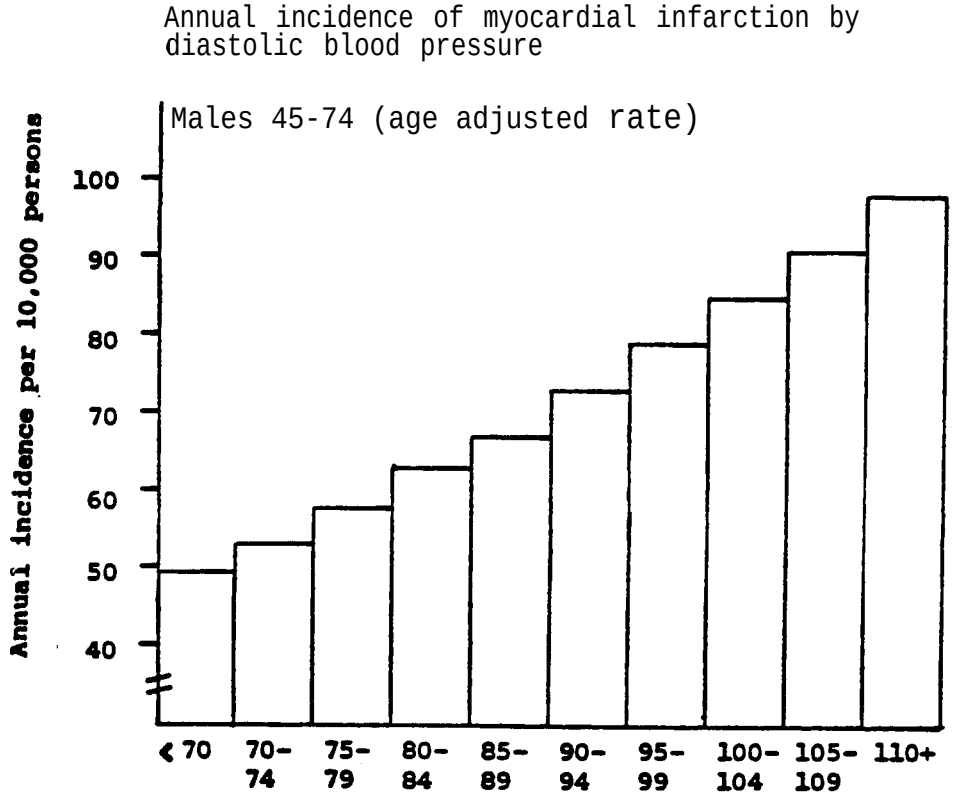
The relationships between blood pressure and cardiovascular diseases are well established by several large, long-term epidemiological studies. The classic study, which was important in establishing cholesterol as a major factor in the risk of heart disease, was the Framingham study (McGee et al., 1976). Extensive analyses of these data have yielded estimates of cardiovascular risks associated with several variables, including blood pressure. Figure V-3 shows the age-adjusted rates of death and heart attacks as functions of blood pressure from that study.

In the 1970s, the National Institutes of Health funded the Pooling Project (The Pooling Project Research Group, 1978), which combined the Framingham data with data from five other long-term studies to improve the accuracy of the risk coefficients for heart attacks. The Pooling Project tested the Framingham coefficients

FIGURE V-3

Adjusted Rates of Death and Heart Attacks versus Blood Pressure: Framingham Data





against the other study results and found that their predictive power was good. It then analyzed the first occurrence of myocardial infarctions (serious heart attacks) in white men who entered the studies at ages 40 to 59 and who were followed for at least 10 years. Our estimates of the numbers of first-time myocardial infarctions under alternative standards employ the Pooling Project's coefficients.

In addition to estimating the risk of heart attacks, the Framingham study estimated regression equations for the risks of stroke and death as functions of blood pressure and other variables. Because the Pooling Project did not include those endpoints, we used the Framingham study coefficients. As with heart attacks, the estimates for strokes cover only first-time events; thus, our estimates for strokes and myocardial infarctions are biased downwards because they exclude second and subsequent heart attacks and strokes associated with elevated blood pressure. The regression equation for deaths covers all causes of death; it includes deaths not just from myocardial infarctions and strokes, but also from other causes associated with blood pressure (e.g., heart diseases other than myocardial infarctions).

Levy et al. (1984) recently tested the Framingham study regression coefficients to see how well they explained the observed decrease in cardiovascular mortality in the United States from 1970 to 1980. They found that the coefficients, when coupled with changes in blood pressure and other cardiovascular risk factors over that same period, were able to explain about 80 percent of the drop in cardiovascular mortality.

The Hypertension Detection and Follow-up Program (NEJM, 1983) found that intervention leading to about a 5mm Hg change in diastolic blood pressure produced a 20 percent reduction in overall mortality. The Australian National Trial on mild hypertension also found reductions in morbidity and mortality resulted from lowered blood pressure (Lancet, 1980). The Multiple Risk Factor Intervention Trial found that drug therapy to lower blood pressure reduced cardiovascular disease in persons with normal resting electrocardiograms (ECGs), but increased it in persons with abnormal resting ECGs (JAMA, 1982). This suggests an adverse affect of the drugs used.

To produce estimates for all 40 to 59 year old white males, the individual risk of each person sampled in the NHANES II was summed and then averaged. Since the sampled individuals represent the U.S. population for their specific age-race-sex category, their average risk represents the average risk for all 40 to 59 year old white men. Because blood lead levels have dropped since the NHANES II period, we corrected for that change and then evaluated the effects of the new lead-in-gasoline limits. Again, only white men were examined because there were too few blacks in the Framingham study, and their risk might be different from whites.

The fact that gasoline lead levels would slowly decline even without new EPA actions created a slight complication. Because gasoline lead levels fall over time in both our base case and the low-lead case, the difference in blood lead levels resulting from the rule will change over time. Therefore, we recalculated the

risk estimates for each year, assessing the annual change in blood lead levels due to reductions in gas lead.

The three cardiovascular-risk regression equations all predict risk over the next 10 years, given current blood pressure, age, and other characteristics. Presumably, the risk in years 2-10 was affected by blood pressure in those years, as well as by initial blood pressure. Because blood pressure levels over time in the same individual are positively correlated, it is likely that the regression coefficient in part picked up the effect of future blood pressure levels. Lacking any data with which to estimate the pure effect of a one-year change in blood pressure, we divided the coefficient for 10-year risk by 10. The adjusted coefficient was then used with the year-by-year predicted changes in blood pressure to estimate risk reductions. This procedure almost certainly overcompensates, lending a downward bias to the results, because current blood pressure is not perfectly correlated with future blood pressure.

We adjusted the population at risk for the increases in the U.S. population of white males aged 40 to 59. The regression from the Framingham study predicting deaths for men aged 40 to 54 was extended to 40 to 59 for data comparability and uniformity. Because the death rate actually increases with age, this also will bias the results downward.

Table V-8 reports the resulting year-by-year estimates of reduced myocardial infarctions, strokes, and deaths for the three phasedown schedules, as was done in Table V-7 for hypertension. As before, these estimates assume that misfueling is completely

TABLE V-8. Reductions in Numbers of Cases of Cardiovascular Disease and Deaths in White Males Aged 40 to 59, Assuming No Misfueling

Condition / Rule	1985	1986	1987	1988	1989	1990	1991	1992
Myocardial infarctions								
Proposed	0	5,350	5,156	4,956	4,726	4,531	4,274	4,289
Alternative	1,829	4,467	4,750	4,956	4,726	4,531	4,274	4,289
Final	1,829	5,350	5,156	4,956	4,726	4,531	4,274	4,289
Strokes								
Proposed	0	1,115	1,074	1,032	984	943	889	892
Alternative	382	932	990	1,032	984	943	889	892
Final	382	1,115	1,074	1,032	984	943	889	892
Deaths								
Proposed	0	5,160	4,971	4,778	4,556	4,367	4,119	4,132
Alternative	1,766	4,310	4,581	4,778	4,556	4,367	4,119	4,132
Final	1,766	5,160	4,971	4,778	4,556	4,367	4,119	4,132

eliminated. They indicate that the rule being promulgated will have a large impact on the incidence of cardiovascular disease. We estimate that the reduction of lead in gasoline in 1986 alone will result in 5,350 fewer myocardial infarctions; 1,115 fewer strokes; and 5,160 fewer deaths from all causes among white males aged 40 to 59. Extending the analysis to men of other ages and to nonwhites would substantially increase these estimates.

V.B.2. Monetized Benefit Estimates

Valuing reductions in morbidity and mortality is a difficult and, to say the least, controversial task. For morbidity, we have restricted our estimates to avoided medical costs and foregone earnings associated with diseases. These estimates clearly are too low, for they fail to account for other important losses associated with disease, including pain and suffering (e.g., the paralysis that often follows a stroke). For valuing the reduction in mortality risk, we have chosen a fairly conservative estimate ($1 million) from the large range obtained from studies of occupational risk premiums.

V.B.2.a. Hypertension

Whether or not it results in coronary or cerebrovascular disease, high blood pressure is a significant chronic illness. It also generates economic costs, in the form of drugs, physicians' visits, hospitalization, and work loss. We used data from the NHANES II and from the National Institutes of Health to estimate the value of avoiding a case of high blood pressure.

The NHANES II ascertained how many times per year a person saw a physician because of high blood pressure. The weighted average, for males 40 to 59 years old with diastolic blood pressure over 90mm, was 3.27 visits per year. We assumed a cost of $35 per visit, for an annual total of $114.

The same population was forced to remain in bed an average of 0.41 days per year because of high blood pressure. At the average daily wage ($80), that translates to $33 per year. NHANES II also found that 29 percent were on medication for hypertension; assuming a drug cost of $200 per year for those on medication yields an annual cost of $58.

The National Hospital Discharge Survey (1979) found that, excluding those with heart disease or cerebrovascular disease, people with high blood pressure used 3.5 million of the occupied hospital bed-days that year; dividing by the 60 million people the NHANES II identified as having high blood pressure gives a rate of 0.058 hospital bed-days per person per year. We have assumed that these results apply to the 40 to 59 year old age group, as well. Using a daily hospital cost of $400 yields an annual cost per hypertensive of $23.

Summing these estimates yields a total of $228 per hypertensive per year. It should be noted that only 29 percent of the people with blood pressure above 90mm in the NHANES II were on medication, in part because some of them had not previously been detected as having high blood pressure. Therefore, the average cost for a detected case will be higher. For example, Weinstein and Stason (1977) used an average cost of $200 in 1975

dollars, or about $450 in 1983 dollars, for treatment of patients undergoing medical care for hypertension. Nevertheless, we have conservatively used $220 as the value of avoiding one case of high blood pressure for one year.

V.B.2.b. Myocardial Infarctions

Our estimate of the benefits of reducing the incidence of myocardial infarctions relies heavily on Hartunian et al. (1981), who estimated the medical expenses and lost wages associated with a variety of diseases. Under the category of myocardial infarctions (MI), Hartunian et al. examined three types of cases: sudden death, fatal MI, and nonfatal MI. ("Sudden death" was classified as a myocardial infarction in the Pooling Project regression coefficients we used.)

For each category and each age group, Hartunian et al. obtained data on the type of medical services needed (e.g., ambulance and coronary intensive care unit), the fraction of cases using each service, and the costs in 1975 dollars. They also determined the annualized recurrence and follow-up costs, by age, for each condition. These were then discounted (using a 6 percent real discount rate) to the time of initial occurrence to estimate the cost, in current dollars, of each new case. The resulting estimates were $96 for sudden death and $7,075 for both fatal and nonfatal MIs.

We have adjusted these 1975 estimates in three ways to reflect 1983 conditions. First, we inflated them to 1983 dollars. Because most of the costs were hospital-related, with the rest

principally being physicians' fees, we inflated the Hartunian et al. cost estimates by a weighted average of 80 percent of the change in the Consumer Price Index (CPI) for hospital rooms and 20 percent of the change in the CPI for physicians' charges. Approximately 90 percent of the Hartunian et al. MI costs were hospital-related, not physicians' fees, and hospital costs rose faster than physicians' fees, so this approach is conservative.

The second adjustment in the 1975 estimates involves changing cost indices. Because cost indices only account for increased costs of the same procedure, in this case principally the initial hospitalization for a heart attack, they do not reflect the cost of new or different procedures. Since 1975, the fraction of people suffering coronary heart disease who subsequently undergo coronary bypass operations has increased substantially. The number of bypass operations tripled in seven years, from 57,000 in 1975 to 170,000 in 1982, while the number of cases of coronary heart disease has remained relatively constant (National Centers for Health Statistics, Hospital Discharge Survey, and unpublished data). Based on the Hartunian et al. data, 7.1 percent of MI cases in 1975 had subsequent bypass operations. Assuming that they shared proportionately in the tripling of the bypass-operation rate, we estimated that an additional 14 percent of MIs now result in a bypass operation. Hartunian et al. estimated the cost of bypass operations at $6,700 in 1975 dollars, or $16,800 in 1983 dollars. Adding 14 percent of this cost to the other direct costs yields an estimate of the total direct costs in 1983 dollars of $20,100 for an MI and $240 for sudden death.

Our third adjustment involved discount rates. Hartunian et al. used a 6 percent real discount rate to present value the future year costs, whereas this analysis employs a 10 percent discount rate. Fortunately, Hartunian et al. performed sensitivity calculations for other discount rates, including 10 percent. Making all of these adjustments, the costs per case are $18,100 for an MI and $216 for sudden death.

Hartunian et al. also obtained data indicating the probability distribution of cases among the different categories. Of the total number of cases in these three categories, about 22.5 percent were sudden deaths and the remaining 77.5 percent were fatal or nonfatal MIs. Applying those percentages to the medical-cost estimates derived above yields a weighted average of $14,076 per myocardial infarction.

Hartunian et al. calculated the present value of foregone earnings based on reduced labor force participation using data on each type of heart disease, broken down by sex and 10-year age categories. We have used those results, with several modifications. First, we excluded foregone earnings for fatal heart attacks, because we valued the reduction in mortality risks separately (see Section V.B.2.d., below). Second, we adjusted for the increase in average non-farm compensation from 1975 to 1983, using information from Data Resources, Incorporated. Finally, we again used a discount rate of 10 percent, rather than the 6 percent used by Hartunian et al. in their base case analysis.

The resulting estimates of foregone earnings are $90,000 for heart attack victims under 45; $47,000 for those between 45 and 54; and $22,000 for those over 55. Based on data from the Pooling Project and NHANES II, 16.1 percent of nonfatal heart attacks in men between 40 and 59 occur in those under 45, 50.9 percent occur in those between 45 and 54, and 33 percent in those 55 and older. Using those percentages yields a weighted average for lost earnings of $45,670 per attack. Combining that earnings estimate with the earlier one for medical costs yields a total benefit per myocardial infarction avoided of about $60,000.

V.B.2.c. Strokes

Our estimates of the benefits of avoiding strokes also rely on Hartunian et al., with similar adjustments. (Unlike myocardial infarctions, we have not adjusted their medical cost estimates for strokes to reflect any changes in medical treatment since 1975.) Table V-9 presents the estimates for three types of stroke -- hemorrhagic, infarctive, and transient ischemic attacks (TIA) -- by age. The averages are based on the distribution of types of strokes and incidence of strokes by age. The overall average is $44,000 per stroke avoided.

We have been unable to estimate a value for avoiding the loss in quality of life that occurs in stroke victims. This is a significant omission. For example, of the people in the NHANES II who reported having had a stroke in the past, 45 percent suffered paralysis in the face and 13 percent still had at least partial facial paralysis, 54 percent suffered paralysis in at

TABLE V-9. Benefits of Reducing Strokes (dollars per case)

Type of Stroke Age	Medical Expenses	Foregone Earnings	Total
Hemorrhagic			
35-44	12,600	41,000	53,600
45-54	13,300	26,000	39,300
55-64	17,200	11,000	28,200
Infarctive			
35-44	17,600	71,000	88,600
45-54	18,100	43,000	61,100
55-64	23,600	14,000	37,600
Transient ischemic attacks			
35-44	3,184	1,114	4,298
45-54	3,184	3,076	6,260
55-64	3,184	8,280	11,464
Weighted average			44,000

least one arm and 21 percent remained paralyzed, 59 percent had numbness in arms or legs and 28 percent had remaining numbness, 30 percent had vision impairment and 13 percent remained visually impaired, and 50 percent had speech impairment with 22 percent continuing to suffer from speech impairment. While we have no estimates of people's willingness to pay to avoid the risk of these profound injuries, common sense suggests that it is high.

V.B.2.d. Mortality

Valuing reductions in mortality is highly controversial. Over the past decade or so, a substantial literature has developed on the subject. Economists are in general agreement that the best conceptual approach to use is the willingness-to-pay (WTP) of the individuals involved. The appropriate value is not the amount that an individual would pay to avoid certain death, but rather the total sum that a large group of individuals would pay to reduce small risks that sum to one; for example, the amount that 10,000 people would pay to reduce a risk to each of them of one in ten thousand.

Several studies have estimated WTP based on implicit tradeoffs between risk and dollars revealed in market transactions. Most of these studies (e.g., Thaler and Rosen, 1976; Smith, 1974 and 1976; Viscusi, 1978) have studied labor markets, based on the premise that, all else being equal, workers must receive higher wages to accept a higher risk of being injured or killed on the job. Such studies typically regress wages on risk and a variety of other explanatory variables (e.g., levels of education required,

worker experience, whether or not the industry is unionized, location, and non-risk working conditions). In such regressions, risk might be measured as the number of fatalities per 1,000 workers per year. The coefficient for that variable is then interpreted as the amount of extra wages needed to compensate for a 0.001 risk of death. Dividing the coefficient by the unit of risk yields the estimate of WTP to avoid a statistical death. For example, if the coefficient is $500, the estimated WTP is $500,000 (= $500/0.001).

A few studies have estimated WTP in nonoccupational settings. Blomquist (1977), for example, estimated the implicit cost-risk tradeoffs that individuals make in deciding whether or not to take the time to put on seat belts.

None of these studies yields definitive answers. All suffer from data limitations (e.g., incomplete information on possible confounding variables and on the extent to which individuals perceive the risks they face). Not surprisingly, given these problems, the studies also yield a wide range of estimates. A recent survey of the literature prepared for EPA found a range of $400,000 to $7 million per statistical life saved (Violette and Chestnut, 1983). Based on that survey, EPA's RIA guidelines do not attempt to set any specific value, but rather recommend that range. To simplify the presentation of the results, this RIA uses a single value from the lower end of that range, $1 million per statistical life saved. Although we do not present any formal sensitivity analyses on this value, the results in Chapter VIII show that the net benefits are so large that they

would remain positive whatever part of that broad range were used; even at $400,000 per statistical life saved, the estimated benefits would be many times higher than the costs.

V.B.3. Summary of Blood Pressure Benefits

Table V-10 summarizes the benefits of reducing the numbers of cases of hypertension, myocardial infarctions, strokes, and deaths due to high blood pressure. As in earlier tables, these estimates assume that misfueling is eliminated. These are conservative estimates for several reasons:

(1) The hypertension estimate covers only males aged 40 to 59.

(2) The other estimates cover only white males aged 40 to 59.

(3) We have not assigned any value to reduced pain and suffering associated with hypertension, myocardial infarctions, and strokes.

(4) We have not estimated any health benefits for adults other than those related to blood pressure.

In addition, of course, some readers may quarrel with the value assigned to reduced risk of mortality; we have chosen a single value for convenience, not because we believe any particular value can be defended strongly. Despite these limitations, the estimated benefits of the final phasedown rule are large, totaling $5.9 billion in 1986.

As discussed at the beginning of this chapter, these estimates should be treated as tentative. Although the two key studies (Pirkle et al., 1985; Harlan et al., 1985) recently have been published in peer-reviewed journals, they have not yet been

TABLE V-10. Year-by-Year Estimates of Blood Pressure Benefits, Assuming No Misfueling (millions of 1983 dollars)

Rule	1985	1986	1987	1988	1989	1990	1991	1992
Proposed	0	5,927	5,707	5,484	5,227	5,008	4,722	4,736
Alternative	2,033	4,955	5,262	5,484	5,227	5,008	4,722	4,736
Final	2,033	5,927	5,707	5,484	5,227	5,008	4,722	4,736

widely available for public review. As a result, EPA has not relied on blood-pressure-related health effects in reaching a decision on the final phasedown rule. These potentially serious health effects will be considered by EPA, however, in connection with a possible ban on lead in gasoline, and extensive review and comments will be sought on them.

CHAPTER VI

BENEFITS OF REDUCING POLLUTANTS OTHER THAN LEAD

Decreasing the amount of lead in gasoline will reduce emissions of several pollutants in addition to lead. Most of these reductions will result from decreased “misfueling,” the misuse of leaded fuel in vehicles equipped with pollution-control catalysts. In such vehicles, leaded gasoline poisons the catalysts, greatly reducing their effectiveness in controlling emissions of hydrocarbons (HC), nitrogen oxides (NO_x), and carbon monoxide (CO). Reducing lead in gasoline should affect emissions from misfueling in two ways. First, it will be more expensive to produce 89 octane leaded gasoline at 0.10 gplg than to produce 87 octane unleaded gasoline. This change in relative manufacturing costs should alter retail price differentials (although, as discussed in Chapter 2, it may not make unleaded cheaper than leaded at the pump), thus reducing the incentive to misfuel. In addition, even for those vehicles that continue to be misfueler it will take considerably longer to destroy the effectiveness of catalysts with 0.10 gplg leaded gasoline than it does now with 1.10 gplg.

All three of these pollutants have been associated with damages to health and welfare, and contribute to ambient air pollution problems covered by National Ambient Air Quality Standards (NAAQS). CO is itself a “criteria pollutant,” covered by a NAAQS. NO_x is the composite formula for nitrogen oxide (NO) and nitrogen dioxide (NO_2); NO_2 is covered by a NAAQS. Although most NO_x is emitted as NO, some of it is chemically transformed

in the atmosphere to NO_2. NO_x and HC both contribute to the formation of ozone (O_3), another criteria pollutant. In addition, certain hydrocarbons (in particular, benzene) have been linked to cancer.

Independent of its effect on misfueling, reducing lead in gasoline also will reduce emissions of ethylene dibromide (EDB), which is added to leaded gasoline as a "scavenger" to reduce the build-up of lead deposits in engines. Because EDB is added in proportion to the amount of lead, tightening the lead standard will reduce the amounts added to gasoline. EDB has been linked to increased risk of cancer.

This chapter examines the impacts of reducing these pollutants, focusing on the three associated with misfueling: HC, NO_x, and CO. Section A estimates the emissions caused by misfueling. Section B addresses the ozone-related health and welfare effects, which account for the vast majority of the benefits that we were able to quantify. Section C discusses the health and welfare gains associated with pollutants other than ozone. We have tried to estimate as many of the effects of these pollutants at ambient concentrations as possible, but our quantitative estimates are subject to considerable uncertainty and provide incomplete coverage of the potential effects of emission reductions.

In both Sections B and C, the estimates of health effects are presented in physical rather than monetary units, but non-health effects (such as crop losses) are estimated in dollars. Finally, in Section D, we estimate the monetized economic benefits

of eliminating misfueling using two methods. The first values directly the health and welfare effects estimated in Sections B and C. That method is conceptually the more appropriate one, but it omits some important categories because of incomplete quantification. These emissions are likely to lend a downward bias to the direct estimates. This downward bias is most obvious in the case of CO, for which we have not monetized any benefits, but also is of major concern for NO_x, for which we have identified but have been unable to quantify, several potentially significant benefit categories, in particular health effects and damages from acid deposition. Even in the case of HC, for which we have quantified significant ozone-related benefits, the estimates may be biased downward significantly because of our inability to quantify ozone's impacts on chronic health conditions and forests, nor have we estimated direct health effects of any HCs other than benzene. Further, there is considerable uncertainty in those categories we have included.

The second method monetizes the emission reductions using the values implied by the cost of the pollution control equipment needed to meet the emission limits set by Congress. Our final monetized benefit estimates, used in later chapters to compute the total and net benefits of alternative rules, are the averages of these two methods.

The basic methodology used in this chapter is the same as that employed in Schwartz et al. (1984) and in the preliminary RIA. The analysis has been refined in several areas, however.

First, to estimate emissions associated with misfueling, we have used the results of EPA's 1983 tampering and misfueling survey, the results of which were not available for use in the earlier documents. Second, the estimates of the health effects associated with ozone exposure rely on an updated statistical model. Third, in this document we do not rely on a property-value-based estimate of NO_x estimates, because closer examination of the underlying study suggested that it had not adequately accounted for confounding factors, in particular other pollutants whose concentrations may covary with NO_x. Finally, and most importantly, additional and more detailed analysis of the impact of HC and NO_x emissions on ozone has caused us to revise downwards significantly our estimates of the rule's impact on ozone concentrations.

Throughout this chapter, we estimate the effects and monetized benefits of eliminating misfueling altogether. For lead levels other than a complete ban on all leaded gasoline, this is probably an overly optimistic assumption. Chapter VIII presents estimates based on a broader range of alternative assumptions about the impacts of different rules on misfueling.

VI.A. Emissions Associated with Misfueling

"Misfueling" or "fuel switching" refers to the use of leaded gasoline in a vehicle originally designed and certified to use unleaded gasoline. Because leaded regular gasoline is cheaper and higher in octane than regular unleaded, some drivers deliberately misfuel their vehicles in an attempt to reduce expenses or to improve vehicle performance.

Misfueling can occur by removing or damaging the nozzle restrictors installed in the fuel inlets of vehicles with catalytic converters, by using an improper size fuel nozzle, or by funneling leaded fuel into the tank. Sometimes gasoline retailers sell gasoline that is mislabeled or contaminated (U.S. EPA, 1983a), but this accounts for less than 1 percent of misfueling.

It is illegal for service stations or commercial fleet owners to misfuel or to allow the misfueling of vehicles originally equipped with catalytic converters. Federal law does not apply to individuals who misfuel their own vehicles, however.

Using leaded gasoline in vehicles with catalytic converters damages this pollution control equipment, and can increase emissions of HC, CO, and NO_x by as much as a factor of eight. Table VI-1 shows the emissions increases caused by misfueling on a per-mile basis. The estimates distinguish between pre- and post-1981 vehicles because emission standards changed in that year, leading to changes in the design of catalysts and other emission control devices. In vehicles manufactured before 1981, misfueling has no effect on NO_x emissions, but does cause relatively large increases in HC and CO emissions.

Misfueling is a significant problem. Several recent surveys by EPA have shown that a substantial number of vehicles are misfueled with leaded gasoline. According to the 1983 survey (U.S. EPA, 1984e), about 15.5 percent of light-duty vehicles designed to use unleaded gasoline are misfueled with leaded. The 1982 survey (U.S. EPA, 1983a), showed a lower rate, about 13.5

TABLE VI-1. Increase in Emissions Due to Misfueling (grams/mile)

Light-Duty Vehicle Model Years	HC	CO	NO_x
1975 to 1980	2.67	17.85	0.0
1981 and later	1.57	11.07	0.71

Source: U.S. EPA, Office of Mobile Sources, "Anti-Tampering and Anti-Misfueling Programs to Reduce In-Use Emissions from Motor Vehicles," May 23, 1983.

percent. Misfueling rates apparently vary by the age of the vehicle, by whether it is in an area with an Inspection and Maintenance (I/M) mobile source enforcement program, by whether it is part of a commercial fleet, and other factors. Table VI-2 provides 1983 misfueling rates by model year of vehicle and by I/M status. We assumed for our analysis that the rates of misfueling by age of vehicle would stay constant in the absence of new regulations.

The EPA surveys probably underestimate real misfueling rates by a significant margin, primarily because vehicle inspections for misfueling are voluntary, which would bias the results downward (assuming that misfuelers are less likely to agree to have their vehicles tested). In some areas, the rates of drivers refusing inspections were very high. In the 1982 survey, the refusal rates ranged from 1 to 8 percent in I/M areas, and from 3 to 44 percent in non-I/M areas.

To estimate the reduction in emissions that would be achieved by eliminating misfueling, we combined the data in Tables VI-1 and VI-2 with our fleet model (described in the Appendix), which projected the number of vehicles of each model year and their annual mileage. For each year of our projection, we first estimated the number of vehicles that would have misfueled for the first time in that year. (We assumed that no emission reductions would result from stopping the misfueling of vehicles that already had their catalysts destroyed by misfueling in earlier years.) We then projected over the remaining lifetime of the vehicle the expected excess pollutants it would have emitted due to misfuel-

TABLE VI-2. Misfueling Rates in 1983 (percent)

Model Year	Overall Misfueling Rates	I/M Areas	Non-I/M Areas
1984	1.6	8.7	0.0
1983	5.1	4.8	5.2
1982	8.0	3.2	9.0
1981	10.0	7.9	10.5
1980	9.0	6.2	9.7
1979	19.6	17.9	20.0
1978	19.0	7.3	21.6
1977	22.6	23.2	22.4
1976	25.5	13.9	28.0
1975	25.9	16.0	28.0
Weighted Average:*	15.5		

* This weighted average does not account for the number of miles driven by each model year.

ing. The projections account for the facts that older vehicles drive fewer miles per year, that survival rates decline with age, and that the efficiency of emission control devices deteriorates with age. These projected emission streams were then discounted (at a 10 percent real rate) back to the year in question. Table VI-3 presents the resulting year-by-year estimates of the discounted emissions avoided by eliminating misfueling. Note that the estimate for each year is not of actual emission reductions achieved in that year, but rather the discounted value of emission reductions due to stopping the first-time misfueling of vehicles in that year. The numbers of tons of HC and CO controlled remain fairly constant from 1986 through 1992, at over 300,000 tons of HC and more than 2.5 million tons of CO. The estimates of reduced NO_x emissions increase, from 94,000 tons in 1986 to 150,000 tons in 1992, because of the increase in the proportion of post-1981 vehicles. (Recall that misfueling does not increase NO_x emissions in vehicles manufactured before 1981.)

VI.B. Health and Welfare Effects Associated with Ozone

Hydrocarbons and nitrogen oxides react photochemically to form ozone ("smog"). Ozone in turn affects health, materials damage, and vegetation. To estimate the ozone-related effects of reducing emissions of HC and NO_x, we employed a two-step process. First, as described below in Section VI.B.1, we estimated the health and welfare effects of a 1 percent change in ozone. Second, as discussed in Section VI.B.2, we estimated the relationship between reducing HC and NO_x and the subsequent decrease in ozone.

TABLE VI-3. Year-by-Year Estimates of Reductions in Emissions, Assuming No Misfueling (thousands of metric tons)

Pollutant	1985	1986	1987	1988	1989	1990	1991	1992
Hydrocarbons	155	305	303	303	303	308	320	331
Nitrogen Oxides	40	94	107	119	130	139	145	150
Carbon Monoxide	1,067	2,116	2,114	2,122	2,131	2,174	2,255	2,333
Total	1,262	2,515	2,524	2,544	2,564	2,621	2,720	2,814

The material in these sections closely parallels McGartland and Ostro (1985). Several of the studies relied upon are EPA contractor reports in progress or in draft; as such they have not undergone full peer review and should be considered preliminary.

VI.B.1. Effects of a 1 Percent Reduction in Ozone

In calculating the effects of a 1 percent change in ozone, we relied primarily on dose-response estimates. That is, we applied change in ambient levels. Occasionally, as a validity check of the benefit estimates, we interpolated from existing aggregate damage estimates to project the impacts of a single pollutant or of a given change in ambient levels. Regardless of the approach, the benefit estimates are uncertain and should be interpreted with caution. Unless noted otherwise, we assumed a constant benefit per ton of pollution control over the relevant range.

The effects of ozone on human health, vegetation, materials, and ecosystems were summarized in the EPA Air Quality Criteria for Ozone and Other Photochemical Oxidants (U.S. EPA, 1978). In addition, we have relied on the considerable amount of research that has become available since that document was finished. As part of EPA's periodic review of the ozone NAAQS, the Office of Research and Development currently is updating the Criteria Document. Nothing in this report is intended to prejudge or supercede the outcome of that process.

VI.B.1.a. Health Effects of Reducing Ozone

Studies of the effects of ozone on human health have investigated the relationships between changes in ozone concen-

trations and changes in lung function; decrements in physical performance; exacerbation of asthma; incidence of headaches; respiratory symptoms, such as coughing and chest discomfort; eye, nose, and throat irritation; and changes in blood parameters (U.S. EPA, 1978; Goldstein, 1982; Ferris, 1978).

Uncertainty remains about whether a threshold level exists for ozone and, if so, at what level. For example, McDonnell et al. (1983) found a nonlinear relationship between health and ozone exposure that "flattened" at ozone levels below 0.18 parts per million (ppm) -- a level above the ambient concentrations in most metropolitan areas. If such a threshold exists, the health benefits of reducing ozone from its current levels would be minimal. Population studies by Zagraniski et al. (1979) and Lebowitz et al. (1984), however, suggest effects may be occurring at ambient levels as low as 0.08 ppm. Moreover, other studies (Portney and Mullahy, 1983; Hasselblad and Svendsgaard, 1975) do not support the existence of any threshold for health effects.

Hammer et al. (1974) found associations between increased oxidants and respiratory symptoms (such as cough and chest discomfort) and other symptoms (such as eye irritation and headache) in young, healthy adults. They obtained the symptom rates from daily diaries and adjusted them by excluding days on which subjects reported fevers. Makino and Mizoguchi (1975) found a correlation between oxidant levels and eye irritation and sore throats in Japanese school children. Lippmann et al. (1983) and Lebowitz et al. (1982, 1983, 1984) found evidence of decreased athletic performance, increased prevalence of acute symptoms, and dysfunction of pulmonary systems resulting from ozone exposure.

In addition to these studies of the general population, Whittemore and Korn (1980), Linn et al. (1981), Bates and Sizto (1983), and others have shown that asthmatics and people with other chronic respiratory diseases may be particularly sensitive to ozone or other oxidants. Even low levels of exposure to photochemical oxidants have been shown to provoke respiratory symptoms in individuals with predisposing factors, such as smoking or respiratory illness (Zagraniski et al., 1979).

There is also evidence linking reduced respiratory function -- measured as Forced Expiatory Volume (FEV) and Forced Ventilating Capacity (FVC) -- to ozone exposure. For example, McDonnell et al. (1983) reported an association for normal subjects while exercising. Folinsbee et al. (1984), Horvath et al. (1979), and Adams and Schelegle (1983) found an association between decrements in FEV and ozone exposure.

Unfortunately, it is difficult to estimate from these studies the potential economic benefits of reducing health effects related to ozone exposure because they did not estimate dose-response functions. Most were designed to investigate potential thresholds, or simply to determine if any relationship existed between ozone and particular effects. In addition, studies using lung function changes as the health endpoint fail to provide a measure that can be valued in economic terms.

Recent work by Portney and Mullahy (1983, 1985) at Resources for the Future (RFF) is an exception. They considered the effect of alternative levels of ozone on various health measures, combining individual health data from the Health Interview Survey (HIS)

with data on pollution concentrations during the same period covered by the survey. In the HIS, interviewees provided information on their health status during the two weeks preceding the survey. As their health measure, Portney and Mullahy focused on the number of days of restricted activity due to a respiratory condition (RADRESP). The RADRESP measure included days when the symptoms were relatively minor, as well as those when they were serious enough to confine individuals to bed or to make them miss work.

Portney and Mullahy regressed RADRESP on a dozen or more independent variables, including socioeconomic and demographic factors, chronic health status, urban variables, and other pollutants, as well as ozone. As their ozone measure, they used the daily maximum one-hour concentration (measured in parts per million) averaged over the two-week period covered for the individual. They considered several different specifications and functional forms. In their ordinary least squares (OLS) regressions, they tried various forms of the ozone measure, including the square and the square root as well as the untransformed variable. The first part of Table VI-4 summarizes the OLS results. In all three specifications, the ozone coefficient was positive, but not significant at the 95 percent confidence level. For the specifications using the linear ozone term, the coefficient on ozone represents the average change in RADRESPs per person per two weeks for a one ppm change in ozone. Thus, for example, that coefficient predicts that reducing the average daily maximum

TABLE VI-4. Regression Results for Portney and Mullahy Study on Respiratory Symptoms Related to Ozone

Ozone Specification	Ozone Coefficient	t-statistic	F-statistic
OLS			
Linear	1.2185	1.13	2.743
Square root	0.8076	1.66	2.867
Squared	0.4667	0.07	2.636
Poisson	6.8827	1.97	N.A.
	(log-likelihood ratio = -1395.4)		

ozone concentration by 0.01 ppm for one year for a population of 1 million adults would decrease the number of RADRESPs by 316,800 (= 0.01 X 1.22 X 1,000,000 X 52/2).

In subsequent analysis, Portney and Mullahy (1985) estimated the relationship using a Poisson model, which can be written as:

$$E(RADRESP) = \exp(XB),$$

where E(RADRESP) is the expected number of RADRESPs and XB is the vector of independent variables and their coefficients. The second part of Table VI-4 summarizes the results of the Poisson model. Because the Poisson model is nonlinear, the ozone coefficient is slightly harder to use for extrapolation. The Poisson model, however, appears to fit the data better; RADRESPs have a Poisson-like distribution.

In separate models estimating RADRESPs for children, Portney and Mullahy (1983) did not find any consistently significant effects. As a lower bound, therefore, we assumed no affect on RADRESP for children in the general population. However, incomplete data for children and the reliance on parents to report child-related health effects may explain this result; a restriction in activity probably was less likely to be reported for a child. Public health scientists continue to debate whether children are as susceptible to ozone as adults. Older children, for example, may not be as susceptible because they typically have large excess lung capacity. On the other hand, damages to the lungs of a child may result in a chronic respiratory condition

in adulthood. Therefore, to place a plausible upper bound on our estimates, we applied the adult coefficients to children as well.

To estimate the change in RADRESPs due to a 1 percent change in ozone, we simulated the change using the data on individual exposures and characteristics constructed by Portney and Mullahy. Their study matched the 1979 HIS with air quality data, weather stations, and other area-specific data. Using the estimated regressions, we rolled back the exposure of each person in the data base by 1 percent. Using data from the Census Bureau, we assumed a population of 230 million in 1984, with 70 percent of the total above age 17.

The changes in total annual RADRESPs for adults predicted by the Poisson and linear models were quite similar, 2.1 and 2.4 million, respectively. Because the Poisson model provided a better fit of the data, we used it as the basis for our estimates. For our high and low estimates of adult effects, we used plus or minus one standard deviation of the ozone coefficient. For children, our low estimate was zero and our high estimate was 0.90 million cases (based on the adult coefficient). For our medium or point estimate, we used the midpoint of those extremes, 0.45 million cases. These results should be interpreted cautiously, because cross-sectional studies of this type can be extremely sensitive to model specification, functional form, omitted and confounding variables, and the ambient air monitors used.

Portney and Mullahy (1985) also used a multinominal logit model to estimate the marginal impact of ozone on the two types of RADRESPs -- the more serious ones resulting in a day of bed

rest or of lost work, and the less serious ones that resulted in a more minor restriction of normal activity. That analysis suggested that a marginal change in ozone was three times as likely to cause a day of minor restricted activity than a day of bed rest or lost work.

To provide an alternative estimate of respiratory conditions and a separate estimate of nonrespiratory irritations, we used the results of a statistical reanalysis of the Hammer et al. (1974) study discussed earlier. In an unpublished paper, Hasselblad and Svendsgaard (1975) fit simple logistic regressions to estimate the relationship between ozone concentration (measured as a daily maximum hourly concentration) and eye irritation, headache, coughing, and chest discomfort. The probability of a response at an ozone level, X, measured in parts per hundred million (pphm), was given as:

$$p(X) = C + (1 - C)/[1 + \exp(-A - BX)]$$

Table VI-5 presents the estimates of the parameters for the four outcome measures. These coefficients must be interpreted cautiously, because Hasselblad and Svendsgaard did not control for some possible confounding variables, in particular temperature and humidity. In a later, published paper, Hasselblad (1981) fit multiple logistic regression models to these same data, but that study paper does not report the regression coefficients we needed to make our estimates.

TABLE VI-5. Regression Coefficients Relating Respiratory and Non-respiratory Symptoms to Ozone

Condition	A	B	C
Respiratory Effects			
Cough	-2.98	0.0092	0.0450
Chest discomfort	-3.53	0.0023	0.0166
Non-respiratory Effects			
Eye irritation	-4.96	0.0907	0.0407
Headache	-4.88	0.0470	0.0976

We used these estimated dose-response functions with the individual information in the Portney and Mullahy data set to simulate the effects of a 1 percent reduction in ozone. For coughs and chest discomfort, the Hasselblad and Svendsgaard coefficients yielded a total of 1.54 million adult cases per year for a 1 percent reduction in ozone, compared to the estimate of 2.1 million based on the Portney and Mullahy Poisson model. These estimates are remarkably consistent, as the RADRESP measure used by Portney and Mullahy included other symptoms besides cough and chest discomfort. In addition, the Hammer et al. sample used by Hasselblad and Svendsgaard consisted of student nurses, who were young and generally healthy, while the Portney and Mullahy sample was more representative of the general population. For those reasons, our estimates for respiratory conditions rely solely on Portney and Mullahy's results.

The results of Hasselblad and Svendsgaard also can be used to estimate the number of nonrespiratory conditions, such as eye irritation and headache, possibly related to exposure to ozone and other photochemical oxidants. There is evidence suggesting that these symptoms are not related to ozone per se, but rather to other oxidants, such as peroxyacetyl nitrate (PAN), whose production may be proportional to that of ozone.

To account for this possibility, we used the estimates based on Hasselblad and Svendsgaard as point estimates for nonrespiratory irritations (headache and eye irritation). Unfortunately, these researchers did not report the standard errors, so a confidence interval could not be determined.

These ozone health benefits reflect the likely acute effects generated by intense, short-term exposure to ozone. Long-term exposure to ozone also may affect the health of some people, but the epidemiological evidence on chronic ozone effects is sparse. One of the available studies, Detels et al. (1979), compared the effects of prolonged exposure to different levels of photochemical oxidants on the pulmonary functions of both healthy individuals and individuals with chronic obstructive pulmonary disease. Persons exposed to an annual mean of 0.11 ppm of oxidant, compared to a control group exposed to 0.03 ppm of oxidant, showed statistically significantly increased chest illness, impairments of respiratory functions, and lower pulmonary function.*

While the epidemiological evidence of the effects of long-term exposure to ozone is sparse, several animal experiments have demonstrated effects on lung elasticity, blood chemistry, the central nervous system, the body's ability to defend against infection, and the rate at which drugs are metabolized (U.S. EPA, 1983f). Unfortunately, it is not possible to extrapolate those results to humans. Therefore, we could not quantify the chronic health effects attributable to ozone, but we believe that some of these effects may be present at current ambient levels.

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* At workshops related to the development of the Criteria Document for ozone, some shortcomings in this analysis were noted. For example, the study group was also exposed to higher levels of $N0_2$ and $S0_4$, and there were some, questions about the adequacy of the measurement of ozone exposure, about the subject selection, and about the test measures. Although it is both reasonable and likely that long-term exposures affect health, the failure to correct for the effects of other pollutants raises uncertainties about the specific findings.

Table VI-6 summarizes our estimates of the acute health effects of a 1 percent change in ozone. The estimates of respiratory effects are based on the Portney and Mullahy results, while the nonrespiratory effects are derived from Hasselblad and Svendsgaard's analysis. In the latter case, high and low estimates are not reported because Hasselblad and Svendsgaard did not calculate standard errors for their estimates. As discussed above, these estimates omit any quantification of chronic health effects.

VI.B.1.b. Ozone Agricultural Effects

Ozone, alone or in combination with sulfur dioxide and nitrogen dioxide, is responsible for most of the U.S. crop damage attributed to air pollution (Heck et al., 1983). Ozone affects the foliage of plants by biochemical and cellular alteration, thus inhibiting photosynthesis and reducing plant growth, yield, and quality.

Early studies of ozone-related damages used generalized relationships between ozone concentrations, yield, and economic loss. Insufficient information precluded the construction of an economic model with credible dose-yield data. Thus, for example, Freeman (1982), in a general survey of the literature, could only conclude that the total agricultural damages from ozone ranged from $1.0 to $4.0 billion in 1978 dollars.

Recent work by the National Crop Loss Assessment Network (NCLAN) suggests that prior studies have underestimated ozone-related damages. NCLAN'S estimated dose-yield functions for soybeans, wheat, corn, peanuts, cotton, barley, and sorghum have

TABLE VI-6. Estimated Health Effects of a 1 Percent Reduction in Ozone (millions of days per year)

Condition	Low Estimate	High Estimate	Medium Estimate
Respiratory Effects			
Bed Rest/Work Loss			
Adults	0.36	1.00	0.53
Children	0.00	0.23	0.11
Subtotal	0.36	1.23	0.64
Minor Restrictions			
Adults	1.07	2.93	1.57
Children	0.00	0.67	0.34
Subtotal	1.07	3.60	1.91
Nonrespiratory Effects			
Headaches	N.A.	N.A.	1.25
Eye Irritation	N.A.	N.A.	3.12
Subtotal			4.37

provided more accurate information on ozone's effects on crops. Kopp (1983, 1984) and Adams et al. (1984) incorporated these functions into models of agricultural production and demand to estimate the benefits of ozone reduction strategies.

Kopp constructed a detailed macroeconomic model of farm behavior for over 200 producing regions in the United States. The NCLAN dose-yield functions are directly incorporated in Kopp's model of the supply side of each crop for each region. Because estimates of the demand and supply elasticities for these crops are used in the analysis, it gives a good indication of the actual change in economic welfare. Kopp's simulations suggest that a 1 percent reduction in ozone would produce total benefits of roughly $110 million (1983 dollars) per year for the seven major crops covered by NCLAN, as shown in Table VI-7. These seven crops accounted for only about 80 percent of the total value of U.S. crop production (USDA, 1982). If we increase Kopp's estimate by assuming that ozone damages to all other crops occur in the same proportion as their relative value, we conclude that the benefits of a 1 percent change in ozone are roughly $137 million annually.

Adams et al. (1984) used a different approach. By incorporating the NCLAN dose-yield functions into an existing quadratic programming model, they calculated ozone benefits for six of the seven crops covered by Kopp (they did not include peanuts). They estimated that a 10 percent reduction in rural ozone would result in annual benefits of roughly $674 million (1983 dollars). Assuming linearity and increasing the estimate to account for omitted crops, we estimated $90 million in benefits

TABLE VI-7. Annual Agricultural Benefits of a 1 Percent Ozone Reduction (millions of 1983 dollars)

Crop	Estimate
Soybeans	50.8
Corn	7.6
Wheat	21.1
Cotton	20.4
Peanuts	5.0
Sorghum	4.6
Barley	0.2
Total assessed	109.8

for a 1 percent change in ozone levels. Unfortunately, Adams et al. did not calculate the benefits on a crop-by-crop basis, so it is not possible to make a detailed comparison with Kopp's estimates.

For our best estimates, we used Kopp (1983, 1984) and Adams et al. (1984), because they used the superior NCLAN data and based their estimates on economic measures of welfare loss. The two estimates are fairly close. We concluded that a 1 percent reduction in rural ozone would produce $90 to $140 million in annual agricultural benefits per year, with a point estimate of $114 million. We qualify these numbers by noting that the estimates do not reflect unreported small "truck farm" sales or any averting activities that farmers may undertake, such as planting pollutant resistant crops; we believe that these categories are likely to be small. On the other hand, the welfare measures do not reflect either the effects of crop subsidy programs at the state and federal levels, or the effects of drought, both of which are likely to reduce the marginal welfare impacts of ozone. The effects of subsidies are likely to be particularly significant.

VI.B.1.c. Ozone Effects on Nonagricultural Vegetation

Forests and ornamental plants also may suffer substantial damages from exposure to ozone. The preliminary draft of the Ozone Criteria Document discusses the issue:

> The influence of 03 on patterns of succession and competition and on individual tree health is causing significant forest change in portions of the temperate zone . . . . Long-term continual stress tends to decrease the total foliar cover of vegetation, decrease species richness and increase the concentrations of species

> dominance by favoring oxidant-tolerant species. These changes are occurring in forest regions with ozone levels (l-hour maximum) ranging from 0.05 ppm (111 ug/m^3) to 0.40 ppm (785 ug/m^3) (U.S. EPA, 1983).

Additional evidence of significant damages from ozone associated with nonagricultural vegetation is provided by McLaughlin et al. (1984).

Unfortunately, no careful quantitative studies of the type done by NCLAN have been performed for nonagricultural vegetation. Heintz et al. (1976) have estimated losses to ornamental plants of $100 million per year in 1973 dollars. Inflating to 1983 dollars using the Farm Products Index, and assuming linearity, yields estimated annual benefits of $1.4 million for the reduction in damages to ornamental from a 1 percent reduction in ozone.

Damage to forests is potentially a much larger concern in terms of reduced production and decreases in recreation and aesthetic values. In a very small contingent valuation study, Crocker and Vaux (1983) found that the shift of an acre of the current mix of severely, moderately, and unharmed timberland in the San Bernardino National Forest into the unharmed category would generate additional annual recreational benefits of between $21 and $68 per acre per year. These findings are difficult to generalize for the rest of the nation because ambient ozone levels are unusually high in the San Bernardino area (and the authors provide no dose-response function for extrapolating to areas with lower concentrations), and because other site attributes and visitors' socioeconomic characteristics have very large and significant effects on users' willingness to pay to reduce damages to forests.

We also lack data on the impact of ozone on commercial forests. Most commercial forests are located in areas with relatively low ozone concentrations, so damages may be small. Weyerhauser Corporation staff recently reported “negligible or nonexistent” damages from ozone (personal communication from Jack Larson of Weyerhauser). In light of the NCLAN findings with respect to crops, however, the possibility of significant ozone damage to forests cannot be dismissed.

VI.B.1.d. Ozone Materials Damage

Ozone directly damages many types of organic materials, including elastomers, paint, textile dyes, and fibers. It can increase the rigidity of rubber and synthetic polymers, causing brittleness, cracking, and reduced elasticity. Ozone exposure also can generate other effects, such as avoidance costs (purchase of specially resistant materials) and aesthetic losses. Only the direct costs are incorporated in this analysis, however.

In his survey of the literature, Freeman (1982) suggested that annual materials damages from oxidants and NO_x amount to approximately $1.1 billion (1978 dollars). We updated that estimate using the U.S. Government price indices for rubber and textile products and the index of personal consumption expenditures for durable goods; this yielded an estimate of $2.25 billion for 1983. Assuming linearity, a 1 percent ozone reduction generates a benefit of roughly $22.5 million annually.

We obtained an alternative estimate of the benefits of reduced material damage by using dose-response information incorporated in the 1978 Criteria Document for ozone. The text contains per capita economic damage functions for elastomers, textiles, industrial maintenance, and vinyl paint. We used a population-weighted mean value of ozone of 0.03 ppm (60 ug/m^3) from the draft of the new Criteria Document (U.S. EPA, 1984), a population estimate of 230 million, and the indices cited above. This method yielded annual benefits of $15 million (in 1983 dollars) for a 1 percent reduction in ozone. Averaging these two estimates (with slightly more weight given to the lower estimate which does not include any NO_x-related damages) yields a point estimate of $18 million annually, with a range of $15 to $22.5 million.

VI.B.1.e. Summary of Benefits of a 1 Percent Change in Ozone

Table VI-8 summarizes our estimates of the effects of a 1 percent reduction in ozone. The estimated health effects include roughly 2.5 million days of respiratory symptoms resulting in some restriction in activity, and 4.4 million days of non-respiratory irritations (headaches and eye irritation). On the nonhealth side of the ledger, increases in agricultural crop production dominate, with a total of $114 million. Materials damage is next, with $18 million, while ornamental plants contribute $1.4 million. In all cases, the estimates are subject to considerable uncertainty; the “high” and “low” estimates provide only a partial indication of that uncertainty, as they reflect only the ranges in available

TABLE VI-8. Summary of Estimated Effects of a 1 Percent Reduction in Ozone

Effect	Low Estimate	High Estimate	Medium Estimate
Health (millions of cases)			
Respiratory Symptoms			
Bed rest/Work loss	0.36	1.23	0.64
Minor restrictions	1.07	3.60	1.91
Nonrespiratory Symptoms	N.A.	N.A.	4.37
Other (millions of dollars)			
Agricultural crops	90.0	140.0	114.0
Ornamental plants	N.A.	N.A.	1.4
Materials damage	15.0	22.5	18.0
Unquantified Benefit Categories			
Chronic health effects			
Forest damage			

estimates or statistical uncertainty in the parameter estimates from particular studies. A more complete accounting for uncertainty -- which would include different functional forms for the dose-response functions, omitted categories, etc. -- would yield substantially broader ranges. The omitted categories, in particular chronic health effects and damage to forests, however, suggest that these estimates are too low, perhaps by a substantial margin.

VI.B.2. Linking NO_x and HC Reductions to Ozone Effects

T-he previous section estimated the effects of reducing ozone. Linking those effects to emissions of HC and NO_x requires estimating the relationships between those pollutants and ozone concentrations. Section VI.B.2.a describes the general process of ozone formation, while Section VI.B.2.b presents rough quantitative estimates of the impact of HC and NO_x emissions on ozone concentrations in both urban and rural areas.

VI.B.2.a. The Process of Ozone Formation

Ozone changes are influenced by the amount of solar radiation and the ratio of, and changes in, the concentrations of NO_x and HC. The general process by which ozone (O_3) is formed is illustrated by the NO_2-NO-O_3 cycle. These reaction equations can be written as:

(VI-1) NO_2 + sunlight --> N O + O

(VI-2) O + O_2 ----------> O_3

(VI-3) O_3 + NO ----------> NO_2 + O_2

Ozone is not emitted in any measurable quantity. The first two reactions represent its only significant source. However, these reactions are fully reversed by the third one. Because NO comprises roughly 90 percent of man-made NO_x emissions, reaction (VI-3) suggests that additional NO_x emissions would reduce ozone concentrations by scavenging the ozone for one of the oxygen atoms, thereby producing O_2 and NO_2. Therefore, increases in NO_x cannot increase ozone, unless some other species oxidizes NO to NO_2 without scavenging ozone for the necessary oxygen atom. This additional reaction is:

(VI-4) Organic radicals (from HC) + NO --> NO_2 + miscellaneous products

With reactions (VI-1), (VI-2), and (VI-4), NO_2 is recycled, allowing for the generation of excess O_3. That is, HC permits ozone formation while bypassing the ozone destructive step of NO + O_3 --> NO_2 + O_2. In short, with a plentiful supply of HC, NO_x will not have to scavenge ozone to form NO_2. Instead, NO_x combines with the HC to form additional NO_2, which in turn makes more ozone. Therefore, in areas with “excess” HC, NO_x control will reduce ozone, but controlling HC will be relatively ineffective. However, when there is not enough HC to react with available NO_x, NO_x will scavenge ozone to form NO_2. In this situation, NO_x control will result in less ozone scavenging (and more ozone). HC control, on the other hand, will be effective in reducing ozone because the greater the reduction in HC, the more NO_x is forced to scavenge ozone.

Atmospheric scientists have shown that the HC/NO_x ratio is relatively small in metropolitan areas. Thus, in such areas, a reduction in HC will reduce ozone, but depending on the specific HC/NO_x ratio, controlling NO_x may increase ozone. In rural areas, HC/NO_x ratios are larger. Therefore, if the ozone in such areas results primarily from local emissions, controlling local NO_x emissions may be a more effective way of reducing ozone than local HC controls.

VI.B.2.b. Quantitative Estimates of Impacts of HC and NO_x on Ozone

Several models have been developed to simulate the effect of changes in HC and NO_x on ozone (U.S. EPA, 1983f, 1984b; Systems Applications, Inc. [SAI], 1984). EPA's Office of Air Quality Planning and Standards (OAQPS) also has developed three sets of regional Empirical Kinetic Modeling Approach (EKMA) isopleths for use in the ozone NAAQS review. Finally, the data base obtained from state implementation plans (SIPS) for ozone summarizes all of the modeling results from the SIPS. A review of these models and their general results is provided in McGartland and Ostro (1985). From these models, we generated estimates of the "average" impact of changes in HC and NO_x on urban and rural peak ozone concentrations (relevant for health effects and materials damage) and on rural average ozone concentrations (more relevant for agricultural effects), measured in terms of elasticities (i.e., the percent change in the ozone variable with respect to a 1 percent change in emissions of the pollutant). The results are summarized in Table VI-9; the discussion of their derivation follows.

TABLE VI-9. Estimated Ozone Reductions from 1 Percent Reduction in Rural and Metropolitan HC and NO_x (percent reduction)

	Reduction in Peak Urban Ozone	Reduction in Peak Rural Ozone	Reduction in Average Rural Ozone
HC			
1 Percent Reduction in Urban HC	0.60	0.05	0.13
1 Percent Reduction in Rural HC	0	0.36[d]	0.36[d, e]
NO_x			
1 Percent Reduction in Urban NO_x	-0.3 to -0.05 (-0.l)a	-0.1 to +0.1 (0.04)b	-0.09
1 Percent Reduction in Rural NO_x	0	0.05 to 0.2[d] (0.15)[c]	0.05 to 0.2[d, e] (0.15)[c]

[a] Estimates varied by a fairly wide range. We used the point estimate of -0.1.

[b] Sparse evidence does not permit the estimation of a single point estimate with a reasonable degree of certainty. In many areas (e.g., the Northeast Corridor), wind trajectories would probably transport metropolitan ozone to other urban centers or over the ocean. In these cases, rural ozone would not change. In other areas, however, the increased ozone from urban NO_x reductions would travel to rural areas, but lower rural NO_2 levels would reduce ozone. Therefore, the overall effect could be positive or neqative. As a conservative estimate, we assumed that a 1 percent uniform reduction in metropolitan NO_x reduces rural ozone by 0.04 percent.

[c] Estimates varied. We used a conservative high point estimate of 0.15.

[d] Results from ongoing analysis at SAI will soon narrow the rate of estimates.

[e] The lack of evidence made it necessary to assume that average rural ozone changes by the same percent as peak rural ozone.

The ozone-SIP data base presents estimates of how urban HC affects peak urban ozone for every city not attaining the ozone standard. Analysis of these data by OAQPS (U.S. EPA, 1984) suggests that a 1 percent reduction in hydrocarbon emissions will reduce metropolitan ozone on average by 0.6 percent. This estimate is based on many EKMA urban simulations throughout the U.S.; as such, it represents the best available estimate available of the percent reduction in urban ozone from a 1 percent reduction in HC. However, there is considerable variance from area to area.

Estimates of how urban HC affects rural average and peak ozone levels are presented by SAI (1984). SAI simulated regional air quality under four basic scenarios -- mornings and afternoons, winter and summer. The two summer scenarios (relevant for estimating agricultural benefits) indicate that average ozone changes by 0.13 percent for every 1 percent change in urban HC during the agricultural growing season. Peak rural ozone changed by much less: roughly 0.05 percent for every 1 percent change in urban HC. This is mainly because the HC and ozone plumes are widely dispersed by the time they reach rural areas.

Estimates of how rural HC affects rural ozone generally were not available. Therefore, we used the three sets of regional EKMA isopleths developed by OAQPS. By estimating data points at HC/NO_x ratios typical of rural areas, we estimated that, on average, a 1 percent reduction in rural HC will reduce peak rural ozone by 0.36 percent. EKMA models are not, however, suitable for estimating changes in average ozone. As our best estimate, we assumed that average ozone changes by the same percentage as the peak.

For rural areas this may be a reasonable approximation, as rural ozone levels do not fluctuate as much as urban ozone levels and the plumes would still be highly concentrated in the same area.

To estimate the effects of NO_x reductions on urban and rural ozone, we used SAI (1984) and the EKMA isopleths developed by OAQPS. By taking an average of data points from the isopleths, we calculated that, on average, a 1 percent reduction in metropolitan NO_x will increase ozone by 0.1 percent.

SAI estimated how rural ozone is affected by changes in NO_x emissions. A 1 percent reduction in urban NO_x increases average rural ozone by 0.09 percent. Peak rural ozone would probably not change as much because the urban plume is widely dispersed when it reaches the rural areas. The SAI analysis showed average rural ozone increasing by a small amount in some situations, but, on average, there probably is a small decrease in rural ozone given a reduction in urban NO_x emissions. As a conservative estimate, we assumed that a 1 percent reduction in urban NO_x reduces peak rural ozone by 0.04 percent.

We again relied on the EKMA isopleths generated by OAQPS to estimate how rural NO_x emissions affect peak rural ozone. On average, the isopleths indicate that a 1 percent reduction in rural NO_x will reduce peak rural ozone by 0.1 to 0.2 percent. We used an estimate of a 0.15 percent decrease in peak ozone for every 1 percent decrease in rural NO_x. We also used that estimate to quantify how average ozone reacts to changes in rural NO_x. (The rural plumes would still be concentrated in a small area.)

Four caveats should be considered in connection with the results summarized in Table VI-9:

1. Many of the results are based on averages of different simulation results.

2. Our estimates represent how ozone is affected on average across the U.S. They should not be used for estimating ozone changes for a specific area.

3. Most of the literature reports estimates of changes in peak ozone. Peak ozone is the appropriate measure for most of our benefit categories, but for one important benefit category (agriculture), changes in daily maximum 7-hour average ozone levels are needed. For rural emission changes, we assumed that the measures of average ozone move in direct proportion to changes in peak ozone.

4. Some of our estimates of how rural emissions affect ozone were derived from models better suited to represent urban areas (e.g., EKMA).

Because of these qualifications, the estimates in Table VI-9 should be regarded as highly uncertain. The estimates for NO_x are particularly uncertain, because much less modeling has been done of the NO_x-ozone relationship than for HC.

We next calculated the population-weighted effects on ozone of 1 percent changes in national emissions of HC and NO_x. (We used population as the weighting factor because the nonagricultural benefits are proportional to it.) We calculated weighted average elasticities using the estimates in Table VI-9 and the fact that about two-thirds of the non-agricultural benefits are attributed to metropolitan area and one-third to non-metropolitan areas (based on relative populations). For hydrocarbons, the estimates in Table VI-9 suggest that a 1 percent reduction in emissions would reduce metropolitan ozone by 0.60 percent (= 0.60 + 0); similarly, non-metropolitan ozone would fall by

0.41 percent (= 0.05 + 0.36). Weighting these two changes by the split in non-agricultural benefits between metropolitan and non-metropolitan areas yields an overall benefit-weighted elasticity of :

(VI-5) $$0.67\ (0.60) + 0.33\ (0.41) = 0.537$$

Multiplying this weighted elasticity times the national effects of a 1 percent change in ozone yields the same result as computing separately, and then summing, the metropolitan and non-metropolitan effects of 1 percent changes in hydrocarbons. Similarly, for NO_X, the estimated benefit-weighted average elasticity for non-agricultural benefits was:

(VI-6) $$0.67\ (-0.1 + 0) + 0.33\ (0.04 + 0.15) = -0.004$$

Note that reductions in NO_x are predicted, on average, to cause a slight increase in peak ozone levels. Using the full range of elasticity estimates in Table VI-9, however, this weighted elasticity ranges from -0.22 to +0.066.

For agricultural crops, we assumed that the relevant measure would be the change in average rural ozone levels; we gave no weight to changes in urban ozone. For HC, the estimated elasticity of average rural ozone with respect to national emissions was:

(VI-7) $$0.13 + 0.36 = 0.49$$

Thus, we estimate that 1 percent reductions in metropolitan and non-metropolitan HC emissions reduce average rural ozone by 0.49 percent. For the NO_x point estimates, the elasticity was:

(VI-8) $-0.09 + 0.15 = 0.06$

Note that this estimate suggests that nationwide NO_x reductions will slightly decrease rural ozone levels, despite the fact that urban NO_x control, on average, is predicted to increase ozone; the increase in urban ozone that is transported to rural areas does not offset the reduction in ozone produced in rural areas. Again, however, the range of uncertainty is large. Using the ranges in Table V-9 generates elasticity estimates for the impact of national NO_x control on average rural ozone ranging from -0.04 to +0.11.

VI.B.2.c. Ozone-Related Effects Per Ton of HC and NO_x Controlled

We used the estimated weighted average elasticities, together with the estimates of reductions in HC and NO_x emissions to predict changes in ozone. We needed to predict changes in two ozone measures: average rural ozone (for the agricultural benefits) and changes in peak ozone levels, averaged over the nation (for the other ozone benefit categories).

To apply the elasticities, we needed to convert the emission reduction to percentages; because our two largest ozone-related benefit categories (agriculture and restricted activity days) were based on 1978 and 1979 air-quality data, we computed the changes as percentages of 1979 emissions, which were about 21.9 million tons for HC and 21.3 million tons for NO_x. Thus, the predicted emissions due to misfueling in 1986 (from Table VI-3) translate to 1.4 percent (= 305,000/21.9 million tons x 100%) and 0.44 percent (= 94,000/21.3 million x 100%) for NO_x. Using these

percentages together with the estimated elasticities for peak ozone (equations VI-5 and VI-6) yields the following estimated percentage change in peak ozone due to emission reductions in 1986:

(VI-9) (0.537)(1.4%) + (-0.004)(0.44%) = 0.75 percent.

Similarly, for average rural ozone, the estimated decline in ozone due to emission reductions in 1986 is:

(VI-10) (0.49)(1.4%) + (0.60)(0.44%) = 0.71 percent.

Table VI-10 presents the year-by-year estimates of changes in ozone due to eliminating misfueling.

Finally, the predicted changes in the two ozone measures were combined with the estimates in Table VI-8 to predict the year-by-year ozone-related effects of the rule. The changes in average rural ozone were used for agricultural crops, while the changes in peak ozone were used for the other benefit categories. Table VI-11 presents the results for 1986. It is important to remember that these estimated effects are subject to all of the uncertainties and omissions discussed earlier in connection with our estimates of the benefits of a 1 percent reduction in ozone. In addition, however, they also reflect the uncertainties in the estimates relating HC and NO_x emissions to ozone; these uncertainties are particularly great in the case of NO_x.

TABLE VI-10. Estimated Ozone Reductions due to Eliminating Misfueling (percent of 1979 level)

Ozone Measure	1985	1986	1987	1988	1989	1990	1991	1992
National peak	0.38	0.75	0.74	0.74	0.74	0.75	0.78	0.81
Rural average	0.36	0.71	0.71	0.71	0.71	0.73	0.76	0.78

TABLE VI-11. Quantified Ozone-Related Effects Due to Elimination of Misfueling in 1986

Effect	Low Estimate	High Estimate	Medium Estimate
Health (millions of cases)			
Respiratory			
Bed rest/Lost work	0.27	0.92	0.48
Minor restrictions	0.80	2.69	1.43
Nonrespiratory	N.A.	N.A.	3.26
Other (millions of dollars)			
Agricultural crops	63.80	99.25	80.81
Ornamental plants	N.A.	N.A.	1.04
Materials damage	11.19	16.79	13.43
Unquantified Benefit Categories			
Chronic health effects			
Forest damage			

VI. C. Health and Welfare Effects Not Related to Ozone

Thus far, our analysis has focused only on HC and NO_x, and only on their ozone-related effects. In this section, we discuss other health and welfare effects associated with those pollutants and with CO and EDB. In most cases, the discussion is only qualitative because we have been unable to make quantitative estimates.

VI.C.1. Hydrocarbons

In addition to the benefits associated with reduced ozone, reductions in hydrocarbon emissions may affect sulfate concentrations and will reduce exposure to benzene. Although quantitative estimates are not possible for the former, we can make some rough estimates of the health benefits of reduced benzene exposure.

VI.C.1.a. Impact on Sulfates

Hydrocarbons are a factor in the formation of sulfates. In particular, SO_2 oxidizes faster when the amount of hydroxide radicals in the atmosphere increases (which is, in turn, a function of the amount of HC in the atmosphere). However, the ability to quantify these complex relationships has just been developed, and experts at SAI and EPA's Office of Research and Development believe that the total change in sulfates is highly dependent upon many factors (e.g., cloud cover, current HC and NO_x concentrations, and oxidant and sulfur dioxide levels) for which we have only limited data.

A recent modeling analysis by SAI (1984) indicated that a 10 percent reduction in HC could reduce sulfates in urban areas during certain times of the year by about 2 percent. However,

because of the uncertainty surrounding this estimate, and the uncertainty in interpolating this to much smaller changes in HC, we did not try to quantify the reduction in sulfates in this analysis.

VI.C.1.b. Impact on Benzene and Other Aromatics

Reducing lead in gasoline will affect benzene emissions in two ways. First, because benzene is a hydrocarbon, reducing misfueling will reduce benzene tailpipe emissions along with other hydrocarbons. Second, to increase octane with less lead, refineries will increase the severity of their catalytic reforming, which in turn increases the amount of benzene in reformate, one of the components blended into gasoline. Benzene emissions are of particular concern because benzene is believed to be a leukemogen, and has been listed by EPA as a hazardous air pollutant under Section 112 of the Clean Air Act.

Benzene comprises about 4 percent of tailpipe hydrocarbon emissions (U.S. EPA, 1983c). Assuming that catalysts are as effective in eliminating benzene as other hydrocarbons, that would imply that benzene would be roughly 4 percent of the reduced HC emissions from eliminating misfueling. Thus, for example, in 1986, we estimate that eliminating misfueling would eliminate 0.04 (305,000) = 12,200 tons of benzene.

Reducing lead is likely to increase the overall benzene content of gasoline because additional reforming creates more benzene in the reformate. Benzene (along with all other aromatics) has a a poor octane response to lead, however, so as the

aromatic content of reformate increases it will be more economical for refiners to increasingly direct such stocks to unleaded gasoline, and use less reformate and more of other higher lead-response components (e.g., alkylate) in leaded gasoline. Based on the DOE refining model, which finds the blending pattern that gives the least cost, we estimated that the benzene content of gasoline will rise from about 1.5 percent to 1.6 percent in unleaded, but will fall from 0.62 percent to 0.38 percent in leaded gasoline (at 0.10 gplg).

Raising the overall benzene content of the gasoline pool will increase evaporative emissions of benzene from gasoline marketing facilities (service stations, etc.), but based on EPA analyses of the impacts of regulating gasoline marketing (U.S. EPA, 1984a), we estimated the increase to be less than 400 tons per year. Moreover, because of the shift of benzene from leaded to unleaded gasoline, evaporative emissions from vehicles should fall by about the same amount, because unleaded gasoline is used in vehicles with evaporative control devices. In addition, switching benzene from leaded to unleaded should reduce tailpipe emissions of benzene because unleaded gasoline is used in vehicles with catalytic converters, which oxidize about 90 percent of the unburned hydrocarbons in the exhaust. In 1986, this should reduce benzene tailpipe emissions by almost 7,000 tons.

Table VI-12 summarizes our estimates of reductions in emissions of benzene for 1986, assuming that misfueling is eliminated. They total about 18,000 tons, of which about two-thirds are due to the elimination of misfueling and one-third are due

TABLE VI-12. Reductions in Benzene Emissions in 1986, Assuming No Misfueling (tons)

Sources of Emissions	Amount
Reduced Misfueling	
Tailpipe emissions	12,200
Changes in fraction of benzene in leaded and unleaded gasoline	
Marketing evaporative	-366
Vehicle evaporative	377
Tailpipe	6,230
TOTAL	18,441

to reduced tailpipe emissions resulting from the change in the relative benzene contents of leaded and unleaded gasoline. Evaporative emissions account, on net, for less than 0.1 percent of the change.

EPA's Carcinogen Assessment Group (CAG), using a "plausible upper bound" unit-risk estimate, estimated that in 1976, automobile emissions of benzene resulted in up to 50.9 deaths from leukemia (U.S. EPA, 1979a). Since then, however, the CAG has revised its unit-risk estimate for benzene downwards by slightly more than 8 percent, yielding an estimate of 47.3 leukemia cases for 1976. The CAG estimate was based on an emissions estimate of 202,000 tons of benzene. Scaling down the CAG risk estimate of 47.3 cases by emissions yields 4.4 (= 47.3 x 18,441/202,000) cases of leukemia eliminated in 1986.

We also have performed similar calculations for emissions of total aromatics, with similar results. Reducing lead in gasoline should reduce overall emissions of aromatics, both because of reduced misfueling and because of shifts in the relative fractions of aromatics in leaded and unleaded gasolines. On net, emissions of aromatics should fall by several tens of thousands of tons per year (Schwartz, 1984, in Docket EN-84-05) as a result of reducing lead in gasoline to 0.10 gplg. We have not included any benefits for these reductions, however, except as they relate to benzene specifically.

VI.C.2. Nitrogen Oxides

Besides the ozone-related effects discussed above, nitrogen oxides also may affect health and welfare directly. NO_x emissions

are believed to affect health and materials, to contribute to reductions in visibility, and are associated with acid deposition. In addition, damage to vegetation has been demonstrated experimentally. Unfortunately, specific dose-response information related to NO_x is sparse. As a consequence, there is great uncertainty in the benefit estimates and many effects cannot be quantified. However, a few of the benefit calculations are presented to provide a partial measure of the effects of NO_x emissions on economic welfare. For health effects, only qualitative evidence is provided. While there may be acid rain benefits as well, we have not included them because of uncertainties about the role of NO_x in acid deposition.

As discussed earlier, NO_x represents the composite formula for NO (nitric oxide) and NO_2 (nitrogen dioxide). NO is the dominant oxide released initially, but atmospheric interactions result in the conversion of NO to NO_2. Based on the results of smog chamber tests and modeling experiments, Trijonis (1978, 1979) concluded that maximum and average NO_2 concentrations tend to be proportional to initial NO_x contributions.

VI.C.2.a. Visibility Benefits from Reduced NO_x

NO_2 is a reddish-brown gas that reduces visibility by absorbing and discoloring light. In contrast, particulate matter scatters light to reduce visual range. While particulate matter accounts for almost all of the damage to visibility in the East, for some western regions NO_2 may play a significant role in determining visual range.

To bound the value of improved visibility per ton of NO_x reduced, we used the results of Brookshire et al. (1976). Their contingent valuation study showed that recreators were willing to pay $1.2 million per year (annualized) to avoid visibility reductions that would result from a planned Kaiparowits power plant in southern Utah. Similar studies, by Randall et al. (1974) and by Blank et al. (1977) produced comparable results for the value of visibility benefits in the Four Corners region of the Southwest. However, these estimates apply only to this particular region, known for its scenic vistas.

In the Kaiparowits study, climatic conditions, emission controls, and other factors allowed the investigators to assume that the major visibility-related impact would be the coloration of the sky by NO_2. Given the projected power plant emissions of 80,000 tons of NO_x per year, the estimate of $1.2 million of potential damage translates to about $2.1 million in 1983 dollars (based on Consumer Price index). For 1986, we project a 94,000 ton reduction in NO_x emissions if misfueling is eliminated. Using the Brookshire et al. estimate and assuming linearity yields a benefit estimate of about $2.4 million. We stress that this figure is probably an upper bound, even for sensitive regions. Although the Kaiparowits area is not densely populated, a large number of recreators use the site and they typically place a high value on protecting clean areas. Further, it is doubtful that NO_2 has a noticeable impact on eastern visibility, where

range is limited by buildings and largely influenced by particulate matter (based on a conversation with Shep Burton of Systems Applications, Inc.).

VI.C.2.b. Health Benefits of Reducing NO_x

Evidence of health effects related to NO_x is provided in the Air Quality Criteria for Oxides of Nitrogen (EPA, 1982b), the OAQPS staff paper on the NAAQS for nitrogen oxides, the Clean Air Science Advisory Committee's (CASAC'S) cover letter (EPA, 1982c), the NO_x Regulatory Impact Analysis (EPA, 1982e), and the published literature. These studies can be divided into four classes:

(1) Animal toxicology studies. Animals are exposed to controlled levels of NO_2. Researchers have the option of using invasive techniques to investigate the effects of NO_2.

(2) Controlled human-exposure studies. These are clinical studies in which humans are exposed to NO_2 in enclosed chambers. They are limited typically to examining the effects of a single, short-term (acute) exposure.

(3) Outdoor epidemiological studies. Health indicators of cross-sectional groups are statistically related to real-world outdoor ambient concentrations. This class of studies is generally most appropriate to assess the benefits of controlling outdoor air pollution, since health effects are related directly to the control variable of interest.

(4) Indoor epidemiological studies. Health measures of cross-sectional qroups are statistically related to indicators of indoor pollutant concentrations. For example, the "gas stove" studies investigate the effect of indoor air pollution on individuals living in homes with gas stoves (a significant source of NO_2), compared with people living in homes with electric stoves.

Most animal toxicology studies involving NO_2 emphasize peak exposure, with concentrations (2 ppm to 20 ppm) roughly 40 to 400 times the annual average ambient NO_2 standard of 0.05 ppm. An

additional limitation of these studies in assessing benefits is that there is no generally accepted method for extrapolating exposure response results from animal studies to humans. Thus, these studies may lend support to the human studies, but are of little help in quantifying health effects.

The majority of the controlled human experiments have examined the effects of NO_2 on healthy adults by exposing them to single, short-term concentrations in enclosed chambers, A very limited number of clinical studies also have examined some potentially sensitive populations (asthmatics and chronic bronchitis), although others (children) have yet to be tested.

In general, these studies, as summarized in the OAQPS staff paper, indicate that healthy adults are not affected by concentrations of 1 ppm or less. When considering the effect on more sensitive individuals, small reductions in pulmonary function may appear in the range of 0.5 to 1.5 ppm. Also, some studies have shown increased sensitivity to agents inducing bronchoconstriction at 0.1 to 0.2 ppm levels of NO_2. Unfortunately, dose-response functions relating NO_x to economically-significant health endpoints do not exist.

Successful outdoor epidemiological studies are scarce because investigators must separate many confounding effects and health hazards. Moreover, some of the studies used in setting the existing annual standard of 0.053 ppm (100 ug/m^3) have been criticized because of NO_2 measurement problems. Therefore, this class of studies does not provide any adequate basis for estimating a dose-response function.

Indoor epidemiological studies have focused on comparisons of homes with gas stoves (which emit NO_x) to those with electric stoves. Some of these studies reported an association between nitrogen dioxide and either lung function changes or respiratory effects (EPA, 1982), but the most recent studies, including Ware et al. (1984), no longer find a statistically significant relationship between children living in homes with gas stoves and the incidence of respiratory illness. Ware et al., however, continue to find small statistically-significant decreases in in pulmonary function associated with NO_x, although the estimates may be confounded by the influence of parental education.

Because of the mixed results concerning respiratory symptoms and the lack of a dose-response estimate for pulmonary function changes, no health benefits are quantified for the anticipated reductions in NO_x emissions.

VI.C.2.c. NO_x Effects on Vegetation

Data concerning the effects of NO_x on plant growth and yield also are limited, Nevertheless, it it reasonable to assume that NO_x- induced reductions in the assimilative capacity of plants through altered metabolism, leaf injury, or abscission affect plant growth. MacLean (1975), however, concluded that average NO_2 concentrations are well below the threshold curve for damage to growth. In fact, the maximum NO_2 concentrations recorded in Los Angeles for 1966 would just begin to damage growth. Even in Los Angeles, however (the only major city in the U.S. that exceeds the NO_2-NAAQS), average NO_2 concentrations are below the likely threshold when averaged over longer periods.

Although NO_2 by itself is unlikely to damage plants at existing outdoor levels, several studies have demonstrated synergistic and antagonistic effects. The Criteria Document (EPA, 1982) concluded that "concentrations of NO_2 between 0.1 ppm and 0.25 ppm can cause direct effects on vegetation in combination with certain other pollutants" (pp. 12-44). But these data indicate that rural areas are still not at risk, because, in general, concentrations will not rise above 0.1 ppm for sufficient time for damages to occur. Therefore, we limit our estimate of benefits to account for vegetation damages in urban areas.

The plants most in danger would be ornamental vegetation, but even these damages are likely to be small. Leighton et al. (1983) concluded from their review that "ozone appears to account for more than 90 percent of total vegetation damages" (p. 60; see also Heck et al., 1982, and Page et al., 1982). If NO_x accounted for the other 10 percent of the total ornamental vegetation damage, as an upper-bound we could use the high estimate of ozone ornamental vegetation damages of $140 million to impute total NO_x damages of roughly $16 million. For the 0.44 percent reduction in NO_x predicted for 1986, that would imply benefits of only $62,000.

VI.C.2.d. NO_x Effects on Materials

Field studies and laboratory research have demonstrated that nitrogen oxides can significantly fade textile dyes. Barrett and Waddell (1973) estimated damages at $280 million (inflated to 1983 dollars using the Textile Products and

Apparel Index) for NO_x damage in this category. The basis for the estimates included not only the reduced wear life of textiles of moderate fastness due to NO_x, but also the costs of research and quality control. The major share of the costs is the extra expense of using dyes of higher NO_x resistance and of using inhibitors. Additional costs are also incurred for dye application and increased labor expenditures. The factors relating higher costs in the textile industry to NO_x are discussed in Chapter 8 of a report of the National Academy of Sciences (1976).

For 1986, assuming linearity, the Barret and Waddell estimate implies that materials benefits related to NO_x control would be about $1.2 million (0.44% x $280 million).

VI.C.2.e. Acid Deposition Benefits

Acid deposition occurs when NO_x and SO_2 emissions are chemically altered into acids in the atmosphere and transported over long distances, or when the precursor emissions are acidified after being deposited in dry form on plant, soil, or building surfaces. Both wet and dry forms of acidic deposition are harmful to aquatic, terrestrial, and material resources. While these damages are potentially important in some regions, basic scientific understanding of the effects on resources and transport processes is quite limited.

The computation of damages requires several crucial pieces of information. Unfortunately, many of these data are so uncertain that confidence intervals of damage estimates are exceptionally wide. Dollar estimates of damage require concentration-response functions and a tabulation of the resources at risk.

Reliable concentration-response data are lacking in nearly all resource areas, especially forestry and materials, and inventory data are unavailable for most material resources. Finally, the benefit estimate requires information on the relative contribution of nitrates and sulfates to total damages, as well as the relationship between the tonnage of NO_x emissions and nitrate deposition. All of these are the subjects of intensive ongoing research. Until more of this research is completed, quantitative damage estimates will not be accurate enough to be useful in a policy context.

Damage from acid deposition may be significant, although there is much uncertainty. Nitrates account for roughly 30 percent of total acidic loadings, but the damage information available indicates that the proportion of nitrate to total damages may be less than that implied by the relative emittant loadings.

VI.C.3. Carbon Monoxide

At current ambient concentrations, exposure to CO may cause health effects in some individuals. Persons with cardiovascular disease appear to be at highest risk, but those with chronic respiratory disease, pregnant women, and the elderly also are believed to be sensitive to CO exposure. Unfortunately, clinical dose-response functions relating low-level CO exposure to particular health effects, when estimated, have not been conclusive. Therefore, it is not possible to estimate the impact of reduced CO emissions on health endpoints. We have, however, been able

to estimate roughly the impact of reducing CO emissions on the numbers of people with carboxyhemoglobin (COHb) levels that may pose some risk.

VI.C.3.a. Health Effects of CO

Probably the greatest concern about CO exposure is its effect on the cardiovascular system. At moderate levels of exposure, CO reduces exercise time before the onset of angina pectoris. This clinical phenomenon is believed to result from insufficient oxygen supply to the heart muscle, and is characterized by spasmodic chest pain, usually precipitated by increased activity or stress, and is relieved by rest. Typically, atherosclerosis, which causes a narrowing of the arteries in the heart (coronary heart disease), predisposes a person to attacks of angina.

Angina pectoris, by definition, is not associated with permanent anatomical damage to the heart. Nonetheless, the discomfort and pain of angina can be severe, and each episode of angina may carry some risk of a myocardial infarction. However, epidemiological studies have not provided conclusive results on the association between CO exposure and the incidence of myocardial infarction.

The health effects from exposure to CO are associated with the percentage of total blood hemoglobin that is bound with CO, producing COHb, which reduces the oxygen-carrying capacity of the blood. The median concentrations of COHb in blood are about 0.7 percent for nonsmokers and about 4 percent for smokers. At least one clinical study (Anderson et al., 1973) associated reduced

exercise time until the onset of pain with COHb levels of 2.9 percent in patients with angina pectoris. At 4.5 percent COHb, this same study reported an increased duration of angina attacks.

The potential health improvements from reduced CO may he significant, for two reasons. First, there are many people in the population believed to be sensitive. EPA has estimated that 5 percent of the U.S. adult population -- roughly 11.5 million people -- has definite or suspected coronary heart disease. Of this group, as many as 80 percent have suspected or definite angina pectoris (U.S. EPA, 1980). Additional large subgroups of the population may be particularly sensitive to exposure to CO, including individuals with pre-existing conditions that compromise oxygen delivery to various tissues, enhance oxygen need, or elevate the sensitivity of tissues to any oxygen imbalance . Other sensitive groups may include:

- people with peripheral vascular diseases, such as atherosclerosis and intermittent claudication (0.7 million people);
- people with chronic obstructive pulmonary diseases (17 million people);
- people with anemia or abnormal hemoglobin types that affect the oxygen-carrying capacity of the blood (0.1245 million people);
- people drinking alcohol or taking certain medications (e.g., vasoconstrictors);
- the elderly;
- residents of and visitors to high altitude areas; and

- fetuses and infants (3.7 million total live births per year).*

The second reason for potentially significant impacts of reduced CO exposure is that the blood of many people shows concentrations of COHb above 2.9 percent, the lowest level of COHb where adverse effects are believed to occur. Data from the second National Health and Nutrition Examination Survey (NHANES II) indicated that for the U.S. population over twelve years of age, 2 percent of those who have never smoked, 3 percent of former smokers, and 66 percent of current smokers exceeded 2.9 percent COHb at the time of the survey (U.S. DHHS, 1982).

Other health effects have been reported at comparable or higher COHb levels. For example, several. investigators have found statistically significant decreases in work time until exhaustion in healthy young men with COHb levels at 2.3 to 4.3 percent (Horvath et al., 1975; Drinkwater et al., 1974; Raven et al., 1974). At higher COHb levels (5.0 to 7.6 percent and above), investigators have reported impairment in visual perception, manual dexterity, ability to learn, and performance Of complex sensorimotor tasks in healthy subjects. Finally, Klein et al. (1980) showed that, at 5.0 to 5.5 percent COHb, healthy young men had decreased maximal oxygen consumption and decreased time at strenuous exercise before exhaustion.

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* Animal studies showed that pregnant females exposed to CO had lower birth weights and increased newborn mortality, and their newborns had lower behavioral levels, even when no effects on the mother were detected. In addition, a possible association has been reported between elevated CO levels and Sudden Infant Death Syndrome (Hoppenbrouwers et al., 1981.)

VI.C.3.b. Change in Numbers of People Above 2.9 Percent COHb

For three specific subgroups of the U.S. population -- current smokers, ex-smokers, and never-smokers -- we calculated the number of people who would move below 2.9 percent COHb (the lowest level frequently associated with specific health effects) because of the reduction in CO emissions. We also calculated the number of "sensitive" individuals who would shift below 2.9 percent COHb. This 2.9 percent level should not be construed as a "threshold" level, hut rather as a level at which some increased health risk has been detected.

We considered several complex relationships to make these rough estimates. Because of resource and data limitations, we assumed simple one-to-one relationships in some of the linkages between changes in CO emissions and the ultimate changes in COHb levels. Specifically, we assumed that a given percent change in CO emissions would generate a similar percent change in ambient CO. We also assumed a linear relationship between changes in ambient CO and the mean percent COHb level in blood. For small changes, these assumptions may be fairly accurate. For larger changes in CO emissions, there is greater uncertainty concerning the impact on COHb levels. For example, it is well known that the binding affinity of hemoglobin may be nonlinearly related to the level and change in CO. Mage et al. (1984), however, found a very high correlation ($R^2 = 0.967$) between maximum COHb levels and five-hour averages of CO concentrations, which suggests that our assumption of linearity is a good approximation.

In estimating the number of people whose COHb levels would exceed 2.9 percent, we assumed that the geometric standard deviation of the distribution of COHb levels was roughly constant for small changes in mean COHb levels; this implies that changes in CO emissions will shift the whole distribution proportionately. Subject to these simplifying assumptions, the results are indicative of the potential magnitude of the effect of reduced CO emissions on COHb levels.

To estimate the reduction in COHb levels, we had to (1) calculate the average annual CO emissions from auto and residential sources* for 1976-1980; (2) project 1988 emissions from these two sources; (3) determine the shape (mean and standard deviation) of the frequency distribution of COHb levels for 1976-1980 from the NHANES II for current smokers, former smokers, and never-smokers; (4) adjust the mean of this distribution for 1988, based on the changes in CO emissions; (5) calculate the change in mean COHb levels with and without misfueling; and (6) multiply the change in the probability distribution by the appropriate population group to predict the number of people now expected to be below 2.9 percent COHb.

The average annual CO emissions from automobiles and residences during 1976 to 1980 were 73.3 and 3.9 million tons, respectively, for a total of 77.2 million tons (U.S. EPA, 1982f).

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* We focused on auto emissions and residential sources (gas ovens, heating, etc.) because they have been identified as the most serious sources of human exposure. Cigarette smoking patterns and rates are assumed to be unaffected by any misfueling policy.

The estimate of auto emissions for 1988 is 36.5 million tons.* To estimate residential CO emissions, we determined the relationship of residential CO emissions over time (U.S. EPA, 1982f), and projected that relationship into the future. Based on this analysis, residential CO emissions would be 8.3 million tons in 1988. Thus, CO emissions from these two sources were estimated to drop to 44.8 million tons in 1988, a 42 percent decrease.

Data on blood carboxyhemoglobin levels were collected as part of the NHANES II study. Because of the seasonal and geographic pattern of the survey during the four-year period, it may not reflect the full range of potential exposure. Specifically, the Northeast was not sampled during the winter, and no high-altitude cities were sampled. Therefore, the survey results may somewhat understate total CO exposure.

For each of the subgroups -- current smokers, ex-smokers, and never-smokers -- we fit the observed COHb levels with a lognormal distribution. We took the log of each observation in the NHANES and calculated the mean and the standard deviation of log(COHb)

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* The baseline projections for CO for 1988 were calculated as follows:

For CO we started with EPA emission factors generated in the draft model of MOBILE III for on-road vehicles in 1988 of approximately 26.65 g/mi. The emission factor was reduced by 0.75 (to 20 g/mi) to adjust for I/M areas and the state of California, which has its own, more stringent, emission controls. We assumed 159.6 million on-road vehicles traveling an average 11,436 miles (see Appendix B). Multiplying:

$$\frac{20 \text{ g/mi} \times 11{,}436 \text{ mi/vehicle} \times 159.6 \times 10^6 \text{ vehicles}}{1 \times 106 \text{ g\metric ton}}$$

$$= 36.5 \times 10^6 \text{ metric tons}$$

for each of the three groups. The mean COHb level (M) is then:

$$M = \exp (u + s^2/2),$$

where u is the mean of the lognormal distribution and s^2 is the variance. To estimate the 1988 COHb baseline, we reduced the mean COHb level by the predicted change in COHb (P) over time. We calculated the difference, log(M) - log(M-P), for each subgroup. This difference was then subtracted from the log(COHb) for each person in the subgroup to obtain a new distribution. This procedure shifts the distribution down while keeping the geometric standard deviation constant. Thus, it assumes constant proportional changes, rather than equal absolute reductions, in COHb levels across subgroups.

To calculate P, we needed to determine the impact of the reduction in CO emissions from 1978 to 1988 on mean COHb levels. We assumed a linear relationship between ambient CO and percent COHb, with a slope coefficient of 0.16 (adapted from Ferris, 1978; U.S. EPA, 1979a; and Jourmard et al., 1981). We used an average ambient CO level of 3.127 ppm for 1978 (Council on Environmental Quality, 1980). Thus, the projected 42 percent change in ambient CO between 1978 and 1988 would reduce mean percent COHb levels, on average, by 0.210 percentage points (0.42 X 3.117 X 0.16 = 0.210).

Given the 1988 baseline distribution of COHh levels, we determined the effect of eliminating all misfueling and reduced

the mean by 5 percent, the estimated CO reduction.* We used the same procedure of converting to a log change, reducing the COHb level of each population subgroup by that amount, and re-averaging. From this new distribution, we could then calculate the change in the number of each subgroup under 2.9 percent COHb due to this reduction in the mean. Since the NHANES II data are weighted to represent the population of the United States, the weighted average gives us the change in the total number of people in each group who would have COHb levels above 2.9 percent. The results indicated that over 400,000 people would shift below the 2.9 percent COHb level if we could eliminate misfueling: 112,000 current smokers: 62,000 exsmokers; and 227,000 never-smokers.

Another indication of the impact of the reduction in CO is generated by considering the reduction in COHb levels for some of the sensitive subgroups identified above. This includes those with suspected and definite angine (9.2 million), peripheral vascular disease (0.7 million), chronic obstructive pulmonary disease (17 million), anemia (0.1245 million), and fetuses (3.7 million). Totalling our projections for 1988, we estimated that there will be 31 million sensitive individuals. In addition, we included a subset of the elderly, who may have an increased risk

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* The actual change in CO was calculated using the estimates of avoided emissions in Table VI-3 and projections of total emissions in 1988. Assuming motor vehicles emit 86 percent of all CC) from transportation and residential fuel combustion, the reduction of 2.2 million metric tons is 5 percent (2.2 x 0.86/ 36.5) of the total.

but who may not have been counted within these other groups. As an approximation, we added 25 percent of those age 65 and above, to obtain a total population at risk of 39 million people.

Since we did not have data on the COHb distribution for these sensitive subgroups (including the elderly and those with coronary heart disease), we assumed their COHb distribution was similar to that of never-smokers. This assumption was made because some in this sensitive group probably take measures to reduce the impact of air pollution and other irritants on their health. However, it probably generates a low estimate because many in this group continue to smoke, and others are former smokers. Nevertheless, we applied the previously determined change in the portion of never-smokers who would shift below 2.9 percent COHb because of the reduction in misfueling (0.000865) to the group considered sensitive. It suggested that 33,700 sensitive people will shift below the 2.9 percent COHb level in 1988.

VI.C.4. Ethylene Dibromide Emissions

Most of the ethylene dibromide (EDB) manufactured in the United States is added to leaded gasoline as a scavenger for the lead. Reducing the lead concentration of gasoline would result in an equal reduction of EDB use, which is of concern to EPA because it is a potential human carcinogen. EDB from leaded gasoline enters the air through three routes: tailpipe emissions, evaporative emissions from cars, and evaporative emissions from the retail and distribution chain of gasoline.

Sigsby et al. (1982) estimated EDB emissions from tailpipes and evaporative emissions from cars. They concluded that EDB emissions at the tailpipe were approximately 0.37 percent of the amount in gasoline. However, all of the emissions tests were done on retuned and adjusted cars, and under somewhat artificial test procedures; the 0.37 percent survival was the average of the tests. The EPA federal test procedure showed an average EDB survival of 0.69 percent. Since EPA test of on-the-road vehicles have generally shown substantially higher actual emissions than in retuned cars undergoing the federal test procedure, we used the 0.69 percent survival factor in this analysis. Multiplying this by the projected leaded gasoline demand in 1986 yields an estimated reduction in EDB tailpipe emissions of 143 metric tons.

Shed tests of the evaporative emissions of EDB generally indicate that the EDB evaporative emissions were 1/20,000 of the hydrocarbon evaporative emissions (Sigsby et al., 1982). In the absence of better data, we assumed the same ratio of refueling evaporative emissions. This results in an estimated 34 additional metric tons of EDB emissions, based on our fleet model.

Finally, EPA estimated (EPA, 1984a) that hydrocarbon emissions in the retail and distribution chain of gasoline were 407,000 metric tons in 1982. Based on our estimate of 37.5 percent leaded gasoline demand in 1986, we estimated that leaded gasoline would produce 153,000 metric tons of hydrocarbon emissions. Using the ratio of EDB to hydrocarbon emissions, this suggests 7.6 metric tons of EDB emissions from the retail and distribution stages that would be reduced if all misfueling was eliminated. In

total, we estimate 185 metric tons of EDB emissions from all three sources (tailpipe, evaporative, and marketing emissions) would be avoided by reducing lead in gasoline in 1986.

In addition, EDB from gasoline storage tanks has been leaking into underground aquifers. Data do not exist, however, to estimate the magnitude of the benefit of reducing the contamination of ground water by EDB.

VI.D. Monetized Benefit Estimates

To estimate the monetized benefits of reducing emissions of pollutants other than lead, we used two different methods. The first used the direct estimates of health and welfare effects, combining the nonhealth estimates with monetized estimates of the health effects. As discussed earlier, that method is conceptually correct, but suffers from substantial uncertainties and from our inability to quantify some potential significant benefit categories. Our second method uses the cost of pollution control equipment destroyed by misfueling as a proxy for the benefits of eliminating misfueling.

VI.D.1. Value of Quantified Health and Welfare Benefits

Our direct approach yields monetary estimates for only a limited number of benefit categories, most of them related to ozone. The ozone-related benefits fall into two major categories: health-related and other. The second category already has been estimated in monetary terms (Table VI-11) for 1986. The ozone-related health effects, however, thus far have been stated only in physical terms.

For the more serious category of respiratory conditions (days of bed rest or work loss), we used the average daily wage ($80) as a lower bound. This is the value of the lost output to society for an employed individual. Because some of these conditions also involve additional medical expense, we used an upper bound of $120, giving a mean value of $100 per day.

To value a day of more minor restrictions in activity due to respiratory conditions, we relied upon Loehman et al. (1979), whose survey results suggested a willingness to pay of $2.31 to prevent a day of minor coughing, $4.90 to prevent minor shortness of breath, and $8.17 to prevent minor head congestion. We converted these estimates from 1978 dollars to 1983 dollars to yield values of $3.50 to $12.50 for avoiding a minor restricted day, with a point estimate of $8.

We were unable to find estimates in the literature for the value of avoiding headaches or eye irritation. These conditions, however, seemed less serious than the respiratory effects discussed above, so we used a value of $3 per case, just below the lower end of the range from the Loehman et al. study.

In addition to the ozone-related benefits, we were able to place rough monetary estimates on three additional benefit categories: (1) reduction in leukemia cases associated with benzene exposure; (2) NO_x-related visibility benefits; and (3) NO_x-related materials damages. The monetary estimates for the last two categories already have been discussed. For the

leukemia cases, we note that the types of leukemia associated with benzene exposure are almost invariably fatal. As discussed in the previous chapter, valuing reductions in risks to life is controversial, with a wide range of values found in the literature. For our high and low estimates, we used the range in the EPA guidelines, $400,000 to $7 million per statistical life saved. For our point estimate, we used the same value employed in the previous chapter for blood-pressure-related fatalities, $1 milliom per case.

Table VI-13 summarizes the monetized benefit estimates for 1986. The total covers a broad range, from $113 million to $305 million, with a "medium" estimate of $171 million. For the reasons discussed earlier, plus uncertainties about the valuation of health effects, even this broad range does not capture the full extent of the uncertainty with respect to the categories estimated. Moreover, it has not been possible to monetize many of the possible benefits; the bottom of Table VI-13 presents a partial list of those omitted categories.

VI.D.2. Implicit Value Based on Cost of Control Equipment

Our second method of valuing reduced emissions of HC, NO_x, and CO is based on the cost of pollution control equipment destroyed by misfueling. This method assumes, implicitly, that the benefits of controlling mobile-source emissions are at least equal to the cost of the equipment needed to meet emission

TABLE VI-13. Monetized Benefit Estimates Due to Elimination of Misfueling in 1986 (millions of dollars)

Effect	Low Estimate	High Estimate	Medium Estimate
Quantified Benefits, Ozone-Related			
Health			
Respiratory			
Bed rest/Lost work	21.5	110.1	47.8
Minor restrictions	2.8	33.6	11.4
Nonrespiratory	9.8	9.8	9.8
Other			
Agricultural crops	63.8	99.2	80.8
Ornamental plants	1.0	1.0	1.0
Materials damage	11.2	16.8	13.4
Quantified Benefits, Not Ozone-Related			
Leukemia (benzene)	1.8	30.8	4.4
Visibility (NO_x)	0.0	2.4	1.2
Materials damage (NO_x)	1.2	1.2	1.2
TOTAL MONETIZED BENEFITS	113.1	305.0	171.0

Unquantified Benefit Categories

Chronic health effects due to ozone
Forest damage due to ozone
Direct health effects of HCs other than benzene
Sulfate-related damages due to HC
Acid-precipitation damages due to NO_x
Health effects due to NO_x
Vegetation damages due to NO_x
Health effects due to CO
Health effects due to EDB

standards. Emissions control equipment costs about $283 per vehicle (U.S. EPA, 1981).*

One complication for this method of valuing emissions is that many catalysts are not destroyed by misfueling until they have been in use for several years. Thus, it was necessary to prorate the cost of the pollution control devices. To do so, we first estimated the tons of emissions that would be controlled over the life of the pollution control device. These estimates accounted for declines in the efficiency of the devices over time, declines in annual miles driven as vehicles age, and scrappage rates. We then discounted the emissions controlled back to the first year (to make them comparable to the cost estimate), and divided them into the $283 cost of pollution control equipment. That yielded a cost of $153 per ton controlled.** Note that this

*The costs are "retail price equivalents," which are 30 percent to 50 percent of the manufacturers' suggested retail price of the components (catalysts, oxygen sensors, etc.) as replacement parts. There may be a small upward bias in this estimate, but we used the lower estimate in the cited report ($250, compared to a $425 upper bound). Converting to 1983 dollars gave a cost of $283. (About half of the cost of oxygen sensors and other equipment was allocated to fuel efficiency, not pollution control.)

**Using 1981 emissions standards of 0.41 grams per mile for HC, 3.4 g/mi for CO, and 1.0 g/mi for NO_x gave us 4.81 g/mi for all pollutants in each future year. We multiplied 4.81 g\mi by E/(1-E) (where E equals the catalytic converter efficiency in that year) and by 10,000 mi/yr, and divided by 1000 g/Kg to get kilograms controlled in each year by one catalytic converter. We then discounted the estimate for each year back to the first year of the catalytic converter's life. Summing these present values gave us an estimate of 1.848 tons controlled by each car's catalytic converter over a 10-year life. A cost of control equipment of $283, divided by 1.848 tons, gave us $153 per ton.

estimate covers the sum of HC, NO_x, and CO emissions; because the devices control all three pollutants simultaneously, it was not possible to generate pollutant-by-pollutant estimates using this method.

We then calculated year-by-year benefits by multiplying that estimate of $153 per ton of pollutant controlled times the projected reduction in total emissions that would be achieved in each year if misfueling were totally eliminated. For example, as shown in Table VI-3, we estimate that eliminating misfueling in 1986 would reduce the present value of emissions of the three pollutants by 2.515 million tons. Thus, our estimate by this method of the benefits for 1986 is $385 million ($153 x 2.515 million tons).

VI.D.3. Summary of Benefits of Controlling Pollutants other than Lead

In this chapter, we have developed two approaches to valuing the reductions in emissions that would be achieved by eliminating misfueling. The first method, based on direct estimates of health and welfare benefits, yielded an estimate of $171 million for 1986. To generate estimates for other years, we scaled the 1986 estimates in Table V-13, with the scaling factor depending on the benefit category. For the ozone-related agricultural benefits, we scaled the estimates in proportion to the estimated change in average rural ozone levels (from Table VI-10). For the other ozone-related benefits, we used the predicted change in peak ozone levels (also from VI-10). For the benzene-related

leukemia cases, we scaled by hydrocarbon emissions (from Table VI-3), because most of the reductions in benzene result from reduced emissions of hydrocarbons from misfueled vehicles. Finally, the two NO_x-related categories, visibility and materials damage, were scaled by NO_x emission reductions (also from Table VI-3). This scaling procedure assumed that benefits are proportional to emission reductions over the relevant ranges. The results are shown in the first line of Table VI-14.

The caveats discussed earlier with respect to the direct estimates for 1986 apply to these estimates as well. They include not only the significant uncertainties associated with the estimated categories, but also the omission of several benefit categories due to limited data. For ozone, the omissions include chronic health effects and forest damage. For HC we have not estimated any direct health effects for hydrocarbons other than benzene, nor have we quantified the link between hydrocarbons and sulfate formation. For NO_x, we have not quantified benefits related to acid precipitation or to vegetation, nor have we quantified any health effects. We also have been unable to generate monetized benefit estimates for CC), although we have made made some rough estimates of the numbers of people whose COHb concentrations may fall below a potential harmful exposure to EDB. As a result of all of these omissions, it is likely that the direct estimates are too low, perhaps by a substantial margin.

The second method, based on the implicit cost per ton of pollutants controlled by catalysts, yielded a value of $153 per ton for the sum of HC, NO_x, and CO emissions. The second line of Table VI-14 shows the estimates based on that method. The last line shows the averages of two methods; the summary estimates in Chapter VIII are based on those averages. In that chapter, we also estimate the benefits if misfueling is only partially eliminated by the rule.

TABLE VI-14. Year-by-Year Monetized Estimates of Benefits of Reduced Emissions of Conventional Pollutants, Assuming No Misfueling (millions of 1983 dollars)

Method	1985	1986	1987	1988	1989	1990	1991	1992
Direct Estimate	86	170	169	170	170	173	180	186
Control Device	193	385	386	389	392	401	416	431
Average	140	278	278	280	282	288	299	310

CHAPTER VII

VEHICLE MAINTENANCE, FUEL ECONOMY, AND ENGINE DURABILITY BENEFITS

Switching from leaded to unleaded gasoline, or using fuel with a lower lead content, provides benefits to vehicle owners. The principal benefits are lower maintenance costs from lead-induced corrosion of exhaust systems and engines. Reducing lead in gasoline is also likely to increase fuel economy. Eliminating lead altogether, however, may cause premature valve-seat recession in a few engines designed to rely on lead as a valve lubricant.

Recognizing the problem of excessive deposits of lead in engines, refiners add scavengers to leaded gasoline to prevent such deposits. These scavengers -- primarily ethylene dibromide (EDB) and ethylene dichloride (EDC) -- form compounds (e.g., halogen acids and lead salts) that accelerate corrosion of exhaust systems and engine components. Section A of this chapter discusses the maintenance benefits associated with reducing lead (and its scavengers) in gasoline, and presents monetary estimates for three categories: exhaust systems, spark plugs, and oil changes.

Reducing or eliminating lead in gasoline increases fuel economy. The refining processes used to produce octane without lead yield gasoline that IS “denser” (i.e., has a higher energy content per gallon). Lead fouls oxygen sensors in newer cars that are misfueled by their owners; this also reduces fuel efficiency. Section B presents the methods used to estimate these fuel economy benefits.

Section C addresses the issue of engine durability. Most modern engines have hardened valve seats or other features designed to minimize valve-seat wear. However, many older (pre-1971) cars, designed to operate on leaded gasoline, do not have hardened valve seats. In such engines, lead can play a positive role, forming a protective veneer that “lubricates” the exhaust-valve seat, thus reducing abrasive and adhesive wear that can erode the seat, requiring major engine repairs.

Concern about potential valve-seat damage in some engines was the primary reason EPA proposed reducing lead to 0.10 gplg, and did not propose banning it altogether until 1995; tests indicate that 0.10 gplg provides a margin of safety to protect against premature valve-seat wear. As discussed in Section C, however, it appears that valve-seat recession may be less of a problem than the Agency believed at the time of the August proposal.

Large studies of vehicles in use have not detected significant valve-wear problems when older engines are switched to unleaded gasoline. Moreover, studies also indicate that lead can cause other potentially serious problems that reduce the useful lives of engines, such as accelerated ring and bearing wear and increased rates of valve burnout. In part because of reduced concern about potential valve-seat wear, EPA is now considering a ban on lead in gasoline to the take effect as early as 1988.

VII.A. Maintenance Benefits

Reducing lead in gasoline can result in less frequent replacement of exhaust systems and spark plugs and less frequent oil changes. Our estimates of maintenance savings are based primarily on tests of vehicles in use, either commercial fleets (e.g., taxis) or vehicles owned by individuals for personal use. In such tests, most of which were performed in the late 1960s or the early 1970s, the maintenance records of vehicles operated on unleaded gasoline were compared to those of vehicles using leaded fuel. In addition, we have supplemented such data with the results of laboratory tests and, in a few cases, theoretical calculations reported in the literature.

Estimating the maintenance benefits was complicated by the fact that in most of the studies, the leaded gasoline averaged 2.3 gplg or more (the levels that were typical when the tests were performed), but current leaded gasoline averages only 1.10 gplg. In addition, the tests usually examined the benefits of switching to unleaded gasoline, not to a very low-lead gasoline of the type permitted by the rule being promulgated. As a result, we were forced to interpolate from limited data, first to estimate how many of the benefits already have been reaped in reducing leaded gasoline to 1.10 gplg, and second to predict what additional benefits could be reaped by further reducing, but not eliminating, lead in gasoline.

VII.A.1. Exhaust Systems

Vehicles experience fewer exhaust system failures using unleaded gasoline than leaded because of the difference in

acidity in exhaust gas condensates. In cars using leaded fuel, these condensates have a PH ranging from 2.2 to 2.6, while for unleaded cars the range is 3.5 to 4.2 (Weaver, 1984b). This higher acidity accelerates corrosion in mufflers and tailpipes.

Table VII-1 summarizes the results of four studies, involving nine different fleets of vehicles, that examined the effects of leaded gasoline on exhaust-system replacement rates. (The effects on spark plug replacements were also studied; this is discussed in Section VII.A.2.) All of the studies found demonstrable increases in expected lifetimes (measured in miles) of exhaust systems in unleaded vehicles when compared with comparable leaded vehicles. The minimum increase in average life of the exhaust system for unleaded vehicles was 86.5 percent (the Wintringham et al., 1972, Detroit fleet).

The average exhaust-system replacement rates for leaded cars varied greatly among the different studies, ranging from 1 per 20,500 miles (Gray and Azhari, 1972, model year 1967 vehicles) to 1 per 58,800 miles (Pahnke and Bettoney, 1969). (The rate per mile for the Gray and Azhari, 1972, study of consumers' personal use cannot be computed because of inadequate data.) Four of the fleets showed virtually no replacements of exhaust systems in the vehicles using unleaded gasoline. Averaging the results of all these studies, we found about one exhaust system replacement every 56,000 miles for cars using leaded fuel, and essentially none for vehicles using unleaded fuel during the test periods.

TABLE VII-1 Summary of On-Road Studies of Spark Plugs and Exhaust Systems

STUDY	REPLACEMENT RATES PER 11,000 MILES (OR PER 1 YEAR) SPARK PLUGS		EXHAUST SYSTEMS		AVG. MPV/YR	ACCUMULATED AVG. MVP	TYPE OF SERVICE	# OF VEHICLES LENGTH OF TEST	LOCATION
	UNLEADED	LEADED	UNLEADED	LEADED					
Pahnke & Bettoney (DuPont, 1969)	.534	.726	.0333	.187	11,400	65,000	Personal Use	59 matched pairs/ 4.7 years	South New Jersey and Wilmington, Delaware
Gray & Azhari (1972) (Amoco)							Commuting and business use	12 matched pairs 2 and 3 years	Chicago and suburbs
MY 1967:	.373	.840	.149	.535	7,500	24,000			
MY 1968:	.307	1.085	0	.217	7,500	17,000			
Gray & Azhari (1972) (Amoco)	.247 weighted avg.	.295	.004 weighted avg.	.071	Not reported	1 to 6 yrs.	Personal Use (Consumer Panel)	151 matched pairs/1-5 years	Eastern states concentrated in Mid-Atlantic
Wintringham et.al., (Ethyl, 1972)							Employee Fleet (Business and Personal Use)		Detroit Baton Rouge
Detroit:	.440	.677	.155	.289	14,575	72,883		31 matched pairs	
Baton Rouge:	.347	.519	.004	.358	16,850	84,260		33 matched pairs, 5 years	
Hickling Partners (Environment Canada) (1981) Municipal Fleets	2.9 times as many w/leaded vehicles		2.4 times as many for leaded vehicles (they exclude Toronto fleet)		(Unknown)	23,810 leaded 24,990 unleaded	Municipal Service	835/5 years	Montreal Edmonton Toronto

It is useful to look most closely at the Ethyl Corporation (Wintringham et al., 1972) findings, as vehicles in that study had the greatest mileage and there is a clear geographic distinction between the fleets, which highlights the effects of climate. The Baton Rouge fleet, after an average of over 84,000 miles of travel per car (compared to a projected lifetime of 100,000 miles), had virtually no exhaust system repairs for unleaded vehicles, but a rate of about 1 per 31,000 miles for leaded vehicles. By comparison, the Ethyl Detroit fleet, after about 73,000 miles of travel per vehicle, had a rate for unleaded vehicles of one exhaust system replacement per 46,000 miles, but a rate of 1 per 24,000 miles for leaded vehicles. The main reason for the different experiences in Baton Rouge and Detroit, the authors concluded, was the greater external corrosion due to road salts in the colder climate.

The Detroit results are consistent with the Environment Canada findings (Hickling Partners, 1981) for two municipal fleets, which had 42 percent fewer exhaust system replacements (at equivalent mileage) for cars using unleaded fuel in a cold climate. On the other hand, the DuPont (Pahnke and Bettoney, 1971) and Amoco (Gray and Azhari, 1972) findings, conducted in the mid-Atlantic region, in Chicago, and in the eastern U.S., were closer to Ethyl's in Baton Rouge; there were virtually no exhaust repairs for vehicles using unleaded fuel. However, the evidence suggests that exhaust system corrosion rates do vary with climate and we have incorporated that variation into our estimates.

Weighting Ethyl's findings for Detroit and Baton Rouge according to the portion of registered cars in Sunbelt versus Snowbelt states in 1982 (43 percent and 57 percent, respectively, according to the Motor Vehicle Manufacturers Association [MVMA], 1983), mufflers nationally would last an average of three times longer with unleaded fuel than with leaded. Unfortunately, these studies were conducted on fleets of vehicles over several years, but for less than the lifetimes of the vehicles. It is possible, therefore, that the studies ended shortly before many of the unleaded vehicles required exhaust system replacements. Perhaps the replacement rates for unleaded vehicles would have increased significantly had the fleets traveled another 10,000 to 20,000 miles. The reported findings, thus, may have overestimated the differences between unleaded and leaded vehicles. Because of this concern, we have assumed that mufflers on vehicles using unleaded fuel would last only twice as long (in miles) as those on vehicles using leaded fuel.

We assumed mufflers on vehicles using leaded gasoline would last about 50,000 miles; in the studies we reviewed, the leaded fleets averaged about 20,000 to 60,000 miles between exhaust system replacements.* Applying our factor of two yielded an

* Passing references in the literature and several commenters have suggested that the metallurgy of exhaust systems was upgraded during the 1970s, e.g., changing from cold-rolled milled steel to chromium stainless steel. Since the more durable metal would corrode less easily, the commenters suggested, this design improvement might affect performance and our estimates of benefits might be substantially overstated. However, on the improved exhaust systems, only the parts from the exhaust manifold to the catalytic converter are stainless steel. The remaining components of the exhaust system (exhaust pipe, muffler, and tailpipe) are generally still made of rolled steel. These are the parts that we estimated would corrode from leaded gasoline. Thus, this technology change should have no effect on our estimates of savings.

expected lifetime of 100,000 miles for vehicles using unleaded gasoline. Based on a cost of $120 per exhaust system replacement, the savings per mile are ($120)(1/50,000 - 1/100,000) = $0.0012/mile, or $12.00 per year for a vehicle driven 10,000 miles annually.

This estimate must be applied with care, because it is based on comparisons of vehicles operated on leaded gasoline with a lead content over 2 gplg to vehicles operated on unleaded gasoline. We were uncertain as to how much, if any, of the benefits of reduced exhaust system corrosion might already have been reaped as a result of reducing lead to its current level of 1.1 gplg. Fortunately, Gray and Azhari (1971) examined exhaust system corrosion rates using leaded gasoline at both 2.3 gplg and 0.5 gplg; they found no difference between the two types of leaded gasoline. (Both showed corrosion rates 10 to 20 times higher than those with unleaded.) This finding suggests that no reductions in exhaust system corrosion are reaped until the lead content falls below 0.5 gplg. Thus, we estimated that vehicle owners switching from leaded gasoline at 1.1 gplg to unleaded will experience savings of $0.0012/mile. This estimate applies to misfuelers who are deterred by the rule, and in the case of a complete ban.

Estimating the exhaust system benefits for vehicle owners who use low-lead (0.10 to 0.50 gplg) gasoline was more problematic. For lack of better information, we assumed linearity between zero and 0.5 gplg; e.g., at 0.10 gplg, owners would get 80 percent of the benefits, or 0.8(0.0012) = $0.00096/mile. This translates to $9.60 per year for a vehicle owner driving 10,000 miles yearly.

VII. A.2. Reduced Fouling and Corrosion of Spark Plugs

The corrosive effects of lead and its scavengers also reduce the useful life of spark plugs. As shown in Table VII-1, all of the fleet studies showed longer intervals between spark plug changes for vehicles operated on unleaded than on leaded. The increases ranged from 19 percent (Gray and Azhari, 1972, consumer use study) to 350 percent (Gray and Azhari, 1972, for model year 1968 vehicles); on average, the gain was about 80 percent.

To estimate benefits, we assumed that the average interval between spark plug changes with leaded gasoline at 2.3 gplg would be 10,000 miles.* That is roughly consistent with manufacturers' recommendations in the early 1970s, before new cars used unleaded. Applying the 80 percent improvement estimated above for users of unleaded would allow an interval of 18,000 miles between changes.

As with the exhaust system data, these tests used leaded gasoline at about 2.3 gplg and unleaded, so we had to make adjustments to account for savings due to the change from 2.3 to 1.1 gplg. In 1971, Toyota reported (Champion, 1971) that fouling

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* In practice, consumers appear to have changed spark plugs less frequently; the average for leaded vehicles in the fleet tests was about 15,000 miles. Owners who delay spark plug changes, however, suffer losses due to decreased fuel economy; which usually exceed the cost of replacing spark plugs at the appropriate interval. For consumers who change spark plugs less frequently than optimal, the benefits of unleaded or reduced lead gasoline will come through added fuel economy (since the spark plugs will degrade less on the unleaded gasoline) rather than reduced spark plug changes. For example, Craver et al. found that spark plugs with the wrong gap and orientation led to decrements of up to 7 percent in fuel economy. In general these benefits will be higher, so our use of the replacement costs is conservative.

of spark plugs occurred at the same rates with leaded gasoline at 0.2 gplg as with unleaded gasoline. In 1972, Union Oil reported (Champion, 1972) that spark plug performance was similar for unleaded and leaded gasoline at 0.5 gplg. With either type of gasoline (unleaded or 0.5 gplg), Union reported spark plugs lasted four times longer than with leaded gasoline containing 3.0 gplg. These findings suggest that there is a threshold below which further reductions in lead yield no additional gains in spark plug life.

For lack of better information, we assumed that the threshold occurred at 0.5 gplg, and that the relationship between lead and spark plug life from that level to 2.3 gplg was linear. Thus, we assumed that the reduction in lead from 2.3 gplg to 1.1 gplg increases the interval by (2.3-1.1)/(2.3-0.5) (80 percent) = 53 percent, or from 10,000 miles to 15,333 miles. At $18 per spark plug change, we estimated that reducing lead from 1.1 gplg to 0.5 gplg or below will provide benefits of (1/15,333 - 1/18,000)($18) = $0.000174/mile. The annual benefit for a car owner driving 10,000 miles per year would be $1.74.* Note that this estimate applies both to those who switch from leaded to unleaded and to those who use low lead gasoline.

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* By contrast,if car owners replace their spark plugs less frequently than they should, and the fuel economy penalty of increased spark plug degeneration from leaded fuel is only 0.5 percent, the benefits would be about double this estimate.

VII.A.3. Extended Oil Change Intervals

The combustion products that deposit on engine surfaces cause corrosion and rusting. Engine oil accumulates much of the debris from this corrosion, as well as some portion of the gasoline lead. According to at least one estimate, up to 10 percent of the lead in gasoline ends up in the used oil, comprising up to 50 percent of the weight of engine oil sludge (Gallopoulos, 1971).

The particles and corrosive products that accumulate in the oil cause substantial wear in the engine, and the internal engine rust may cause hydraulic valve lifter sticking (Cordera et al., 1964). Besides the long-term engine wear that reduces the durability of the engine, the vehicle driver may also experience excessive valve noise and other performance degradation due to this premature contamination of oil. Although rusting can occur even in the absence of the halogen acids derived from lead salts, these compounds are the major cause of internal rusting under normal driving conditions (Weaver, 1984b).

The fleet studies summarized in Table VII-1 generally did not consider oil changes or, if they did, found little difference between the behavior of drivers using leaded and those using unleaded. This result should not be surprising, as it is unlikely that the vehicle owners in the studies were aware of the impact of eliminating lead on engine oil. Presumably most drivers today are similarly unaware, and follow the recommendations in their owners' manuals (or habit) in changing their oil. If unleaded gasoline increases the useful life of engine oil, however, switching to

unleaded will yield benefits in the form of improved engine durability, even if oil change intervals do not change. Thus, in these cases, our estimates may be viewed as a proxy for improved engine durability.

Gallopoulos (1971), of the General Motors Corporation, was one of the first people to investigate the potential impacts of unleaded gasoline on oil-change intervals. He concluded that with unleaded gasoline it might be possible to decrease the frequency of oil changes from 2 or 3 per year to only 1 per year, but added that further investigation was needed.

Pless (1974), also of General Motors, reported more conclusive results based on experiments with taxicabs under conditions that took an unusually severe toll on oil quality. In a group of 20 taxis (1970 model year), Pless found less piston varnish, ring wear, and used-oil insoluble for the unleaded vehicles after 16,000 miles of stop-and-go service.

On a fleet of 1972 taxis, Pless (1974) compared unleaded vehicles after 16,000 miles without an oil change with leaded vehicles (2.7 grams of lead per gallon) after 8,000 miles. The results indicated less sludge, oil ring deposits, compression ring wear, cam and lifter wear, and oil degradation for the unleaded vehicles with extended oil change intervals, compared to the leaded taxis with “normal” oil changes. While the unleaded vehicles had somewhat greater plugging of oil filters, Pless concluded that this was not a significant finding. Finally, another fleet traveling predominantly short trips (closer to typical consumer driving patterns) led Pless to conclude:

> A combination of unleaded gasoline and doubled oil change interval allowed significantly less ring wear, and directionally less sludge, varnish, and cam and lifter wear than did the combination of leaded gasoline and "standard" oil-change interval.

Subsequent to these findings, both General Motors and Chrysler recommended lengthened periods between oil changes.

Gergel and Sheahan (1976), of the Lubrizol Corporation, reported results similar to those of Pless, but did not find any significant plugging of oil filters. They concluded that engine wear was the limiting factor in extending oil change intervals, suggesting a maximum of 12,000 miles between changes for unleaded gasoline engines.

The evidence indicates that there is a relationship between lead additives and oil change intervals shown through reduction in engine and engine-parts wear (from reduced abrasive lead particles or reduced rust), oil degradation, and general engine and engine-part cleanliness (e.g., lack of deposits and sludge). One indication of this relationship is the fact that manufacturers' recommended intervals between oil changes have more than doubled since the introduction of unleaded gasoline, and in a recent statement to EPA the MVMA stated that using unleaded gasoline allows the doubling of oil change intervals. (Some of the increase, however, has reflected improved oil quality.)

To estimate the benefits of increased oil change intervals, we assumed an interval of 5,000 miles with leaded gasoline (at 2.3 gplg) and, following Pless' results, a doubling of the interval to 10,000 miles with unleaded. Based on a cost per oil

change of $10.50,* the estimated benefit is then (1/5,000 - 1/10,000) ($10.50) = $0.00105/mile. The annual benefit to a car owner would be about $10.50, based on 10,000 miles per year.

As before, this estimate is based on changing from gasoline containing about 2.3 gplg of lead to gasoline with no lead. To estimate what the benefits would be in going from 1.1 gplg to lower levels, we relied on Cordera et al. (1964), who examined the effects on engine rust of varying concentrations of lead (and its scavengers, EDB or EDC) in gasoline. They evaluated valve-lifter rusting at O, 0.53, and 3.2 gplg; rusting decreased nonlinearly with reductions in lead (and its scavengers), with the sharpest declines occurring at low lead levels. Fitting a smoothed curve to their data suggests that about 12.7 percent of the total reduction in rusting would occur in going from 2.3 gplg to 1.1 gplg, leaving 87.3 percent, or 0.873($0.00105/mile) = $0.00092/mile in benefits for switching from leaded at 1.1 gplg to unleaded. Based on that same curve, we estimated that going from 1.1 to 0.10 gplg yielded 58.3 percent of the total benefit, or 0.583(0.00105) = $0.00061/mile.

VII.A.4. Summary of Maintenance Benefits

Table VII-2 summarizes our maintenance estimates on a per mile basis. They total $0.00038/mile for changing from 1.1 gplg

* This assumes four quarts of oil at $1.50 each, plus half an oil filter (assuming the filter would be replaced every other oil change) at $4 each, plus 15 minutes of labor at $10 per hour.

TABLE VII-2. Estimated Maintenance Benefits Per Mile (cents/mile)

Category	Standard (gplg)		
	0.50	0.10	0
Exhaust systems	0.000	0.096	0.120
Spark plugs	0.017	0.017	0.017
Oil changes	0.021	0.061	0.092
Total	0.038	0.174	0.229

to 0.5 gplg, $0.00174/mile for tightening to 0.10 gplg, and $0.00229/mile for eliminating lead altogether.

To calculate the benefits in each year, we combined those estimates with estimates from our fleet model of the numbers of miles driven by light-duty vehicles of different types. For 1986, for example, we estimate that legal leaded users of light-duty vehicles will travel 307 million miles and that misfuelers will travel 174 million miles. For a standard of 0.1 gplg, assuming that it eliminates all misfueling, the estimated benefit is then ($0.00174/mile)(307 million miles) + ($0.00229/mile)(174 million miles) = $933 million. Table VII-3 presents year-by-year estimates for the three alternative schedules presented in earlier chapters. As before, the estimates assume that all misfueling is eliminated; alternative assumptions are explored in Chapter VIII.

Note that these monetized estimates of maintenance savings apply only to light-duty vehicles (cars and light-duty trucks), because we did not have data on such savings for other classes of vehicles, such as heavy-duty trucks and busses. It is likely, however, that such vehicles would also reap maintenance savings. In 1986, we estimate that they will account for about one-quarter of the demand for leaded gasoline; their share will grow in later years. Consequently, these estimates understate the benefits in these categories.

TABLE VII-3. Year-by-Year Estimates of Maintenance Benefits, Assuming No Misfueling (millions of 1983 dollars)

Category / Rule	1985	1986	1987	1988	1989	1990	1991	1992
Spark Plugs								
Proposed	0	84	77	73	69	67	65	64
Alternative	46	84	77	73	69	67	65	64
Final	46	84	77	73	69	67	65	64
Exhaust Systems								
Proposed	0	503	473	450	433	422	415	412
Alternative	95	356	411	450	433	422	415	412
Final	95	503	473	450	433	422	415	412
Oil Changes								
Proposed	0	347	330	318	309	303	301	301
Alternative	112	267	287	318	309	303	301	301
Final	112	347	330	318	309	303	301	301
Total								
Proposed	0	933	880	840	811	792	780	776
Alternative	252	706	775	840	811	792	780	776
Final	252	933	880	840	811	792	780	776

VII.B. Improved Fuel Economy

Reducing the lead content of gasoline should improve fuel economy in three ways: by increasing the energy content of gasoline through more intense processing, by reducing the fouling of oxygen sensors in misfueled late-model vehicles, and by reducing the fouling of spark plugs. Energy content and oxygen sensor benefits are discussed and monetized below. The third source of benefits was covered, at least in part, by our estimate of increased intervals between spark plug changes, and hence is not monetized in this section.

VII.B.1. Energy Content

Increased reforming and isomerization of gasoline to replace the octane lost through lead reductions increases the density (energy content) of gasoline. Unleaded gasoline also generates more deposits in engine combustion chambers, which increases compression and engine efficiency slightly. Exxon (1978) has estimated that these effects could cause a 1 to 1.5 percent improvement in fuel economy.

To estimate the benefits of increased fuel economy from denser gasoline, we computed the changes in density predicted by the DOE refinery model at different lead levels. Because the predicted change represents a relatively small difference between two estimated large numbers and depends on the precise methods used by refiners to raise octane, these estimates are subject to substantial uncertainty. We used a formula developed by the Society of Automotive Engineers (1979) to estimate the change in fuel efficiency as a function of density. Finally, we multiplied

the estimated savings by the retail price of a gallon of gasoline. (For this calculation, we used the retail price, $1.10 per gallon in 1983 dollars, rather than the refinery gate price because a reduction in gasoline consumption -- which would result from greater fuel economy -- yields savings in distribution and retailing costs, as well as refining costs.)

VII.B.2. Reduced Fouling of Oxygen Sensors

For vehicles with oxygen sensors and closed-loop catalyst systems, reducing lead in gasoline offers additional gains in fuel efficiency to the extent that it reduces misfueling. In such vehicles, lead fouls the oxygen sensor, thus reducing its ability to optimize engine performance for maximum fuel economy. In a recent paper, Armstrong (1984) presented data showing that replacing the oxygen sensor as well as the catalyst in a misfueled vehicle reduced hydrocarbon emissions, indicating that leaded gasoline causes the oxygen sensor to require a fuel mixture that is too rich. She found that replacing the oxygen sensor reduced tailpipe emissions by an average of 0.13 grams per mile. Because the catalyst oxidizes most of the extra hydrocarbons that the engine wasted with a lead-fouled sensor, it is necessary to divide that number by (1 - catalyst efficiency) to estimate the reduction in wasted hydrocarbons. In Armstrong's sample, the average efficiency of the catalysts was 83.4 percent, so the excess consumption of hydrocarbons in the misfueled vehicles was 0.13/(1 - 0.834) = 0.783 grams per mile. If the sensor is functioning properly, these hydrocarbons are burned in the engine, increasing fuel economy.

To estimate the benefits associated with reduced hydrocarbon consumption, we estimated the number of post-1981, sensor-equipped vehicles that would be misfueled for the first time in each year from 1985 through 1992. We then computed the discounted (at a real rate of 10 percent) number of miles that such vehicles would travel, on average, over their remaining lifetimes, multiplied by 0.783 grams per mile and converted the resulting grams of hydrocarbon to equivalent gallons of gasoline. Finally, we multiplied total gallons of gasoline for each year by the price of gasoline ($1.10).

VII.B.3. Summary of Fuel Economy Benefits

Table VII-4 presents the year-by-year estimates of fuel economy benefits, assuming, as in past chapters, no misfueling. The estimates are dominated by the savings due to higher fuel density. These savings fall over time because as demand for leaded fuel declines, the baseline fuel density rises. The savings due to reduced oxygen sensor fouling increase over time because the number of misfueled post-1981 vehicles would grow in the absence of the new rule.

VII.C. Engine Durability

Lead in gasoline can have both positive and negative effects on the durability of engines. The primary concern with unleaded gasoline has been premature valve-seat wear in engines designed to use leaded gasoline. Such effects have been demonstrated in laboratory and track tests, although tests of vehicles in normal use have failed to find any significant acceleration of valve-seat recession. Section VII.C.1 focuses on an evaluation of valve

TABLE VII-4. Year-by-Year Estimates of Fuel Economy Benefits, Assuming No Misfueling (millions of 1983 dollars)

Category / Rule	1985	1986	1987	1988	1989	1990	1991	1992
Fuel Density								
Proposed	0	168	150	97	114	113	148	140
Alternative	57	106	106	97	114	113	148	140
Final	60	168	150	97	114	113	148	140
Oxygen Sensors								
Proposed	0	22	25	27	30	32	34	35
Alternative	11	22	25	27	30	32	34	35
Final	11	22	25	27	30	32	34	35
Total								
Proposed	0	190	175	124	144	145	182	175
Alternative	68	128	132	124	144	145	182	175
Final	68	190	175	124	144	145	182	175

seat recession.

Tests indicate that lead and its scavengers can increase the wear of other major engine components, and consequently, shorten the useful lives of engines using leaded gasoline. These negative effects of lead are discussed in section VII.C.2.

VII.C.1. Valve-Seat Recession

We reviewed two types of research in evaluating the potential for valve-seat recession. The first type of study was engine tests on dynamometers, done using either unusually high engine loads to test valve durability, or cycles that simulated typical driving patterns, or a combination of the two. The second type of study involved on-road vehicle tests, ranging from high-load studies to surveys of consumers' experiences. The advantage of engine tests is their greater measurement precision and control over test conditions. The on-road studies, on the other hand, are more likely to reflect "real world" effects.

VII.C.1.a. Laboratory and Track Studies of Valve-Seat Recession

Laboratory studies suggest that exhaust-valve recession results from abrasion and adhesion on the valve seat when engines operate continuously under high temperatures, loads, or speeds. (For detailed discussions of the mechanisms of valve wear, see Godfrey and Courtney, 1971; Giles, 1971; or Kent and Finnigan, 1971.)

Several researchers have examined rates of valve recession as a function of engine operating variables and the amount of lead in the fuel. Giles (1971) and Godfrey and Courtney (1971)

were consistent in finding that recession rates were a function mostly of engine speeds. The shape of this function apparently varied significantly by vehicle models and years.

Table VII-5 summarizes the available laboratory and track studies of valve recession as a function of lead concentrations. Note that most engine studies of valve recession were conducted at speeds and loads much greater than normal driving patterns. For example, Giles and Updike (1971), of TRW's Valve Division, conducted six dynamometer tests simulating vehicle speeds from 50 to 100 mph. These tests, combined with the other evidence, led them to conclude that:

> exhaust valve recession in engine I accelerates rapidly above 70 mph . . . . The data shown here also indicate that the average driver, who seldom exceeds 70 mph, should not experience significant engine deterioration while using lead-free gasoline. The salesman, however, who drives 15,000 turnpike miles per year at 80 mph, may well expect valve train problems. (p. 2369)

Their data showed the rate of valve-lash loss actually decreased slightly between 50 and 70 mph (wide open throttle at 2000 and 2800 rpm, respectively). Felt and Kerley (1971), of Ethyl Corporation, also found that valve recession (using unleaded gasoline) was about two-thirds lower for vehicles traveling at 60 mph than for those traveling at 70 mph, despite going 22 percent to 280 percent more miles.

These studies were designed either to investigate the mechanisms causing exhaust-valve-seat recession, or to show the importance of leaded fuel combustion products in reducing valve wear. They did not usually test for the likelihood of

TABLE VII-5. Summary of Findings of Track and Dynamometer Studies of Lead Levels and Valve Recession

Study	Findings and Conditions
Doelling, 1971	Engine tests at about 65 mph showed that between 0.04 and 0.07 gplg was sufficient protection. Lead levels of 0.14, 0.07, 0.04, and 0.0 gplg.
Felt and Kerley, 1971	Excess wear in continued high speed operation (70-95 mph). Much lower wear rates in intermittent operation using oil with metal additives.
Fuchs, 1971	Engine tests showed 0.5 gplg virtually eliminated valve recession. Lead levels of 0.5 and 0.0.
Giles et al., 1971	Rapid wear on engines with unhardened valve seats at engine speeds typical of 80 mph or greater. Little or no excess wear on unleaded with hardened valve seats at maximum engine speed. Limited testing of heavy-duty truck engines with inserts showed no increased wear.
Giles and Updike, 1971	No excess wear at less than 3000 rpm, excess wear on unhardened valve seats above that. No excess valve wear at 3500 rpm with hardened valve seats.
Godfrey and Courtney, 1971	High load and speed are cause of valve recession on unleaded.
Kent and Finnegan, 1971	High load engine tests showed 0.20 gplg was sufficient protection. Lead levels of 3.0, 0.5, 0.2, and 0.0 gplg.
Pahnke and Bettoney, 1971	High load engine tests showed severe valve recession at 0.0 gplg, none at 0.5 gplg.
U.S. Army, 1971	Dynamometer test of three vehicles and three stationary generators showed no excess valve-seat wear. Generators at maximum rpm and load; had hard valve-seat inserts. Vehicle engines at maximum torque, wide open throttle, and 3200 rpm; one each with unhardened, hardened, and hardened inserts

valve recession under normal driving conditions. In particular, intermittent operation at high speeds may well result in substantially different test patterns than continuous operations. Felt and Kerley, for example, found no significant protective effect of metal additives in engine oil in a continuous high engine speed test, but that metal additives reduced wear rates by a factor of 10 in a test on the same engine with operations alternating between 50 mph and high speed operation.

Overall, the laboratory studies implied that using unleaded gasoline exclusively in vehicles with unhardened valves designed for leaded fuel could risk premature valve failure under severe engine loads. These studies indicated that such severe recession is most likely to occur in engines operating at high loads or speeds, which, for light-duty vehicles, would involve vehicle speeds well above the legal speed limit of 55 mph for extended periods of time (tens or hundreds of hours).

The evidence indicated that conditioning a vehicle on leaded gasoline helped to prevent valve recession during subsequent use of unleaded gasoline for a limited time, but did not lower the longer-term risk. Giles (1971) measured valve wear during and after “break-in” periods of an engine running on leaded gasoline. He demonstrated that recession rates were high initially, even using leaded gasoline. But, as the leaded gasoline combustion products built up on the valve seat, recession rates dropped to very low levels. Giles showed that, after switching the engine to unleaded gasoline, recession rates continued to be low until

the lead deposits wore away (after about 10 hours of high engine speed operation). Recession then rose again to high rates.

Giles and Updike also showed that vehicles with hardened valves had no more wear on unleaded gasoline than vehicles with unhardened valves had on leaded. This result was confirmed by the Army dynamometer studies.

For engines equipped with hydraulic valve lifters (the vast majority of on-highway engines), the amount of valve-seat recession that can be tolerated before serious problems appear is about 0.07 to 0.15 inches. Engines without hydraulic lifters will require adjustment after a much shorter time. The results of the laboratory and test-track studies discussed above indicate that this amount of wear can be experienced in as little as 100 hours of <u>continuous</u> 70 mph freeway driving (7,000 miles) in a light-duty vehicle with unhardened valve seats. At 60 mph (a more typical speed for the present day), this limit could still be reached in as little as 18,000 miles of <u>continuous</u> high speed operation. Miles accumulated at 55 mph and less are unlikely to contribute to seat recession. Heavy-duty gasoline engines, which operate at higher rpm and higher load levels, could be affected even sooner, but most engines of that type have hardened valve seats, or valve-seat inserts, and may have speed governors that restrict engine rpms. These studies were done before modern engine oils, which contain additives to reduce such wear.

Most of the laboratory studies compared valve wear with unleaded gasoline to that with leaded at the levels typical of

the late 1960s and early 1970s (about 2.3 gplg). Several studies, however, also tested the effects on valve wear of using gasoline with a lower lead content. At least four studies concluded that 0.5 gplg of lead would provide sufficient valve-seat protection, even under severe conditions (Kent and Finnigan, 1971; Pahnke and Bettoney, 1971; Felt and Kerley, 1971; Fuchs, 1971). Kent and Finnigan (1971) also found that "as little as 0.2 g/gal of lead was sufficient to reduce wear to substantially zero."

Only one study examined valve wear at very low lead concentrations to discover how little lead was necessary to eliminate valve recession. Doelling (1971) conducted tests at lead levels of 0.04, 0.07, and 0.14 gplg for 100 hours each. Focusing on the maximum recession of any one of the valves, Doelling found no recession at 0.07 or 0.14 gplg, but found excess wear at 0.04 gplg . He thus concluded that leaded gasoline would protect exhaust valves beginning at levels between 0.04 and 0.07 grams of lead per gallon.

Based primarily on concerns raised by these studies, EPA's proposed rule allowed the continued use of low-lead gasoline through the mid-1990s. The standard of 0.10 gplg was chosen to provide a margin of safety in protecting against valve wear.

VII.C.1.b. Fleet Studies of Valve-Seat Recession

The laboratory tests discussed above suggest that premature valve-seat recession in some engines with unhardened valve seats that operate at high speeds could be a serious cost of eliminating lead in gasoline altogether, though it should not be a significant

problem with the 0.10 gplg standard being promulgated. Studies of engines under normal operating conditions, however, suggest that even a ban on lead might not have major impacts on valve-seat durability. The available studies are summarized in Table VII-6.

Several of these fleet studies found little or no incidence of valve-seat problems with unleaded gasoline (Pahnke and Conte, 1969; Orrin et al., 1972; Gray and Azhari, 1972). Other fleet studies were inconclusive concerning the relative incidence of valve-seat problems for unleaded vehicles (Pahnke and Bettoney, 1971; Crouse et al., 1971; Pless, 1974). Wintringham et al. (1972) also noted that reported incidents of valve problems were rare among users of unleaded gasoline in the late 1960s (when at least one major oil company sold a premium unleaded grade). Two studies, however, cited more valve-seat problems for unleaded than for leaded vehicles (Wintringham et al., 1972; Felt and Kerley, 1971). Recently, EPA has become aware of a very large test by the U.S. Army in the mid-1970s, which found no problems using unleaded in a wide range of vehicles. These and other studies are discussed below.

In the middle and late 1960s, Ethyl Corporation carried out an extensive five-year study of leaded versus unleaded gasoline use (Wintringham et al., 1972). This study included 64 matched pairs of cars, owned and driven by Ethyl Corporation employees. One vehicle in each pair used leaded gasoline, the other used unleaded exclusively. The cars averaged more than 15,000 miles per year (an average of 78,749 miles per car for the unleaded

TABLE VII-6. Summary of Findings of Consumer and Fleet Studies of Lead Levels and Valve-Seat Recession

Study	Findings and Conditions
Crouse et al., 1971	A 50,000 mile test of matched pairs found an insignificant decline in valve wear on unleaded. Lead levels of 2.6 and 0.0 gplg.
Crouse et al., 1971	A severe service test using a state police patrol fleet found valve recession after 10 to 15 thousand miles. Lead levels of 3.1 and 0.0 gplg.
Felt and Kerley, 1971	An employee fleet test found more valve problems at 0.0 than at 0.5 gplg.
Gray and Azhari, 1971	No additional valve problems found with employee fleet test or in a consumer survey. Lead levels of 2.8 and 0.0 gplg.
Orrin et al., 1972	No extra valve problems in a study of taxi fleets. Lead levels of 2.8 and O gplg.
Pahnke and Bettoney, 1971	A consumer survey found no clear difference but somewhat more valve problems. Lead levels of 2.3 and O gplg.
Pahnke and Conte, 1969	No additional valve problems for employee cars in personal use. Lead levels of 2.8, 0.1, and 0.0 gplg.
Pless, 1974	No severe valve problems, but some valve-stem wear with unleaded in one of the taxi fleets.
U.S. Army, 197.5	Conversion of six Army bases to unleaded produced a valve recession rate of 1 per 10 million VMT* for commercial vehicles. No valve recession in other vehicles.
U.S. Post Office, 1983	Conversion of 1,562 1975-model Ford trucks with valve seat inserts to unleaded produced valve recession rate of 1 per 15 million VMT*. 152 International Harvester trucks experienced no valve-seat failures on unleaded.
Wintringham et al., 1972	An employee fleet test found more expensive valve problems-with unleaded; about 1 per million VMT*.

*Vehicle miles traveled.

group over the life of the test). At that time, speed limits on the interstate highway system were 65 or 70 miles per hour. Despite this, only four unleaded vehicles (6 percent) required cylinder head replacements. One vehicle in the leaded group also required a new cylinder head during the same period. In addition, the absence of lead showed a beneficial effect in reducing the number of burned and damaged valves -- only six vehicles in the unleaded group required valve jobs, compared with sixteen of the vehicles using leaded gasoline.

Three other studies, conducted about the same time, gave similar results. Gray and Azhari (1972) reported the results of a small fleet test and a consumer use survey, neither of which indicated any particular problems with valve-seat recession. Overall, engine repair costs for the unleaded group were lower than for the leaded group, exactly the opposite of what would have been expected if valve-seat recession were widespread. However, no details of repair records were provided, so the data must be interpreted cautiously.

Crouse et al. (1971) provide data on four cars used in a comparison of leaded and unleaded gasoline effects on lubricants. The cars were operated on a more-or-less normal schedule, involving home-to-work driving on weekends and turnpike driving on weekends. Three cars completed 50,000 miles successfully on this schedule; the fourth suffered from valve-seat recession and had to be dropped from the test after 34,000 miles. None of these cars had operated on anything but unleaded fuel. This is signi-

ficant, since the researchers found that preconditioning on leaded fuel at least doubled the mileage obtained in another test fleet (operating under very severe patrol-service conditions) before valve recession became a problem.

Schwochert (1969) operated an experimental catalyst-equipped car for 50,000 miles on unleaded gasoline in a test cycle that simulated typical city and highway driving (the Auto Manufacturers Association's mileage accumulation cycle). Valve-seat recession in this cycle did not exceed 0.02 inches. Subsequent operation in a very high-speed cycle (70 to 90 mph) destroyed the valve seats in less than 12,000 miles.

All of the tests discussed above involved light-duty vehicles. Heavy-duty vehicles, since they often have lower power-to-weight ratios and higher rpm at highway speeds, may suffer more severely from valve recession with unleaded gasoline if they have unhardened valve seats. These concerns are also applicable to a wide range of farm, construction, and industrial equipment, much of which also operates at high average power ratings and rpm.

In this regard, it is instructive to consider tests conducted by the U.S. Army. These involved some 7,600 vehicles -- including light-duty cars and trucks, heavy-duty trucks, tractors, jeeps, tactical and combat vehicles, and some motorized heavy equipment -- and lasted for three years, with about half of the vehicles being added during the last year. Table VII-7 lists the types of vehicles and other engines involved. The average commercial vehicle in the study accumulated over 10,000 miles per year, and many accumulated more. One class-6 truck put on 34,000 miles in

the first year alone, and several pick-ups accumulated over 30,000 miles in the first year. Military vehicles accumulated lower mileage, but generally operate under high load conditions. The study is documented in a series of reports by the Army Fuels and Lubricants Research Laboratory (Moffit, 1972; Russel and Tosh, 1973; Tosh et al., 1975; Tosh, 1976). Given the the broad assortment and diverse ages of the vehicles involved, it seems likely that many of these vehicles did not have hardened valve seats.

The results of this test were negative -- no untoward maintenance problems that could be attributed to the use of unleaded gasoline were experienced. Overall, an engine failure rate of 0.5 percent was experienced. This rate was stated as being comparable to experience with leaded gasoline. Only three cases of valve-seat recession were reported, all in light-duty vehicles. This is especially significant because the test was conducted before the imposition of the 55 mph speed limit, and many of the posts were located in remote areas, so that considerable highway driving would be expected. The conclusions of the Army study are worth quoting:

> From the evaluation results, it can be concluded that commercial, tactical and combat vehicles, and all other equipment used in this program can operate satisfactorily during their normal day-to-day activities without any fuel economy penalties and with no apparent increases in vehicle maintenance or operating costs so long as unleaded gasoline meeting VV-G-00169A Federal specification is used. (Tosh, 1976, p. 34; emphasis in original)

The Federal specification cited is essentially that for present-day commercial unleaded gasoline.

TABLE VII-7 Vehicle and Engine Types in U.S. Army Unleaded Gasoline Test

Commercial Vehicles		Tactical Vehicles	
Cars	445	1/4 ton trucks*	2785
		3/4 ton trucks	8
Light-Duty Trucks		1 1/4 ton trucks	919
		Other tactical trucks	83
0-6,000 pounds	1,003		
6,000-10,000 pounds	429		
Medium and Heavy-Duty Trucks			
10,000-14,000 pounds	68		
14,000-16,000 pounds	57		
16,000-19,500 pounds	163		
19,500-26,000 pounds	43		
26,000-33,000 pounds	63		
33,000 pounds plus	28		
Unclassified cars and trucks	411		
Buses	87		
Tractors	84		
Construction and Other Equipment			
Cranes	38		
Graders	5		
Fork Lifts	256		
Generators	527		
Miscellaneous Construction Equip.	255		
Other Vehicles and Engines			
Scooters	40		
Outboard motors	41		
Lawn mowers	225		
Motorcycles	7		

*These vehicles did not have hardened valve seats as of 1971; status of valve seats for other tactical vehicles unsure.

In 1975, shortly after this test, all branches of the U.S. Armed Services converted completely to unleaded gasoline wherever it was available. A monitoring system was set up to detect subsequent problems and no special vehicle maintenance or other problems were experienced since this conversion (M. DePara, U.S. Army, Belvoir Research and Development Center, personal communication).

Data provided by the U.S. Postal Service tell a very similar story for heavy-duty trucks in their service. The Postal Service has operated some 1,562 1975-model Ford heavy-duty trucks (22,000 pounds) on unleaded gasoline since 1980. These trucks were originally purchased in 1975, and travel about 50,000 miles per year, on average. By 1980, most of them were on their second or third engine rebuild or replacement, so that there was a wide variety of engine mileages -- from zero to about 100,000 miles -- represented in the fleet. All of the new and rebuilt engines in the fleet used hardened valve seat inserts (as do most heavy-duty trucks).

In the 42 months or so since switching to unleaded, the Postal Service has recorded 69 instances of valve problems (a valve failure rate of 4.4 percent) and 18 cases of valve-seat problems (a failure rate of 1.2 percent), while operating these trucks for an average of about 175,000 miles each on unleaded gasoline. This would normally include at least one full engine rebuild (M. Sanders, U.S. Postal Service, personal communication). For comparison, Ford indicated that its warranty data for the same types of engines -- presumably run primarily on leaded fuel --

showed higher valve and cylinder head failure rates (Ford Automotive Emissions Office, personal communication). The Postal Service has experienced no significant mechanical or operating problems as a result of using lead-free gasoline in its fleet.

VII.C.1.c. Other Types of Engines

The studies described above generally involved on-road vehicles (cars and trucks), although the Army study also included some construction equipment, stationary generators, motorcycles, and outboard engines. To investigate possible valve-seat damage in smaller engines, such as those used in lawnmowers, snowblowers, garden tillers, and snowmobiles, we contacted three manufacturers of small engines (Briggs, Tecumseh, and Kohler). All said either that their engines could almost always use either leaded or unleaded, or that they specifically recommend unleaded. Representatives from these companies also stated that they believed that this would be true for all of the engines that they had manufactured for at least the last 10 years, and were not aware of any design changes that would have made this untrue even for earlier engines.

Marine engines are generally of two types: inboard and outboard. Inboard engines typically are adapted from automobile or truck engines, so we would expect the data on light-duty vehicles to apply to them as well. Outboard engines are almost all two-stroke engines, for which the fuel is mixed with a special type of oil. For most such engines, unleaded does not appear to cause any serious problems; however, for high-output two-stroke

engines (125 hp and above), cylinder-wall scoring and premature bearing failures can occur (Weaver, 1984b). Several solutions are possible for this problem, if lead is banned. One would be to allow lead to remain in gasoline for marine use. The other would be to allow lead or another additive to be added to the oil that is mixed with gasoline for two-stroke engine use.

Another engine class of potential concern is farm equipment. Although diesel engines now dominate the market for tractors and other large pieces of farm equipment, there are many older gasoline-powered engines still in use on farms. The Army tests involved some farm as well as non-farm tractors that are likely to be used under conditions similar to those on farms. That study also included portable generators, which should be similar to many small engines used on farms to power stationary equipment. To the extent that the Army data are applicable, it seems that the Agency's action should not have a significant impact on the durability of engines used on farms.

VII.C.1.d. Alternatives to Lead to Avoid Valve Recession

As noted earlier, most engines manufactured over the last decade have used induction-hardened valve seats, hardened valve-seat inserts, or other mechanisms to eliminate potential valve-seat recession problems without lead. It is not feasible to modify existing engines in those ways, however, except during major engine rebuilds. Thus, the most promising way of coping with potential valve-seat durability problems in the total absence of lead would be alternative additives.

Relatively little research has been done on such alternatives, presumably because there is little incentive to develop and market them so long as lead remains available. (Because lead is a relatively cheap octane booster, it is a "free" valve lubricant.) Limited work on the subject, however, suggests several possible alternatives, the most promising of which is phosphorus.

Several experiments suggest that phosphorus in unleaded gasoline could reduce or eliminate the risk of valve recession at high speeds. Specifically, at about 0.06 or 0.07 grams of phosphorus per gallon, valve wear proceeds at one-half to one-third the rate occurring with no additives (Giles and Updike, 1971; Kent and Finnigan, 1971; Felt and Kerley, 1971; Wagner, 1973). The tests were run primarily under unusually high loads or speeds, similar to conditions used in the previously-described studies of valve recession.

Amoco (Wagner, 1971) reported that its road tests of heavily loaded 1970-vintage cars, for 20,000 to 30,000 miles at average speeds of 60 mph (and up to 70 mph), found that 0.07 g/gal of phosphorus was effective in controlling valve recession for nearly 90 percent of the cars tested. The phosphorus more than halved the rates of recession for the cars that, without lead or phosphorus, had experienced sinkage rates of more than 0.01 inches per 10,000 miles. Kent and Finnegan found, however, that at lower load conditions and 2300 rpm for 80 hours, phosphorus was fully protective against any valve-seat widening or oxidation. Cordera et al. (1964) found the presence of phosphorus in the gasoline was critical to exhaust valve-life durability. All of these results

indicated that adding phosphorus to unleaded gasoline would substantially reduce the risk of valve recession for those vehicles at risk. Because phosphorus has a negative impact on catalysts, however, it would be necessary either to have a special grade of unleaded gasoline with phosphorus for older engines, or to make phosphorus available as a separate additive.

In considering a possible ban on leaded gasoline, EPA is soliciting comments on phosphorus and other possible additives to deal with potential valve-seat recession. The Agency is asking for comments also on other possible approaches, such as making leaded gasoline available on a very limited basis (e.g., at marine terminals).

VII.C.2. Negative Effects of Lead on Engine Durability

Wear in engines may be due either to physical processes (abrasive wear) or to chemical effects (corrosive wear). Abrasive wear results from the rubbing contact between two parts. Corrosive wear is a phenomenon akin to engine rusting -- it occurs where chemicals can attack a surface subject to wear, and either dissolve it directly, or combine with it to form a less wear-resistant material.

Lead and its salts are effective solid lubricants. Thus, it might be expected that engine components exposed to lead deposits might suffer less abrasive wear. However, discussions with TRW (H. McCormick, TRW Piston Ring Division, personal communication) indicate that lead deposits may actually increase abrasive wear of piston rings. In addition, the acid combustion products of

lead scavengers contribute to corrosive wear, especially if water is present. Hudnall et al. (1969) have commented that corrosive wear can be much greater than abrasive wear, especially in cold operation. Heavy-duty engines, however, which are less subject to rusting due to their higher operating temperatures, also suffer less from corrosive wear (Hudnall et al., 1969).

Several investigators have compared the levels of wear observed with leaded and unleaded gasolines. Cordera et al. (1964) compared wear results with the standard scavenger mixture containing both chlorine (from EDC) and bromine (from EDB) with wear using only bromine. They found that eliminating the chlorine reduced wear rates by about 40 percent. In another test, they examined the effects of different lead (and lead-scavenger) concentrations on wear rates. They found that going from 3.0 to 1.5 grams of lead per gallon reduced wear rates by around 40 percent, with a small additional improvement at 0.5 grams per gallon. Going to unleaded gasoline from 0.5 grams actually increased wear rates, although wear was still lower than at 3 grams. Gagliardi and Ghannam (1969) obtained similar results in an 18,000-mile fleet test. They found that piston ring wear was lowest at 0.5 grams per gallon of lead, and increased slightly for both zero and 1.5 grams. Wear at 3 grams per gallon was 70 to 200 percent greater than at 0.5 grams.

The reduction in wear with low-lead gasoline is not surprising, but the observed increase in wear when going from low-lead to unleaded gasoline is. One reasonable explanation for this increase would be the solid lubricating effects of lead deposits

on the cylinder walls, which would be present with low-lead gasoline but not with unleaded. Alternatively, some differences in combustion or lube-oil chemistry due to the presence of lead might account for the difference in wear. The available laboratory data give conflicting impressions as to the degree of increased wear in changing from low-lead to unleaded gasoline. Cordera and coworkers found a rather large increase, while Gagliardi and Ghannem reported only a small effect. To better evaluate the magnitude of this effect in actual use, it is instructive to consider the results of in-use fleet testing.

Orrin et al. (1972) and Carey et al. (1978) have reported the results of two tests of leaded vs. unleaded gasoline in taxi fleets. One fleet operated in Oakland, California for 48,000 miles, and the other operated in Montreal, Canada for 80,000 kilometers. These tests would be expected to favor leaded gasoline. As taxis generally operate nearly continuously for 8 to 24 hours per day, they spend a comparatively small amount of time in warm-up and cold operation -- the conditions that tend to favor corrosion. Despite this, the results of these tests showed a distinct advantage for unleaded gasoline.

In each case, wear measurements in taxis using leaded fuel were 70 to 300 percent greater than those for taxis using unleaded. Piston-ring and cylinder-bore wear, perhaps the most critical areas, ranged from 70 percent to 150 percent greater with leaded than with unleaded fuel. Neither fleet experienced any overt problems with rust, possibly indicating that significant corrosive wear can occur even in the absence of visible rust. Alternatively,

some of the difference might be due to increased abrasive wear due to lead deposits, as suggested by TRW.

These data, which closely match those of Gagliardi and Ghannam (1969) in laboratory tests, appear to indicate that the actual decrease in wear with unleaded gasoline is almost as great as that found with low-lead fuel. Since lead deposits appear to form similarly at 0.1 and 0.5 grams per gallon, it is probable that wear at 0.1 gram per gallon would be similar to or lower than that at 0.5. The data also indicate that the recent reduction to 1.10 grams per gallon should have produced a significant decrease in corrosive wear, at least with regular oil changes. However, oil changes are frequently irregular, and blowby volume in worn engines is much greater than in the new engines on which these test were conducted. Both of these factors would tend to increase corrosive wear rates, even at the current 1.10 gram per gallon level. Thus, either lower-lead (0.5 and 0.1 gplg) or unleaded fuel could be expected to produce a significant reduction in wear rates from those observed with regular leaded gasoline, even at 1.1 gram per gallon.

The economic significance of reduced wear rates would be considerable. At present, worn-out piston rings and cylinder bores are one of the major causes of failure in gasoline engines. They result in poor fuel economy, poor performance, and increased emissions. Repairing this condition requires an engine overhaul, at a cost of $500 to more than $1,000, depending on the engine. Many older vehicles with these problems are simply junked and not repaired.

In new engines, the use of unleaded gasoline can extend piston-ring lives significantly -- by as much as a factor of two (H. McCormick, TRW Piston Ring Division, personal communication). It is not clear, however, how to extrapolate from this finding to estimate the effects on the service lives of engines now in use. If corrosive wear is the major factor in piston-ring wear, then one would expect it to get worse over time as blowby rates increase and, generally, maintenance practices degrade. The general shift to shorter trips and less annual mileage with increasing age would also increase corrosion. Thus, a car that had run for half of the expected lifetime of its piston rings would probably have accumulated somewhat less than half its lifetime wear, and a radical decrease in wear might have more than proportional benefits.

On the other hand, if abrasive wear due to lead deposits is the dominant factor, these deposits would last for some time after the switch to unleaded, and would thus result in less than proportional increases in service life. Overall, the effect of unleaded gasoline in increasing the remaining service life of the piston rings is probably best estimated as being linearly proportional to the remaining life. The typical service life for piston rings in cars using leaded gasoline is about 70,000 to 80,000 miles. A car driven 50,000 miles on leaded gasoline could expect perhaps another 25,000 miles before needing an overhaul. Switching to unleaded would probably increase this by 70 to 150 percent, giving a new expected time-to-overhaul of about 43,000 to 67,000 miles. Since parts other than piston

rings can fail, the actual increase in engine life would probably be closer to 70 percent than 150 percent. Because a substantial part of the oil change benefits that have been monetized may reflect reduced engine wear rather than longer oil change intervals, we have not included any monetized benefits for this category.

VII.C.3. Summary of Engine Durability Effects

We have made no attempt to monetize the potential engine durability benefits or costs of reducing or eliminating lead in gasoline. The net impact of lead on engine durability is unclear. For most engines, it appears that lead does substantially more harm than good. For some, however, lead may play an important role in reducing the risk of valve-seat recession at high loads and speeds, although tests of vehicles in use suggest that few engines need lead under normal operating conditions. It appears that the low valve-seat wear rates in in-use fleets are due both to the low proportion of time spent at high rpm, and the ability of engine oil additives to build up a protective coating during the low rpm use which then protects the engine during intermittent high speed operation. The 0.10 gplg standard provides a margin of safety to protect against potential recession at high loads and speed since it also has been shown to build up a protective layer during low and moderate rpm use.

For the longer run, when EPA proposes to ban lead in gasoline, several solutions may be possible for those few engines that need protection against valve-seat recession. First, phosphorus or some other additive may prove to provide effective protection.

Second, it may be possible to make leaded gasoline available on a very limited basis, so that its use is restricted to those engines that truly need it.

If acceptable alternatives are not developed, the Agency may be forced to accept some increased risk of premature valve-seat recession in some engines as the price of eliminating the severe health and environmental consequences associated with lead in gasoline. As part of its continued deliberations on a possible ban, the Agency will attempt to develop quantitative estimates of the magnitude of this problem.

CHAPTER VIII

COST-BENEFIT ANALYSIS OF ALTERNATIVE PHASEDOWN RULES

EPA considered many alternative phasedown schedules before deciding on the final rule. This chapter compares the costs and benefits of those alternatives, based on the methods and results described in earlier chapters. Section A of this chapter summarizes the estimates of benefits and costs, and compares them to the estimates contained in the Preliminary Regulatory Impact Analysis (RIA) issued when the rule was proposed in August 1984. Section B presents the cost and benefit estimates for different lead standards under various assumptions about the impact of the rule on misfueling. Section C examines the impact of the proposed banking rule on the costs and benefits of the final phasedown rule. Finally, Section D summarizes the conclusions and EPA's rationale for selecting the final phasedown schedule.

Throughout this chapter, we present benefit estimates with and without adult blood-pressure-related benefits. The estimation of a dose-response relationship between blood lead and blood pressure is very recent. The paper presenting those results (Pirkle et al., 1985) has just been published in a peer-reviewed journal. A summary of the results of that study and their application to this rule was also placed in the docket for this rulemaking several weeks before it closed for public comment. Until the scientific community has had an opportunity for more intensive review, however, EPA is not relying on these results to reach final regulatory decisions on lead in gasoline. As the results in this chapter show, these blood-pressure-related benefits

greatly increase the total estimated benefits, but even when they are not included the benefits of the rule exceed the costs by a large margin.

VIII.A. Summary of Cost and Benefit Estimates

Table VIII-1 summarizes the cost and benefit estimates for the 0.10 gplg standard in 1986; these estimates assume that the rule would eliminate all misfueling. As shown in the table, the benefits total $7.9 billion, while the estimated cost is only $607 million, resulting in net benefits of $7.3 billion. About 75 percent of the estimated benefits are attributable to reductions in cardiovascular diseases associated with elevated blood pressure. Even if these benefits associated with adult health are excluded from the calculation, however, the benefits still exceed the costs by more than a three-to-one margin.

Table VIII-1 also presents the cost and benefit estimates contained in the Preliminary RIA that accompanied the proposed rule. The most striking difference, of course, is in adult blood pressure benefits, which were not included in the Preliminary RIA.

All of the other categories show some changes as well, reflecting changes made in response to comments or to newly available information. The higher cost estimates reflect several ‘changes in base-case assumptions. The two most important are: reduced yields from reformers operated at high severity and reduced segregation of naphthas for optimal allocation to processing units. Partly offsetting those changes are the use of newer, more efficient catalysts in FCC units, and lower oil prices, as discussed in Chapter II.

TABLE VIII-1. Costs and Monetized Benefits of 0.10 gplg in 1986, Assuming No Misfueling: Comparison of Current and Draft RIA Estimates (millions of 1983 dollars)

	Current	Draft RIA
MONETIZED BENEFITS		
Children's health effects	602	271
Adult blood pressure	5,927	N.A.
Conventional pollutants	278	348
Maintenance	933	840
Fuel economy	190	360
TOTAL MONETIZED BENEFITS	7,930	1,819
TOTAL REFINING COSTS	607	575
NET BENEFITS	7,323	1,244
NET BENEFITS EXCLUDING BLOOD PRESSURE	1,396	1,244

The category of "Children's Health Effects" is higher for several reasons. First, the CDC recently reduced the blood lead and FEP levels that define lead toxicity from 30 ug/dl to 25 ug/dl; this greatly increases the number of children requiring at least some follow-up medical testing or treatment. Second, in estimating medical costs, we have relied on recently published recommendations that call for more extensive testing and treatment than assumed in the Preliminary RIA. Finally, because of the change in the CDC definition of lead toxicity and the greater weight that the most recent draft of the Lead Criteria Document gives to cognitive effects at blood lead levels in the range of 30 to 50 ug/dl blood lead, we have increased our estimate of the number of children likely to warrant compensatory education in the absence of further reductions in gasoline lead.

As discussed earlier, the estimates of the benefits of reduced emissions of conventional pollutants are the average of two estimation methods: the value implied by the cost of the pollution control equipment destroyed by misfueling and a direct valuation of some of the health and welfare effects associated with these pollutants. (The second method, direct valuation, is based on an incomplete quantification of these health and welfare effects.) The reduction in the overall estimates compared to the Preliminary RIA reflects a decrease in the direct estimate, as discussed in Chapter VI.

The changes in the maintenance estimates reflect refinements in the fleet model used to estimate the number of miles traveled

by different classes of vehicles. The reduction in the fuel-economy estimate is caused by changes in the predicted fuel density, which in part results from changes in the inputs to the refinery model.

Table VIII-2 presents some important non-monetary measures of the estimated benefits of the 0.10 gplg standard in 1986 (again, assuming that the rule eliminates all misfueling). Note that we estimate that the rule will reduce by 172,000 the number of children above the new CDC blood-lead limit of 25 ug/dl. The reductions in the numbers of children at lower, but still possibly harmful, blood-lead levels are even greater; we estimate that 1.7 million fewer children will experience blood lead levels over 15 ug/dl in 1986 as a result of the rule.

If the rule eliminates misfueling, we estimate that it will eliminate over 2.5 million tons of excess emissions of HC, NO_x, and CO. The current estimates are higher than those made in the preliminary RIA because we have used the results of the 1983 EPA tampering and misfueling survey, which show higher rates than the 1982 survey employed earlier, and because of refinements in the fleet model used to estimate the number of miles driven by different classes of vehicles.

Table VIII-2 also reports estimates of the numbers of reduced health effects among adults in 1986. As already discussed, these estimates are restricted to males aged 40 to 59, and the estimates for myocardial infarctions, strokes, and deaths apply only to white males in that age range. Despite these limitations, the estimated benefits are large, ranging from 1.8 million fewer cases of hypertension to 5,160 fewer deaths from all causes.

TABLE VIII-2. Nonmonetary Measures of Health and Environmental Benefits of 0.10 gplg in 1986, Assuming No Misfueling: Comparison of Current and Draft RIA Estimates

	Current	Draft RIA
Reductions in thousands of children above selected blood lead levels		
30 ug/dl	52	52
25 ug/dl	172	172
20 ug/dl	563	563
15 ug/dl	1,726	1,726
Reductions in thousands of tons of pollutants		
Hydrocarbons	305	247
Nitrogen oxides	94	81
Carbon monoxide	2,116	1,646
Reductions in adult male health effects		
Thousands of hypertensives	1,804	N.A.
Myocardial infarctions	5,350	N.A.
Strokes	1,115	N.A.
Deaths	5,160	N.A.

VIII.B. Comparisons of Alternative Lead Levels

In the August 1984 proposal, EPA discussed a range of alternative schedules and presented two specific possibilities: (1) a one-step reduction to 0.10 gplg starting January 1, 1986 and (2) a phasedown with several steps -- 0.50 gplg on July 1, 1985; 0.30 gplg on January 1, 1986; 0.20 gplg on January 1, 1987; and 0.10 gplg on January 1, 1988. The final rule imposes the 0.10 gplg as of January 1, 1986, and also requires a reduction to 0.50 gplg as of July 1, 1985. In addition to these three schedules, however, the Agency considered many other possibilities.

For 1986, the Agency considered levels between 0.1 gplg and 0.5 gplg. Table VIII-3a presents the cost and benefit estimates for those alternatives, assuming in each case that all misfueling would be eliminated. Net benefits are maximized at the tightest of those limits, 0.10 gplg, whether or not adult blood pressure benefits are included. Table VIII-3b shows the estimates assuming that the rule fails to have any impact on misfueling; again, net benefits are maximized at 0.10 gplg.

Neither of these two extreme assumptions about misfueling (that it will be eliminated entirely or that it will continue unabated, even at very low lead levels) appears realistic. EPA believes it is more likely that the number of misfuelers is a declining function of the lead level, primarily because the manufacturing cost differential between leaded and unleaded declines and then reverses as the lead limit is tightened. We expect changes in manufacturing costs to be at least partly reflected in retail prices. Although we cannot be certain that

TABLE VIII-3a. Costs and Monetized Benefits of Alternative Lead Levels in 1986, Assuming No Misfueling (millions of 1983 dollars)

	Lead Level (gplg)				
	0.50	0.40	0.30	0.20	0.10
MONETIZED BENEFITS					
Children's health effects	466	504	539	571	602
Adult blood pressure	4,018	4,483	4,955	5,436	5,927
Conventional pollutants	278	278	278	278	278
Maintenance	517	608	706	808	933
Fuel economy	119	119	128	136	190
TOTAL MONETIZED BENEFITS	5,398	5,992	6,606	7,229	7,930
TOTAL REFINING COSTS	243	305	386	472	607
NET BENEFITS	5,155	5,687	6,220	6,757	7,323
NET BENEFITS EXCLUDING BLOOD PRESSURE	1,137	1,204	1,265	1,321	1,396

TABLE VIII-3b. Costs and Monetized Benefits of Alternative Lead Levels in 1986, Assuming Full Misfueling (millions of 1983 dollars)

	Lead Level (gplg)				
	0.50	0.40	0.30	0.20	0.10
MONETIZED BENEFITS					
Children's health effects	403	455	504	550	592
Adult blood pressure	3,328	3,920	4,526	5,144	5,778
Conventional pollutants	0	0	0	0	0
Maintenance	186	329	482	642	838
Fuel economy	44	97	97	115	177
TOTAL MONETIZED BENEFITS	3,961	4,801	5,609	6,451	7,385
TOTAL REFINING COSTS	178	260	350	460	627
NET BENEFITS	3,783	4,541	5,259	5,991	6,758
NET BENEFITS EXCLUDING BLOOD PRESSURE	455	621	733	847	980

unleaded will be priced below leaded at the retail level even if its manufacturing cost is lower, we do expect the price differential between leaded and unleaded to decline and that the decline will cause some misfuelers to switch to unleaded gasoline.

Figure VIII-1 plots three possibilities for how the percentage of current misfuelers might decline as a function of the lead limit. Each of the three curves assumes that 50 percent of misfueling would be eliminated at 0.25 gplg, the estimated point at which the manufacturing costs of leaded and unleaded intersect. They differ, however, in the assumed rates at which misfueling changes. Curve A assumes that misfueling declines linearly from 0.50 gplg to 0 gplg; at 0.10 gplg, misfueling is 20 percent of its base level. Curve B also assumes that the decline in misfueling begins at 0.50 gplg and ends at 0 gplg, but that the rate of decline is most rapid over the intermediate range. Curve C is similar to B, but the change is compressed to the range 0.40 gplg to 0.10 gplg; it assumes no misfueling at 0.10 gplg.

Figure VIII-2 plots the net benefits of the alternative lead limits for all five assumptions about misfueling: no misfueling, full misfueling, and the three intermediate cases. In all five cases, not surprisingly given the results in Tables VIII-3a and VIII-3b, the net benefits peak at 0.10 gplg. Figure VIII-3 presents similar estimates, but excludes the adult blood pressure benefits; again, net benefits peak at 0.10 gplg, though they are much smaller than with the inclusion of the adult health effects.

It is impossible to determine which assumption about

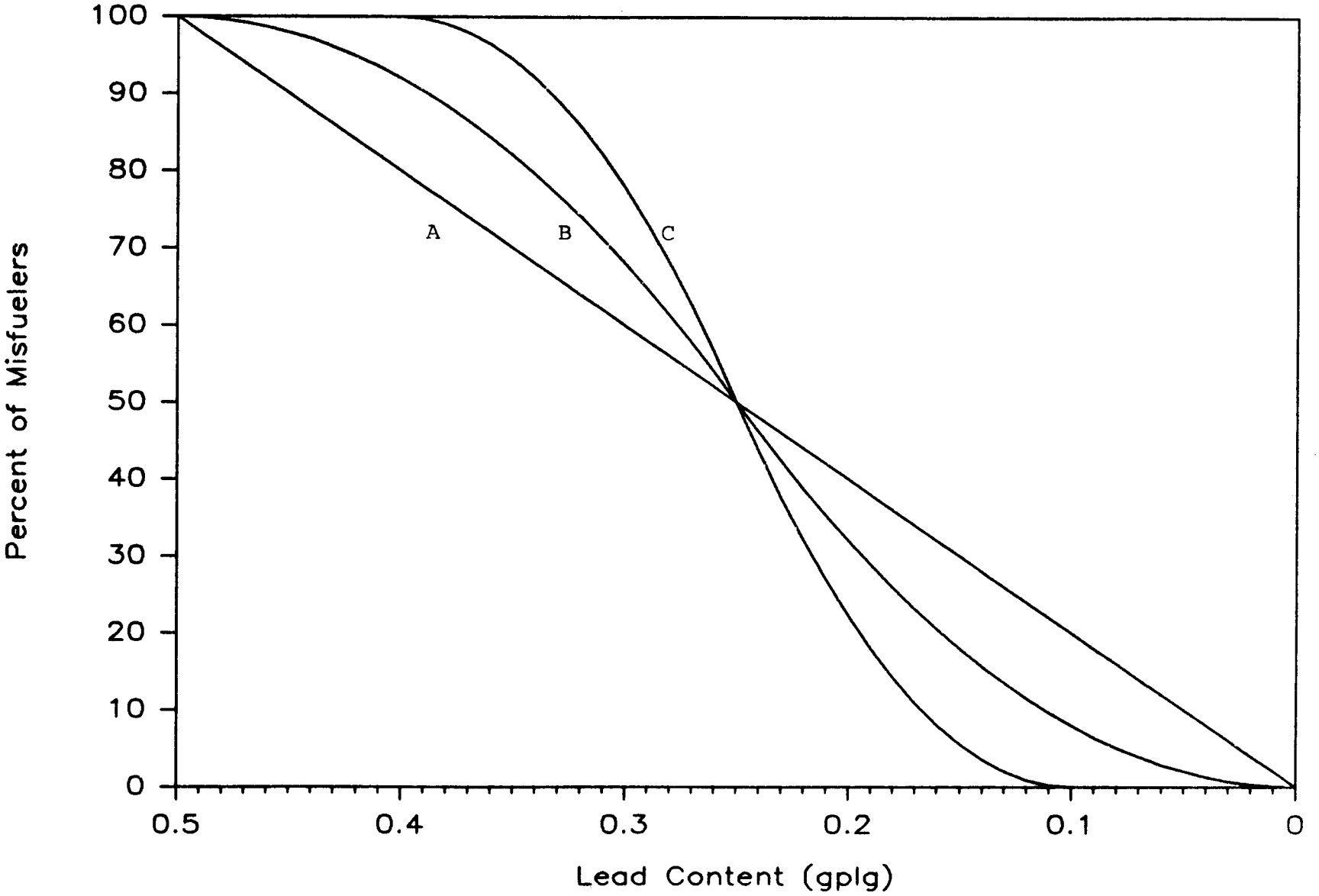

FIGURE VIII-1. Impact of Lead Levels on Misfueling Under Three Assumptions

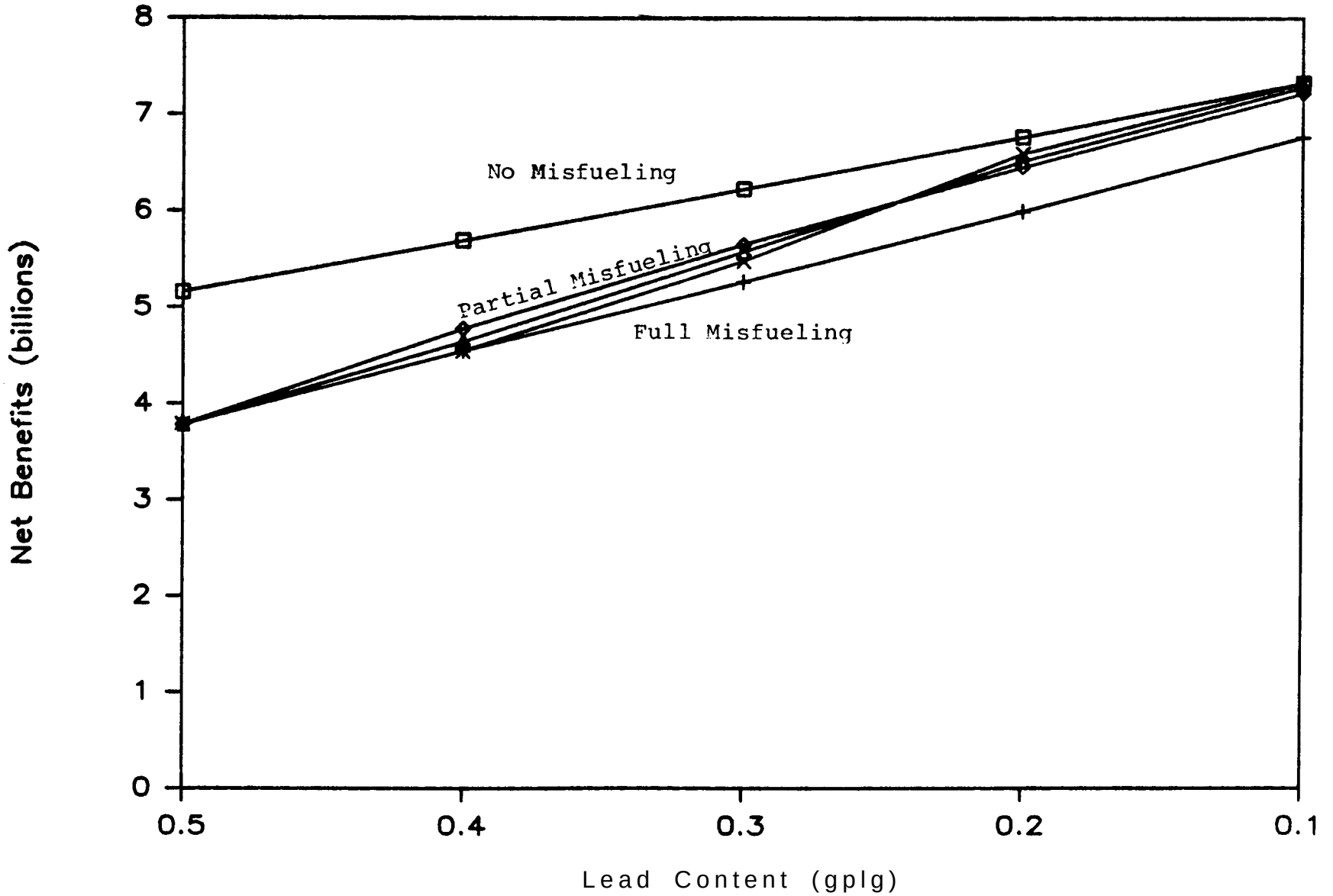

FIGURE VIII-2. Net Benefits (Including Blood-Pressure-Related Effects) as Functions of Lead Level and Misfueling

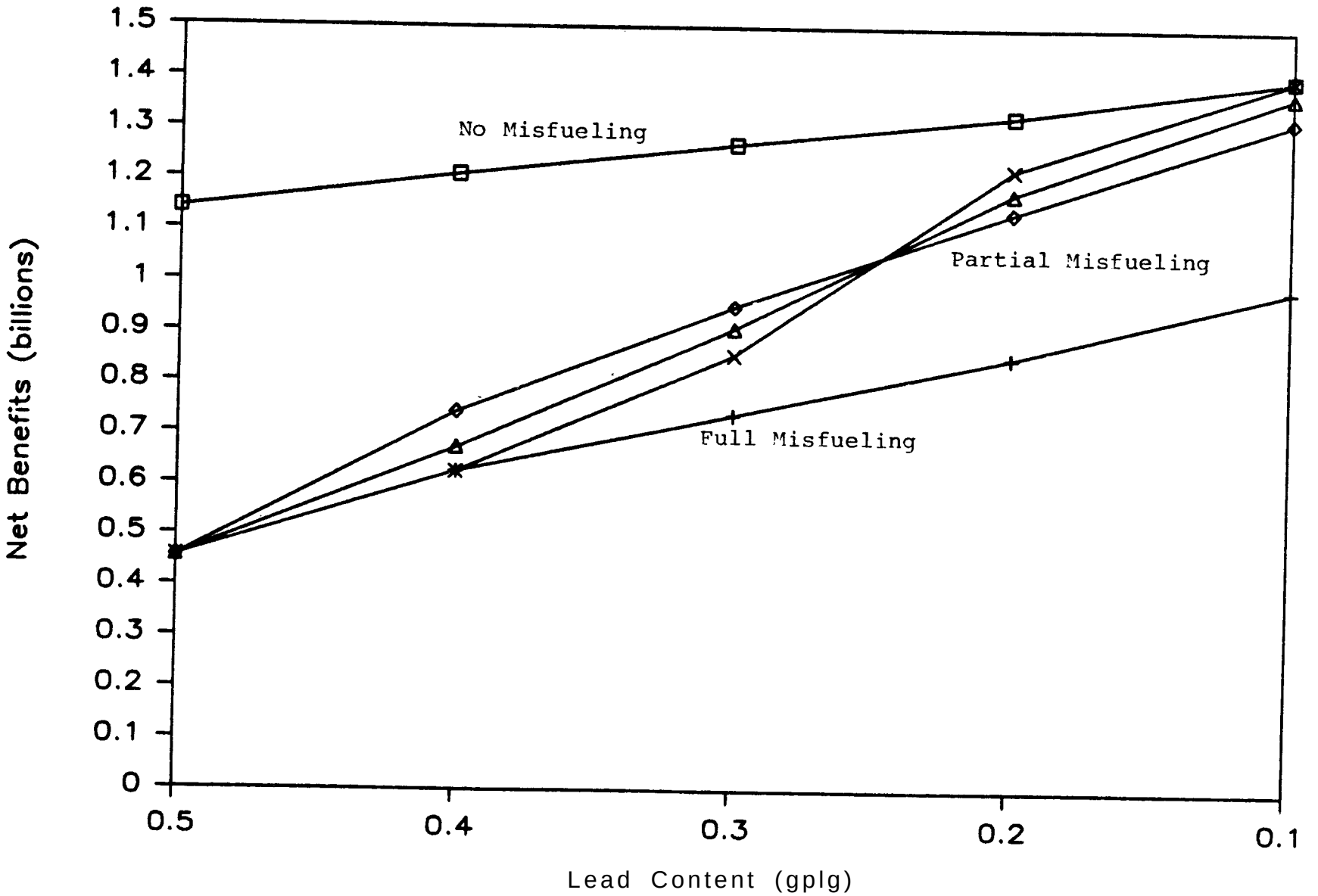

FIGURE VIII-3. Net Benefits (Excluding Blood-Pressure-Related Effects) as Functions of Lead Level and Misfueling

misfueling is most accurate. To do so would require knowing how gasoline retail prices will respond to changes in production costs, and how misfuelers will respond to changes in prices. Thus, most of our estimates in this chapter and the next one present both extreme possibilities with respect to misfueling.

It is useful, however, to have a standard "partial misfueling" case for making comparisons. For that case, we have settled on the simplest of the three curves in Figure VIII-1, curve A, which assumes that misfueling declines linearly from 100 percent of its current level at 0.50 gplg and above, to zero at O gplg. Under that assumption, 20 percent of the misfuelers continue to use leaded gasoline at 0.10 gplg. We believe this is a reasonable estimate, as some gasoline stations are likely to continue to sell leaded gasoline at a lower price than unleaded, and some misfuelers may continue to buy leaded even if it costs more than regular unleaded, either because they desire higher octane or because they mistakenly believe that leaded gasoline is better for their engines. Table VIII-3c presents the cost and benefit estimates under this assumption, i.e., that misfueling declines from 100 percent of its current level at 0.50 gplg to 20 percent at 0.10 gplg.

For 1987, EPA considered two alternative levels: 0.20 gplg and 0.10 gplg. Tables VIII-4a through VIII-4c present the cost and benefit estimates for the no-misfueling, full-misfueling, and partial-misfueling cases, respectively. As in the earlier tables, net benefits are maximized at 0.10 gplg, whether or not adult blood pressure benefits are included.

TABLE VIII-3c. Costs and Monetized Benefits of Alternative Lead Levels in 1986, Assuming Partial Misfueling (millions of 1983 dollars)

	Lead Level (gplg)				
	0.50	0.40	0.30	0.20	0.10
MONETIZED BENEFITS					
Children's health effects	403	465	518	563	600
Adult blood pressure	3,328	4,033	4,698	5,319	5,897
Conventional pollutants	0	56	111	167	222
Maintenance	186	385	572	742	914
Fuel economy	44	101	109	128	187
TOTAL MONETIZED BENEFITS	3,961	5,039	6,008	6,918	7,821
TOTAL REFINING COSTS	178	269	364	467	608
NET BENEFITS	3,783	4,770	5,643	6,451	7,213
NET BENEFITS EXCLUDING BLOOD PRESSURE	455	738	946	1,131	1,316

TABLE VIII-4a. Costs and Monetized Benefits of Alternative Lead Levels in 1987, Assuming No Misfueling (millions of 1983 dollars)

	Lead Level (gplg)	
	0.20	0.10
MONETIZED BENEFITS		
Children's health effects	522	550
Adult blood pressure	5,262	5,707
Conventional pollutants	278	278
Maintenance	775	880
Fuel economy	132	175
TOTAL MONETIZED BENEFITS	6,968	7,590
TOTAL REFINING COSTS	452	553
NET BENEFITS	6,516	7,037
NET BENEFITS EXCLUDING BLOOD PRESSURE	1,255	1,330

TABLE VIII-4b. Costs and Monetized Benefits of Alternative Lead Levels in 1987, Assuming Full Misfueling (millions of 1983 dollars)

	Lead Level (gplg)	
	0.20	0.10
MONETIZED BENEFITS		
Children's health effects	501	539
Adult blood pressure	4,940	5,543
Conventional pollutants	0	0
Maintenance	596	777
Fuel economy	106	150
TOTAL MONETIZED BENEFITS	6,143	7,009
TOTAL REFINING COSTS	441	578
NET BENEFITS	5,702	6,431
NET BENEFITS EXCLUDING BLOOD PRESSURE	762	888

TABLE VI II-4c. Costs and Monetized Benefits of Alternative Lead Levels in 1987, Assuming Partial Misfueling (millions of 1983 dollars)

	Lead Level (gplg)	
	0.20	0.10
MONETIZED BENEFITS		
Children's health effects	513	547
Adult blood pressure	5,133	5,675
Conventional pollutants	167	222
Maintenance	703	859
Fuel economy	122	170
TOTAL MONETIZED BENEFITS	6,638	7,474
TOTAL REFINING COSTS	448	558
NET BENEFITS	6,191	6,916
NET BENEFITS EXCLUDING BLOOD PRESSURE	1,058	1,241

For 1985, EPA considered five alternative levels: 1.10 gplg (i.e., no change), 0.80 gplg, 0.70 gplg, 0.60 gplg, and 0.50 gplg. Table VIII-5 presents the estimated costs and benefits, assuming full misfueling. (Estimates are not presented for the no-misfueling case, as we doubt that standards above 0.50 gplg will have enough impact on the price of leaded gasoline to make a significant difference in misfueling.) Note that the estimates cover only half a year, as none of the standards considered for 1985 would take effect until the middle of that year (July 1). Again, net benefits are maximized at the tightest standard discussed in the August proposal, 0.50 gplg.

The net benefits of the 0.50 gplg standard in 1985 are substantial: $264 million if blood-pressure-related benefits are not included, and $2.0 billion if they are. Moreover, as discussed in Chapter II, all available measures indicate that the refining industry can comply easily with that portion of the rule; reducing lead to 0.50 gplg should require minimal adjustments in refinery operations.

Table VIII-6 compares the present values of the costs and benefits of three phasedown schedules over the period 1985-1987: the original primary proposal; the more gradual, illustrative phasedown presented in the Notice of Proposed Rulemaking; and the schedule in the final rule. In all cases, the costs and benefits have been discounted at 10 percent (real) to the beginning of 1985. (The 1985 estimates were discounted for half a year, the 1986 benefits for a full year, and the 1987 benefits for two years.) All of the schedules yield substantial

TABLE VIII-5. Costs and Monetized Benefits of Alternative Lead Levels in 1985, Assuming Full Misfueling (millions of 1983 dollars)

	Lead Level (gplg)			
	0.80	0.70	0.60	0.50
MONETIZED BENEFITS				
Children's health effects	124	159	193	223
Adult blood pressure	837	1,126	1,423	1,724
Conventional pollutants	0	0	0	0
Maintenance	37	54	80	102
Fuel economy	-5	0	31	35
TOTAL MONETIZED BENEFITS	993	1,339	1,727	2,084
TOTAL REFINING COSTS	44	56	75	96
NET BENEFITS	949	1,283	1,652	1,988
NET BENEFITS EXCLUDING BLOOD PRESSURE	112	157	229	264

TABLE VIII-6. Present Values of Costs and Monetized Benefits: Comparison of Proposed, Alternative, and Final Schedules for 1985-1987 (millions of 1983 dollars)

	Proposed	Alternative	Final
With No Misfueling			
Costs	1,009	845	1,130
Benefits	13,482	14,377	16,095
Net benefits	12,473	13,532	14,965
Net benefits, excluding blood pressure	2,368	2,743	2,924
With Full Misfueling			
Costs	1,048	774	1,139
Benefits	12,506	12,160	14,490
Net benefits	11,459	11,386	13,351
Net benefits, excluding blood pressure	1,625	1,547	1,876
With Partial Misfueling			
Costs	1,014	793	1,105
Benefits	13,287	12,932	15,271
Net benefits	12,273	12,139	14,166
Net benefits, excluding blood pressure	2,222	1,985	2,473

net benefits, in excess of $11 billion with blood-pressure-related benefits and over $1.5 billion without them. Compared to the other schedules, the final rule has higher costs but even higher benefits (whether or not blood-pressure-related benefits are included), with the result that it has the highest net benefits of the three schedules.

Tables VIII-7a through VIII-7c present year-by-year estimates of the costs and benefits of the final rule over the period 1985 to 1992, under the three assumptions about misfueling. The costs fall from 1986 to 1992 because we project that the demand for leaded gasoline would fall even in the absence of the rule, as a result of retirement of older cars. For that same reason, most of the estimated annual benefits also decline over time. The major exception is conventional pollutants, because we expect the amount of misfueling to increase in the absence of the rule, as the number and average age of catalyst-equipped vehicles increase.

Table VIII-8 shows the present values of the final rule under the different assumptions about misfueling. The estimated net benefits, not including blood-pressure-related benefits, range from $4.1 billion if the rule has no impact on misfueling, to $6.7 billion if the rule eliminates all misfueling. Under the more realistic “partial misfueling” assumption, the present value of the net benefits is $5.9 billion. If the adult blood pressure benefits are included, the present value of the net benefits is much higher, $33.4 billion under the partial misfueling assumption.

TABLE VIII-7a. Year-by-Year Costs and Monetized Benefits of Final Rule, Assuming No Misfueling (millions of 1983 dollars)

	1985	1986	1987	1988	1989	1990	1991	1992
MONETIZED BENEFITS								
Children's health effects	251	602	550	504	455	417	371	361
Adult blood pressure	2,033	5,927	5,707	5,484	5,227	5,008	4,722	4,736
Conventional pollutants	140	278	278	280	282	288	299	310
Maintenance	252	933	880	840	811	792	780	776
Fuel economy	68	190	175	124	144	145	182	175
TOTAL MONETIZED BENEFITS	2,744	7,930	7,590	7,232	6,919	6,649	6,354	6,358
TOTAL REFINING COSTS	127	607	553	530	502	468	442	440
NET BENEFITS	2,617	7,323	7,037	6,702	6,417	6,181	5,912	5,918
NET BENEFITS EXCLUDING BLOOD PRESSURE	584	1,396	1,330	1,218	1,190	1,174	1,190	1,182

TABLE VIII-7b. Year-by-Year Costs and Monetized Benefits of Final Rule, Assuming Full Misfueling (millions of 1983 dollars)

	1985	1986	1987	1988	1989	1990	1991	1992
MONETIZED BENEFITS								
Children's health effects	223	592	539	494	445	406	361	350
Adult blood pressure	1,724	5,778	5,543	5,303	5,031	4,798	4,521	4,512
Conventional pollutants	0	0	0	0	0	0	0	0
Maintenance	102	838	777	730	694	668	650	640
Fuel economy	35	177	150	70	96	113	131	122
TOTAL MONETIZED BENEFITS	2,084	7,385	7,009	6,597	6,266	5,985	5,663	5,624
TOTAL REFINING COSTS	96	627	578	539	514	485	451	443
NET BENEFITS	1,988	6,758	6,431	6,058	5,752	5,500	5,212	5,181
NET BENEFITS EXCLUDING BLOOD PRESSURE	264	980	888	755	721	702	691	669

TABLE VIII-7c. Year-by-Year Costs and Monetized Benefits of Final Rule, Assuming Partial Misfueling (millions of 1983 dollars)

	1985	1986	1987	1988	1989	1990	1991	1992
MONETIZED BENEFITS								
Children's health effects	223	600	547	502	453	414	369	358
Adult blood pressure	1,724	5,897	5,675	5,447	5,187	4,966	4,682	4,691
Conventional pollutants	0	222	222	224	226	230	239	248
Maintenance	102	914	859	818	788	767	754	749
Fuel economy	35	187	170	113	134	139	172	164
TOTAL MONETIZED BENEFITS	2,084	7,821	7,474	7,105	6,788	6,517	6,216	6,211
TOTAL REFINING COSTS	96	608	558	532	504	471	444	441
NET BENEFITS	1,988	7,213	6,916	6,573	6,284	6,045	5,772	5,770
NET BENEFITS EXCLUDING BLOOD PRESSURE	264	1,316	1,241	1,125	1,096	1,079	1,090	1,079

TABLE VIII-8. Present Values of Costs and Benefits of Final Rule, 1985-1992 (millions of 1983 dollars)

	No Misfueling	Full Misfueling	Partial Misfueling
MONETIZED BENEFITS			
Children's health effects	2,582	2,506	2,546
Adult blood pressure	27,936	26,743	27,462
Conventional pollutants	1,525	0	1,114
Maintenance	4,331	3,634	4,077
Fuel economy	856	643	788
TOTAL MONETIZED BENEFITS	37,231	33,526	35,987
TOTAL REFINING COSTS	2,637	2,678	2,619
NET BENEFITS	34,594	30,847	33,368
NET BENEFITS EXCLUDING BLOOD PRESSURE	6,658	4,105	5,906

VIII.C. Impact of Banking on Costs and Benefits of Final Rule

The analysis thus far in this chapter has assumed that refineries will follow the phasedown schedule being promulgated in this final rule. EPA has proposed the use of "banking" of lead rights during 1985 for use in 1986 and 1987. Under banking, refineries would have the option of using less lead than allowed in 1985 and "banking" it for use in 1986 or 1987. As discussed in Chapter II, this provision would give individual refineries extra flexibility in reducing lead, without increasing the total allowable amount of lead in gasoline between 1985 and 1987. Although refiners would be under no obligation to use the right to bank, EPA expects that most would, because the marginal value of lead to refiners will be higher in 1986 and 1987 than in 1985, for the reasons discussed in Chapter II. This section briefly examines the impact that banking may have on the costs and benefits of the phasedown rule. This analysis is for illustrative purposes only, as EPA has determined that the final rule is feasible and not unduly expensive without banking, and should be promulgated independent of the banking rule.

Table VIII-9 compares the schedule without banking to the two possible alternatives with banking that were presented in Chapter II. Both alternatives are based on the partial misfueling assumption; i.e., that misfueling continues unabated through 1985 and is reduced by 80 percent in 1986 and 1987.

Alternative 1 assumes that refiners would not start banking until the second quarter of 1985, at which point they would use an average of 0.60 gplg, thus banking 0.50 grams, on average, for

TABLE VIII-9. Alternative Phasedown Patterns with Banking (gplg)

Alternative	1985 (by quarter I	II	III-IV)	1986	1987
Without Banking	1.10	1.10	0.50	0.10	0.10
With Banking					
Alternative 1	1.10	0.60	0.40	0.25	0.19
Alternative 2	0.80	0.60	0.45	0.30	0.21

each gallon of leaded produced.* Under alternative 1, we also assume some banking in the last half of the year, with leaded gasoline averaging 0.40 gplg, slightly below the limit of 0.50 gplg. A total of 7.0 million grams of lead (about 22 percent of the total allowed in 1985) would be banked during 1985, allowing refiners to average 0.25 gplg in 1986 and 0.19 gplg in 1987. Note that shaving 0.10 gplg from the annual average in 1985 translates into a larger per gallon increase in 1986 or 1987, because the amount of leaded gasoline produced in the later years is smaller; the total amount of lead use allowed over the three years, however, would be the same as without banking.

Alternative 2 assumes that some refiners would be able to reduce lead more quickly, so that banking would begin in the first quarter of 1985. Compared to Alternative 1, those extra banked rights from the first quarter would be used to reduce banking slightly in the last half of 1985 and to achieve slightly higher lead levels in 1986 and 1987. The amount banked would be 9.1 million grams. Again, the total amount of lead used would be the same as without banking.

Table VIII-10 presents the year-by-year cost and benefit estimates for the two alternatives with banking. Compared to the estimates without banking (see Table VIII-7c), the benefits and costs would be higher in 1985, but lower in 1986 and 1987.

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* Note that in California, where a state-imposed rule will limit leaded gasoline to 0.8 gplg in the first half of 1985, refiners producing 0.60 gplg leaded gasoline would only be able to bank 0.20 gplg in the second quarter; refiners would not be allowed to take credit for reductions mandated by state laws.

TABLE VIII-10. Costs and Monetized Benefits of Alternative (Banking) Phasedown Patterns, Assuming Partial Misfueling (millions of 1983 dollars)

	Alternative 1*			Alternative 2*		
	1985	1986	1987	1985	1986	1987
MONETIZED BENEFITS						
Children's health effects	347	550	522	395	532	515
Adult blood pressure	2,745	5,122	5,244	3,008	4,870	5,162
Conventional pollutants	0	222	222	0	222	222
Maintenance	110	716	752	97	661	732
Fuel economy	95	123	126	87	114	126
TOTAL MONETIZED BENEFITS	3,297	6,733	6,866	3,587	6,399	6,757
TOTAL REFINING COSTS	176	420	463	170	378	452
NET BENEFITS	3,121	6,313	6,403	3,417	6,021	6,305
NET BENEFITS EXCLUDING BLOOD PRESSURE	376	1,191	1,159	409	1,151	1,143

*See Table VIII-9 for description of alternatives.

Table VIII-11 compares the present values of the costs and benefits with banking to those without. In present value terms, banking reduces refining costs by $173 million under alternative 1 and $226 million under alternative 2, a reduction of over 20 percent. As discussed in Chapter II, this is likely to be an underestimate of the real savings that can be achieved with banking, as it does not account for the extra flexibility banking allows in meeting unexpected problems. It is also interesting to note that the present value of the cost of the final rule with banking is actually lower than the cost of the August proposed rule without banking (cf. Table VIII-6), despite the fact that the final rule eliminates significantly more lead.

Banking has a slight negative impact on the present value of estimated benefits. This reduction reflects several factors. The most important of these is that our estimates of the relationship between blood pressure and blood lead employs the logarithm of blood lead; thus it predicts slightly higher benefits from concentrating the reduction in lead in 1986 and 1987 rather than spreading it over 1985-1987. The same holds true for maintenance benefits, because exhaust system corrosion appears to fall most sharply as lead is reduced at low levels. In contrast, children's health benefits are higher with banking than without, primarily because the lead reductions are achieved earlier with banking.

Overall, banking has virtually no impact on net benefits. The small differences shown in Table VIII-11 are well within the "noise" of the estimates and should be regarded as insignificant.

TABLE VIII-11. Present Values of Costs and Benefits of Alternative Phasedown Patterns, 1985-87, Assuming Partial Misfueling (millions of 1983 dollars)

	Without Banking	With Banking	
		Alt. 1	Alt. 2
MONETIZED BENEFITS			
Children's health effects	1,210	1,262	1,285
Adult blood pressure	11,693	11,605	11,558
Conventional pollutants	385	385	385
Maintenance	1,638	1,377	1,298
Fuel economy	344	307	291
TOTAL MONETIZED BENEFITS	15,271	14,936	14,818
TOTAL REFINING COSTS	1,105	932	879
NET BENEFITS	14,166	14,007	13,939
NET BENEFITS EXCLUDING BLOOD PRESSURE	2,473	2,399	2,381

Coupled with the important, but nonmonetized, cost saving that banking provides in the form of flexibility to meet unexpected refining problems, these results indicate that banking is a desirable policy.

VIII.D. Summary

The results presented in this chapter indicate that the final phasedown rule being promulgated has the highest net benefits of the alternatives considered. This conclusion holds whether or not the recently developed estimates of blood-pressure-related benefits are included, and whether or not it is assumed that the rule will eliminate misfueling. Although many of the estimates are subject to uncertainty, the magnitude of the estimated monetized benefits relative to the costs indicates that this conclusion is very robust. Moreover, the monetary benefit estimates represent an incomplete tabulation of the benefits likely to result from the rule; in short, the benefits are under-estimated.

Three limitations deserve particular notice. First, we have not estimated any benefits for children at blood lead levels below the CDC cutoff of 25 ug/dl, and our monetized estimates for children above that level cover only medical and compensatory education costs. Second, the direct estimates of benefits associated with reduced conventional pollutants omit several important categories, including benefits associated with ozone's effects on nonagricultural vegetation, chronic health effects related to ozone, and the effects of nitrogen oxides and carbon

monoxide on health. Last, the estimates of adult health benefits cover only blood-pressure-related effects, and only males aged 40-59. In the case of myocardial infarctions, strokes, and deaths, only whites males in this age range are included. (This is because of limited data on the cardiovascular risks associated with high blood pressure in nonwhites.) These unquantified benefits add further strength to the conclusion that rapid reductions of lead in gasoline are amply justified.

REFERENCES

Abell RF. Operation and application of hydraulic valve lifters. SAE Paper #690347, Society of Automotive Engineers, Warrendale, PA 1969.

Adams R, McCarl B. Assessing the benefits of alternative oxidant standards on agriculture: the role of response information. Prepared for U.S. EPA, 1983 September.

Adams R, et al. Economic effects of ozone on agriculture. Final Report to U.S. EPA, 1984.

Adams WC, Schelegle ES. Ozone toxicity effects consequent to prolonged, high intensity exercise in trained endurance athletes. J App Physiol Respir Environ Exercise Physiol 1983; 55: 805-12.

Agerty HA. Lead poisoning in children. Med Clin North Am 1952; 36: 1587-97.

American Petroleum Institute. Unpublished Carcinogenicity Study on Unleaded Gasoline in Vapor in Fischer 344 Rats and B6C3F1 Mice. 1982.

Amoco, submission to EPA Central Docket EN-84-OS.

Anderson E, et al. Effect of low-level carbon monoxide exposure on onset and duration of angina pectoris: a study on 10 patients with ischemic heart disease. Annals of Internal Medicine 1973; 79: 46-50.

Anderson G, et al. Development and application of methods for estimating: effects of industrial emission controls on air quality impact of reactive pollutants. U.S. EPA 1984 July.

Angle CR, McIntire MS, Swanson MS, Stohs SJ. Erythrocyte nucleotides in children - increased blood lead and cytidine triphosphate. Pediatric Research 1982; 16: 331-4.

Annest JL, Pirkle JL, Makuc D, Neese JW, Bayse DD, Kovar MG. Chronological trends in blood lead levels between 1976 and 1980. New England Journal of Medicine 1983; 308: 1373-7.

Armstrong JA. Investigations into the emissions effects of vehicle misfueling. Presented to: The North American Motor Vehicle Emission Control Conference. New York, 1984 April 2.

Aub JC, Fairhall Lt, Minot AS, Reznikoff P, Hamilton A. Lead Poisoning in Baltimore, MD. The Williams and Wilkins Company (Medicine monographs: v.7.)

The Australian Therapeutic Trial in Mild Hypertension: Report by the Management Committee. Lancet 1980; 2: 1261-7.

Barrett LB, Waddell T. The Cost of Air Pollution Damages: A Status Report. U.S. EPA; Research Triangle Park, NC 1973

Barton JC, Conrad ME, Nuby S, Harrison L. Effects of iron on the absorption and retention of lead. Journal of Laboratory and Clinical Medicine 1978; 92: 536-47.

Bascunana J, Stahman R. Impact of gasoline characteristics on fuel economy and its measurement. Report 76-10, Technology Assessment and Evaluation Branch, ECTD, EPA, 1976 December.

Bates D, Sizto R. Relationship between air pollution levels and hospital admissions in southern Ontario. Canadian J of Pub Health 1983 March/April; 79.

Batuman V, Landy E, Maesaka JK, Wedeen RP. Contribution of lead to hypertension with renal impairment. N Engl J Med 1983; 309: 17-21.

Beevers DG, Erskine E, Robertson M, Beattie AD, Campbell BC, Goldberg A, Moore MR, Hawthorne VM. Blood-lead and hypertension. Lancet 1976; 2(7975): 1-3.

Beevers DG, Cruickshank JK, Yeoman WB, Carter GF, Goldberg A, Moore MR. Blood-lead and cadmium in human hypertension. J Environ Pathol Toxicol 1980; 4: 251-60.

Bell AG, Keene JA, Reders K. Road antiknock performance of low-lead and nonleaded gasoline in european cars. SAE Paper #710625, Society of Automotive Engineers, Warrendale, PA 1971.

Bellinger D, Needleman H, Leviton A, Waternaux C, Rabinowitz M, Nichols M. Early sensory-motor development and prenatal exposure to lead. Neurobehavioral Toxicology and Teratology 1984; 6: 387-402.

Benignus VA, Otto DA, Muller KE, Seiple KJ. Effects of age and body lead burden on CNS function in young children: II. EEG Spectra. Electroencephalog Clin Neurophysiol 1981; 52:240-8.

Bennett PA. Engine rusting in service and dynamometer tests. Lubrication Engineering 1960 November: 529.

Benson JD. Some factors which affect octane requirement increases. SAE Paper #750933, Society of Automotive Engineers, Warrendale, PA 1975.

Benson JD. Manganese fuel additive (MMT) can cause vehicle problems. SAE Paper #770655, Society of Automotive Engineers, Warrendale, PA 1977.

Bernard JP. Short-trip engine oil rust testing. SAE Paper #710839, Society of Automotive Engineers, Warrendale, PA 1971.

Bert JA, Gething JA, Hansel TJ, Newhall HK, Peyla RJ, Voss PA. Gasoline additive concentrate removes combustion chamber deposits and reduces vehicle octane requirement. SAE Paper #831709, Society of Automotive Engineers, Warrendale, PA 1983.

Betts PR, Astley R, Raine DN. Lead intoxication in children in Birmingham. Br Med J 1973; 1(5850): 402-6.

Bhalla AK, Amento EP, Clemens T, Holick MF, Krane SM. Specific high-affinity receptors for l,25-dihydroxyvitamin D_3 in human peripheral blood mononuclear cells: presence in monocytes and induction in T lymphocytes following activation. J Clin Endocrinol Metab 1983; 57: 1308-10.

Bigley HA Jr, Keller BD, Kloppe MG. Octane requirement increase in 1971 model cars with leaded and unleaded gasolines. SAE Paper #710675, Society of Automotive Engineers, Warrendale, PA 1971.

Bigley HA Jr, Benson JD. Octane requirement increases in 1971 model cars - with and without lead. SAE paper #730013, Society of Automotive Engineers, Warrendale, PA 1973.

Billick IH, Curran AS, Shier DR. Analysis of pediatric blood lead levels in New York City for 1970-1976. Environmental Health Perspectives 1979: 31:183-90.

Billick IH, et al. Predictions of pediatric blood lead levels from gasoline consumption. U.S. Department of Housing and Urban Development 1982.

Blank FM, et al. Valuation of aesthetic preferences: A case study of the economic value of visibility. Draft report to the Electric Power Research Institute, Palo Alto, Ca 1977.

Blomquist G. Valuation of Life: Implications of Automobile Seat Belt Use. Ph.D. Dissertation. University of Chicago, 1977.

Brennan MJW, Cantrill RC. y-Aminolevulinic Acid is a potent agonist for GABA auto receptors. Nature 1979; 280: 514-5.

Brookshire DS, et al. The valuation of aesthetic preferences. J Environ Econ Manag 1976; 3: 325-46.

Brown C. Equalizing differences in the labor market. Quarterly Journal of Economics 1980; 94.

Bryant EC. Statistical Analyses (2nd Edition) New York: McGraw Hill 1966.

Bryce-Smith D, Deshpunde RR, Hughes Jr Waldron HA. Lead and cadmium levels in still births. Lancet 1977; 1: 1159.

Bull RJ, Stanaszek PM, O'Neill JJ, Lutkenhoff SD. Specificity of the effects of lead on brain energy metabolism for substrates donating a cytoplasmic reducing equivalent. Environ Health Perspect 1975; 12:89-95.

Bull RJ. Effects of trace metals and their derivatives on the control of brain energy metabolism. Lee SD ed. Biochemical Effects of Environmental Pollutants. Ann Arbor, Mi: Ann Arbor Science 1977: 425-40.

Bull RJ, Lutkenhoff SD, McCarty GE, Miller RG. Delays in the postnatal increase of cerebral cytochrome concentrations in lead-exposed rats. Neuropharmacology 1979; 18: 83-92.

Bull RJ. Lead and energy metabolism. Singhal PL, Thomas JA eds. Lead Toxicity. Baltimore: Urban and Schwarzenberg, Inc 1980: 119-68.

Bull RJ, McCauley DT, Tayler DH, Crofton KM. The effects of lead on the developing central nervous system of the rat. Neurotoxicology 1983; 4(l): 1-17.

Bureau of Food and Drug Administration Compliance Program Report of Findings, Total Diet Studies, 1973-1980 reports.

Byers RK. Lead poisoning, review of the literature and report on forty-five cases. Pediatr 1959; 23:585-603.

Caracciolo F, Sperot JA. The effects of phosphorus-containing engine oil additives on exhaust oxidation catalyst degradation. SAE Paper #760562, Society of Automotive Engineers, Warrendale, PA 1976.

Carey LR, Stover WH, Murray DW. Extended drain passenger car engine oils. SAE Paper #780952, Society of Automotive Engineers, Warrendale, PA 1978.

Champion Spark Plug Co. Champion Ignition and Engine Performance Conferences. Volumes 1971-1976.

Chance RL, Ceselli RG. Corrosiveness of exhaust gas condensates. SAE Paper #830585, Society of Automotive Engineers, Warrendale, PA 1983.

Cherrie JM. Factors influencing valve temperature in passenger-car engines. SAE Paper #650484, Society of Automotive Engineers, Warrendale, PA 1965.

Chesney RW, Rosen JF, DeLuca HF. Disorders of calcium metabolism in children. Chiumello, Sperling M eds. Recent Progress in Pediatric Endocrinology. New York, NY: Raven Press 1983: 5-24.

Children living in the vicinity of a primary lead smelter. Environmental Research 1980; 22: 81-94.

Chisolm JJ Jr, Harrison HE. The exposure of children to lead. Pediatrics 1956; 18: 943-58.

Chisolm JJ Jr. The use of chelating agent in the treatment of acute and chronic lead intoxication in children. J Pediatr (St. Louis) 1968; 73: 1-38.

Chisolm JJ Jr, Barltrop D. Recognition and management of children with increased lead absorption. Arch Dis Child 1979; 54: 249-62.

Chock D, et al. Effect of NO_x emission rates on smog formation in the California south coast air basin. Environmental Science and Technology 1981 August: 15(8) .

Cohen C, et al. Respiratory symptoms, spirometry, and oxidant air pollution in nonsmoking adults. Amer Rev Resp Disease 1972; 105: 251-61.

Cohen GJ, Ahrens WE. Chronic lead poisoning: a review of seven years' experience at the Children's Hospital, District of Columbia. J Pediatr (St Louis) 1959; 54: 271-84.

Colyer C, Harding K. The 1980s -- a decade of challenge for engine oils. SAE Paper # 811410, Society of Automotive Engineers, Warrendale, PA 1981.

Cooper WC, Gaffey WR. Mortality of lead workers. Cole JF ed. Proceedings of the 1974 Conference on Standards of occupational Lead Exposure; February 1974; Washington, DC. J Occup Med 1975; 17: 100-7.

Cooper WC. Mortality in employees of lead production facilities and lead battery plants, 1971-1975. Lynam Dr, Piantanida LG, Cole JF Eds. Environmental Lead: Proceedings of the second international symposium on environmental lead research; Cincinnati, OH, 1978 December. New York: Academic Press 1981.

Cordera FJ, et al. TEL scavengers in fuel affect engine performance and durability. SAE Paper #877A, Society of Automotive Engineers, Warrendale, PA 1964.

Council on Environmental Quality. Environmental Quality - 1980. Government Printing Office, 1980 December.

Craver RJ, Podiak RS, Miller RD. Spark-plug design factors and their effect on engine performance. SAE paper #700081, Society of Automotive Engineers, Warrendale, PA 1970.

Crocker T, Vaux H. Some economic consequences of ambient oxidant impacts on a natural forest. Prepared for U.S. EPA, Office of Policy Analysis, 1983 August.

Crofton KM, Taylor DH, Bull RJ, Snulka DJ, Lutkenhoff SD. Developmental delays in exploration and locomotor activity in male rats exposed to low level lead. Life Science 1980; 26: 823-31.

Crouse WW, Johnson RH, Reiland WH. Effect of unleaded fuel on lubricant performance. SAE Paper #710584, Society of Automotive Engineers, Warrendale, PA 1971.

Danis LJ. Engine valve cooling. SAE Paper #730055, Society of Automotive Engineers, Warrendale, PA 1973.

De la Burde B, Choate MS Jr. Does asymptomatic lead exposure in children have latent sequelae. Journal of Pediatrics 1972; 81: 1088-91.

De la Burde B, Choate MS Jr. Early asymptomatic lead exposure and development at school age. Journal of Pediatrics 1975; 87: 638-42.

Delves HT. A micro-sampling method for the rapid determination of lead in blood by atomic absorption spectrophotometry. Analyst 1970: 431-3.

DePalma TV, Bailey CH. Application of catalytic converters to heavy-duty gasoline powered trucks. SAE Paper #750902, Society of Automotive Engineers, Warrendale, PA 1975.

De Para M, Belvoir Research and Development Center, U.S. Army. personal communication.

Detels R, et al. The UCLA population studies of chronic obstructive respiratory disease. American Journal of Epidemiology 1979; 109.

Dingwall-Fordyce I, Lane RE. A follow-up study of lead workers. Br J Ind Med 1963; 20: 313-5.

Doelling RP. Engine's definition of unleaded gasoline. SAE Paper 8401, Society of Automotive Engineers, Warrendale, PA 1971.

Dresner DL, Ibrahim NG, Mascarenhas BR, Levere RD. Modulation of bone marrow heme and protein synthesis by trace elements. Environ Res 1982; 28: 55-66.

DRI. The Data Resources Inc review of the U.S. economy. Various issues, 1983 and 1984. DRI, Lexington, MA.

Drinkwater B, et al. Air pollution, exercise, and heat stress. Archives of Environmental Health 1974; 28: 177-81.

Durand D. Stable Choas. Morristown, NJ: General Learning Corporation 1971.

Eccleston BH, Hum RW. Comparative emissions from some leaded and prototype lead-free automobile fuels. Report of Investigation #7390, U.S. Department of the Interior, Bureau of Mines Petroleum Research Center, Bartlesville, OK 1970.

Ellis JC. Future automotive fuels. SAE paper #720617, Society of Automotive Engineers, Warrendale, PA 1972.

Ellis JC. Gasoline for low-emission vehicles. SAE Paper #730616, Society of Automotive Engineers, Warrendale, PA 1973,

Energy and Environmental Analysis, Inc. Historical and projected emissions conversion factor and fuel economy for heavy-duty trucks 1962-2002. Motor Vehicle Manufacturers Association, Detroit, Michigan 1983.

Ennis JM, Harrison HE. Treatment of lead encephalopathy with BAL (2,3-dimercaptopropanol). Pediatrics 1950; 5: 853-68.

Erickson MM, Pokus A, Canter CE, Dickson AW, Hillman LS. Tissue mineral levels in victims of sudden infant death syndrome. I. toxic metals - lead and cadmium, Pediatric Research 1983; 17: 784-99.

ETA Engineering, Inc. Assessment of benefits from new source performance standards for volatile organic compounds. Prepared for U.S. EPA, Office of Policy Analysis, 1983 September.

Ethyl Corporation. Monthly Retail Gasoline Sales Bulletins, 1976-1982.

Evans, et al. Ozone measurement from a network of remote sites. Journal of the Air Pollution Control Association 1983 April; 32(4).

Expert Committee on Trace Metal Essentiality. Independent peer review of selected studies by Drs. Irchgessner and Reichlmayr-Lias concerning the possible nutritional essentiality of lead: official report of findings and recommendations of an interdisciplinary expert review committee, 1983. Available for inspection at: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, Research Triangle Park, NC.

Exxon Memo, Re: Gulf/East Coast Gasoline. 1978 January.

Fachetti S, Geiss F. Isotopic lead experiment status report. Luxembourg: Commission of the European Communities 1982; Publication No. EUR8352EN.

Faggan JE, et al. An evaluation of manganese as an antiknock in unleaded gasoline. SAE paper #750925, Society of Automotive Engineers, Warrendale, PA 1975.

Fahim MS, Fahim Z, Hall DG. Effects of subtoxic lead levels on pregnant women in the state of Missouri. Res Commun Chemical Pathol Pharmacol 1976; 13(2): 309.

Faucett Associates, Draft Report: Review and Critique of Previous OMSAPC Cost-Effectiveness Analysis, 1983 March.

Felt AE, Kerley RV. Engines and effects of lead-free gasoline. SAE Paper #710367, Society of Automotive Engineers, Warrendale, PA 1971.

Ferris B Jr. Health effects of exposure to low levels of regulated air pollutants. Journal of the Air Pollution Control Association 1978 May; 28(5).

Fisher RA. Statistical Methods for Research Workers. New York: Hafner Press 1970.

Florey C du V, et al. The relation between respiratory illness in primary schoolchildren and the use of gas for cooking. III. nitrogen dioxide, respiratory illness and lung infection. Int J Epidemiol 1979; 8: 347-53.

Folinsbee LJ, et al. Effect of 0.62 ppm NO_2 on cardiopulmonary function in young male non-smokers. Environmental Research 1978; 15: 199-205.

Folinsbee M, Bedi JF, Horvath SM. Pulmonary function changes in trained athletes following l-hour continuous heavy exercise while breathing 0.21 ppm ozone. J Appl Physiol Respir Environ Exercise Physiol (in press) 1984.

Ford Automotive Emissions Office, personal communication.

Forthofer RN. Investigation of non-response bias in NHANES II. American Journal Of Epidemiology (Bult.) 1983

Fouts PJ, Page IH. The effect of chronic lead poisoning on arterial blood pressure in dogs. Am Heart J 1942; 24: 329-31.

Fouts PJ, Page IH. The effect of chronic lead poisoning on arterial blood pressure in dogs. American Heart Journal 1942; 24: 329-31.

Fowler BA. General subcellular effects of lead, mercury, cadmium, and arsenic. Environ Health Perspect 1978; 22: 37-41.

Fowler BA, Kimmel CA, Woods JS, McConnell EE, Grant LD. Chronic low-level lead toxicity in the rat: III. an integrated assessment of long-term toxicity with special reference to the kidney. Toxicol Appl Pharmacol 1980; 56: 59-77.

Freas W. A digital solution to city-specific ekma isopleth diagrams in ozone regulatory analysis. U.S. EPA, Research Triangle Park 1983 July.

Freeman AM III. Air and Water Pollution Control: A Benefit-Cost Assessment. New York: John Wiley and Sons 1982.

Freeman R. Reversible myocarditis due to chronic lead poisoning in childhood. Arch Dis Child 1965; 40: 389-93.

Friedlander SK. Chairman, Clean Air Scientific Advisory Committee (CASAC). Review and Closure of the OAQPS Staff Paper for Nitrogen Oxides, 1982 July 6.

Fuchs EJ. Unleaded versus leaded fuel results in laboratory engine tests. SAE Paper #710676, Society of Automotive Engineers, Warrendale, PA 1971.

Gagliardi JC. Effect of fuel antiknock compounds and deposits on exhaust emissions. SAE paper #670128, Society of Automotive Engineers, Warrendale, PA 1967.

Gagliardi JC, Ghannam F. Effects of tetraethyl lead concentration on exhaust emissions in customer-type vehicle operation. SAE Paper #690015, Society of Automotive Engineers, Warrendale, PA 1969.

Gallopoulos NE. Projected lubricant requirement of engines operating with lead-free gasoline. SAE Paper #710585, Society of Automotive Engineers, Warrendale, PA 1971.

General Motors Corporation. General Motors Corporation comments on EPA report 'Costs and Benefits of Reducing Lead in Gasoline' letter from T.M. Fisher (GM) to R.D. Morgenstern (EPA/OPA), 1984 June 18.

Gergel WC, Sheahan TJ. Maximizing petroleum utilization through extension of passenger car oil drain periods - what's required? SAE Paper #760560, Society of Automotive Engineers, Warrendale, PA 1976.

Gerking S, Stanley L, Weirick W. An economic analysis of air pollution and health: the case of St. Louis. Prepared for U.S. EPA, Office of Policy Analysis, 1983 July.

Giles WS. Valve problems with lead free gasoline. SAE Paper #710368, Society of Automotive Engineers, Warrendale, PA 1971.

Giles WS, Updike SH. Influence of low lead fuels on exhaust valve performance. SAE Paper #710674, Society of Automotive Engineers, Warrendale, PA 1971.

Glasson WA, Tuesday CS. Environ Sci Technol 1970; 4: 37.

Glasson W. Effect of Hydrocarbons and NO_x on photochemical smog formation under simulated transport conditions. Journal of the Air Pollution Control Association 1981 November 11; 31(11).

Godfrey D, Courtney RL. Investigation of the mechanism of exhaust valve seat wear in engines run on unleaded gasoline. SAE Paper #710356, Society of Automotive Engineers, Warrendale, PA 1971.

Goldberg AM, Meredith DA, Miller S, Moore MR, Thompson CG. Hepatic drug metabolism and heme biosynthesis in lead-poisoned rats. British Journal of Pharmacology 1978; 62: 529-36.

Goldstein BD. The relation between respiratory illness in school-children and the use of gas for cooking. II. Factors affecting nitrogen dioxide levels in the home. Int J Epidemiol 1979; 8: 339-45.

Goldstein E, et al. Fate and distribution of inhaled nitrogen dioxide in rhesus monkey. Am Rev Respir Dis 1977; 115: 403-12.

Goldstein E, Moderator. Photo Chemical Air Pollution. Inter-departmental Conference sponsored by the Department of Medicine, University of California, School of Medicine; Davis, California 1982.

Goth G. Recent developments in hardfacing alloys for internal combustion engines. SAE Paper #831287 in State-of-the-Art on Design and Performance of Diesel Power Cylinder Components, SP-552 , Society of Automotive Engineers, Warrendale, PA 1983.

Goyer RA, Rhyne BC. Pathological effects of lead. Internal Rev Exp Pathol 1973; 12: 1-77.

Gray DS, Azhari AG. Saving maintenance dollars with lead-free gasoline. SAE Paper #720084, Society of Automotive Engineers, Warrendale, PA 1972.

Green AES, et al. An interdisciplinary study of the health, social, and environmental economics of the sulfur oxide pollution in Florida. Interdisciplinary Center for Aeronomy and (other) Atmospheric Sciences, Univ of Florida; Gainesville, FL 1978.

Gross-Selbeck E, Gross-Selbeck M. Changes in operant behavior of rats exposed to lead at the accepted no-effect level. Clinical Toxicology 1981; 18: 1247-56.

Gunter EW, et al. Laboratory Procedures Used by the Clinical Chemistry Division, Centers for Disease Control for the Second National Health and Nutrition Examination Survey. Atlanta Centers for Disease Control 1981.

Hackney JD, et al. Experimental studies on human health effects on air pollutants. II. Four hour exposure to ozone alone and in combination with other pollutants. Arch Environ Health 1975a; 33: 379-84.

Hackney JD, et al. Experimental studies on human health effects on air pollutants. III. Two hour exposure to ozone alone and in combination with other gases. Arch Environ Health 1975b; 30: 176-81.

Hackney JD. Effects of atmospheric pollutants on human physiologic function. Final report, U.S. EPA 1976$_0$

Hackney JD, et al. Experimental studies on human health effects of air pollutants. IV. Short-term physiological and clinical effects of nitrogen dioxide exposure. Arch Environ Health 1978; 33: 176-81.

Hall CA, Felt AE, Brown WJ. Evaluating effects of fuel factors on stabilized exhaust emissions levels. SAE Paper #690014, Society of Automotive Engineers, Warrendale, PA 1969.

Hammer D, et al. Los Angeles student nurse study. Daily symptom reporting and photochemical oxidants. Archives of Environmental Health 1974; 28.

Hare CT, Springer KJ, Huls TA. Exhaust emissions from farm construction, and industrial engines and their impact. SAE Paper #750788, Society of Automotive Engineers, Warrendale, PA 1975.

Harlan WR, Landis JR, Schmouder RL, et al. Relationship of blood lead and blood pressure in the adolescent and adult U.S. population. JAMA 1985 January 25.

Harrison. Principles of Internal Medicine. Ninth Edition.

Hartunian NS, Smart CN, Thompson MS. The Incidence and Economic Costs of Major Health Impairments. Lexington, MA: Lexington Books 1981.

Harvey P, Hamlin M, Kumar R. The Birmingham blood lead study. Presented at: annual conference of the British Psychological Society, Symposium on lead and health, 1983. Available for inspection at: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, Research Triangle Park, N.C.

Hasselblad V. Modeling dose response relations for health effects data. Environmetrics 81: Selected Papers, SIAM, Philadelphia, 1981.

Hasselblad V, Svendsgaard D. Reanalysis of the Los Angeles student nurse study. Unpublished paper, U.S. EPA, Health Effects Research Lab, Research Triangle Park, NC 1975 August.

Heck W, et al. Nitrogen dioxide: time-concentration model to predict acute foliar injury. U.S. EPA, Corvallis, Oregon. Pub. No. 600/3-79-057, 1979.

Heck W, et al. A reassessment of crop loss from ozone. Environmental Science and Technology 1983; 17(12).

Heintz HT, Hershaft A, Horak GC. National Damages of Air and Water Pollution. Report submitted to U.S. EPA 1976.

Hickling Partners, Inc. Final Report on the Assessment of the Economic Impact on the Automotive Parts/Service Industry of Proposed Gasoline Lead Content Regulations. submitted to Policy Planning and Assessment Directorate, Environment/Canada 1981 March.

Hernberg S, Nikkanen J. Enzyme inhibition by lead under normal urban conditions. Lancet 1970; 1: 63-4.

Holtzman D, Shen Hsu J. Early effects of inorganic lead on immature rat brain mitochondrial respiration. Pediatric Res 1976; 10: 70-5.

Holtzman D, Shen Hsu J, Desautel M. Absence of effects of lead feedings and growth-retardation on mitochondriae and microsomal cytochromes in the developing brain. Toxicol Appl Pharmacol 1981; 58: 48-56.

Hoppenbrouwers T, et al. Seasonal relationships of sudden infant death syndrome and environmental pollutants. American Journal of Epidemiology 1981; 113(6).

Hornbeck DD, et al. Advantages of lead in gasoline for european cars - the lead road bonus. SAE Paper #750936, Society of Automotive Engineers, Warrendale, PA 1975.

Horvath S, et al. Maximal aerobic capacity at different levels of carboxyhemoglobin. Journal of Applied Physiology 1975; 38: 300-3.

Horvath SM, Folinsbee LJ. Effects of pollutants on cardiopulmonary function. Report to U.S. EPA 1979.

Hospadaruk V. Fundamental mechanisms of automobile corrosion. SAE Paper #780909, Society of Automotive Engineers, Warrendale, PA 1978.

Hudnall JR, et al. New gasoline formulations provide protection against corrosive engine wear. SAE Paper #690514, Society of Automotive Engineers, Warrendale, PA 1969.

Hudson PJ. Fitting segmented curves whose join points have to be estimated. Journal of the American Statistical Association 1966; 61: 873-80.

Hypertension Detection and Follow-up Program Cooperative Group. The Effect of Treatment on Mortality in Mild Hypertension. N Eng J Med 1983; 307: 976-80.

Iannacone A, Carmigmani M, Boscolo P. Cardiovascular reactivity in the rat following chronic exposure to cadmium and lead. Ann 1st Supr Sanrta 1981; 17: 655-60.

Innes WB. Environmental Science and Technology 1981; 15: 933.

Isringhaus EA Jr, Henderson BM. Engine rusting in short-run passenger-car service. SAE Paper #650868, Society of Automotive Engineers, Warrendale, PA 1965.

Jacobs KM. Energy usage and other comparisons between gasoline and diesel medium duty trucks. SAE Paper #770757, Society of Automotive Engineers, Warrendale, PA 1977.

Janney A. The relationship between gasoline lead emissions and blood poisoning in Americans. Prepared for U.S. EPA, Office of Policy Analysis, 1982.

Joumard R, et al. Mathematical models of the uptake of carbon monoxide on hemoglobin at low carbon monoxide levels. Environmental Health Perspectives 1981; 41.

Journal of Pediatrics (Editorial). New approaches to screening for iron deficiency. 1977; 90: 678.

Kagawa J, et al. Photochemical air pollution: its effects on respiratory function of elementary school children. Arch Environ Health 1975; 30: 117-22.

Kakalik JS, et al. The Cost of Special Education. Rand Corporation Report N-1791-ED, 1981.

Kammholz LP, Thatcher LG, Blodgett FM, Good TA. Rapid protoporphyrin quantitation for detection of lead poisoning. Pediatrics 1972; 50: 625-31.

Keller MD, et al. ORI of today's vehicles. SAE Paper #760195, Society of Automotive Engineers, Warrendale, PA 1976.

Keller MD, et al. Respiratory illness in household using gas and electricity for cooking. II. Symptoms and objective findings. Environ Res 1979; 19: 504-15.

Kent WL, Finnigan FT. Effect of some fuel and operating parameters on exhaust valve seat wear. SAE Paper #710673, Society of Automotive Engineers, Warrendale, PA 1971.

Kerr HD, et al. Effects of nitrogen dioxide on pulmonary function in human subjects. Environmental Chamber Study. U.S. EPA Research Triangle Park, NC 1978.

Kerr JCS, et al. Effects of nitrogen dioxide on pulmonary function in human subjects. Environmental Chambers Study. Environ Res 1979; 19: 392-404.

Killus JP, et al. Application of a regional oxidant model to the northeast United States. Presented at the International Conference on Long Range Transport Models for Photochemical Oxidants and Their Precursor. U.S. EPA; Research Triangle Park, NC, 1983 April 12-14.

Kim Y, Nickola R. A heat-resistant aluminized steel for high-temperature applications. SAE Paper #800316, Society of Automotive Engineers, Warrendale, PA 1980.

Kinosian J. Ozone precursor relationships from EKMA diagrams. Environmental Science and Technology 1982; 16 (12).

Kipp KL, et al. Ability of gasoline additives to clean engines and reduce exhaust emissions. SAE Paper #700456, Society of Automotive Engineers, Warrendale, PA 1970.

Klein JP, et al. Hemoglobin affinity for oxygen during short-term exhaustive exercise. Journal of Applied Physiology 1980; 48.

Kleinbaum DG, Morgenstern H, Keyser LL. Epidemiologic Research. 1982.

Kopp R, Vaughan W. Agricultural benefit analysis: alternative ozone and photochemical oxidant standards. Resources for the Future 1983 June 30; and discussion with authors, 1984.

Kopp SJ, Glonek T, Erlanger M, Perry EF, Perry HM Jr, Barany M. Cadmium and lead effects on myocardial function and metabolism. J Environ Pathol Toxicol 1980; 4: 205-27.

Kromhout D, Coulande CL. Trace metals and CHD risk indicators in 152 elderly men (the Zutphen Study). Eur Heart J 1984; 5 (Abstr Suppl 1): 101

Kuroishi N. Recent technology of sintered metals for automotive use. SAE Paper #811416, Society of Automotive Engineers, Warrendale, PA 1981.

Lamola A-A, Joselow M, Yamane T. Zinc protoporphyrin (ZPP): a simple, sensitive, fluorometric screening test for lead poisoning. Clin Chem (Winston Salem, NC) 1975a; 21: 93-7.

Lamola A-A, Piomelli S. Poh-Fitzpartick MB, Yamane T, Harber LC. Erythropoietic protoporphyria and lead intoxication: the molecular basis for difference in cutaneous photosensitivity: II. different binding of erythrocyte proteporphyrin to hemoglobin. J Clin Invest 1975b; 56: 1528-5.

Landrigan PJ, Baker EL, Feldman RG, Cox DH, Eden KV, Orenstein WA, Mather JA, Yankel AJ, von Lindern IH. Increased lead absorption with anemia and slower nerve conduction in children near a lead smelter. Journal of Pediatrics 1976; 89: 904-10.

Larson J, of Weyerhauser. personal communication, 1984.

Lauwerys R, Buchet JP, Reels H, Hubermont G. Placental transfer of lead, mercury, cadmium, and carbon monoxide in women. I. Comparison of the frequency distributions of the biological indices in maternal and umbilical cord blood. Environmental Research 1978; 15: 278-89.

Lave L, Seskin E. Air Pollution and Human Health. Baltimore: Johns Hopkins University Press 1977.

Lax D. Analysis of fuel economy and vehicle use data for the 1977 truck inventory and use survey. SAE Paper #810388, Society of Automotive Engineers, Warrendale, PA 1981.

Layland D, Cole H. A review of recent applications of the SAI urban airshed model. U.S. EPA; Research Triangle Park, NC 1983 December.

Lebowitz MD, et al. The effect of air pollution and weather on lung function in exercising children and adolescents. American Review of Respiratory Diseases 1974; 109.

Lebowitz MD, O'Rourke MK, Dodge R. Holberg CJ, Corman G, Hoshaw RW, Pinnas JL, Barbe RA, Sweller MR. The adverse health effects of biological aerosols, other aerosols, and indoor microclimate on asthmatics and nonasthmatics. Environ Int 1982; 8: 375-80.

Lebowitz MD, Holberg CJ, Dodge RR. Respiratory effects on population from low level exposures to ozone. Presented at: 34th annual meeting of the Air Pollution Control Association, 1983 June, Atlanta, GA. Pittsburgh, PA: Air Pollution Control Association, paper no 83 - 12.5.

Lebowitz, MD. The effects of environmental tobacco smoke exposure and gas stoves on daily peak flow rates in asthmatic families. Eur T Respir Dis 1984; 64 (133): 90-7.

Leighton J, Shehadi A, Wolcott R. Aggregate benefits of air pollution control. prepared for U.S. EPA, Office of Policy Analysis, by Public Interest Economics Foundation; Washington, DC 1983 June.

Leikkanen HE, Beckman EW. Effect of leaded and unleaded gasolines on exhaust emissions as influenced by combustion chamber deposits. SAE Paper #710843, Society of Automotive Engineers, Warrendale, PA 1971,

Lenane DL. MMT - a further evaluation. SAE paper #770656, Society of Automotive Engineers, Warrendale, PA 1977.

Levy RI. The decline in coronary heart disease mortality: Status and perspective on the role of cholesterol. American Journal of Cardiology 1984 August 27; 54 (4).

Lewis BW, Collin RJ, Wilson HS. Seasonal incidence of lead poisoning in children in St. Louis. Southern Medical Journal 1955; 48: 298-301.

Lewis RA, Newhall HK, Peyla RJ, Voss DA, Welstand JS. A New concept in engine deposit control additives for unleaded gasoline. SAE Paper #830938, Society of Automotive Engineers, Warrendale, PA 1983.

Lilienfeld AM, Lilienfeld DE. Foundations of Epidemiology. New York, 1980.

Lincoln RH. written statement before the Senate Committee on Environmental and Public Works in Opposition to Senate Bill 2609, 1984.

Lin-Fu JS. Vulnerability of children to lead exposure and toxicity: parts one and two. New England Journal of Medicine 1973; 289: 1229-33, 1289-93.

Linn WS, et al. Respiratory function and symptoms in urban office workers in relation to oxidant air pollution exposure. Amer Rev Resp Disease 1976; 114: 477-83.

Linn W, et al. Human respiratory effects of heavy exercise in oxidant-polluted ambient air. American Review of Respiratory Disease 1981; 123 (4).

Lippmann M, et al. Effects of ozone on the pulmonary function of children. Lee S, et al. eds, The Biomedical Effects of Ozone and Related Photochemical Oxidants. Princeton Scientific Publishers, Inc; Princeton, NJ: Advances in Modern Environmental Toxicology 1983; V: 423-46.

Litman DA, Corriea MA. L-tryptophan: a common denominator of biochemical and neurological events of acute hepatic porphyrias? Science 1983; 222: 1031-3.

Loehman E, et al. Distributional Analysis of regional benefits and cost of air quality control. Journal of Environmental Economics and Management 1979 September; 6(3).

Love GJ, et al. Acute respiratory illness in families exposed to nitrogen dioxide ambient air pollution in Chattanooga, TN. Arch Environ Health 37: 75-80.

MacLean DC. Stickstoffoxide ALS phytotoxis CHE Luftuerurein 16 UNGEN. Staub-Reinhalt Luft 1975; 35(5).

Mage D, et al. A sensitivity analysis of the effects of CO time patterns on computer blood carboyhemogologin levels. presented at: Air Pollution Control Association Meetings 1983, Atlanta, Ga.

Mahaffey KR, Annest JL, Roberts J, Murphy MS. National estimation of blood lead levels: United States (1976-1980). New England Journal of Medicine; 307: 573-9.

Mahaffey KR, Rosen JF, Chesney RW, Peeler JT, Smith CM, Deluca HF. Association between age, blood lead concentrations and serum 1,25-dihydroxycholecalciferol levels in children. Amercian Journal of Clinical Nutrition 1982; 35: 1327-31.

Makino K, Mizoguchi I. Symptoms caused by photochemical smog. Japan Journal of Public Health 1975; 22(8).

Manton WI. Sources of lead in blood: identification by stable isotopes. Archives of Environmental Health 1977; 32:149-59.

McBride WG, Black BP, English BJ. Blood lead levels and behavior of 400 preschool children. Med Journal Aust 1982; 2: 26-9.

McCauley PT, Bull RJ. Lead-induced delays in synaptogenesis in the rat cerebral cortex. Fed Proc Fed Am Soc Exp Bio 1978; 37: 740.

McCormick H. TRW Piston Ring Division, personal communication.

McDonnel TF. The effects of engine oil additives on vehicle fuel economy, emissions, emission control components and engine wear. SAE Paper #780962, Society of Automotive Engineers, Warrendale, PA 1978.

McDonnell WF, Hortsmann DH, Hazucha DH, Hazucha MJ, Seal E Jr, Haak ED, Salaam S, House DE. Pulmonary effects on ozone exposure during exercise: dose-response characteristics. J Appl Physiol Respir Environ Exercise Physiol 1983; 54: 1345-52.

McGartland A, Ostro B. Benefits Analysis of New Source Performance Standards: The control of HC and NO_x. U.S. EPA, Office of Policy Analysis, Draft reported 1984 October.

McGartland A, Ostro B. Benefit Analysis of New Source Performance Standards: The Control of VOC and NO_x. U.S. EPA, Office of Policy analysis, 1985.

McGee D, Gordon T. The results of the Framingham Study applied to four other U.S. - based epidemiologic studies of coronary heart disease. The Framingham Study. Section 31. DHEW Pub No. (NIH) 76-1083. National Institutes of Health, Washington, D.C. U.S. Government Printing Office 1976.

McLaughlin S, et al. Measuring effects of air pollution stress on forest productivity. Tapp Journal 1984; 67: 74.

Melia RJW, et al. Association between gas cooking and respiratory disease in children. Br Med J 1977; 2: 149-52.

Melia RJW, et al. Differences in $N0_2$ levels in kitchens with gas or electric cookers. Atm Environ 1978; 12: 149-52.

Melia RJW, et al. The relation between respiratory illness in primary schoolchildren and the use of gas for cooking. I - Results from a national survey. Int J Epid 1979; 8: 333.

Mellins RB, Jenkins CD. Epidemiological and psychological study of lead poisoning in children. Journal American Medical Association 1955; 158: 15-20.

Mentzer WC. Differentration of iron deficiency from thalessemia trait. Lancet 1973; 1: 882.

Menzel DB. The role of free radicals in the toxicity of air pollutants (nitrogen oxides and ozone). Bryor WA ed. Free Radicals in Biology, vol III. New York: Academic 1976: 181-202.

Meredith PA, Moore MR, Campbell BC, Thompson GG, Goldberg A. Delta-animolaevulinic acid metabolism in normal and lead-exposed humans. Toxicology 1978; 9: 1-9.

Milar CR, Schroeder SR, Mushak P, Dolcourt J, Grant LD. Contributions of the caregiving environment to increased lead burden of children. American Journal Mental Deficiency 1980; 84: 339-44.

Milar CR, Schroeder SR, Mushak P, Boone L. Failure to find hyperactivity in preschool children with moderately elevated lead burden. Journal Pediatric Psychology 1981; 6: 85-95.

Moffitt JV. Compatability of military standard engines with leaded and low-leaded fuels. AFLRL-19, Southwest Research Institute, San Antonio, Texas (NTIS #AD-756 511) 1972.

Monthly Energy Review. U.S. Department of Energy, various issues, 1978 - 1982.

Moore MR, Meredith PA. The association of delta aminolevulinic acid with the neurological and behavioral effects of lead exposure. Hemphill DD ed. Trace Substances in Environmental Health - X. Columbia, MO: University of Missouri - Columbia 1976.

Moran JB. The environmental implications of manganese as an alternative antiknock, SAE Paper #750926, Society of Automotive Engineers, Warrendale, PA 1975.

Morbidity and Mortality Weekly Reports, various issues, 1976-1981, U.S. Public Health Service.

Morbidity and Mortality Weekly Reports: Reports of the Lead Point Poisoning Prevention Screening Program 1973-1981.

Morgan JM. Hyperkalemia and acidosis in lead neuropathy. South Med J 1976; 69(7): 881-3.

Moreau T, Orssaud G, Juguet B, Busquet G. Plombemie et pression arterielle: premiers resultats d'une enquete transversable de 431 sujets de sexe masculin. [Blood lead levels and arterial pressure: initial results of a cross sectional study of 431 male subjects.] [Letter] Rev Epidemol Sante Fpulique 1982; 30: 395-7.

Morris WE, Rogers JD Jr, Poskitt RW. 1971 cars and the 'new' gasolines. SAE Paper #710624, Society of Automotive Engineers, Warrendale, PA 1971.

Motor Vehicle Manufacturing Association. Motor Vehicle Facts and Figures "83". Detroit 1983.

Motor Vehicle Manufacturing Association. Incentives for Proper Usage of Unleaded Fuel. Detroit, 1984.

Multiple Risk Factor Intervention Trial: Risk factor changes and mortality results. Multiple Risk Factor Intervention Trial Research Group. JAMA 1982; 248: 1465-77.

National Academy of Sciences. Lead: airborne lead in perspective. Washington, DC: National Academy of Sciences 1972. (Biologic effects of atmospheric pollutants.)

National Academy of Sciences. Air quality and automobile emission control, Vol 4. prepared for the Committee on Public Works, U.S. Senate, U.S. Government Printing Office 1974.

National Academy of Sciences. Nitrogen oxides: medical and biologic effects of environmental pollutants. National Research Council, Washington, DC 1976.

National Academy of Sciences. Nitrogen oxides. National Academy of Sciences, Washington, DC 1977: 197-214.

National Center for Health Statistics. National Hospital Discharge Survey, various issues.

National Center for Health Statistics. Plan and Operation of the Second National Health and Nutrition Examination Survey 1976-1980. National Center for Health Statistics 1981 (Vital and Health Statistics Series 1, No. 15)

National Petroleum Council. Refinery Flexibility. an interim report of the National Petroleum Council, 1979 December.

National Research Council. Air quality and automobile emisison control, Vol 4: The costs and benefits of automobiles emission control. A report by the coordinating Committee on Air Quality Studies of the National Academy of Sciences and the National Academy of Engineering. Prepared for the Committee on Public works, United States Senate, 93rd Congress, 2nd Session. Washington, DC: U.S. Government Printing Office 1974.

National Research Council. Nitrogen oxides: medical and biological effects of environmental pollutants. National Academy of Sciences, Washington, DC 1977: 1-333.

National Research Council. Nitrates. an environmental assessment. National Academy of Sciences, Washington, DC 1978: 723.

National Research Council. Acid deposition: atmospheric processes in eastern North America. NRC , National Academy Press, Washington DC 1983.

Needleman HL, Gunnoe C, Leviton A, Reed R, Peresie H, Maher C, Barret P. Deficits in psychological and classroom performance of children with elevated dentine lead levels. New England Journal of Medicine 1979; 300: 689-95.

Needleman HL, Rabinowitz M, Leviton A, Linn S, Schoenbaums S. The relationship between prenatal exposure to lead and congenital anomalies. Journal of the American Medical Association 1984; 251: 2956-9.

New approaches to screening for iron deficiency. Editorial. Journal of Pediatrics; 90: 678.

Nisbet ICT. Sulfates and acidity in precipitation: their relationship to emissions and regional transport of sulfur oxides. Air quality and stationary source emission control, a report by the Commission on Natural Resources of the National Research Council. prepared for the Committee on Public Works, U.S. Senate, 94th Congress, 1st Session. Washington, DC: U.S. Government Printing Office 1975.

Odenbro A, Greenberg N, Vroegh K, Bederka J, Kihlstrom J-E. Functional disturbances in lead-exposed children. Ambio 1983; 12: 40-4.

Orehek J, et al. Effect of short-term, low-level nitrogen dioxide exposure on brochial sensitivity of asthmatic patients. J Clin Invest 1976; 57:37

Orrin DS, Miner WR, Kipp KL. Unleaded gasoline - lubricant requirements and fuel additive performance. SAE Paper #720689, Society of Automotive Engineers, Warrendale, PA 1972.

Otto DA, Benignus VA, Muller KE, Barton CN, Effects of age and body lead burden on CNS function in young children. I: slow cortical potentials. Electroencephalog Clin Neurophysiol 1981; 52: 229-39.

Otto, et al. Effects of low to moderate lead exposure on slow cortical potentials in young children: two year follow up study. Neurobehav Toxicol Teratol 1982; 4:733-7.

Otto D, Rovinson G, Baumann S, Schroeder S, Kleinbaum D, Barton C, Mushak P, Boone L. Five-year follow-up study of children with low-to-moderate lead absorption: electrophysiological evaluation. presented at second international conference on prospective lead studies 1984 April; Cincinnati, OH. available for inspection at: U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office, Research Triangle Park, NC.

Page WP. Estimation of Economic losses to the agricultural sector from airborne residuals in the Ohio River Basin. Journal of the Air Pollution Control Association, 1982; 32: 151-4.

Paglia DE, Valentine WN, Dahlgren JG. Effects of low level lead exposure on pyrimidine 5′ nucleotidase and other erythrocyte enzymes: possible role of pyrimidine 5′ nucleotidase in the pathogenesis of lead induced anemia. Journal of Clinical Investigation 1975; 56:1164-9.

Pahnke AJ, Conte JF. Effects of combustion-chamber deposits and driving conditions on vehicle exhaust emissions. SAE Paper #690017, Society of Automotive Engineers, Warrendale PA 1969.

Pahnke AJ, Bettoney WE. Role of lead antiknocks in modern gasoline. SAE Paper #710842, Society of Automotive Engineers Warrendale, PA 1971.

Patterson WR. Materials, design and corrosion effects on exhaust-system life. SAE Paper #780921, Society of Automotive Engineers, Warrendale, PA 1978.

Perlstein MA, Attala R. Necrologic sequelae of plumbism in children. Clinic Pediatric (Philadelphia) 1966; 5: 292-8.

Perry HM Jr, Erlanger MW. Pressor effects of chronically feeding cadmium and lead together. Hemphill DD ed. Trace Substances in Environmental Health-XII: [proceedings of University of Missouri's 12th annual conference on trace substances in environmental health] ; 1978 June, Columbia, MO. Columbia MO: University of Missouri-Columbia: 268-75.

Pitts JN Jr, et al. Comment on effect of nitrogen oxide emissions on ozone levels in metropolitan regions effects of NO_x emission rates on smog formation in the California south coast air basin, and effects of hydrocarbon and NO_x on photochemical smog formation under simulated transport conditions. Environ Sci Technol 1983; 54-7.

Piomelli S, Davidow B, Guinee VF, Yound P, Gay G. The FEP (free erythrocyte porphyrins) test: a screening micromethod for lead poisoning. Pediatrics 1973; 51: 254-9.

Piomelli S, Seaman C, Zullow D, Curran A, Davidow B. Metabolic evidence of lead toxicity in "normal" urban children. Clinic Res 1977; 25: 459A.

Piomelli S, Seaman C, Zullow D, Curran A, Davidow B. Threshold for lead damage to heme synthesis in urban children. Proceeding of the National Academy of Sciences in 1982 May; 79: 3335-9.

Piomelli S, Rosen JF, Chisolm JJ, Graef JW. Management of childhood lead poisoning. Journal of Pediatrics 1984; 4:105.

Pirkle JL, Annest JL. Blood lead levels 1976-1980 - Reply (letter). N Eng J Med 1984; 310(17): 1125-6.

Pirkle JL, Schwartz J, Landis JR, Harlan WR. The relationship between blood lead levels and blood pressure and its cardiovascular risk implications. American Journal of Epidemiology, 1985; 121(2): 246-58

Platts Oilgram Price Report, Price Average Supplement. December 1983 Monthly Averages. 1984 January 26.

Pless LG, Bennett PA. Effects of some engine, operating, and oil variables on engine rusting in short-trip service. Lubrication Engineering 1969 October; 384.

Pless LG. Effects of some engine, fuel, and oil additive factors on engine rusting in short-trip service. SAE paper #700457, Society of Automotive Engineers, Warrendale, PA 1970.

Pless LG. Interactions among oil additive and engine operating parameters affecting engine deposits and wear. SAE Paper #720686, Society of Automotive Engineers, Warrendale, PA 1972.

Pless LG. A study of lengthened engine oil-change intervals. SAE Paper #740139, Society of Automotive Engineers, Warrendale, PA 1974.

Pless LG. Deposits, wear, and catalyst performance with low ash and ashless engine oils. SAE Paper #750900, Society of Automotive Engineers, Warrendale, PA, 1975.

Pocock SJ, Ashby D. Environmental lead and children's intelligence: a review of recent epidemiological studies. Statician (in press).

The Pooling Project Research Group. Relationship of blood pressure, serum cholesterol, smoking habit, relative weight and ECG abnormalities to incidence of major coronary events: Final report of the pooling project. J Chron Dis 1978; 31: 201-306.

Port CD. A comparative study of experimental and spontaneous emphysema. J Toxicol Environ Health 1977; 2:589-604.

Portney P, Mullahy J. Ambient ozone and human health: an epidemiological analysis. prepared for U.S. EPA, Office of Air Quality Planning and Standards, 1983 September.

Portney P, et al. A multinominal model of the air pollution respiratory health relationship. Resources for the Future 1984.

Portney P, Mullahy J. Urban air quality and acute respiratory illness. Journal of Urban Economics forthcoming 1985.

Posin C, et al. Nitrogen dioxide and human blood biochemistry. Arch Environ Health 1978 Nov/Dee; 318-24.

Provenzano G. The social costs of excessive lead-exposure during childhood. HL Needleman ED. Low Level Lead Exposure: The Clinical Implications of Current Research. New York: Raven Press 1980.

Provvedini DM, Tsoukas CD, Deftos LJ, Manolagas SC. l,25-dihydroxy-vitamin D_3 receptors in human lukocytes. Science (Washington, DC) 1983; 221: 1181-2.

Public Use Data Tape Documentation, Hematology and Biochemistry. Second National Health and Nutrition Examination Survey 1976-1980. Catalog number 5411, U.S. Public Health Service, National Center for Health Statistics.

Rabinowitz WB, Needleman H. Petrol lead sales and umbilical cord blood lead levels in Boston, Massachusetts. Lancet 1983.

Rabinowitz WB, Wetherill GW, Kopple JD. Kinetic analysis of lead metabolism in human health. Journal of Clinical Investigation 1976; 58:260-70.

Ramirez-Cervantes B, Embree JW, Hine CH, Nelson KM, Varner MO, Putnam RD. Health assessment of employees with different body burdens of lead. Journal Occupational Medicine 1978; 20: 610-7.

Randall A, et al. Bidding games for valuation of aesthetic environmental improvements. J Environ Econ Manag 1974; 1: 132-49.

Rasmussen H, Waisman DM. Modulation of cell function in the calcium messenger system. Rev Physiol Biochemical Pharmacol 1983; 95: 111-48.

Raven P, et al. Effect of carbon monoxide and peroxyacetylitrate on man's maximal aerobic capacity. Journal of applied physiology 1974; 36.

Raymond L. Today's fuels and lubricants and how they got that way. SAE Paper #801341, Society of Automotive Engineers, Warrendale, PA 1980.

Richet G, Albahary C, Morel-Maroqer L, Guillaume P, Galle P. Les alterations renales clans 23 cas de saturnisme professionnel. [Renal changes in 23 cases of occupational lead poisoning] Bull Memorial Society Medical Hop Paris 1966; 117: 441-66.

Reels H, Buchet JP, Lauwerys R, Hubermont G, Braux P, Claeys-Thoreau F, LaFontaine A, Van Overschelde J. Impact of air pollution by lead on the heme biosynthetic pathway in schoolage children. Arch Environ Health 1976; 31: 310-6.

Rogers JJ, Kabel RH. The sequence IID engine oil test. SAE Paper #780931, Society of Automotive Engineers, Warrendale, PA 1978.

Rosen JF, Chesney RW, Hamstra A, De Luca HF, Mahaffey KR. Reduction in 1,25-dihydroxy vitamin D in children with increased lead absorption. New England Journal of Medicine 1980a; 302: 1128-31.

Rosen JF, Chesney RW, Hamstra A, Deluca HF, Mahaffey KR. Reduction in 1,25-dihydroxyvitamin D in children with increased lead absorption. Brown SS, Davis DS ed. Organ Directed Toxicity Chemical Indices and Mechanisms. Pergamon Press 1980b.

Rosen JF, Chesney RW. Circulating calcitriol concentrations in health and disease. Journal Pediatric (St. Louis) 1983; 103: 1-7.

Rowley DW. Exhaust system considerations for heavy duty trucks. SAE Paper #770893, Society of Automotive Engineers, Warrendale, PA 1977.

Royal Comission on Environmental Pollution. Ninth report - lead in the environment. Cmnd 8852, Her Majesty's Stationery Office, London, England 1983.

Rowe R, Chestnut L. Ozone and asthmatics in Los Angeles: a benefits analysis. Report for Office of Policy Analysis by Energy and Resource Consultants, 1984.

Rummo JH, Routh DK, Rummo NJ, Brown JF. Behavioral and neurological effects of symptomatic and asymptomatic lead-exposure in children. Arch Env He 1979; 34(2): 120-4

Russell JA, Tosh JD, Johnson AA. Performance of army engines with leaded and unleaded gasoline. AFLRL-21, Southwest Research Institute, San Antonio, Texas (NTIS #AD-766 760) 1973.

SAI. Simulations of the Regional Air Quality Impacts of Industrial Emission Controls. Final report to U.S. EPA, Economic Analysis Division 1984.

Sanders M. U.S. Postal Service, personal communication.

SAS Users Guide. Statistical Analysis System, Inc. Cary, NC 1982.

Sassa S, Garnick JL, Garnick S, Kappas A, Levere RD. Studies in lead poisoning. I: Microanalysis of erythrocyte protoporphyrin levels by spectrofluorometry in the detection of chronic lead intoxication in the subclinical range. Biochem Medical 1973; 8: 135-48.

Schwartz J, Leggett J, Ostro B, Pitcher H, Levin R. Costs and Benefits of Reducing Lead in Gasoline. U.S. EPA, Office of Policy Analysis 1984a.

Schwartz J, Janney A, pitcher H. The relationship between gasoline lead and blood lead. U.S. EPA, Office of Policy Analysis 1904b.

Schwartz J. Blood lead and blood pressure. Memo to EPA Central Docket EN-84-O5, 1984c September 7.

Schwartz J. Change in aromatics emissions due to lead phasedown. Memo to EPA Central Docket EN-84-05, 1984d September 17.

Schwartz J. Availability and severity of catalytic reforming equipment. Memo to EPA Central Docket EN-84-05, 1984e. September 11.

Schwochert HW. Performance of a catalytic converter that operates with nonleaded fuel. SAE paper #690503, Society of Automotive Engineers, Warrendale, PA 1969,

Seigneur C, Saxena P, Roth P. Preliminary results of acid rain modeling. submitted at a Specialty Conference on Atomspheric Deposition sponsored by the Air Pollution Control Association; Detroit, Michigan 1982 November 7-10.

Seigneur C, et al. Modeling studies of sulfate and nitrate chemistry: the effect changes in sulfur dioxide, nitrogen oxide, and reactive hydrocarbon levels. final report 1984.

Shah BV, Surregr: Standard Errors of Regression Coefficients from Sample Survey Data. Research Triangle Park, NC, Research Triangle Institute 1982.

Shepard CD. Instrumentation and computer techniques aid automotive exhaust system design for low noise levels; materials to reduce system corrosion. SAE Paper #690006, Society of Automotive Engineers, Warrendale, PA 1969.

Sherwin RP, et al. Sequestration of exogenous peroxidase in the lungs of animals exposed to continuous 0.5 ppm nitrogen oxide. Fed Proc 1977; 36:1091.

Shier DR, Hall A. Analysis of housing and data collected in a lead based paint survey in Pittsburgh. National Bureau of Standards 1977.

Shlossman M, Brown M, Shapiro E, Dziak R. Calcitonin effects on isolated bone cells. Calcif Tissue Int 1982; 34: 190-6.

Shy CM, et al. The Chattanooga school children study: effects of community exposure of nitrogen dioxide. I Methods, description of pollutant exposure and results of ventilator function testing. J Air Pollut Control Assoc 1970; 20 (8):539-45.

Shy CM, Love GJ. Recent evidence on the human health effects of nitrogen dioxide. Proceedings of the Symposium on Nitrogen Oxides; Honolulu, Hawaii, 1979 April 4-5.

Sigsby J, et al. Automotive emissions of ethylene dibromide. SAE Paper #820786. Society of Engineers, Warrendale, PA 1982.

Silbergeld EK, Adler HS. Subcellular mechanisms of lead neurotoxicity. Brain Research 1978; 148: 451-67.

Silbergeld EK, Hruska RE, Miller LP, Eng N. Effects of lead in vivo and in vitro on GABAergic neurochemistry. J Neurochem 1980; 34:1712-8.

Silver W, Rodriguez-Torres R. Electrocardiographic studies in children with lead poisoning. Pediatrics 1968; 41: 1124-7.

Smith M, Delves T, Lansdown R, Clayton B, Graham P. The effects of lead exposure on urban children: the Institute of Child Health/ Southampton study. United Kingdom Department of the Environment 1983.

Smith RS. The feasibility of an injury tax approach to occupational safety. Law and Contemporary problems 1974; 38: 730-44.

Smith RS. The Occupational Safety and Health Act. Washington, DC: American Enterprise Institute for Public Policy Research, 1976.

Society of Automotive Engineers. Fuel Economy Measurement - Road Test Procedure. SAE Standard J 1082, Warrendale, PA 1980 September.

Soloman and Associates. Letter to the National Petroleum Refiners Association. Available in the U.S. EPA Central Docket EN-84-05, 1984.

Sorem SS. Effects of fuel factors on emissions. SAE Paper #710364. Society of Automotive Engineers, Warrendale, PA 1971.

Speizer FE, et al. Respiratory disease rates and pulmonary function in children associated with NO_2 exposure. Am Rev Resp Dis 1980; 121:3-10.

Spengler JD, et al. Sulfur dioxide and nitrogen dioxide levels inside and outside homes and the implications on health effects research. Environ Sci Techn 1979; 13:1271-6.

Spengler JD. Nitrogen dioxide inside and outside 137 homes and implications for ambient air quality standards and health effects research. Environ Sci Technol 1983; 17:164-8.

SRI International. An estimate of the nonhealth benefits of meeting the Secondary National Ambient Air Quality Standards. a final report to the National Commission on Air Quality 1981.

Stambaugh RL, Kopko RJ, Franklin TM. Effects of unleaded fuel and exhaust gas recirculation on sludge and varnish formation. SAE Paper #720944, Society of Automotive Engineers, Warrendale, PA 1972.

Sugiura K. Present status of Japanese motor oils and extension of oils drain intervals. SAE Paper #780953, Society of Automotive Engineers, Warrendale, PA 1978.

Supplemental Analysis of Refining Costs. Submitted to U.S. EPA Central Docket EN-84-05, 1984 September 19.

Tera, et al. Identification of gasoline lead in childrens' blood using isotopic analysis. Arch Env Health 1985 January.

Thaler R, Rosen S. The value of saving a life: evidence from the labor market. Taleckji NE ed. Household Production and Consumption. New York: Columbia University Press 1976.

Thomas HV, et al. Lipoperoxidation of lung lipids in rats exposed to nitrogen dioxide. Science 1968; 159:532-4.

Thompson CR, et al. Effects of continuous exposure of navel oranges to NO_2. Atm Environ 1970; 4:349-55.

Tosh JD, Johnson AA, Frame EA. Performance of army engines with leaded and unleaded gasoline. AFLRL-54, Southwest Research Institute, San Antonio, Texas (NTIS #AD-AO05 577) 1975.

Tosh JD. Performance of army engines with unleaded gasoline--field study. AFLRL-82, Southwest Research Institute, San Antonio, Texas (NTIS #AD-A032 075) 1976.

Trijonis JC. Empirical Relationships Between Atmospheric Nitrogen Dioxide and Its Precursors. U.S. EPA, Office of Research and Development. Environmental Sciences Research Laboratory, Research Triangle Park, NC, 1978 February.

Trijonis JC, et al. The Relationship of Ambient NO_2 to Hydrocarbon and NO_x Emissions. Draft report from Technology Service Corporation to U.S. EPA under Contract NO_x 68-02-2299. U.S. EPA, Office of Research and Development. Triangle Park, NC 1979.

Turner, Mason and Associates. Reply on behalf of the Lead Industries Association, July 1984. Available in the EPA Central Docket EN-84-05.

Union Carbide, personal communication, 1984.

Unzelman GH, Michalski GW. Octane improvement update -- refinery processing, antiknocks, and oxygenates. NPRA paper #AM-84-43, National Petroleum Refiners Association, Washington, DC 1984.

Urban CM, Springer KJ, McFadden JJ. Emissions control of gasoline engines for heavy-duty vehicles. SAE Paper #750903, Society of Automotive Engineers, Warrendale, PA 1975.

Urban CM, Kaupert AW. Evaluation of emission control technology approaches for heavy-duty gasoline engines. SAE Paper #780646, Society of Automotive Engineers, Warrendale, PA 1978.

U.S. Department of Agriculture. Agricultural Statistics, 1980. Washington, DC: U.S. Government Printing Office 1982.

U.S. Department of Energy, Energy Information Administration. Annual Report to Congress. 1983.

U.S. Department of Energy, Energy Information Administration. Annual Report to Congress. 1984a.

U.S. Department of Energy, Energy Information Administration, Office of Oil and Gas Refinery Evaluation Modeling System (REMS), model documentation. 1984b July.

U.S. Department of Energy. Refinery Evaluation Modeling System (REMS), 1983 Analysis of Verification. 1985 January.

U.S. Department of Health, Education, and Welfare. Limitations of activity and mobility due to chronic conditions. Public Health Service, Vital and Health Statistics Series 10, 1973a; (96).

U.S. Department of Health, Education, and Welfare. prevalence of selected chronic respiratory conditions: United States - 1970. Public Health Service, Vital and Health Statistics Series 10, 1973b; (84).

U.S. Department of Health and Human Services, Public Health Service. Blood carbon monoxide levels in persons 3-74 years of age: United States, 1976-80. Advance Data 1982 March 17; (76).

U.S. Environmental Protection Agency. Ozone SIP Data Base and Summary Report. Office of Air Quality Planning and Standards, Research Triangle Park, NC.

U.S. Environmental Protection Agency. Air Quality Criteria for Ozone and Other Photochemical Oxidants. Office of Research and Development, 1978 April.

U.S. Environmental Protection Agency, Carcinogen Assesmment Group. The Carcinogen Assessment Group's Final Report on Population Risk to Ambient Benzene Exposure. 1979a January 10.

U.S. Environmental Protection Agency, Office of Mobile Source Air Pollution Control. Draft Regulatory Analysis: Heavy-Duty Diesel particulate Regulations. U.S. EPA; Ann Arbor, Michigan 1979b.

U.S. Environmental Protection Agency. Regulatory Impact Analysis of the National Ambient Air Quality Standards for Carbon Monoxide. Office of Air Quality Planning and Standards, 1980.

U.S. Environmental Protection Agency. The Costs of Controlling Emissions of 1981 Model Year Automobiles. Office of Mobile Source Air Pollution Control, 1981 June.

U.S. Environmental Protection Agency. 1982 NCLAN Annual Report. Environmental Research Lab, Corvallis, Oregon l982a.

U.S. Environmental Protection Agency. Air Quality Criteria for Oxides of Nitrogen (Criteria Document). Research Triangle Park, NC 1982b.

U.S. Environmental Protection Agency. Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of Scientific and Technological Information. OAQPS, NC, 1982c August.

U.S. Environmental Protection Agency. NAAQS Environmental Impact Statement for NO_2. Research Triangle Park, NC 1982d.

U.S. Environmental Protection Agency. Cost and Economic Assessment of Regulatory Alternatives for NO_2 NAAQS (DRAFT). Research Triangle Park, NC 1982e.

U.S. Environmental Protection Agency. National Air Pollutant Emissions Estimates, 1940 - 1980. Monitoring and Data Analysis Division 1982f January.

U.S. Environmental Protection Agency. Regulation of Fuel and Fuel Additives. 47 Federal Register 49382, 1982g October 28.

U.S. Environmental Protection Agency. Motor Vehicle Tampering Survey 1982. National Enforcement Investigation Center, 1983a April.

U.S. Environmental Protection Agency, OAR, OMS. Anti-Tampering and Anti-Misfueling Programs to Reduce In-Use Emissions from Motor Vehicles. 1983b December 31.

U.S. Environmental Protection Agency. The API Study and Its Possible Human Health Implications. memo from Al Lorang (Chief, Technical Support Staff) to Charles Gray, Jr. (Director, Emission Control Technology Division), 1983c May 16.

U.S. Environmental Protection Agency, Environmental Criteria and Assessment Office. Air Quality Criteria for Lead. 1983d.

U.S. Environmental Protection Agency, Office of Mobile Source Air Pollution Control, Standards Development and Support Branch. Issues Analysis - Final Heavy-Duty Engine HC and CO Standards. U.S. EPA; Ann Arbor, Michigan 1983e.

U.S. Environmental Protection Agency. Preliminary Draft Revised Air Quality Criteria for Ozone and Other Photochemical Oxidants. Office of Research and Development 1983f.

U.S. Environmental Protection Agency. The Feasibility, Cost, and Cost-Effectiveness of Onboard Vapor Control. prepared by Glenn Passavant, 1984a March.

U.S. Environmental Protection Agency. VOC/Ozone Relationships from EKMA. memo from Warren Freas (Air Management Technology Branch) to Alan McGartland (Benefits Branch), 1984b January 27.

U.S. Environmental Protection Agency. Response to Public Comments on EPA's Listing of Benzene Under Section 112 and Relevant Procedures for the Regulation of Hazardous Air Pollutants. OAQPS, 1984c.

U.S. Environmental Protection Agency, Office of Air, Noise and Radiation. Regulation of Fuels and Fuel Additives: Lead Phase Down. Federal Register 1984d August 2; 31032.

U.S. Environmental Protection Agency. Motor Vehicle Tampering Survey 1983. National Enforcement Investigation Center, 1984e.

U.S. Environmental Protection Agency, Office of Policy Analysis. Preliminary Regulatory Impact Analysis of Proposed Rules Limiting the Lead Content of Gasoline. 1984f July 23.

U.S. Environmental Protection Agency. Regulatory Impact Analysis Guidelines. 1984g.

U.S. Environmental Protection Agency. Draft Revised Air Quality Criteria for Ozone and Other Photochemical Oxidants. Office of Research and Development 1984h.

U.S. Environmental Protection Agency. Regulation of Fuels and Fuel Additives; Banking of Lead Rights. 50 Federal Register 718, 1985 January 4.

U.S. Office of Management and Budget. Interim Regulatory Impact Analysis Guidance. 1981, p. 4.

Victery W, Vander AJ, Shulak JM, Schoeps P, Juluis S. Lead, hypertension, and the renin-angiotensin system in rats. Journal Labortory Clinic Medical 1982: 99: 354-62.

Violette, Chestnut. Valuing Reductions in Risks: A Review of the Empirical Estimates. 1983.

Viscusi WK. Labor market valuations of life and limb: empirical evidence and policy implications. Public Policy 1978; 26: 359-86

Von Nieding G, et al. Minimum concentration of NO_2 causing acute effects on the respiratory gas exchange and airway resistance in patients with chronic bronchitis. Int Arch Arbeitsmed 1971; 27:338-48. U.S. Environmental Protection Agency, Research Triangle Park, NC.

Von Nieding G, et al. Studies of the acute effect of NO_2 on lung function: influence on diffusion, perfusion and ventilation in the lungs. Int Arch Arbeitsmed 1973; 31:61-72.

Von Nieding G, et al. Acute effects of ozone on lung function of men. VDI-Ber 1977; 270:123-9.

Wade WA III, et al. A study of indoor air quality. J Air Pollut Control Assoc 1975; 25-93-939.

Wagner TO. Motor gasoline and automotive air pollution. SAE Paper #710485, Society of Automotie Engineers, Warrendale, PA 1971.

Wagner TO, Russum LW. Optimum octane number for unleaded gasoline. SAE Paper #720552, Society of Automotive Engineers, Warrendale, PA 1973.

Ware JH, et al. Passive smoking, gas cooking and respiratory health of children living in six cities. America Review of Respiratory Diseases 1984; 129.

Weaver C. Particulate control technology and particulate standards for heavy-duty diesel engines. SAE Paper #840174, Diesel Particulate Traps: 140, Society of Automotive Engineers, Warrendale, PA 1984.

Weaver CS. The Effects of Low-Lead and Unleaded Fuels on Gasoline Engines. Prepared for: U.S. Environmental Protection Agency, Office of Policy Analysis. Energy and Resource Consultants, Inc, 1984b September 28.

Webb RC, Winquist RJ, Victery W, Vander AJ. In vivo and in vitro effects on lead on vascular reactivity in rats. AM J Physiol 1981; 24: H211-6.

Wedeen RP, Maesaka JK, Weiner B, Lipat GA, Lyons MM, Vitale LF, Joselow MM. Occupational lead nephropathy. American Journal of Medicine 1975; 59: 630-41.

Weekly Petroleum Status Report. 1984, various issues.

Weinstein MC, Stason WB. Allocation of Resources to Manage Hypertension. N Eng J Med 1977; 296(13): 732-9.

White JM, Harvey DR. Defective synthesis of A and B globin chains in lead poisoning. Nature (London) 1972; 236: 71-3.

Whittemore A, Kern EL. Asthma and air pollution in the Los Angeles area. American Journal of Public Health 1980; 70.

Wibberly DG, Khera AK, Edwards JH, Rushton DI. Lead levels in human placentae from normal and malformed births. Journal of Medical Genetics 1977; 14: 339.

Williams BJ, Griffith WH III, Albrecht CM, Pirch JH, Hejtmancik MR Jr. Effects of chronic lead treatment on some cardiovascular responses to norepinephrine in the rat. Toxicology Appl Pharmacol 1977; 40: 407-13.

Williams BJ, Goldman D, Hejtmancik MR, Ziegler MG. Noradrenergic effects of lead in neonatal rat. Pharmacology 1978; 20(3): 186.

Williams BJ, Hejtmancik M. Time and level of perinatal lead-exposure for development of norepinephrine cardiotoxicity. Res Comm CP 1979; 24(2): 367-76.

Willits N, Ott W. Modeling the dynamic response of an automobile for air pollution exposure studies. Environmetrics, Summaries of Conference Presentations 1981; 81: 104-5.

Winneke G, Brockhaus A, Baltissen R. Nerve behavioral and systemic effects of longterm blood lead elevation in rats. Arch Toxicol 1977; 37: 247-63.

Winneke G. Impaired intelligence in children from environmental lead (letter). Mun Med Woc 1979; 121(26): 865.

Winneke G, Lilienthal H, Werner W. Task dependent nerve behavioral effects of lead in rats. Arch Toxicol 1982a: Supp 5: 84-93.

Winneke G, Hrdina K-G, Brockhaus A. Neuropsychological studies in children with elevated tooth-lead concentrations. part I: pilot study. Int Arch Occupational and Environmental Health 1982b; 51: 169-83.

Winneke G, Kramer U, Brockhaus A, Ewers U, Kujanek G, Lechner H, Janke W. Neuropsychological studies in children with elevated tooth lead concentrations. Part II: extended study. Int Arch Occup Environ Health 1983; 51: 231-52.

Winneke G, Beginn U, Ewert T, Havestadt C, Kramer U, Krause C, Thron HL, Wagner HM. Studie zur erfassung subklinischer bleiwirkungen auf das nervensystem bei kindern mit bekannter pranataler exposition in Nordenham. [Study on the determination of subclinical lead effects on the nervous system of Nordenham children with known pre-natal exposure.] BGA-Berichte 1984.

Wintringham JS, et al. Car maintenance expense in owner service with leaded and nonleaded gasolines. SAE Paper #720499, Society of Automotive Engineers, Warrendale, PA 1972.

Wong GL. Actions of parathyroid hormone and 1,25-dihydroxcholecalciferol on citrate decarboxylation in osteoblast-like bone cells differ in calcium requirement and in sensitivity to trifluoperazinc. Calcif Tissue Int 1983; 35: 426-31.

World Health Organization, United Nations Environmental Program; Lead: Environmental Health Criteria 3, Geneva, Switzerland 1977.

Worthen RP, Tunnecliffe TN. Temperature controlled engine valves. SAE Paper #820501, Society of Automotive Engineers, Warrendale, PA 1982.

WR Grace, Davison Chemical Division. Davison Catalagram #69, 1984.

Yip R, Norris TN, Anderson AS. Iron status of children with elevated blood lead concentrations. Journal Pediatr (St. Louis) 1981; 98: 922-5.

Young CT, Grimes DA. Erosion mechanisms of automotive spark plug electrodes. SAE Paper #780330, Society of Automotive Engineers, Warrendale, PA 1978.

Yule W, Landsdown R, Millar IB, Urbanowicz MA. The relationship between blood lead concentrations, intelligence and attainment in a school population: A pilot study. Dev Medical Child Neurology 1981; 23:567-76.

Yule W, Lansdown R. Lead and children's development: recent findings. presented at international conference: Management and Control of Heavy Metals in the Environment, 1983 September; Heidelberg, West Germany.

Yule W, Urbanowicz MA, Lansdown R, Millar IB. Teacher's ratings of children's behaviour in relation to blood lead levels. British Journal of Developmental Psychology 1984.

Zagraniski R, Leaderer B, Stolwuk J. Ambient sulfates, photochemical oxidants and acute adverse health effects: an epidemiologic study. Environmental Research 1979; 19.

Ziskind RA, et al. Carbon Monoxide Intrusion in Sustained-Use Vehicles, #SAI-068-80-535. U.S. EPA; Research Triangle Park, NC, 1979 November.

APPENDIX A

REFINERY PROCESSES

In refining, crude oil is first separated by molecular size into fractions, each of which can be blended directly into final petroleum products or processed further. In the downstream processing operations, the molecular size and structure of petroleum fractions are altered to conform to desired characteristics of refined products. Table II-1 in the text classifies the various refinery processes according to their principal functions. The actual processing configuration will depend on the characteristics of the crude oil processed and on the desired final product mix. These major processing steps are described briefly below.

Fluid Catalytic Cracking uses high temperature in the presence of a catalyst to convert or "crack" heavier fractions into lighter products, primarily gasoline and distillates. Feed is brought to process conditions (1000° F and 20 pounds per square inch pressure [psi]) and then mixed with a powdered catalyst in a reaction vessel. In the reactor, the cracking process is completed and the hydrocarbon products pass to a fractionating section for separation.

Coke, a coal-like by-product, is formed on the catalyst as a result of the cracking reaction. Coked catalyst is transferred from the reactor to a regenerator vessel where air is injected to burn the coke to CO and CO_2. The regenerator flue gases are passed through cyclones and, sometimes, electrostatic precipitators, to remove entrained catalyst. They are then vented to the

atmosphere or sent to a CO boiler where carbon monoxide is burned to produce CO_2. The regenerated catalyst is returned to the reactor.

Hydrotreating (also known as hydrodesulfurization) is a catalytic process designed to remove sulfur, nitrogen, and heavy metals from petroleum fractions. Feed is heated to process temperatures (650° to 705°F), mixed with hydrogen, and fed to a reactor containing a fixed bed of catalyst. The primary reactions convert sulfur compounds in the feed to hydrogen sulfide (H_2S) and the nitrogen compounds to ammonia. The H_2S and ammonia are separated from the desulfurized product; the H_2S is sent to sulfur recovery facilities.

Catalytic reforming is used to upgrade low-octane naphtha to produce high-octane gasoline blending stocks. The flow pattern is similar to that of hydrotreating except that several reactor vessels are used. The required temperature is about 1000°F and the required pressure is about 200 pounds per square inch. Reforming catalysts are readily poisoned by sulfur, nitrogen, or heavy metals, and therefore the feed is normally hydrotreated before being charged to the reforming unit.

In hydrocracking the cracking reaction takes place in the presence of hydrogen. The process produces high quality desulfurized gasoline and distillates from a wide variety of feedstocks. The process employs one or more fixed bed reactors and is similar in flow to the hydrotreating process. Process conditions are 800° F and 2000 psi. Like hydrotreating, hydro-

cracking produces by-product H_2S, which is diverted to sulfur recovery.

Coking is another type of cracking which does not employ a catalyst or hydrogen. The process is utilized to convert heavy fuel oils into light products and a solid residue (coke). Feed is brought to process conditions (900°F and 50 psi) and fed to the coking vessel. Cracked products are routed to a fractionation section. Coke accumulates in the vessel and is drilled out about once a day. In one version of the coking process, fluid coking, a portion of the coke is used for process fuel and the balance is removed as small particles.

Acid gas treating and sulfur recovery units are used to recover hydrogen sulfide (H_2S) from refinery gas streams and convert it to elemental sulfur. Sour gas containing H_2S is produced in several refinery units, particularly cracking and hydrotreating. In the acid gas treating units, H_2S is removed from the fuel gas by absorbing it in an alkaline solution. This solution, in turn, is heated and steam-stripped to remove the H_2S to form sulfur and water. Sulfur recovery is high but never 100%. The remaining sulfur is incinerated and discharged to the atmosphere or removed by a tail gas treating unit.

The purpose of the tail gas treating unit is to convert any remaining sulfur compounds from the sulfur recovery unit to elemental sulfur. There are several processes available, the most common of which are the Beavon and SCOT processes. In both processes, sulfur compounds in the sulfur unit tail gas are

converted to H_2S. The Beavon process converts H_2S to sulfur through a series of absorption and oxidation steps. The SCOT process concentrates the H_2S and returns it to the sulfur recovery facilities. In both processes, the treated tail gas is virtually free of sulfur compounds when released to the atmosphere.

APPENDIX B

THE FLEET MODEL

We developed a fleet model to predict the number of cars and gasoline powered trucks (for six weight classes) on the road; the model also forecasts the number of miles driven per year for each vehicle type for the years 1985-1992. In this document we have used our fleet model for several different purposes. Estimates of total miles driven per year by vehicle type, age and fuel consumed are used to estimate the maintenance savings attributable to the use of lower lead or unleaded fuel. Estimates of lifetime vehicle-miles traveled by new misfuelers in each year provide the basis for estimating the value of the reduced emissions due to reduced misfueling.

B.1 Vehicles on the Road

The extrapolation procedure for total vehicle miles per year by age and type of vehicle relies on two basic sets of data:

1) Vehicles on-the-road for 1968-1983 as published annually by the Motor Vehicle Manufacturers Association(MVMA) in Motor Vehicle Facts and Figures. Data are available for cars (Table B-1) and trucks (Table B-2) ages 1 to 15 years, as well as 16 and older. No breakdown of trucks by weight and engine type (diesel versus gasoline) is available, however.
2) Data Resources, Inc. (DRI) projections of the total fleet size for cars, as well as annual sales for cars and trucks.

Using these data sets, the fleet model projected how many cars and trucks (by age) would be on the road for each of the years, 1985 through 1992. For trucks, the projections reflect only

gasoline-powered vehicles and the fleet is divided into six weight classes.

The model required two sets of inputs: an inventory of vehicles on the road by age and a set of survival rates for determining how many vehicles in each age category would still be on the road the next year. The initial inventory for vehicles aged 'New' through 15 years of age is taken from the 1983 column in Tables B-1 and B-2. The category of vehicles aged 16 and over is broken into 15 additional age categories. The procedures we used are discussed in sections B.1.a for cars and B.1.b. for trucks.

The survival rates used in the forecast are based on an analysis of historical survival rates. These historical survival rates are derived by dividing the number of vehicles in a model year cohort in one year by the number of vehicles in the same cohort the previous year. For cars, the historical survival rates are given in Table B-3, while Table B-4 presents the rates for trucks. As both tables show, there is significant variation in the rates over time. A statistical analysis of the data showed that both the overall scrappage rate (defined as total vehicle retirements divided by fleet size) and the fraction of the total fleet that each model year comprised were important explanatory variables. So, for the forecast, we adjusted the survival rates to reflect changes in the scrappage rates and the relative size of the cohort in each year. As explained below, the scrappage rates used for cars are derived from the DRI

Table B-1 Cars on the Road 1968-1983

Year	1968	1969	1970	1971	1972	1973	1974	1975
Age of Car								
New	6182	6467	6288	5927	7169	7988	6433	4684
1	8122	8927	9299	8888	8915	10158	11269	9763
2	8836	8054	8816	9280	8851	8715	10147	11332
3	8939	8798	7878	8802	9122	8612	8622	10098
4	7667	8855	8538	7772	8596	8881	8493	8549
5	7058	7532	8506	8313	7499	8291	8615	8341
6	6183	6829	7116	8171	7930	7120	7931	8339
7	4657	5804	6268	6651	7583	7333	6624	7556
8	4615	4087	5058	5624	5920	6715	6531	6113
9	3347	3726	3267	4274	4713	4963	5710	5796
10	1709	2452	2776	2525	3343	3698	3976	4825
11	1990	1188	1692	2035	1824	2470	2824	3234
12	1612	1421	799	1183	1413	1268	1813	2229
13	1496	1139	996	563	805	967	901	1407
14	743	1063	794	730	389	548	682	689
15	623	525	753	580	526	274	391	523
16+	1517	1578	1583	1804	1813	1780	1621	1742
Total	75358	80449	80449	83138	86439	89805	92608	95241

Year	1976	1977	1978	1979	1980	1981	1982	1983
Age of Car								
New	6472	7177	7426	7288	5868	5140	4399	5044
1	7683	9557	10382	10699	10402	8818	8280	7429
2	9746	7477	9483	10219	10483	10245	8825	8273
3	11130	9594	7291	9203	9931	10290	10075	8749
4	9872	10854	9431	6990	8900	9758	10155	10014
5	8249	9563	10559	9004	6682	8735	9661	10038
6	7966	7866	9140	9965	8499	6463	8471	9434
7	7774	7449	7326	8431	9151	8050	6190	8195
8	6856	6963	6784	6573	7544	8458	7498	5867
9	5361	5859	6087	5909	5653	6791	7629	6885
10	4888	4416	4917	5034	4939	4929	5989	6798
11	3923	3887	3589	3999	4049	4238	4243	5239
12	2578	3023	3093	2862	3172	3369	3581	3632
13	1740	1969	2369	2460	2280	2635	2822	3052
14	1083	1315	1545	1874	1969	1910	2208	2395
15	526	818	1021	1223	1516	1654	1609	1869
16+	1943	2093	2496	2930	3514	4346	5220	6037
Total	97818	99904	102957	104677	104564	105839	106867	108961

Table B-2 Trucks on the Road 1968-1983

Year	1968	1969	1970	1971	1972	1973	1974	1975
Age of Truck								
NEW	1058	1262	1263	1193	1637	1883	1834	1326
1	1452	1581	1881	1736	1784	2385	2829	2739
2	1512	1447	1536	1872	1744	1753	2396	2848
3	1380	1495	1428	1496	1858	1709	1742	2384
4	1208	1357	1483	1398	1468	1825	1698	1730
5	1035	1177	1339	1441	1372	1446	1804	1668
6	889	1004	1154	1298	1409	1336	1418	1779
7	692	860	975	1112	1260	1363	1305	1395
8	749	661	826	927	1066	1208	1309	1273
9	682	706	621	774	877	1005	1145	1256
10	461	632	658	585	721	817	941	1085
11	508	421	583	610	525	663	752	884
12	514	459	383	532	544	474	603	697
13	537	459	417	347	471	487	427	554
14	370	480	414	376	303	419	434	388
15	425	330	432	369	328	268	370	391
16+	2182	2242	2278	2383	2380	2356	2289	2393
TOTAL	15685	16586	17686	18465	19773	21412	23312	24813

Year	1976	1977	1978	1979	1980	1981	1982	1983
Age of Truck								
NEW	1893	2177	2533	2402	1362	1244	1291	1564
1	2148	2746	3240	3541	3765	2225	2099	2214
2	2732	2109	2743	3231	3663	3808	2183	2106
3	2799	2689	2076	2679	3332	3633	3817	2160
4	2346	2752	2656	2006	2750	3318	3621	3779
5	1697	2291	2681	2589	2046	2722	3297	3566
6	1635	1639	2227	2587	2641	2005	2689	3230
7	1731	1573	1567	2140	2609	2567	1950	2615
8	1345	1645	1509	1501	2163	2516	2469	1887
9	1220	1267	1554	1435	1540	2078	2399	2368
10	1191	1129	1189	1459	1429	1438	1942	2247
11	1024	1096	1043	1111	1452	1337	1364	1848
12	828	922	998	958	1082	1348	1258	1288
13	642	736	832	906	919	999	1256	1184
14	503	566	663	749	856	842	930	1177
15	351	442	506	595	688	779	779	870
16+	2455	2422	2531	2687	2948	3192	3628	4026
TOTAL	26560	28222	30565	32583	35268	36069	36987	38143

Table B-3 Year to Year Survival Rates for Cars(historical)

Year	1968 -1969	1969 -1970	1970 -1971	1971 -1972	1972 -1973	1973 -1974	1974 -1975	1975 -1976
Age of Car								
New-1	1.242	1.284	1.411	1.161	1.238	1.315	1.470	1.188
1-2	.992	.988	.998	.996	.978	.999	1.006	.998
2-3	.996	.978	.998	.983	.973	.989	.995	.982
3-4	.991	.970	.987	.977	.974	.986	.992	.978
4-5	.982	.961	.974	.965	.965	.970	.982	.965
5-6	.968	.945	.961	.954	.949	.957	.968	.955
6-7	.939	.918	.935	.928	.925	.930	.953	.932
7-8	.878	.871	.897	.890	.886	.891	.923	.907
8-9	.807	.799	.845	.838	.838	.850	.887	.877
9-10	.733	.745	.773	.782	.785	.801	.845	.843
10-11	.695	.690	.733	.722	.739	.764	.813	.813
11-12	.714	.673	.699	.694	.695	.734	.789	.797
12-13	.707	.701	.705	.680	.684	.711	.776	.781
13-14	.711	.697	.733	.691	.681	.705	.765	.770
14-15	.707	.708	.730	.721	.704	.714	.767	.763
15-16+	2.533	3.015	2.396	3.126	3.384	5.916	4.455	3.715

Year	1976 -1977	1977 -1978	1978 -1979	1979 -1980	1980 -1981	1981 -1982	1982 -1983
Age of Car							
New-1	1.260	1.238	1.261	1.299	1.309	1.293	1.241
1-2	.973	.992	.984	.980	.985	1.001	.999
2-3	.984	.975	.970	.972	.982	.983	.991
3-4	.975	.983	.959	.967	.983	.987	.994
4-5	.969	.973	.955	.956	.981	.990	.988
5-6	.954	.956	.944	.944	.967	.970	.977
6-7	.935	.931	.922	.918	.947	.958	.967
7-8	.896	.911	.897	.895	.924	.931	.948
8-9	.855	.874	.871	.860	.900	.902	.918
9-10	.824	.839	.827	.836	.872	.882	.891
10-11	.795	.813	.813	.804	.858	.861	.875
11-12	.771	.796	.797	.793	.832	.845	.856
12-13	.764	.784	.795	.797	.831	.838	.852
13-14	.756	.785	.791	.800	.838	.838	.849
14-15	.755	.776	.792	.809	.840	.842	.846
15-16+	3.979	3.051	2.870	2.873	2.867	3.156	3.752

Table B-4 Truck Year to Year Survival Rates (historical)

Year	1968 -1969	1969 -1970	1970 -1971	1971 -1972	1972 -1973	1973 -1974	1974 -1975	1975 -1976
Age of Truck								
New-1	1.151	1.273	1.278	1.133	1.191	1.171	1.333	1.123
1-2	.997	.972	.995	1.005	.983	1.005	1.007	.997
2-3	.989	.987	.974	.993	.980	.994	.995	.983
3-4	.983	.992	.979	.981	.982	.994	.993	.984
4-5	.974	.987	.972	.981	.985	.988	.982	.981
5-6	.970	.980	.969	.978	.974	.981	.986	.980
6-7	.967	.971	.964	.971	.967	.977	.984	.973
7-8	.955	.960	.951	.959	.959	.960	.975	.964
8-9	.943	.939	.937	.946	.943	.948	.960	.958
9-10	.927	.932	.942	.932	.932	.936	.948	.948
10-11	.913	.922	.927	.897	.920	.920	.939	.944
11-12	.904	.910	.913	.892	.903	.910	.927	.937
12-13	.893	.908	.906	.885	.895	.901	.919	.921
13-14	.894	.902	.902	.873	.890	.891	.909	.908
14-15	.892	.900	.891	.872	.884	.883	.901	.905
15-16+	5.275	6.903	5.516	6.450	7.183	8.541	6.468	6.279

Year	1976 -1977	1977 -1978	1978 -1979	1979 -1980	1980 -1981	1981 -1982	1982 -1983
Age of Truck							
New-1	1.123	1.149	1.132	1.414	1.187	1.233	1.153
1-2	.982	.999	.997	1.034	1.011	.981	1.003
2-3	.984	.984	.977	1.031	.992	1.002	.989
3-4	.983	.988	.966	1.027	.996	.997	.990
4-5	.977	.974	.975	1.020	.990	.994	.985
5-6	.966	.972	.965	1.020	.980	.988	.980
6-7	.962	.956	.961	1.009	.972	.973	.972
7-8	.950	.959	.958	1.011	.964	.962	.968
8-9	.942	.945	.951	1.026	.961	.953	.959
9-10	.925	.938	.939	.996	.934	.935	.937
10-11	.920	.924	.934	.995	.936	.949	.952
11-12	.900	.911	.919	.974	.928	.941	.944
12-13	.889	.902	.908	.959	.923	.932	.941
13-14	.882	.901	.900	.945	.916	.931	.937
14-15	.879	.894	.897	.919	.910	.925	.935
15-16+	6.900	5.726	5.310	4.955	4.640	4.657	5.168

forecast of sales and fleet size for cars. Since DRI does not forecast fleet size for trucks, we modified the adjustment procedure for truck year to year survival rates to use car scrappage rates. A detailed description of the adjustment procedure is given in the next two sections.

B.1.a Projection Model for Cars on the Road

We begin with a description of the projection procedure for cars. Let C_{ij} denote cars of age i in year j. Thus C_{10} 1985 represents the number of ten year old cars in 1985. Then, letting S_{ij} be the probability of a car of age i in year j still being on the road in year j+1, we have $C_{i+1,j+1} = C_{ij} \times S_{ij}$. Further, we have

$$R_j = \underset{i}{SUM}\ C_{ij} \times (1 - S_{ij})$$

where R_j is total retirements in the fleet in year j. However, we also have

$$R_j = F_{j+1} - F_j + N_j,$$

where F_j is the size of the fleet in year j and N_j is new car sales in that year. Thus, we have two equations which determine retirements and these must be consistent if the model is to operate properly. We used the DRI forecast as the source of the overall loss rates and adjusted year to year survival rates until they gave the same total losses as the DRI forecast.

We adjusted age specific survival rates by using the regression coefficient of the scrappage rate in the model

(B.1) $$S_{ij} = a_o + bSR_j + c(C_{ij}/\underset{j}{SUM}\ C_{ij}) - e$$

where SR_j is the overall scrappage rate for year j and

$C_{ij}/SUM_{j} V_{ij}$ is the fraction of the fleet that vintage i is of the fleet in year j. The survival rates were varied by changing the scrappage rate in equation B.1 until the total losses from the survival rates were within 0.01% of those in the DRI forecast.

In equation B.1 the scrappage rate value necessary to achieve equality in total losses with the DRI forecast was typically larger than the DRI scrappage rate. Thus, the survival rate model overpredicts the size of the fleet in the next year when compared to the DRI forecast. However, the advantage of adjusting using the model is that the survival rates maintain a consistent pattern by vehicle age as model forecasts of retirements from each cohort in the fleet are changed in order to obtain equality with the DRI forecast.

For the car fleet, the adjustment equations were estimated using sixteen years of data from the MVMA for cars on the road in July of each year. The resulting estimates are given in Table B-5. For forecasting purposes, the results of the regression analysis were not used for the first two age groups. Because new cars on the road on July 1st of the year fluctuated sharply as a percentage of total car sales for the model year, we assumed they were equal to 75% of total sales for that model year. One year old cars were assummed to be 99.9 percent of the appropriate new car model year sales and two year old cars were assumed to be 99.5 percent of the original sales level. One and two year old cars show little variation in survival rates and the correction mechanism occasionally gave survival rates greater than one -- therefore these rates were imposed.

Table B-5. Regression Coefficients for the Car Year to Year Survival Rates

Survival Rate	Constant	Scrappage Rate	Cohort Weight
New-1	1.426	-.000239	-1.880
1-2	1.009	-.003030	.0721
2-3	.9854	-.003828	.2983
3-4	1.019	-.004151	-.05617
4-5	.9967	-.004715	.1383
5-6	.9857	-.004911	.1298
6-7	1.013	-.006537	-.3060
7-8	1.013	-.006537	-.7581
8-9	.8523	-.01218	1.619
9-10	.5212	-.001298	5.467
10-11	.5580	-.004541	5.969
11-12	.6533	-.008412	5.550
12-13	.6067	-.000991	6.832
13-14	.6810	-.005657	7.202
14-15	.7057	-.004773	7.637

The other two pieces of information necessary to start the forecast procedure were a 1983 inventory of vehicles and an initial set of survival rates. The initial inventory data were extended from the 15 years available in the data to 30 years. The extension was necessary because consumers have been keeping their cars longer, and consequently, older vehicle have been steadily growing as a fraction of the fleet. As Tables B-1 and B-2 indicate, the cohort of vehicles aged 16 years old and greater has been growing in size during the last 15 years, in both absolute and relative terms.

We began the inventory extension by noting, as Tables 3 and 4 show, that the survival rates for vehicles aged 11 through 15 were similar in size and showed a consistent pattern of change from year to year. Therefore, we assumed that the survival rate for older vehicles was equal to the rate for 14 to 15 year-old cars and that it changed in the same way as this rate did in response to a change in the overall scrappage rate. Using this assumption, we constructed our estimates of 15 through 30 year old cars in 1983 iteratively. We began in 1968 with 15 year old cars and estimated the number of 16 year old cars in 1969 using the survival rate for 14 to 15 year old cars for 1968 to 1969. Thus, in 1969 we had an estimate for 15 and 16 year old cars. We then repeated the same process for two cohorts to get estimates of 15, 16 and 17 year-old cars in 1970. A similar process was carried on through 1983 to give us estimates of cars 15 through 30 years of age. In 1983, the total number of cars resulting from our estimate of the individual cohorts was 5 percent less

than the actual total for cars over 15 years of age. We corrected for this error by adjusting each age group by

(B.2) $(1 + r)i - 15,$

where i is the age of the cohort and r was chosen to yield the desired equality. This adjusts older vehicles more than younger vehicles to reflect their longer extrapolation period. The value for r was .0181 for cars. The largest correction, for the oldest cohort, was a 31 percent increase.

The other required set of initial information was the 1983-1984 survival rates. These were the 82 to 83 set with rates for age 15 and older cohorts set to 0.84. This was the rate for 14 year old cars in 1983. This was was used because the assumption had been used in the inventory extension process and had worked quite well.

With these two inputs determined, the model could forecast the total number cars on the road for each year from 1985 through 1992; the results are in Table B-6. As the table shows the number of older vehicles continues to grow. By 1992, cohorts aged 16 and older exceed 12 million cars and are nearly 10 percent of the fleet. While this is to some extent an artifact of the extrapolation procedure, the overall size of the pool of cars over 15 years of age is consistent with existing behavior within the fleet and the DRI forecast of cars on the road.

B.1.b Modification of the Model for Trucks

For trucks, it was necessary to modify the car forecasting procedure because DRI does not provide a forecast of the size of

Table B-6 Predicted Number of Cars on the Road 1984-1992

Year	1984	1985	1986	1987	1988	1989	1990	1991	1992
Age of Car									
NEW	7950	7950	7875	8100	8400	8625	8850	9000	9075
1	9191	10589	10589	10490	10789	11189	11489	11788	11988
2	7392	9145	10536	10536	10438	10735	11133	11432	11729
3	8117	7256	9016	10408	10396	10288	10558	10936	11219
4	8526	7915	7109	8852	10207	10182	10053	10303	10661
5	9620	8196	7651	6888	8565	9862	9812	9673	9901
6	9545	9154	7844	7341	6600	8194	9409	9346	9202
7	8741	8852	8554	7355	6870	6164	7625	8737	8664
8	7345	7842	8007	7765	6663	6211	5550	6850	7835
9	4988	6257	6787	6978	6741	5760	5328	4740	5830
10	5993	4342	5456	5923	6087	5878	5018	4640	4126
11	5715	5042	3675	4631	5019	5150	4959	4226	3903
12	4317	4716	4208	3083	3874	4186	4271	4100	3485
13	3070	3650	3993	3565	2611	3279	3541	3612	3466
14	2525	2543	3046	3344	2980	2178	2725	2937	2990
15	1973	2082	2111	2535	2779	2472	1801	2250	2421
16	1539	1626	1727	1756	2106	2304	2043	1486	1853
17	1326	1268	1349	1437	1459	1746	1904	1685	1224
18	979	1092	1052	1122	1194	1210	1443	1570	1388
19	861	807	906	875	932	990	1000	1190	1293
20	650	709	669	754	727	773	818	825	980
21	422	536	588	557	626	603	639	675	679
22	279	348	445	489	463	519	498	527	556
23	172	230	289	370	406	384	429	411	434
24	86	142	191	240	307	337	317	354	338
25	67	71	118	159	199	255	278	261	292
26	36	55	59	98	132	165	211	229	215
27	18	30	46	49	81	109	136	174	189
28	26	15	25	38	41	67	90	112	143
29	21	21	12	21	32	34	55	74	92
30	20	17	17	10	17	27	28	45	61
Tot	111510	112498	113950	115769	117741	119876	122011	124188	126232

the truck fleet. Rather than attempt to develop an independent estimate of the total size of the fleet, we assumed that the year to year survival rates were a function of the scrappage rates observed in the car fleet. Since these can he derived from the DRI forecast and from historical data, we can estimate the size of the fleet by adding trucks surviving from the previous year to new trucks delivered. The precise details are given below.

We regressed truck survival rates on car scrappage rates and the fraction of the fleet that the truck cohort of age i was of all trucks. Thus,

(B.3) $$S_{ijT} = a + bSR_{j,cars} + c(T_{ij}/\text{SUM}_{i} T_{ij}) + e$$

Because the truck fleet data for 1980 reflect the reclassification of all passenger vans from cars to trucks, we excluded the survival rates from 1979 to 1980 from the data base used for the regressions. The regression results are presented in Table B-7. The regression coefficients for SRj are used to adjust the survival rates in forecast years for each truck cohort. Aggregate fleet size is the sum of the individual cohorts plus new truck sales for the year. As in the car model, new trucks on the road were assumed to be 75 percent of model year sales and one year-old trucks were assumed to be 99.9 percent of original sales. Trucks on the road were extended to 30 years of age using the same process as for cars. The extrapolation resulted in a 17 percent overestimate of the fleet between 16 and 30 years of age. The adjustment coefficient (r) in equation B.2 was -.019, which resulted in a 25 percent reduction in the oldest cohort.

Table B-7 Regression Coefficients for Truck Year to Year Survival Rates

Survival Rate	Constant	Scrappage Rate	Cohort Weight
New-1	1.227	.007742	-1.079
1-2	1.018	-.004245	.1060
2-3	1.000	-.000509	-.08927
3-4	.9843	-.000364	.05054
4-5	.9504	.001117	.2416
5-6	.9824	-.000491	-.02511
6-7	.9799	-.000722	-.06452
7-8	.9589	.000908	.1289
8-9	.9509	-.002385	.2915
9-10	.9503	-.001290	-.07566
10-11	.8771	-.001353	1.326
11-12	.8680	-.002685	1.813
12-13	.8721	-.001531	1.483
13-14	.8759	-.002938	1.756
14-15	.8784	-.003051	1.781

The initial set of survival rates for trucks for 1983-84 was taken to be the same as the 82-83 rates. However, the rates for cohorts age 16 and greater were not set equal to the 14-15 rate for 1983 for two reasons. First, in the inventory extrapolation, it was necessary to revise the older cohorts downward significantly to make them equal to the number of trucks 16 years of age and older in 1983. Secondly, there was no overall forecast to constrain the year to year survival rates, so it was essential to reflect the information from the extrapolation process in the survival rates. The rates were set to decline from 0.93 to 0.82 as cohorts ranged from 16 to 30 years of age. As seen in Table B-8, even with this reduction, older trucks become about one sixth of the fleet by 1992. Further, the total number of trucks increases substantially relative to cars during the forecast period.

Once total trucks on the road were determined, several additional adjustments to the data were made. First, total trucks on the road had to be disaggregated by weight class. Trucks were divided into six weight classes, derived from the usual eight classes by combining classes three through five into one class. We did this because classes three(l0,000 to 14,000 pounds), four (14,000 to 16,000 pounds) and five (16,000 to 19,500 pounds) are very small. Currently, they constitute less than one tenth of 1 percent of all trucks sold.

The adjustment procedure required the weight composition of new trucks on the road in every year from 1954 to 1992. For the years from 1968 through 1983, we used published data on truck

Table B-8 Predicted Trucks on the Road 1984-1992

Year	1984	1985	1986	1987	1988	1989	1990	1991	1992
Age of Truck									
New	3139	3340	3275	3468	3670	3830	3995	4148	4295
1	3139	4081	4299	4243	4499	4757	4963	5171	5368
2	2189	3102	4034	4250	4194	4447	4702	4905	5111
3	2084	2165	3069	3991	4205	4150	4400	4652	4853
4	2130	2058	2137	3029	3939	4150	4096	4343	4592
5	3700	2084	2014	2092	2964	3856	4062	4009	4250
6	3464	3591	2024	1956	2031	2879	3744	3944	3892
7	3122	3345	3469	1955	1890	1962	2780	3616	3809
8	2500	2975	3192	3312	1867	1804	1873	2654	3451
9	1764	2333	2779	2982	3094	1744	1685	1749	2479
10	2249	1673	2214	2638	2830	2936	1655	1599	1660
11	2114	2109	1571	2080	2477	2658	2757	1554	1501
12	1736	1981	1978	1474	1952	2325	2494	2587	1458
13	1202	1613	1845	1843	1373	1818	2165	2323	2409
14	1103	1115	1500	1716	1714	1277	1690	2012	2158
15	1090	1017	1030	1386	1586	1584	1180	1561	1859
16	801	1000	935	947	1274	1458	1456	1084	1435
17	731	731	914	855	866	1165	1333	1330	991
18	591	663	664	831	777	788	1059	1211	1209
19	525	533	600	601	751	703	712	957	1095
20	415	471	480	539	540	676	632	640	861
21	314	371	421	429	482	483	604	565	572
22	232	277	328	372	379	426	427	534	499
23	173	202	243	286	325	332	373	373	467
24	120	149	175	210	248	281	286	322	323
25	117	103	127	149	179	212	240	245	275
26	95	99	87	107	126	151	179	203	206
27	59	79	82	72	90	105	126	149	169
28	61	48	65	68	60	74	87	104	123
29	60	50	39	53	55	48	60	71	85
Tot	41020	43360	45590	47938	50440	53078	55812	58614	61453

sales by weight class. These data were converted to percentages and applied to new trucks on the road to yield estimates of the fleet by weight. For the years before 1968, we assumed a constant composition of the fleet at 1968 levels. For the years after 1983, we used DRI's decomposition of new truck sales into light trucks (those under 14000 pounds) and medium and heavy trucks (those over 14,000 pounds). We broke the light truck category into two classes light duty trucks classes 1 and 2. We used the 1981-1983 average ratio of class 1 to class 2 trucks and applied it to the DRI light trucks forecast to derive these two categories of trucks. We assumed there were no trucks between 10 and 14,000 pounds (1983 sales were 145). Similarly, we applied the average ratios for the other four weight categories to get a breakdown of the DRI forecast of medium and heavy trucks. The resulting percentage composition of the fleet is presented in Table B-9.

The other adjustment that we made in the data was to estimate the fraction of each class of truck that was diesel; we removed these from the fleet. For the years 1968 through 1983, we estimated the fraction of truck sales that were diesel from actual data. Because it is difficult to find a coherent set of data on all aspects of truck sales, we used total factory sales of diesels for the numerator and total sales of trucks for the denominator; this may understate the diesel fraction somewhat. For the years before 1968 we used the data in column two of Table B-10 as the fraction of each of the six truck classes that were diesel. These correspond to the actual ratios in 1965.

Table B-9 Fraction of Truck Fleet in Each Weight Class

Year	LDT1	LDT2	10,000 -19,500	19,500 -26,000	26,000 -33,000	33,000 plus
1995	.4874	.4448	.0007	.0182	.0199	.0291
1994	.4874	.4448	.0007	.0182	.0199	.0290
1993	.4874	.4448	.0007	.0181	.0198	.0290
1992	.4865	.4440	.0007	.0186	.0204	.0298
1991	.4862	.4437	.0008	.0188	.0205	.0300
1990	.4848	.4424	.0008	.0195	.0213	.0312
1989	.4833	.4410	.0008	.0203	.0222	.0324
1988	.4815	.4395	.0008	.0212	.0232	.0338
1987	.4814	.4393	.0009	.0212	.0232	.0340
1986	.4824	.4403	.0008	.0207	.0227	.0331
1985	.4879	.4453	.0007	.0179	.0196	.0286
1984	.4899	.4471	.0007	.0169	.0185	.0270
1983	.4850	.4453	.0005	.0172	.0219	.0301
1982	.4901	.4276	.0011	.0197	.0278	.0337
1981	.4543	.4309	.0014	.0365	.0261	.0509
1980	.4415	.4368	.0027	.0402	.0262	.0526
1979	.3929	.4865	.0065	.0451	.0153	.0536
1978	.3409	.5467	.0209	.0398	.0105	.0413
1977	.3746	.5172	.0128	.0469	.0082	.0404
1976	.4331	.4602	.0172	.0502	.0073	.0320
1975	.4684	.4048	.0142	.0675	.0098	.0354
1974	.5635	.2682	.0097	.0823	.0126	.0636
1973	.5825	.2556	.0239	.0682	.0142	.0557
1972	.5781	.2389	.0336	.0744	.0173	.0577
1971	.5828	.2369	.0442	.0644	.0177	.0539
1970	.5615	.2373	.0452	.0736	.0227	.0597
1969	.5830	.2106	.0514	.0766	.0173	.0610
1968	.5992	.2035	.0536	.0745	.0221	.0472
1967	.5812	.2171	.0500	.0749	.0207	.0560
1966	.5812	.2171	.0500	.0749	.0207	.0560
1965	.5812	.2171	.0500	.0749	.0207	.0560
1964	.5812	.2171	.0500	.0749	.0207	.0560
1963	.5812	.2171	.0500	.0749	.0207	.0560
1962	.5812	.2171	.0500	.0749	.0207	.0560
1961	.5812	.2171	.0500	.0749	.0207	.0560
1960	.5812	.2171	.0500	.0749	.0207	.0560
1959	.5812	.2171	.0500	.0749	.0207	.0560
1958	.5812	.2171	.0500	.0749	.0207	.0560
1957	.5812	.2171	.0500	.0749	.0207	.0560
1956	.5812	.2171	.0500	.0749	.0207	.0560
1955	.5812	.2171	.0500	.0749	.0207	.0560
1954	.5812	.2171	.0500	.0749	.0207	.0560

Table B-10. Fraction of Trucks that are Diesel by Weight Class

Weight Class	Before 1968	After 1983
0-6,000	0.0	0.02
6,000-10,000	0.001	.10 to .135
10,000-19,500	0.05	.00
19,500-26,000	0.06	.24
26,000-33,000	0.45	.54
33,000+	0.80	.975

For the years after 1983, we used the values given in column 3 of Table B-10 as the fraction of the fleet that will be diesel. These forecasts are the result of an inspection of historical data on diesel sales ratios and an overall forecast that the price of gasoline and diesel fuel relative to other goods is not going to rise significantly. Class two trucks are the only class that gives any indication of a growth in diesel penetration at the current time. Therefore, we increased diesel penetration rates by 0.05 per year for this class.

B.2 Miles Driven per Year by Vehicle Class

Data for cars and light duty trucks on miles driven per year are taken directly from the Transportation Energy Data Book (Department of Energy, 1982), For heavier trucks, only average miles per year for all trucks in the class are available in this reference. To develop a representation of how miles per year declined with the age of these vehicles, we used data from

Jambekar and Johnson (1978), and normalized this to the average found in the DOE report.

An important division of gasoline fueled vehicles for our purposes was the extent to which vehicles designed for unleaded fuel were misfueled with leaded gasoline. This was important to compute both the maintenance benefits and the benefits due to the reduction of emissions from engines that would have been misfueled in the absence of the rule. To calculate this, we estimated the extent of misfueling by vehicle age from EPA's 1983 survey of tampering and misfueling. This data was quite variable when broken down by age of vehicle, so we smoothed it by fitting a regression. The resulting regression is

$$MSFR = .0326 + .02314\ T$$

where MSFR is the misfueling rate by vehicle age and T is the age of the vehicle (T = 0 for new vehicles). The truck sample size in the survey was too small to support a separate estimate for them. Therefore, we used the same regression for trucks and cars although the available data suggests that trucks misfuel at a higher rate than cars.

B.3 Total Miles per Year for Maintenance Benefits

For maintenance benefits we had to compute the total mileage driven by both misfuelers and legal leaded-gasoline users in a given year. Miles driven by misfuelers were estimated by computing the fraction of misfuelers in each model year using the regression given above. Cars and light duty trucks over 10 years of age were assumed to experience no further increase in misfueling.

For legal leaded users, we used estimates of the legal leaded fleet and the corresponding estimates of miles driven per year. In Table B-11, we present the estimated miles per year traveled by cars and by light duty trucks classes 1 and 2 by leaded, misfueled, and unleaded status. Because we computed no maintenance benefits for heavier trucks or other gasoline powered vehicles, no estimates of usage are provided in this table. The results reflect the decline in the size of the fleet of legal leaded-gasoline users; the results also show how miles traveled by misfuelers are projected to grow over time.

B.4 Miles per Year for Conventional Pollutant Emissions

Emissions due to misfueling constitute the other major source of benefits calculated on the basis of the fleet model results. For conventional benefits estimates, we had to calculate the increase in misfueling for each model year in a given calendar year. For each year, the incremental increase in misfueling for a given model year is given by the slope coefficient. Since we restricted the increase in overall misfueling so that misfueling was constant for vehicles 10 years of age and older, there is no increase in misfueling for cars more than 10 years of age and, thus, no conventional pollutant benefits are attributed to these vehicles. We then computed the expected number of miles the additional misfuelers will travel each year for the next 20 years. These estimates are corrected to reflect expected loss rates and reductions in miles traveled as the vehicle ages. The resulting estimates are present valued to the year of initial

Table B-11 Mileage Estimates by Misfueling Status and Type of Vehicle (millions of miles per year)

Year	1984	1985	1986	1987	1988	1989
LEGAL LEADED						
CARS	312.454	250.001	199.925	159.257	125.563	98.420
LDT1	50.772	44.513	39.015	34.124	29.796	26.000
LDT2	77.076	71.901	67.040	62.740	59.971	57.729
MISFUELED						
CARS	119.747	132.948	144.940	155.805	165.381	173.989
LDT1	13.264	15.494	17.742	20.020	22.351	24.724
LDT2	6.612	8.413	10.312	12.299	14.185	16.062
UNLEADED						
CARS	861.141	921.376	974.934	1025.857	1074.616	1120.383
LDT1	110.594	128.196	142.936	157.037	171.227	185.365
LDT2	70.709	84.593	96.027	106.681	116.523	126.642

Year	1990	1991	1992
LEGAL LEADED			
CARS	76.682	59.441	46.191
LDT1	22.638	19.645	17.092
LDT2	55.804	54.239	53.122
MISFUELED			
CARS	181.770	189.069	195.837
LDT1	27.194	29.746	32.328
LDT2	17.965	19.874	21.703
UNLEADED			
CARS	1162.103	1200.156	1233.052
LDT1	199.305	213.001	226.255
LDT2	136.531	146.108	154.995

misfueling using a 10 percent discount rate. Total lifetime miles driven by new misfuelers in each year are given in Table B-12. The total miles are then multiplied by estimates of the increase in emissions due to poisoning of the catalyst. These increases vary by model year and are given in Table VI-1. The resulting increases in total emissions are given in Table VI-3.

Table B-12 Discounted Mileage Traveled in Following Twenty Years by Vehicles that Initially Misfuel in Given Year (millions of vehicle miles)

Year	Cars	LDT1	LDT2
1985	132,104	17,917	12,032
1986	135,764	19,309	13,509
1987	139,178	20,812	14,973
1988	143,644	22,398	16,392
1989	147,955	23,899	16,948
1990	152,671	25,710	17,986
1991	157,174	27,478	19,046
1992	161,523	29,134	20,076

APPENDIX C

REGRESSION RESULTS

This appendix consists of four sections. Part C.1 contains of a description of the variables used in the regressions reported in later sections. Part C.2 contains logistic regressions for white and black preteens, obtained using PROC LOGIST, a statistical software package available within the Statistical Analysis System (SAS). The third section, Part C.3, presents linear regression results for whites and blacks obtained by application of SURREGR, a statistical routine available as an adjunct of SAS which corrects regression results for the complex sample survey design used in the NHANES data collection. Part 4 of appendix C contains regression results where the solder content of cans has been added to the variables used in the gas lead blood lead regressions reported in Chapter Three.

C.1 Variable Descriptions

In addition to the regressions shown in Chapter Three, we have used the regressions presented in this appendix in our forecasts of child health effects. We have used the following variables in these regressions:

Variable Name	Description
Gaslead	Lead Used in gasoline, in hundreds of metric tons a day, lagged one month.
Poor	1 if Income 1(see below); 0 otherwise
Age 1	1 is age $\geq$ 6 months and < 2 years; 0 otherwise
Age 2	1 if age $\geq$ 2 years and < 4 years; 0 otherwise

Age 3	1 if age $\geq$ 4 years and < 6 years; 0 otherwise
Age 4	1 if age $\geq$ 6 years and < 8 years; 0 otherwise
Age 5	1 if age $\geq$ 8 years and < 10 years; 0 otherwise
Age 6	1 if age $\geq$ 10 years and < 12 years; 0 otherwise
Age 7	1 if age $\geq$ 12 years and < 14 years; 0 otherwise
Teen	1 if age $\geq$ 14 years and < 18 years; 0 otherwise
Income 1	1 if family income < $6,000; 0 otherwise
Income 2	1 if family income > $6,000 and < $15,000; 0 otherwise
Male	1 if gender is male; 0 otherwise
Teen Male	1 if gender is male and age $\geq$14 and < 18 years; 0 otherwise
Adult Male	1 if gender is male and age $\geq$18 years; 0 otherwise
Small City	1 if residence is in city with population $\geq$ 1,000,000; 0 otherwise
Rural	1 if residence is in a rural area as defined by the Bureau of the Census; 0 otherwise
Drinker	1 if alcohol consumption is $\geq$ 1 drink/ week and < 1 drink/day; 0 otherwise
Heavy Drinker	1 if alcohol consumption is $\geq$ 1 drink/day; 0 otherwise
Northeast, Mideast, South	1 if in this Census Region; 0 otherwise

Education	0 if the person never completed grade school; 1 if grade school was the highest level completed; 2 if high school was the highest level completed; and 3 if college was completed
Kid	1 if age < 6; 0 otherwise

C.2 Logistic Regression Results

Black Preteens = 8-13 years old, 112 observations

Dependent variable: 1 if blood lead is over 20 ug/dl; 0 otherwise

Model Chi-square = 6.42 with 4 D.F.

Variable	Beta	Std. Error	Chi-Square	P-Value
Intercept	-6.0148	2.4044	6.26	0.0124
Gaslead	0.9786	0.4943	3.92	0.0477
Poor	0.2356	0.5289	0.20	0.6560
Age 5	0.6158	0.6304	0.95	0.3286
Age 6	0.2397	0.6208	0.15	0.6994

Fraction of concordant pairs of predicted probabilities and responses = 0.656

White Preteens = 8-13 years old, 660 observations

Dependent variable: 1 if blood lead is over 20 ug/dl; 0 otherwise

Model Chi-square = 21.35 with 4 D.F.

Variable	Beta	Std. Error	Chi-Square	P-Value
Intercept	-8.9395	1.6782	28.38	0.0000
Gaslead	1.0674	0.3374	10.01	0.0016
Poor	0.8355	0.4883	2.93	0.0871
Age 5	1.4199	0.5810	5.97	0.0145
Age 6	1.2041	0.5904	4.16	0.0414

Fraction of concordant pairs of predicted probabilities and responses = 0.710

All logistic regression results were run using PROC LOGISTIC within the Statistical Analysis System (SAS). This procedure uses individual data where the dependent variable is 1 if the individual is above the threshold, and 0 otherwise.

APPENDIX C.3. Linear Regression Results*

Whites:

Dependent variable: individual blood lead levels

Variable	Beta	Std. Error	F-Statistic	P-Value
Intercept	5.4436	1.1842	--	--
Gaslead	2.1835	0.0345	138.19	0.0000
Income 1	0.7675	0.0553	10.65	0.0026
Income 2	0.3381	0.0288	3.97	0.0548
Age 1	3.2352	0.2015	51.95	0.0000
Age 2	4.0452	0.1713	95.51	0.0000
Age 3	3.2020	0.1267	80.91	0.0000
Age 4	2.1818	0.2118	22.48	0.0000
Teen	-0.7386	0.0519	10.52	0.0028
Male	0.5763	0.1040	3.19	0.0834
Teen Male	1.7556	0.2150	14.34	0.0006
Adult Male	3.9812	0.1203	131.72	0.0000
Small City	-0.8490	0.1080	6.67	0.0146
Rural	-1.3215	0.1188	14.70	0.0006
Drinker	0.8582	0.0296	24.84	0.0000
Heavy Drinker	2.0871	0.0889	48.97	0.0000
Northeast	-1.0908	0.1302	9.14	0.0049
Midwest	-1.2243	0.1631	9.19	0.0048
South	-1.0598	0.2493	4.51	0.0416
Education Level	-0.9440	0.0182	48.90	0.0000

* Procedure used is SURREGR in SAS.

Whites:

Dependent variable: individual blood lead levels

Variable	Beta	Std. Error	F-Statistic	P-Value
Intercept	5.4593	1.1766	--	--
Gaslead	2.1821	0.0344	138.53	--
Income 1	0.7542	0.0559	10.17	0.0000
Income 2	0.3386	0.0284	4.04	0.0032
Kid	3.2344	0.0926	112.97	0.0531
Teen Male	2.0860	0.2093	20.79	0.0000
Rural	-1.3350	0.1221	14.59	0.0001
Small City	-0.8443	0.1098	6.49	0.0006
Teen	-1.5987	0.0910	28.08	0.0159
Male	1.1333	0.0348	36.90	0.0000
Adult Male	3.4231	0.0504	232.33	0.0000
Age 4	1.8952	0.2205	16.29	0.0003
Age 5	0.5581	0.1126	2.77	0.1060
Age 6	0.4784	0.1629	1.41	0.2445
Age 7	0.3958	0.0727	2.15	0.1520
Drinker	0.8672	0.0303	24.92	0.0000
Heavy Drinker	2.0789	0.0894	48.35	0.0000
Northeast	-1.0823	0.1312	8.92	0.0054
Midwest	-1.2414	0.1663	9.27	0.0046
South	-1.0619	0.2504	4.50	0.0417
Education Level	-0.9461	0.1808	49.51	0.0000

Blacks:

Dependent variable: individual blood lead levels

Variable	Beta	Std. Error	F-Statistic	P-Value
Intercept	4.8847	2.4116	- -	- -
Gaslead	1.9342	0.1432	26.12	0.0000
Income 1	1.1457	2.2593	5.06	0.0328
Income 2	1.0941	0.2902	4.13	0.0522
Age 1	6.1030	1.3729	27.13	0.0000
Age 2	8.8867	0.5052	156.32	0.0000
Age 3	6.6989	0.4592	97.73	0.0000
Age 4	4.8920	0.7706	31.06	0.0000
Teen	0.6352	0.1869	2.16	0.1533
Male	1.8280	0.3413	9.79	0.0042
Adult Male	4.2469	0.6157	29.29	0.0000
Drinker	1.0359	0.4713	2.28	0.1429
Heavy Drinker	1.4088	1.2531	1.58	0.2190
Education Level	-0.8329	0.0874	7.93	0.0090

Blacks:

Dependent variable: individual blood lead levels

Variable	Beta	Std. Error	F-Statistic	P-Value
Intercept	4.795	2.48	--	--
Gaslead	2.041	0.12	33.84	0.0000
Income 1	1.016	0.26	3.90	0.0587
Income 2	1.063	0.33	3.44	0.0748
Kid	7.204	0.29	180.91	0.0000
Teen	-0.806	0.35	1.84	0.1857
Male	1.860	0.24	14.39	0.0008
Adult Male	4.061	0.48	34.08	0.0000
Age 4	4.869	0.81	29.22	0.0000
Age 5	2.494	1.10	5.67	0.0246
Age 6	2.215	0.44	11.07	0.0025
Age 7	0.417	0.59	0.30	0.5910
Drinker	1.063	0.44	3.03	0.0933
Heavy Drinker	1.386	1.17	1.64	0.2117
Northeast	-1.460	0.84	2.53	0.1230
Midwest	0.145	1.05	0.02	0.8885
South	-0.1173	0.501	0.03	0.8695
Education Level	-0.826	0.086	7.91	0.0091

APPENDIX C.4. Regression Results: Whites with Solder Used in Cans

Effect	Coefficient	Standard Error	P-Value
Intercept	4.16	--	--
Gasoline lead	2.16	0.04	0.0000
Low Income	0.78	0.06	0.0024
Moderate Income	0.36	0.03	0.0383
Child	3.47	0.11	0.0000
Solder	0.74	1.01	0.4651
Teenager	-0.32	0.05	0.1667
Male	0.68	0.08	0.0251
Male Teenager	1.38	0.13	0.0005
Adult Male	3.89	0.11	0.0000
Small City	-0.82	0.11	0.0212
Rural	-1.32	0.12	0.0007
Drinker	0.83	0.03	0.0000
Heavy Drinker	2.09	0.09	0.0000
Northeast	-1.02	0.13	0.0091
South	-1.15	0.17	0.0084
Midwest	0.88	0.29	0.1093
Education Level	-0.93	0.02	0.0000

*U.S. GOVERNMENT PRINTING OFFICE: 1985 526 259 30280