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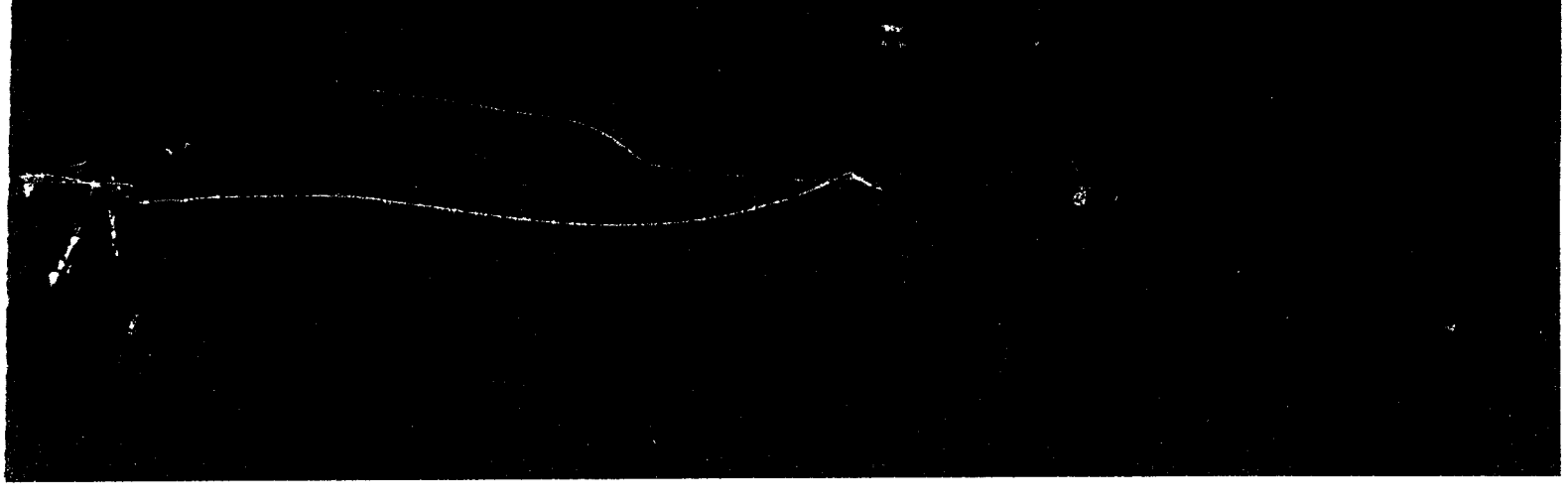
Costs and Benefits of Reducing Lead in Gasoline



COSTS AND BENEFITS OF REDUCING LEAD IN GASOLINE

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CHAPTER VI

EXECUTIVE SUMMARY

COSTS AND BENEFITS OF REDUCING LEAD IN GASOLINE

COSTS

Lead is a relatively inexpensive way to boost gasoline octane, but eliminating or severely limiting lead would increase the manufacturing cost of gasoline by less than 1%. Eliminating lead altogether may result in excessive valve wear in some trucks and older cars; a low-lead fuel of 0.10 grams/gallon would prevent this problem.

BENEFITS

Maintenance Savings: Lead forms corrosive compounds that increase automobile maintenance costs. Cars that use leaded gasoline need more frequent tune-ups, exhaust system replacements, and oil changes.

Misfueling: Recent EPA surveys indicate that over 12% of all cars equipped with catalytic converters to control auto emissions are currently being "misfueled" with leaded gasoline because consumers want either to save money or to obtain higher octane. Misfueling poisons catalysts and substantially increases the conventional auto pollutants: hydrocarbons, carbon monoxide, and nitrogen oxides. Given current misfueling rates, misfueled vehicles will account for one-third of leaded gasoline demand in 1988, significantly increasing our estimates of future lead and conventional pollutant emissions. The impact of these emissions on public health and welfare is substantial.

Health: Our analysis and other major studies both in the U.S. and abroad indicated that the amount of lead in blood is directly related to the amount of lead in gasoline. Lead has long been known to cause pathophysiological changes, including the inhibition of major enzymatic processes, adverse effects on the central nervous system, and decreases in cognitive ability. Children are especially vulnerable to lead, and black children are more severely affected than others. Children with elevated blood lead levels require medical monitoring and/or treatment.

Adverse effects of lead in the blood are now found at levels that were previously thought safe, and additional effects are suspected. The Centers for Disease Control is currently investigating lowering its definition of lead toxicity.

SUMMARY

Our examination of the costs and benefits of two options for further restricting the use of lead in gasoline, summarized in Table 1, on the next page, indicated that the benefits exceed the costs. Although we were able to place dollar values on reduced medical costs and cognitive damage for children with high lead levels we did not monetize other factors affecting this group, such as behavioral and other problems, nor the pain of medical treatment and parents' lost work time. No monetary values at all were estimated for children with lower lead levels, although they suffer some negative effects. These non-monetized benefits are represented by "H" in the table. Table 2 provides a summary of the environmental effects of reducing lead in gasoline.

SUMMARY TABLE 1

Comparison of Benefits and Costs of
Lead Reduction Options in 1988
(millions of 1983 dollars)

	Low-Lead Option*	All Unleaded**
COSTS		
Manufacturing Costs	$503	$691
Non-monetized Valve Damage to Engines that Need Lead	--	D
TOTAL COSTS	$503	$691+D
BENEFITS		
Maintenance Benefits	$660	$755
Environmental and Health Benefits		
Conventional pollutants		
Reduced damage by eliminating misfueling	$404	$404
Non-monetized health benefits†	H_1	H_1
Lead		
Reduced medical care costs	$41	$43
Reduced cognitive damage	$184	$193
Non-monetized health benefits††	H_2	H_3
TOTAL BENEFITS	$1,289+H_1+H_2$	$1,395+H_1+H_3$
NET BENEFITS	$786+H_1+H_2$	$704+H_1+H_3-D$

* This option would make a low lead gasoline (0.10 grams of lead per gallon) available only for those few vehicles that require some lead. It assumes no misfueling.

** All lead in gasoline would be banned by 1988.

† These include chronic health effects of ozone and CO, and any effects of reduced sulfate particulates.

†† Since medical costs and cognitive damage were only monetized for children with high blood lead (>30 ug/dl), H_2 and H_3 represent other benefits for this group (pain, lost work time to parents, etc.) as well as all the benefits (medical, cognitive, behavior, etc.) for the lower lead group (<30 ug/dl). H_2 and H_3 differ because the numbers of children at risk under the two options differ.

SUMMARY TABLE 2

ENVIRONMENTAL EFFECTS IN 1988 OF REDUCED GASOLINE LEAD USE

	LOW-LEAD	ALL UNLEADED
REDUCTIONS IN EMISSIONS (thousands of metric tons)		
Lead	33.4	35.6
HC	314	314
CO	2,202	2,202
NO_x	130	130
Ozone (resulting from reduced HC and NO_x emissions)	1.5% reduction	1.5% reduction
REDUCTIONS IN THE NUMBER OF CHILDREN AT RISK OF ADVERSE HEALTH EFFECTS		
Reduction in number of children at risk of:	4,257,000	4,486,000
- Inhibition of enzyme activity (PY-5-N and ALA-D)		
Reduction in number of children at risk of:	1,475,000	1,553,000
- Changes in EEG patterns - Impairment of heme synthesis - Elevated levels of ALA and possible interference with neurotransmission processes - Impairment of vitamin D activity - Possible adverse cognitive effects		
Reduction in number of children at risk of impaired globin synthesis	476,000	500,000
Reduction in number of children at risk of:	43,000	45,000
- Potentially requiring active medical care - Probable adverse cognitive effects		

CHAPTER I

INTRODUCTION, FINDINGS, AND QUALIFICATIONS

I.A. Background

Since 1973, the U.S. Environmental Protection Agency (EPA) has regulated the use of lead as an additive to gasoline. Section 211 of the Clean Air Act gives the EPA Administrator authority to control or prohibit any fuel or fuel additive that:

- causes, or contributes to, air pollution which may reasonably be anticipated to endanger the public health or welfare, or
- will impair to a significant degree the performance of any emission control device or system which is in general use...

To avoid the adverse effects of lead in the environment and to protect emission control equipment which is rendered ineffective or "poisoned" by lead additives, EPA required that cars, beginning with model year 1975, meet tighter emissions limits. To do this automobile manufacturers installed catalytic converters requiring unleaded gasoline. In several stages during the period 1976-1982, EPA mandated that the lead content of leaded gasoline be reduced from over 2.0 grams per gallon to 1.1 g/gal.

During this period, studies on blood lead levels showed that reducing the lead content of gasoline would also reduce blood lead levels in all major population groups in the United States. It was anticipated that the combination of these two actions would restrict and eventually eliminate the exposure of the general population, especially young children, to airborne lead from mobile sources, as well as reduce health and welfare damage from conventional pollutants.

While EPA's rules have virtually eliminated leaded premium gasoline, consumers of regular gasoline must choose between relatively inexpensive gasoline containing lead additives or more expensive unleaded gasoline. In addition to savings, some consumers want the slightly higher octane of leaded regular. (However, few recognize lead's corrosive effects on their engines or the increased maintenance cost of using leaded gasoline.)

Recently, several EPA and private studies have found widespread "misfueling" (i.e., the use of leaded gasoline in vehicles designed for unleaded gasoline). The studies showed that constant misfueling rates of over 12% have slowed the decline in lead emissions significantly, and challenged the assumption that leaded gasoline would soon be eliminated because of lack of demand. These findings have occurred at the same time that the public health community's long-standing concern about lead has produced a substantial literature about the adverse effects of lower lead exposures. Specifically, recent studies have strengthened the identification of gasoline lead as a major source of blood lead and new information on the effects of lead on physiological functions has become public.

Some of this information began to surface during the hearings and subsequent comment period related to EPA's proposed lead phasedown rule making in 1982. At that time, the Agency had proposed several regulatory alternatives. As a result of the information gained from the public response during the proceedings, EPA tightened the restrictions on the amount of lead permitted in

leaded gasoline. The restriction also set a uniform limit for both small and large refiners.

The growing problem of the misuse of leaded fuel in cars with catalytic converters, the increasing recognition of serious health effects from even low lead levels, and the fact that gasoline has been identified as the major source of environmental exposure to lead all indicated that a simple continuation of current policies needed reexamination. EPA presently has two review processes underway for assessing the effects of lead. The first is the Agency's formal Criteria Document process, which is managed by the Office of Research and Development. The Lead Criteria Document will evaluate all of the environmental effects of lead. A Draft Lead Criteria Document was circulated for comment in October 1983; a final document is expected by August 1984. Nothing in this paper is intended to prejudge or supercede the outcome of that process.

Concurrently, and on a somewhat faster timetable, EPA's Offices of Policy, Planning and Evaluation, and Air and Radiation have been reviewing data from the 1982 phase-down effort to evaluate the costs and benefits of additional restrictions on the amount of lead in leaded gasoline. This paper is primarily an analysis of the monetized costs and health and welfare benefits of reducing the lead content of gasoline.

I.B. Approach

For this paper, we have contrasted the costs and benefits of two hypothetical options against a base case which continues existing regulations and compliance practices. Both the options and the base case are projections for 1988. The first option is a low-lead fuel (0.10 grams of lead per gallon) for the few classes of vehicles, such as trucks and older cars, that may require the valve lubrication that lead provides. Contrary to the current situation, such a low lead fuel would cost more to manufacture than regular gasoline. We assume this cost inversion, coupled with availability restrictions on this fuel, would eliminate misfueling as a practical problem. The second option is the banning of all leaded gasoline.

I.B.1. Base Case

At present, EPA regulations restrict the use of lead in gasoline in two ways. First, beginning with the 1975 model year, almost all light-duty vehicles have been equipped with catalytic converters and require unleaded gasoline. By 1981, virtually all new light-duty vehicles should have been using unleaded gasoline. Second, EPA limits the lead content of leaded gasoline to 1.1 grams per gallon. Because lead is a relatively inexpensive octane enhancer, this is about half of what refiners would use if not statutorily constrained.

The lead standard must be met on a quarterly average, however, not for each gallon produced. In addition, refiners may average their own production or sell off-sets to each other.

That is, two refiners may agree that one of them will produce gasoline with 1.0 grams of lead per gallon and the other will use 1.2 grams of lead (and, presumably, pay the first one an agreed amount). This allows the refinery industry as a whole to optimize its use of octane manufacturing capacity and to minimize the cost of meeting the restrictions on lead use.

I.B.2. Hypothetical Options

To address the problems of misfueling and airborne lead pollution, the first alternative we considered was an outright ban on the use of lead in gasoline. Such a regulation would meet both the public health and misfueling concerns, and we examined this option carefully. However, some vehicles could experience severe valve damage if no leaded fuel were available. We therefore added a second "low-lead" option, which assumed that marketing restrictions would be designed so as to eliminate misfueling. The amount of lead in leaded gasoline would be restricted to 0.10 grams per gallon, which is sufficient to protect valves from undue wear, but which minimizes environmental contamination.

I.C. Summary of Analysis

Our analysis evaluates and compares the costs and benefits in 1988 of reducing or eliminating lead in gasoline. To calculate the costs of restricting lead as an octane-enhancer, we used a linear programming model of the refinery industry.

In the benefits area, we calculated vehicle maintenance savings that would be realized by eliminating the corrosion and engine fouling problems associated with lead in gasoline. We also monetized the benefits of reducing the emissions of conventional pollutants that result from misfueled vehicles, and analyzed the number of children at risk of various health effects from lead exposure.

We have valued the benefits associated with reducing the number of children suffering from "undue lead absorption," currently defined by the Centers for Disease Control as blood lead levels above 30 micrograms per deciliter (ug/dl) and free erythrocyte protoporphyrin (FEP) levels over 50 ug/dl. For children with blood lead levels below 30 ug/dl, we calculated the change in the number of children with blood lead levels above the lowest observed effects level for pathophysiological changes but we did not ascribe any dollar values to reducing their lead exposures. We also estimated the change in the number of children who might suffer small decreases in cognitive ability, but again we attached no monetary value to this.

Chapters II (on costs) and V (on the health effects of blood lead levels over 30 ug/dl) contain detailed sections describing the methods we used in our analysis.

I.C.1. The Costs of Reducing Lead in Gasoline

Lead is added to gasoline because it is the least expensive way for petroleum refiners to boost the octane of fuel. Reducing

or eliminating the lead content of gasoline will require extra energy use (and potentially more equipment) and, consequently, greater resource costs. We estimated the increased costs of raw materials and refining would be less than 1%. As a result, many consumers would pay slightly more for gasoline.

Chapter II contains a description of consumer demand for gasoline, the leaded/unleaded split, and current needs for octane. Based upon our models and projections by the Energy Information Administration and Data Resources, Inc. (DRI), we have projected gasoline demand and the leaded/unleaded split under existing policies and misfueling rates, and under the two hypothetical options: low-lead and all unleaded. The refinery cost figure is an estimate of the extra manufacturing costs incurred by refineries if they must use other octane-producing processes to meet U.S. demand for gasoline. These costs were derived from the same linear program of the refining industry that was used in EPA's economic analysis of the 1982 lead phase-down regulations.

We projected that, meeting current consumer requirements for octane, the 1988 cost to refiners of reducing lead in the low-lead option would be $503 million and the cost of the all unleaded option would be $691 million. Because we could not predict how changes in production costs might affect the marketing strategies of retailers under our two options, we did not attempt to estimate the change in gasoline prices to consumers.

I.C.2. The Benefits of Reducing Lead in Gasoline

Chapters III-VI describe the monetized benefits of reducing the amount of lead in gasoline and some unmonetized health benefits of reducing overall exposure to lead.

Chapter III (Maintenance Savings) describes the vehicle operation and maintenance savings that would result from restricting lead in gasoline. Lead compounds and their associated scavengers foul and corrode the engines and exhaust systems of all vehicles using leaded gasoline, whether designed for it or not.* Operation and maintenance savings come from three primary areas: less frequent tune-ups, less frequent exhaust system replacements, and less frequent oil changes. We estimated that vehicle owners who switch from leaded to unleaded gasoline could save 3-4 cents per gallon of gasoline. The total benefits were computed by multiplying the savings per gallon times the total number of gallons consumed. The estimates of maintenance savings we have included in Table I-1 (on the next page) were at the low end of our range. We also discussed the possibility of valve damage to leaded vehicles, which could occur in our all unleaded option, but not in our low-lead option. We were unable to estimate a monetary value for this because we did not have information on how many vehicles are driven under the conditions where it could occur.

* Scavengers are necessary to remove lead from the engine after combustion. Without these scavengers, engine performance would rapidly deteriorate to complete inoperability.

TABLE I-1

Comparison of Benefits and Costs of
Lead Reduction Options in 1988
(millions of 1983 dollars)

	Low-Lead Option*	All Unleaded**
COSTS		
Manufacturing Costs	$503	$691
Non-monetized Valve Damage to Engines that Need Lead	--	D
TOTAL COSTS	$503	$691+D
BENEFITS		
Maintenance Benefits	$660	$755
Environmental and Health Benefits		
Conventional pollutants		
Reduced damage by eliminating misfueling	$404	$404
Non-monetized health benefits†	H_1	H_1
Lead		
Reduced medical care costs	$41	$43
Reduced cognitive damage	$184	$193
Non-monetized health benefits††	H_2	H_3
TOTAL BENEFITS	$1,289+H_1+H_2$	$1,395+H_1+H_3$
NET BENEFITS	$786+H_1+H_2$	$704+H_1+H_3-D$

* This option would make a low lead gasoline (0.10 grams of lead per gallon) available only for those few vehicles that require some lead. It assumes no misfueling.

** All lead in gasoline would be banned by 1988.

† These include chronic health effects of ozone and CO, and any effects of reduced sulfate particulates.

†† Since medical costs and cognitive damage were only monetized for children with high blood lead (>30 ug/dl), H_2 and H_3 represent other benefits for this group (pain, lost work time to parents, etc.) as well as all the benefits (medical, cognitive, behavior, etc.) for the lower lead group (<30 ug/dl). H_2 and H_3 differ because the numbers of children at risk under the two options differ.

We estimated that total savings from reduced maintenance and operation expenses in 1988 would be $660 million for the low-lead option and $755 million for the all unleaded option.

Chapter IV (Benefits of Avoiding Excess HC, CO, and NO_x Emissions) examines misfueling practices and their consequences for emissions of the conventional auto pollutants: hydrocarbons, carbon monoxide, and nitrogen oxides. As we have noted, using leaded gasoline in vehicles designed to run on unleaded gasoline poisons their catalytic converters, which causes a substantial increase in HC, CO, and NO_x. While all vehicles equipped with catalytic converters are required to use unleaded gasoline, over 12% of all vehicles equipped with catalysts are currently being misfueled with leaded gasoline.

We estimated the excess emissions in grams per mile and computed the increases in total emissions due to poisoned catalysts. Because HC and NO_x combine to form ozone, we also estimated the increase in ozone which formed as a result of more conventional pollution. Our estimates of the size of these changes appear in Table I-2. We used existing literature and data on the negative health and welfare effects of these conventional pollutants to value these changes in emissions.

We used three methods to value the benefits of avoiding these excess emissions in 1988: 1) an estimate valuing the avoided emissions at the average cost per ton of the most cost effective alternative for controlling these pollutants, 2) an estimate valuing the avoided emissions at the average cost per ton of the

TABLE I-2

ENVIRONMENTAL EFFECTS IN 1988 OF REDUCED GASOLINE LEAD USE

	LOW-LEAD	ALL UNLEADED
REDUCTIONS IN EMISSIONS (thousands of metric tons)		
Lead	33.4	35.6
HC	314	314
CO	2,202	2,202
NO_x	130	130
Ozone (resulting from reduced HC and NO_x emissions)	1.5% reduction	1.5% reduction
REDUCTIONS IN THE NUMBER OF CHILDREN AT RISK OF ADVERSE HEALTH EFFECTS		
Reduction in number of children at risk of:	4,257,000	4,486,000
- Inhibition of enzyme activity (PY-5-N and ALA-D)		
Reduction in number of children at risk of:	1,475,000	1,553,000
- Changes in EEG patterns - Impairment of heme synthesis - Elevated levels of ALA and possible interference with neurotransmission processes - Impairment of vitamin D activity - Possible adverse cognitive effects		
Reduction in number of children at risk of impaired globin synthesis	476,000	500,000
Reduction in number of children at risk of:	43,000	45,000
- Potentially requiring active medical care - Probable adverse cognitive effects		

program requiring catalytic converters on cars, and 3) an estimate using econometric damage functions to value the avoided emissions. We used the average of the last two alternative methods, $404 million, as a point estimate in Table I-1.

Chapter V discusses the health benefits of reducing lead in gasoline by valuing the damage resulting from blood lead levels over 30 ug/dl (which is, in combination with elevated FEP levels, currently the Centers for Disease Control's definition of lead toxicity).

Lead emissions from cars increase the blood lead levels of children. We analyzed blood lead data from the Second National Health and Nutrition Examination Survey (NHANES II) and found a strong, statistically significant relationship between blood lead levels and the amount of lead in gasoline, after controlling for age, race, sex, income, degree of urbanization, education, region of the country, and alcohol consumption. The strong relationship is shown graphically in Figure I-1. Inner city black children have the highest rates of elevated blood lead levels, but a substantial number of white children also are affected.

Lead is known to damage the kidney, the liver, the reproductive system, blood creation, basic cellular processes, and brain functions. Using the projected lead reductions from Chapter II, we estimated how many fewer children would be likely to be at risk of undue lead exposure in 1988. From these we estimated the benefits of avoiding the pathophysiological and cognitive and behavioral effects of elevated blood levels.

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

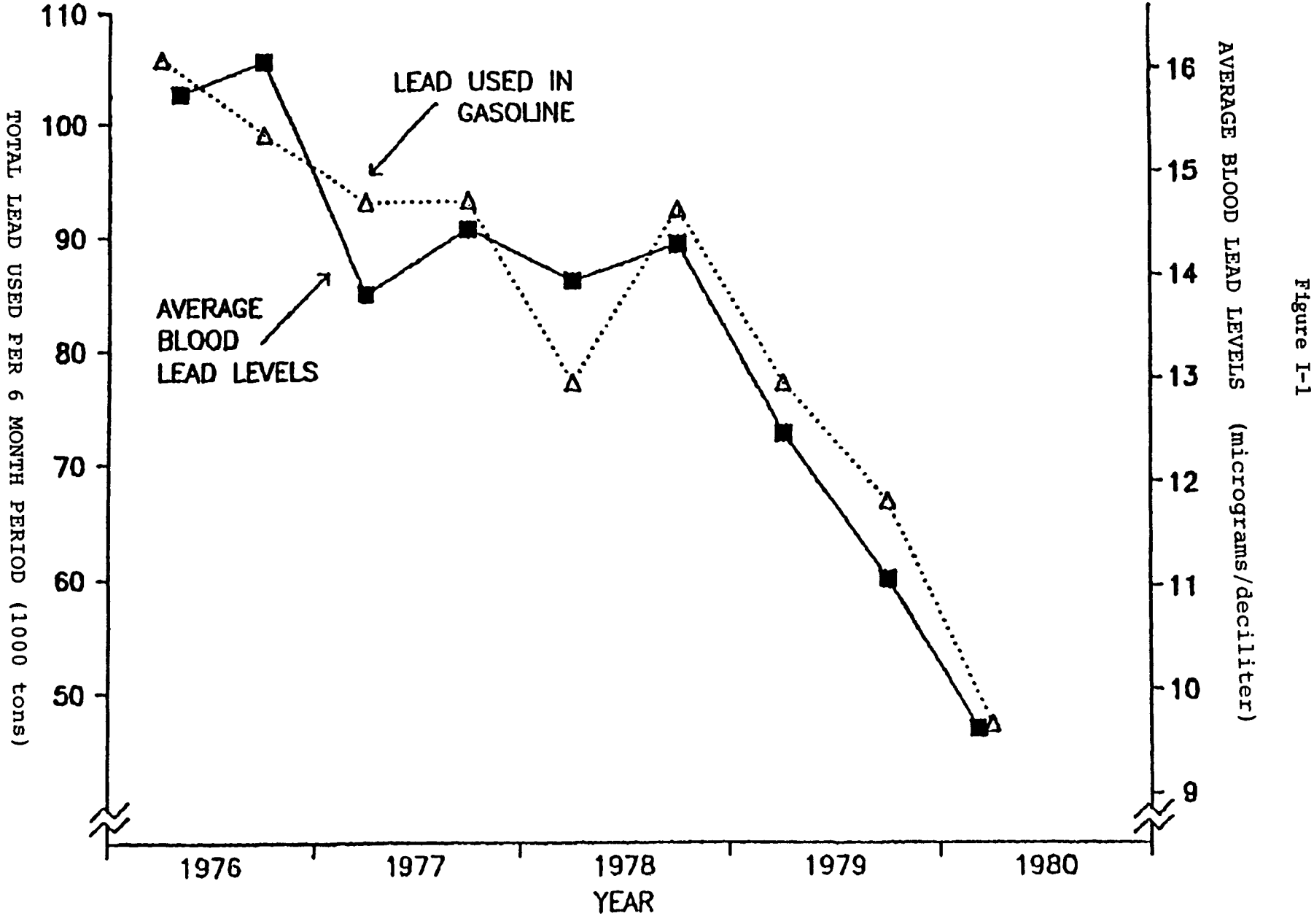

Figure I-1

When children's blood lead levels are over 30 ug/dl, they require follow-up and/or medical treatment. The estimate in Table I-1 was based on a regression that projected the number of additional children who would require medical treatment for elevated blood lead levels as a result of gasoline lead use. It did not include children who would need treatment for lead poisoning because of lead-based paint or other sources of exposure. We included the costs of medical treatment even for the children whom public health officials do not find and treat, because we assumed that the social cost of elevated lead levels for an untreated child was at least as great as what we spend on treatment for those who are identified.

Some of these children have blood lead levels high enough to reduce cognitive performance, including the loss of several IQ points. Researchers have found that these cognitive deficits remain three years later, even after medical attention. Table I-1 also includes the costs of compensatory education to overcome the additional learning difficulties that children with high lead levels incur. As in the case of medical costs, we included costs even for those children who do not receive compensatory education. Again, we assumed that the costs to society of a learning disability were at least as great as the cost of a program to partially compensate for the damage.

We estimated, for the all unleaded case, that the benefits of avoiding medical and associated costs for children with blood lead levels over 30 ug/dl in 1988 were $43 million, and

that the value of avoiding the cognitive damage likely to occur at those levels was $193 million. We estimated that the benefits in the low-lead case are $41 million for medical savings and $184 million for avoided cognitive damage.

Chapter VI discusses the health effects of blood lead levels below 30 ug/dl. As measurement tools have improved, research has detected pathophysiological effects at blood lead levels that were previously thought to be safe, and additional effects are suspected. These results warrant concern about even small changes in the total body lead burden of children, especially those children who are subject to sources of lead exposure in addition to lead from gasoline.

While the full clinical significance of the effects of blood lead levels below those requiring medical management under current practice is not yet clear, the Centers for Disease Control is now considering lowering its current (30 ug/dl of blood lead and FEP levels of 50 ug/dl) criteria for lead toxicity.

Among the recent data on these pathophysiological changes are inhibition of the enzymes Pyrimidine-5'-nucleotidase (PY-5-N) and aminolevulinic acid dehydrase (ALA-D), which begins to be detectable at about 10 ug/dl of blood lead; changes in EEG patterns, detectable at about 15 ug/dl; elevated ZPP or FEP in red blood cells at about 15 ug/dl; inhibition of globin synthesis at about 20 ug/dl; increased risks of abnormally small red blood cells at 20-25 ug/dl; and other disruptions of aminolevulinic acid (ALA) and vitamin D homeostasis at about 15 ug/dl. In addition, our analysis of the combined evidence from all the

relevant studies indicated that mild cognitive effects also occurred at low lead exposure levels.

Our estimates of the reduced number of children at risk of health effects in 1988 are presented in Table I-2. We have not valued these changes monetarily, but crude valuation procedures suggest the benefits are likely to be large.

I.D. Limitations of the Analysis

This paper is a cost-benefit analysis of reducing the lead content of gasoline. To do this, we have proposed two hypothetical options: a low-lead and an all unleaded scenario. Our analysis measured the effects in one year, 1988. With such a far-reaching issue, the limitations of our findings should be clarified.

We have forecast circumstances and events that will occur four years in the future, and the future is, at best, uncertain. One problem is shifts in underlying behavior such as a change in consumer preferences back to large cars or changes in external events (e.g., another big war in the Mideast). In addition, because we are extrapolating from our perceptions and experience to date, any misapprehension of what is will tend to be magnified as we project several years ahead. (An example of this may be the misfueling problem.)

Although we believe that our model of the refinery industry is as accurate as possible, we can not predict marketing behavior. We believe we have estimated real resource costs

fairly accurately, but we can not predict with confidence what would happen to consumer prices.

In the benefits area, we are still learning about the health effects of lead and other criteria pollutants. The body of knowledge is neither well-defined nor unequivocal. While the trend in new findings seems to be uncovering more effects at lower levels, the clinical significance of these findings is not always clear. Also, the distributions of effects that we are predicting, especially at 30 ug/dl of blood lead, are near the tails of the distributions, and therefore, more susceptible to changes and uncertainties. However, we have no indications that our estimation procedure is biased, so the effects are as likely to be larger as smaller. In addition, it is difficult to measure IQ loss, and even more difficult to put a dollar value on lost IQ points.

While we have used accepted state-of-the-art methods for valuing health and environmental effects, there are uncertainties about the health and welfare effects of hydrocarbons, nitrogen oxides, and carbon monoxide; and about the transformation of hydrocarbons and nitrogen oxides into ozone. Finally, there are some uncertainties inherent in the monetary valuation of these effects.

I.E. Quantifying Effects

We have, in the course of this analysis, explored many alternative assumptions and methods for valuing effects. Throughout, our overall results have proven to be very robust to changes in details; that is, small changes did not alter results.

The effects for which we have presented monetary values in Table I-1 have a solid basis. Where the data could not support a point estimate or even a range, we did not provide a monetary value. All significant effects, however, whether monetized or not, are included in Table I-1 to allow the reader to gain a full perception of the problem.

The clear conclusion from the data summarized in Table I-1 is that the benefits of the low-lead option substantially exceed the costs. For the all unleaded case, the issue is less clear because of the unresolved nature of the cost of valve damage. However, as engines which need leaded gasoline retire from the fleet, the issue of valve damage becomes less important. Thus, in the long run, the option of eliminating lead in gasoline appears very attractive.

CHAPTER II

COSTS OF REDUCING LEAD IN GASOLINE

Petroleum refiners add lead to gasoline as the least expensive way to boost octane. There are alternative additives that also help boost octane, but they generally are more expensive and, like lead, can also be toxic.

The most attractive alternative refiners have for raising the octane of unleaded gasoline is additional processing of the gasoline in either catalytic reformers or in isomerization units. Increasing the use of reformers and isomerizers requires more energy consumption, and thus raises the cost of manufacturing gasoline. (This may also increase the density of gasoline which raises slightly the energy content per gallon of gasoline.)

If refiners need to produce more unleaded gasoline but are limited by isomerization or reforming capacity, they can construct more capacity, incurring a capital charge. Alternatively, refiners can purchase either a better grade of crude oil or add other octane boosters, incurring higher operating costs. The sum of all these costs, along with miscellaneous energy costs, etc., is the additional cost of making gasoline with less or no lead.

In this chapter we discuss some of the basic input assumptions we used to estimate the refinery costs of producing gasoline under our two options.

Estimates of the reduction in lead emissions under our two policy options are presented. We then show the costs derived from applying our assumptions to the Department of Energy (DOE) refinery model. A description of the DOE model

and a brief explanation of refinery processes are presented in the last section of this chapter.

II.A. Input Assumptions

The cost to refiners of manufacturing unleaded or low lead gasoline depends principally on three factors:

- o the total gasoline volume produced,
- o the portions of gasoline production that are leaded and unleaded, and
- o the level of octane specified for the gasoline pool.

II.A.1. Gasoline Volume

Table II-1 presents the gasoline demand assumptions that we used to estimate the costs of manufacturing unleaded gasoline for our baseline and two policy options. Gasoline demand estimates are obviously subject to uncertainty. Demand in 1983 was approximately 6.6 million barrels per day. We assumed that demand in 1988 would fall to 6.5 million barrels per day, because newer cars are more fuel efficient (i.e., get more miles to the gallon) than the older vehicles they replace. We believe fuel efficiency effects will slightly outweigh the effects of the growing number of vehicles, even though vehicle miles travelled is also expected to increase. (For comparison with our estimate, a recent Data Resources Inc. [DRI, 1983] model forecast for 1988 is for 3% fewer gallons of gasoline than we assumed; last year's Energy Information Administration [EIA, 1983] estimate was much lower but has been revised upward to about our level.)

TABLE II-1

Projected Gasoline Demands
(millions of barrels per day)

Year	With Misfueling			Without Misfueling		
	Leaded	Unleaded	Total	Leaded	Unleaded	Total
1982 (actual)	3.13	3.41	6.54	N.A.	N.A.	N.A.
1984	2.80	3.78	6.58	2.32	4.26	6.58
1986	2.45	4.09	6.54	1.88	4.66	6.54
1988	2.11	4.39	6.50	1.48	5.04	6.50
1990	1.80	4.66	6.46	1.07	5.39	6.46

*To convert to billions of gallons per year, multiply by 42 gallons/barrel times 365 days/year.

II.A.2. Leaded-Unleaded Split

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% (plus or minus 0.6% at a 95% confidence level) of the gasoline market in 1988. Our vehicle fleet model (described in Chapter III), using historic scrappage rates from DRI, predicted essentially the same unleaded share. For our cost analysis, therefore, we assumed a 67.5% share.

The first set of projections, "with misfueling," is our reference or baseline of current regulations and current misfueling rates. The projections for leaded gasoline "without

misfueling" are used in our first policy option, whereby the lead content of gasoline is reduced to the level necessary to protect older vehicles' valves, but misfueling is eliminated.

We believe this low-lead option will eliminate misfueling because leaded gasoline with only 0.1 grams per gallon will cost more to manufacture than unleaded regular gasoline, so it will no longer be the lowest cost product. Also, restrictions on availability will reduce the incentive to misfuel, for instance, if this grade were limited to full service stations.

II.A.3. Misfueling

While the population of vehicles that legally may use leaded gasoline is shrinking, misfueled vehicles are slowing the decline in demand for leaded gasoline. By 1990, misfueling will account for over one-third of leaded gasoline demand. Table II-2 shows these percentages. (The model that forecast them is discussed in Chapter III.)

TABLE II-2

Leaded Gasoline Demand Due to Misfueling

Year	Billions of Gallons	Percent of Total Gasoline	Percent of Leaded Gasoline
1984	7.36	7.3%	17%
1986	8.72	8.7%	23%
1988	9.97	10.0%	31%
1990	11.19	11.3%	41%

EPA surveys indicated that over 12% of vehicles designed to use unleaded gasoline in fact use leaded (EPA, 1983). Because surveyed motorists may refuse to allow their cars to be inspected, however, these survey results probably greatly underestimate the misfueling rate. Misfueling has significantly increased the current demand for leaded gasoline. Only 52% of gasoline demand in 1982 was unleaded, as opposed to Dupont's 1979 projections of 62%. The two most common reasons given by motorists for misfueling are the price differentials between leaded and unleaded fuel, and driver dissatisfaction with performance resulting from the lower octane generally found in regular unleaded gasoline.

A problem of octane-related performance occurs because regular leaded gasoline generally has 89 octane* while regular unleaded has 87 octane. Some cars designed to use leaded gasoline do not function as well with the lower octane in unleaded regular. In our analysis we addressed this issue by projecting actual octane need and requiring our refinery model to meet that demand. This increased the cost of manufacturing unleaded gasoline. We included the octane-related performance issue as a cost of manufacturing, not as performance degradation. The specific assumptions we made about the distribution of octane requirements for misfuelers and leaded gasoline vehicles are discussed below.

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* In this paper, we define octane to be the average of research and motor octane, commonly expressed as (R + M)/2.

II.A.4. Octane Requirements

If the no lead or low-lead options eliminate misfueling, we must then identify what octane fuel the former misfuelers will choose. If all misfueling resulted from the current seven cent per gallon average price differential, misfuelers would revert to the lowest priced alternative. In our two hypothetical options, regular unleaded would be the least expensive. If about half of misfueling were due to price and half to performance considerations, half of the misfuelers would drop to 87 octane regular unleaded* but the other half would still require 89 octane.** If all misfueling were due to octane needs, all previously misfueled cars would still require 89 octane after reverting to unleaded.

We believe the intermediate case (half misfueling due to price and half to octane) is the most reasonable assumption, but we have calculated the range of costs for the various assumptions.

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* Currently, the average octane of "87 octane" unleaded is really above 87 octane and the average octane of leaded gasoline and premium unleaded is also above the number specified. We have used the real average octanes, not their numerical specifications, for the three gasoline grades in our model, but we refer to them as "87 octane," etc., for convenience.

** An 89 octane unleaded grade need not be specifically manufactured for retail outlets. Most gasoline stations now have three pumps. If they ceased selling leaded, they could attach the third pump to a blend of regular unleaded (87 octane) and premium unleaded (91 octane), thereby producing a mid-grade fuel.

The second policy option we analyzed was eliminating all leaded gasoline. To estimate the costs for this case we used the projected total 1988 demand for gasoline.* Here, again, we had to allocate octane demand. We used the same assumptions about misfuelers as before. We assumed people who owned leaded gasoline vehicles would continue to require an average of 89 octane.

II.B. Reduction in Lead Emissions

Our analysis of reduced lead emissions assumed that every gram of lead entering a car's gas tank came out its tailpipe. In fact, some lead ends up in the oil (and may end up as waste oil recycled for home heating) and some adheres to the exhaust system and tailpipe, eventually flaking off. Ultimately, however, all lead in gasoline ends up in the environment as a potential source of lead contamination.

To estimate the reduction in lead emissions, we first computed the number of tons of lead that would be removed in 1988 under our base case. We used gasoline demands from Table II-1 and assumed 1.1 grams of lead per gallon of gasoline (the amount allowed under current regulations).

To calculate the tons of lead removed under the all unleaded option, we took the volume of leaded gasoline that would be used in 1988 assuming no changes in current rules or practices (i.e.,

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* We may have overestimated costs by assuming that unleaded demand would equal total demand in the all unleaded case. We assumed demand would not change as a result of changing prices, i.e., we assumed no elasticity of demand.

1.1 grams of lead per gallon and continued misfueling). Multiplying that volume (32.4 billion gallons) by the lead content (1.1 g/gal) gave us the total amount of lead reduced in the all unleaded option. The result, shown in Table II-3, was 35,600 metric tons of lead removed.

For the low-lead option we needed to calculate the lead emissions resulting from that reduction option (i.e., demand [22.4 billion gallons] times 0.10 grams per gallon). Subtracting the lead emissions under the low-lead option from 1988 emissions based on no changes in rules gave us emission reductions of 33,400 metric tons, shown in Table II-3.

Table II-3

Metric Tons of Lead Removed in 1988*

Low-Lead	All Unleaded
33,400	35,600

*Computed by assuming 1.1 grams of lead per gallon and using gasoline demands from Table II-1.

II.C. Cost Estimate

To estimate the costs of lowering the lead content of gasoline, we used the Department of Energy's linear programming model of the petroleum refining industry. The model and oil refinery processes are described in greater detail in Section II.F.

Using the DOE refinery model and the assumptions described above, we have estimated the cost differences for our two cases. The costs, and their sensitivities to octane assumptions, are discussed below. These costs have been estimated for several different scenarios that indicate sensitivities to the basic assumptions.

Our cost analysis indicated that reducing the amount of lead in gasoline would involve relatively little capital cost. This is because refiners overbuilt catalytic reforming capacity before the 1978 Iranian revolution and were left with a surplus as oil prices rose and gasoline demand fell. The capital costs of this excess capacity are already sunk.

II.C.1. Incremental Cost of the All Unleaded Case

We computed costs for three different categories of octane demand: a high octane scenario, a low octane scenario, and an intermediate octane scenario. We also looked at how sensitive our cost numbers were to changes in projected demand for gasoline.

We examined one additional factor that influenced costs. There are several octane boosting additives besides lead on the market. One of them, ethanol, receives large government subsidies. If we allowed ethanol demand to vary among our cases, and the model "saved money" by replacing lead with subsidized ethanol rather than using a more expensive alternative, we would be underestimating the cost of removing lead. We avoided this by holding the quantity of ethanol used constant as lead was removed.

Because other additives frequently contain fewer BTUs per barrel than gasoline, whenever additive use increased we readjusted total demand to keep BTUs, rather than volume, constant.

Case 1: High Octane Demand. If we assumed all misfueling was for octane, not price, the annual cost of removing lead from gasoline would be $759 million, of which $104 million was the cost of moving misfuelers back to unleaded and $655 million was the cost of eliminating leaded gasoline.

Case 2: Low Octane Demand. At the other extreme, if we assumed that 50% of the people using 91 octane premium unleaded would be satisfied by an 89 octane mid-grade unleaded, and that 50% of misfueling was due to price, the annual cost would decrease to $538 million, of which $66 million was the cost of moving misfuelers back to unleaded and $482 was the cost of eliminating leaded gasoline. (The petroleum industry's Coordinating Research Council studies of octane satisfaction suggested that about half of the people using 91 octane premium unleaded would be satisfied by 89 octane unleaded.)

Case 3: Intermediate Scenario. If we left all the premium unleaded demand at 91 octane and assumed that half of misfueling was due to price, the annual cost would be $691 million (of which $104 million, as in case 1, was the cost of moving misfuelers). We have used this number in Summary Table 1 because we believe that at least half of misfueling was due to price. Also, we cannot be sure that premium unleaded users will switch

to a lower grade, although we believe that some will. This point estimate represents caution, not expectation.

Case 4: Volume Sensitivity. This was measured against demand in the high octane case (6.5 million barrels), the most expensive case. If gasoline demand were 6.75 million barrels per day, our estimate for the high octane scenario would be $761 million. If gasoline demand were 6.25 million barrels per day, it would be $759 million.

II.C.2. Low-Lead Case

The 0.10 gram of lead per gallon of gasoline case resulted in annual costs of $550 million in 1988, assuming all misfueling were due to octane, and $503 million, if half were due to price. In the low octane demand case, costs would be reduced to $410 million.

TABLE II-4

Cost of Reducing or Banning Leaded Gasoline

	Range	Point Estimate	Misfueling Portion	cents*/ leaded gallon
	(millions of 1983 dollars)			
Low-Lead (0.10 g/gal)	$410-550	$503	$104	1.66¢
All Unleaded	$538-759	$691	$100	2.13¢

* This is the increased cost of making gasoline under the two options divided by the number of gallons of leaded gasoline in the base case.

As a check on the plausibility of the model, we examined the spot price* differential between leaded regular and unleaded regular for barge load quantities of fuel. This differential has been between one and four cents/gallon for the last few years. While spot prices can differ from manufacturing costs, they will not differ for long periods unless there are supply constraints. As the last column in Table II-4 indicates, when we allocated the cost of removing all lead from gasoline to our projected leaded gasoline demand, we obtained a cost per gallon well within the range of market price differences between leaded and unleaded regular gasoline. This confirms that our cost estimates are reasonable.

II.C.3. Cost of Lead Reduction

The low-lead and the all unleaded options would reduce lead emissions by about 33,400 and 35,600 metric tons, respectively, in 1988. The cost per metric ton of avoided lead emissions, therefore, would be about $15,100 for the low-lead option and $19,400 for the all unleaded option. (These figures are not net of vehicle maintenance savings, which we discuss in Chapter III.)

II.D. Price Differentials

Our estimates assessed incremental changes in manufacturing costs; they do not indicate what changes might occur in consumer

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* "Spot price" refers to the price of large quantity purchases on the open market, as compared to long-term supply contracts.

prices. Consumer price differentials between leaded, unleaded regular, and unleaded premium gasoline currently are considerably larger than the differences in manufacturing costs of the three grades and considerably larger than the refiners' price differential to intermediate and bulk purchasers. For example, average spot price differentials between leaded regular and unleaded regular for barge load quantities in New York harbor were 1.29 cents per gallon in December 1983. The differential at the Gulf termini of the pipelines bringing gasoline from the Gulf to the Northeast was 1.1 cents per gallon. Contract price differentials in the Gulf were 2.75 cents per gallon in Houston. (The source of these price differentials is Platts Oilgram.) On the other hand, retail price differentials are usually seven cents per gallon. This indicated that most of the price differential was added at the retail level, and may be part of the retailers' marketing strategy of cross-subsidization, where leaded gasoline serves as a "loss leader" product.

Apparently, price differentials depend on market conditions and oil company marketing strategies as well as costs. For example, most gasoline marketers presently seem to be selling regular leaded gasoline as a very low margin product, and are making their profit on unleaded grades. This situation has occurred in the past with regular or subregular leaded grades vs. premium leaded gasolines. The two most common explanations are that consumers shop on the basis of the lowest cost gasoline offered regardless of whether they purchase that gasoline, and

that the price elasticity of demand for gasoline is higher for users of leaded gasoline, perhaps because they own older cars.

It is difficult and beyond the scope of this analysis to predict what marketing strategies might be adopted if either the low-lead or all unleaded policy options were implemented. Under either of our hypothetical options, however, regular unleaded gasoline would be the lowest cost product. In fact, the model showed that in the 0.10 gram case the marginal cost of making unleaded gasoline would decrease slightly from its cost in the base case, while the costs of leaded gasoline and premium unleaded gasoline would both increase by about one cent per gallon. If marketers continue to make the lowest cost product the "fighting" grade, then the current situation will invert, with regular unleaded gasoline prices falling and leaded and intermediate-grade unleaded becoming the high profit products. The differences in prices that individual consumers pay will depend upon changes in retail marketing strategies.

In this analysis, however, we used the real resource costs of manufacturing to measure economic costs. We expect these to reflect the differences in prices that consumers pay on average. That is, we believe that all manufacturing costs will be passed on to consumers, and that average retail margins will not increase, although their distribution among grades may change.

II.E. Longer Term Projections

The costs for both the 0.10 grams per gallon and the all unleaded cases will decline over time because the total demand

for leaded gasoline will shrink as the fleet of vehicles designed for leaded gasoline retires. Thus, these restrictions will affect fewer gallons of gasoline in later years.

II.F. Refinery Model

Our estimates of the costs of lowering the lead content of gasoline, given these various projections, were calculated using the DOE linear programming model of the petroleum refining industry. The model simulates current and projected U.S. refining capacity, using available crude oil supplies, and projected imports to meet expected U.S. petroleum product demands. The objective function is to minimize costs, subject to constraints on lead usage. The model recently has been subjected to two verification checks by the Department of Energy (DOE), described in Attachment 1.

II.F.1. General Description of DOE Petroleum Refinery Yield Model

The DOE Refinery Yield Model estimates optimal refining industry operations under a range of assumptions and operating conditions. The solution provides "optimum" petroleum flows, prices, investments, etc., for the petroleum refining industry. In addition to the optimal answer, the model provides valuable economic information on important aspects of the refining industry's operations, such as the rate at which costs change (the marginal costs and values of specific refinery processes) as refinery operations are altered to change the yield of products or to accommodate different inputs.

The model contains approximately 350 equations to simulate the process by which crude oil and other inputs are turned into various products and the costs that are thereby incurred. The model can show which products can be made at varying costs in the many different refineries that exist throughout the world. It allows investment in new equipment in later years at a real (constant dollar) capital charge of 15%.

The DOE model is based on many fairly similar models developed and used widely by the petroleum refining industry for years. The refinery industry model was one of the earliest industrial applications of linear programming.

The basic model has been used by EPA in its analyses of the impacts of regulations on the petroleum industry and on petroleum product purchasers, and served DOE in many ways, including:

- evaluating Strategic Petroleum Reserve crude mixes for selections of storage sites,
- 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.

To understand the model, it is useful to describe briefly how refineries work. Exhibit II-1 is a schematic of a very simple refinery, often called a topping plant, which processes low sulphur crude oils. A complex refinery contains distillation units and other types of processing units. Exhibits II-2, II-3, and II-4 (provided by Sobotka and Company, Inc.) illustrate schematics of such refineries. (The model presents

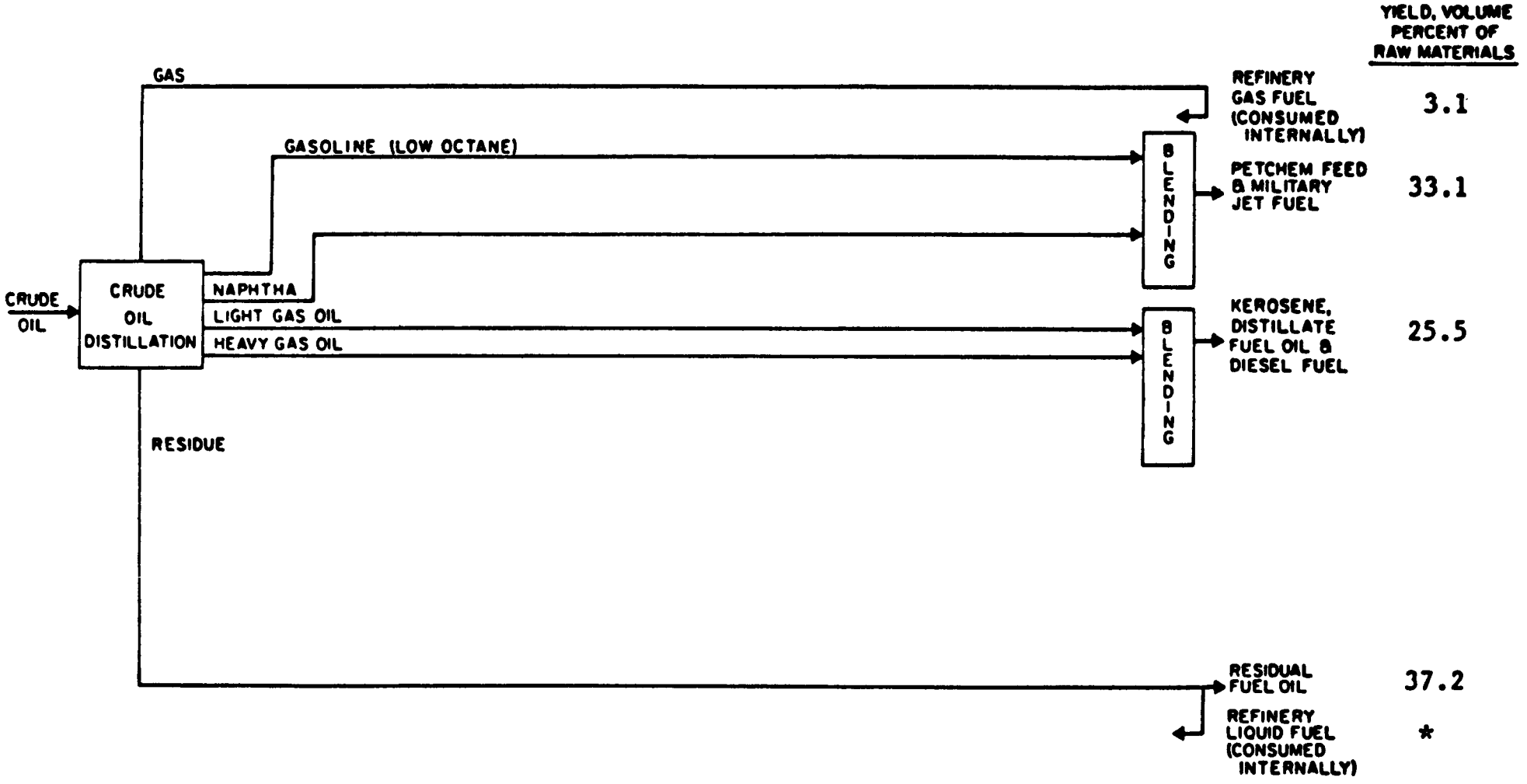
considerably more detail than even these exhibits indicate.) In all refineries, there is a selection of a combination of different process "units" that can be assembled into final structures that accomplish different but related purposes, and that look similar. The basic similarity of process units makes 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 takes in a raw material (crude oil or an intermediate product) and makes one or more intermediate or final products (and often some pollutants). 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. Exhibit II-5 is a summary of the basic types of refinery processes. Attachment 2 to this chapter 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

EXHIBIT II-1

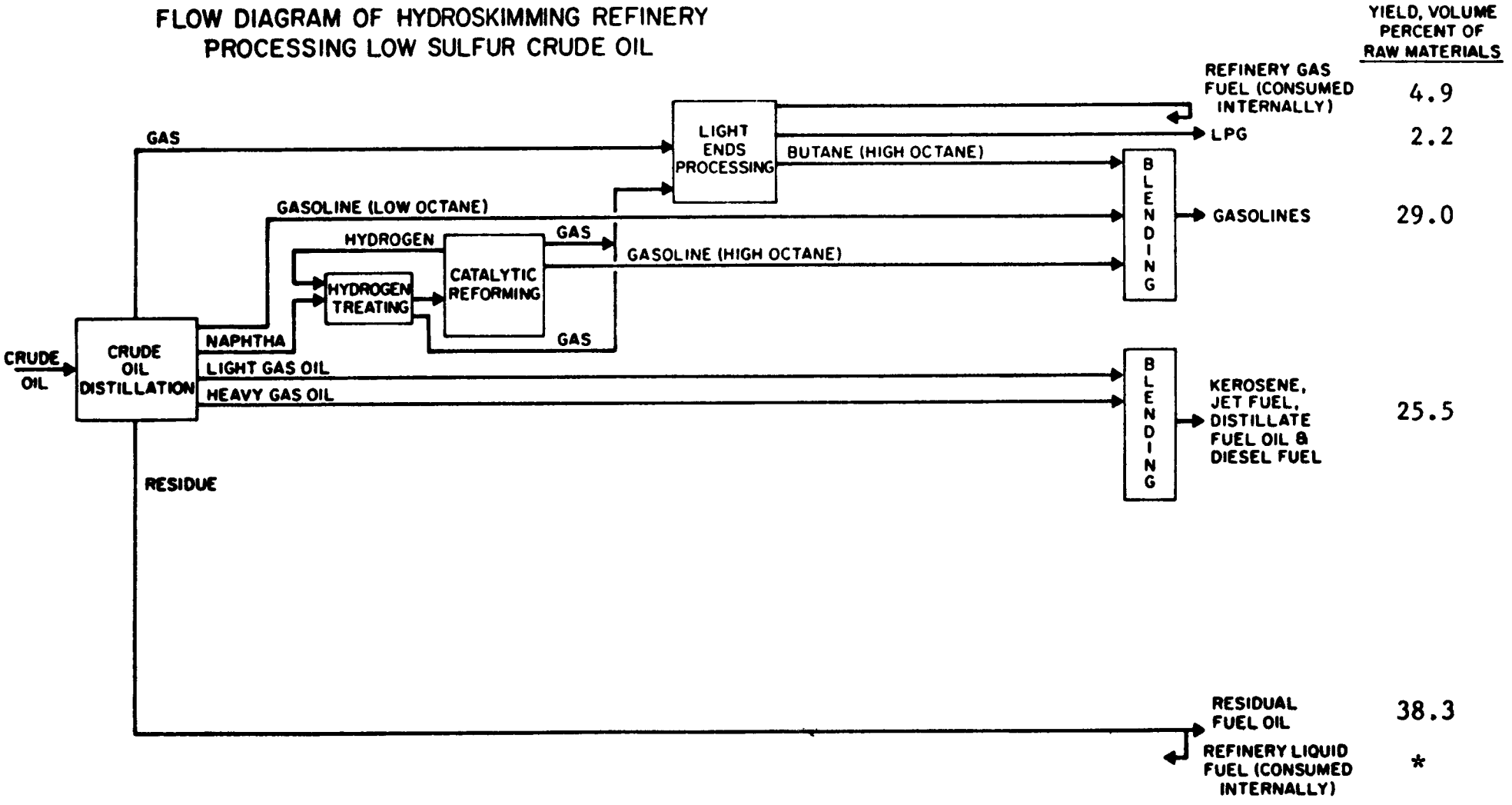
FLOW DIAGRAM OF TOPPING REFINERY
PROCESSING LOW SULFUR CRUDE OIL



*Included with gas fuel

EXHIBIT II-2

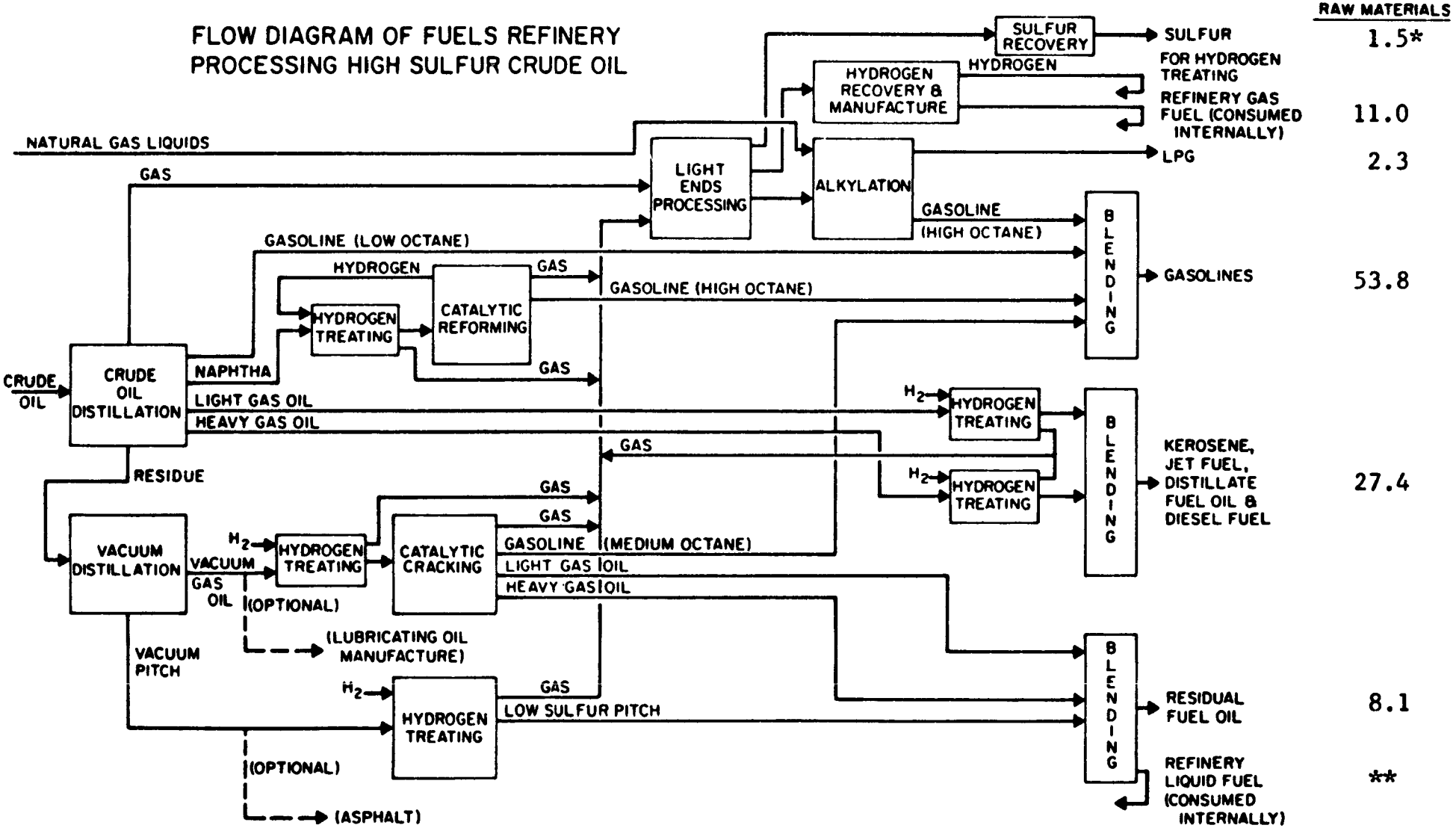
FLOW DIAGRAM OF HYDROSKIMMING REFINERY
PROCESSING LOW SULFUR CRUDE OIL



* Included with gas fuel

EXHIBIT II-3

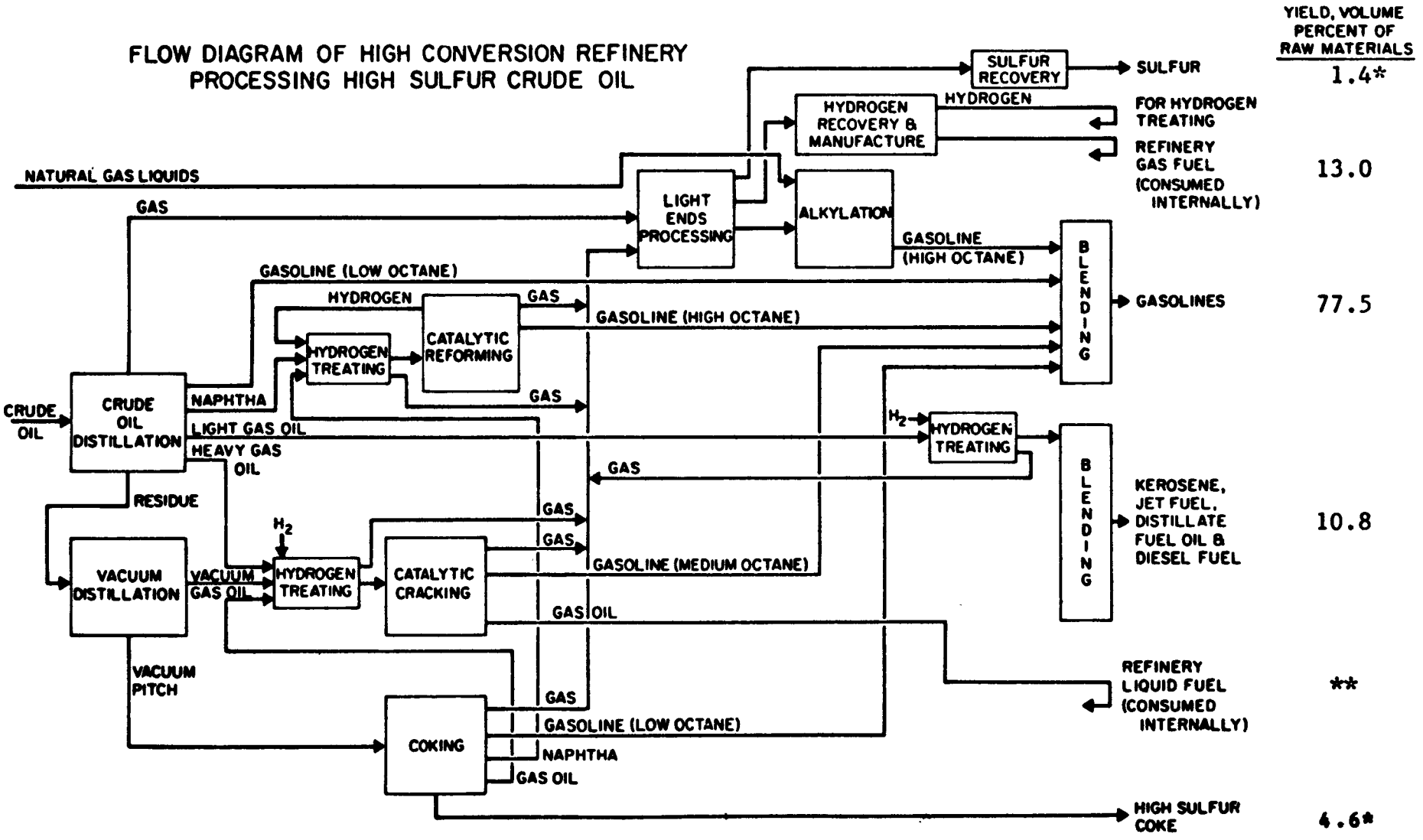
FLOW DIAGRAM OF FUELS REFINERY PROCESSING HIGH SULFUR CRUDE OIL



* Percent by weight
** Included with gas fuel

EXHIBIT II-4

FLOW DIAGRAM OF HIGH CONVERSION REFINERY PROCESSING HIGH SULFUR CRUDE OIL



* Percent by weight
** Included with gas fuel

EXHIBIT II-5

FUNCTIONAL CHARACTERIZATION OF PETROLEUM REFINERY PROCESSES

SEPARATION	ALTERATION (CONVERSION)
A. 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)	
B. 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

function of the material that the unit is "fed." Each unit incurs some costs that vary with how hard it is run -- called "severity".

The 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 large refining regions in which aggregate demands can be forecast.) Alternatively, the simulation can vary product quantities at preselected prices.

The principal purpose of using computer models to simulate petroleum operations is to measure differences between alternative scenarios in order to estimate the changes in petroleum activities when some conditions change. Simulations of petroleum activities are complex. The models are more reliable for determining differences in costs between scenarios than they are for predicting the total costs of manufacturing all petroleum products in the United States. So the major focus of analyses should be differences between alternative model solutions. These practical considerations should be kept in mind in the interpretation of model results.

Exhibits II-6 and II-7 illustrate the basic structure of the linear programming refinery model. All processes consist of a series of linear relationships that describe the process output and operating cost, given a specific input and a set of operating conditions. The relationships are stored in the model in the form of a process data 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 first column in Exhibit II-6 specifies that as one barrel of Saudi Light crude is processed, a mix of fifteen intermediate streams is created. The operation consumes fuel, power, steam, and capacity, and incurs variable operating costs of 9.2 cents per barrel.

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 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.

Exhibit II-8 presents projected capacity in 1988 for various processing units in the model.

EXHIBIT II-6

YIELDS AND OPERATING COST COEFFICIENTS
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	.001	.001	.001
PROPANE	.003	.003	.003
ISOBUTANE	.002	.002	.002
NORMAL BUTANE	.013	.015	.009
LT ST RUN (C5-175) LO OCT	.051		
LT ST RUN (C5-175) INT OCT		.040	.035
LT NAPH (175-250) PARF	.070	.060	.025
LT NAPH (175-250) INTM			.025
NAPH (250-325) PARF	.050	.044	.010
NAPH (250-325) INTM	.020	.011	.050
HVY NAPH (325-375) PARF	.020	.020	.005
HVY NAPH (325-375) INTM	.020	.014	.030
KERO (375-500) JET FUEL QUALITY	.115	.090	.070
KERO (375-500) OTHER	.015	.005	.040
DIST (500-620) HI SULFER	.130	.090	.100
HY GAS OIL (800-BTMS) (2.0% S)	.180	.180	.105
ASPH VERY HI SUL (4.3% S)	.143	.300	.350
	Operating Cost Coefficients (Per Barrel of Throughput)		
FUEL, FUEL OIL EQUIVALENT	-.021	-.022	-.020
POWER, KWH	-0.6	-0.6	-0.6
STEAM, LB	-60.7	-63.4	-57.9
OTH VAR OP COST, $	-.092	-.093	-.092
CAPITAL CHARGE		varies	

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

EXHIBIT II-7

YIELDS AND OPERATING COST COEFFICIENTS
CATALYTIC REFORMING UNIT
(200 PSIG Operating Pressure)

	Paraffinic Feedstocks		Naphthenic Feedstocks	
	90 RON	100 RON	90 RON	100 RON
REF FEED (250-325) PARF	-1	-1		
REF FEED (250-325) NAPH			-1	-1
CAPACITY FACTOR	.95	1.05	.95	1.05
Yields (Fraction of Intake)				
H2 (100 PCT FOE)	.034	.041	.047	.056
STILL GAS	.036	.069	.025	.036
PROPANE	.031	.076	.015	.030
ISOBUTANE	.020	.029	.003	.007
NORMAL BUTANE	.037	.052	.005	.012
REFORMATE (90 RON)	.852		.930	
REFORMATE (100 RON)		.739		.886
LOSS	-.010	-.006	-.025	-.027
Operating Cost Coefficients (Per Barrel of Thoughput)				
FUEL, FUEL OIL EQUIVALENT	-.042	-.045	-.042	-.045
ELECTRICITY, KWH	-2.6	-2.6	-2.6	-2.6
STEAM, LB	-75.	-75.	-75.	-75.
OTH VAR OP COST, $	-.099	-.108	-.099	-.108
CAPITAL CHARGE	varies			

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

EXHIBIT II-8

ESTIMATED U.S. REFINERY PROCESSING UNIT CAPACITIES FOR 1988
(thousands of barrels per day)

PROCESSING UNIT	CAPACITY
CRUDE DISTILLATION	15,900
COKER-DELAYED	1,175
COKER FLUID	170
VISCBREAKER	170
NAPHTHA HYDROTREATER	3,770
DISTILLATE HDS	2,670
FCC FEED HYDROFINER	1,085
RESID DESULFURIZER	580
CAT REFORMER 450 PSI	760
CAT REFORMER 200 PSI	3,145
FLUID CAT CRACKER	5,325
HYDROCRACKER - 2 STAGE	980
ALKYLATION PLANT	960
CAT POLYMERIZATION	77
HYDROGEN PLT, MBPD FOE	115
AROMATICS RECOVERY PL	300
PEN/HEX ISOMERIZATION	140
BUTANE ISOMERIZATION	55
LUBE + WAX PLANTS	240

Attachment 1 to Chapter II

Evolution of DOE Refinery Model and Current Status

In late 1983 Decision Analysis Corporation and Sobotka & Company, Inc., jointly updated the Department of Energy's Refinery Yield Model (RYM) and performed model verification tests for the Energy Information Administration. The recent update involved revisions to the model's raw material availability, product demands, and product specifications to reflect a 1982 environment. Processing capacities were revised to represent operable capacity on January 1, 1983, as reported by DOE. In addition, the model's technical representations were altered to reflect changes or improvements in processing technology that have taken place since the original model development, to update major crude assays, and to expand processing flexibility in the residual fuel portion of the crude oil barrel.

The verification tests of the updated model were conducted to determine how closely the RYM could simulate refinery activities in 1982. The verification test runs on the updated model were designed to verify material balance closure in the model solution and to assess the capability of the models to simulate actual regional refining activities. Each regional model was run with most crude and products specified at actual 1982 levels. The model then simulated the 1982 operations with some flexibility to vary marginal feedstocks and products. After the initial check for overall material balance closure, the model results were compared with actual 1982 refining balances, process utilizations, and economic relationships. The verification tests and results are discussed in more detail below.

Verification Methodology

The Refinery Yield Model (RYM) verification tests consisted of two simulations for each model region, Verification A and Verification B, specified as follows:

Verification A: All crudes except for a marginal high and a marginal low sulfur crude were fixed at the actual 1982 level[1]/ as were natural gasoline, plant condensate, outside fuel and utility purchases, and unfinished oils.[2]/ The marginal crudes were permitted to vary within a range equal to about 2 to 3 percent of actual crude input. Butane purchases were also allowed to vary but were not allowed to exceed actual. Product output was fixed at the 1982 level except for liquefied petroleum gas (LPG), coke, and low and high sulfur residual fuel.

Verification B: All input was specified at the 1982 level. Gasoline, distillate fuel, LPG, coke, and low and high sulfur residual fuels were allowed to vary while all other output was fixed at 1982 volumes.

The primary purpose of the first simulation test, Verification A, was to check the model for face validity. This included first a check for material balance closure in the overall refining operations as well as in each processing and blending operation. The results were then compared against actual operations to check the ability to meet end product demand with available feedstocks and to check the model's calculation of fuel consumption. Finally, the initial simulations were checked to ascertain if model economics and processing operations were within acceptable limits.

The second verification simulation runs, Verification B, allowed for an additional check of model face validity. The refinery material balances and projected economics were again checked against actuals. In this case, the models were allowed more flexibility to optimize and would be expected to operate major conversion processing at maximum. The product prices provided

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1/ Actual crude types were estimated by the contractors, based on available DOE data.

2/ Actual natural gas input was assumed to be equivalent to reported natural gas consumed for fuel. Actually, refiners may use additional natural gas as hydrogen plant feed. In the district 13 model, a large volume of natural gas was routed to the hydrogen plant, and therefore, natural gas purchases were increased about 25 percent.

are those which would result if all these facilities were in short supply (which was the actual situation during 1982).

Verification B runs also provided an assessment of model overoptimization. The volumes of light and heavy products produced from the 1982 volume of feedstocks were compared for each region run to evaluate the impact of overoptimization of product yield capabilities. In this comparison, the sum of gasoline and distillate production was compared to actual rather than production of individual products. The actual gasoline-distillate mix is a function of regional weighted average price differentials for 1982. The available price data are not sufficient to accurately test the model's projection of gasoline-distillate production costs.

Verification A Results: The results of the initial verification showed that the model was able to balance all material and account for all processing streams. The model provides a summary for each processing and blending operation which includes a balance row indicating any stream not accounted for in the model. The balance rows for all regional models were zero.

The model was able to produce a product slate close to actual operations with available raw materials. The flows calculated by the model were very close to actual figures. The model used about two percent less feedstock and produced about two percent less output. The model calculated a four percent loss of petroleum (products excluding refinery fuel) which is exactly the actual 1982 loss. Refinery fuel was about five percent higher in the model, indicating that the process efficiencies within the model may be slightly under-estimated.

Crude and product prices varied from region to region, but in general were reasonable. Gasoline and distillate prices were close, with regular gasoline typically less than one dollar per barrel above middle distillate. Low sulfur residual fuel was $6-8 per barrel below distillates and high sulfur residual around $15 below. These results compare well with 1982 actual price differentials.

Verification B Results: The aggregate U.S. refining balance from Verification B was close to actual 1982 operations. The models overstated the capability to produce light products by about 276,000 barrels per day (i.e., the yield of gasoline plus distillate per barrel of crude was overstated by 2.3%). The Verification B results indicated a large reduction in high sulfur residual versus actual. The high sulfur residual reduction was also due in part to the nature of the test. Refining regions were not required to produce a very low sulfur fuel grade that is typical of some regions, and were thus able to blend a greater volume of high sulfur components to low sulfur residual to meet product demands. Fuel consumption calculated by the model was about 11% higher than actual, but as a percent of total crude input there is less than a 1% difference.

The combination of Verifications A and B provide substantial confidence in the model's ability to predict the changes in costs and in operations that would take place in the domestic petroleum refining industry if gasoline specifications, such as limits on the use of lead additives, were changed. And the model also provides adequate flexibility in combining refinery process units so that the same analytical question can be answered for subsections of the petroleum industry categorized by size of plant or firm, or by the processing complexities of plants, or geographically.

ATTACHMENT 2 to CHAPTER II

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. Exhibit II-5 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.

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CHAPTER III

BENEFITS FROM REDUCED VEHICLE MAINTENANCE REQUIREMENTS

Lead in gasoline has served both beneficial and destructive functions. Refiners add lead because it is the least expensive way to boost the octane of motor gasoline. Thus, for gasolines of equivalent octane, leaded gasoline would be less expensive to make than unleaded gasoline. However, when vehicles burn leaded gasoline, deposits are formed in the engine and exhaust system. To reduce combustion chamber deposits, organic halogens -- primarily ethylene dibromide (EDB) and ethylene dichloride (EDC) -- are added to scavenge the lead.* These compounds react with most of the lead to form compounds more volatile than those formed with lead alone, and are discharged in exhaust gases. While this effectively reduces combustion chamber deposits, a significant portion still deposits on internal engine and exhaust system surfaces. Such deposits (e.g., halogen acids and lead salts) become very corrosive in the humid and warm environments within engines and exhaust systems. For these and other reasons, the use of unleaded gasoline reduces maintenance costs.

The deposits from leaded gasoline form a coating on exhaust valve seats. On pre-1971 and some other vehicles, this thin layer protects against the abrasive and adhesive wear that can occur between the exhaust valve face and valve seat during certain engine

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*Restricting or removing lead from gasoline would restrict or remove EDB and EDC. This would have compounding environmental benefits as EDB, EDC, and lead are substances of concern in leaks from underground storage tanks and in tailpipe and evaporative emissions. We have not included EDB or EDC benefits in our analysis.

operating modes. By 1971, however, several major engine manufacturers were building vehicles with valve seat metallurgy that had minimized or eliminated valve recession with unleaded gasoline.

Because leaded gasoline combustion products form engine deposits and corrode exhaust systems, studies have found four main categories of savings in operating and maintenance costs from switching to unleaded gasoline:

- less corrosion of the exhaust train, requiring fewer muffler and exhaust pipe replacements;
- better engine performance due to less fouling and corrosion of spark plugs;
- less corrosion and rusting in the engine, decreasing engine wear and allowing longer periods between oil changes; and
- better fuel economy, relating partly to better engine performance (from the effects listed above) and partly to the fact that unleaded gasoline contains more energy content per gallon than leaded gasoline. Quantitative estimates of this last benefit, however, are less reliable than the others.

We discuss each of these in the sections under Maintenance Savings (Sections III.A. and II.B. below).

As the remainder of this chapter indicates, switching misfueled and other vehicles currently using leaded gasoline to unleaded would likely produce millions of dollars in vehicle operation and maintenance savings. The fuel economy benefits are less certain and are not included in our summation of monetized benefits. Most cars using regular leaded gasoline would run as well, or better, on unleaded gasoline of the same octane. However, some older engines and non-diesel trucks require the valve lubrication that lead in gasoline provides. Valve recession

can occur in these engines from the inadequate lubrication of exhaust valves, potentially resulting in premature valve failure. The major constraint on an all unleaded policy in this decade is a technical one: some vehicles need the valve lubrication currently provided by lead in gasoline. The cost and practicality of other solutions to this problem (e.g., other protective additives or retrofits with improved valve seats) seem to pose significant obstacles, but more information is needed to evaluate these options.

As an alternative, we examined the maintenance benefits and technical feasibility of a low-lead option (0.10 grams of lead per gallon of gasoline) to lower sustantially the current concentrations of lead in gasoline (1.1 grams per gallon), but still allow sufficient lead to protect valves. The question of the linearity of the maintenance benefits, and an estimate of the dollar savings at 0.1 grams of lead per gallon of gasoline follow the discussion of the all unleaded case.

We have not included three additional adverse effects due to misfueling: plugging of catalytic converters, clogging of exhaust gas recirculation (EGR) valves, and reduced performance from exhaust gas oxygen sensors. In a recent communication with the Motor Vehicle Manufacturers Association (MVMA, 1984), these effects were raised, and subsequent contacts with automotive engineers substantiated the engineering rationales for these effects. The mechanisms for coating engine systems and exhaust systems would also plug catalysts and EGR valves and coat oxygen sensors. Extensive testing on another metal-based fuel additive,

methylcyclopentadienyl manganese tricarbonyl (MMT), clearly demonstrated the existence of catalyst and EGR valve plugging and interference with oxygen sensors.

Catalyst plugging may result in back pressure problems. Interference with oxygen sensors and closed-loop systems will affect fuel metering systems regulating the air-fuel ratio. Both affect driveability and fuel economy. Finally, plugging the EGR valve can also adversely affect fuel economy, knock, and driveability. Some of these effects were observed with MMT use and there is a plausible case for their occurrence with lead use in cars designed for unleaded gasoline.*

MVMA (1984) valued the cost to a single consumer for catalyst plugging at $300, for EGR plugging at $60, and for oxygen sensor disruption at $75, all after 50,000 miles of driving. Given the large number of misfuelers, over 12% of light-duty vehicles (EPA, 1983), the aggregate costs could be large. However, because we do not have the "dose-response" function for these effects, we could not evaluate them under the regulatory options examined here. Therefore, we have not monetized them. Excluding these effects obviously underestimates the maintenance benefits of reducing lead in gasoline.

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* DuPont (1982) has observed severe catalyst plugging due to lead in gasoline (0.5 grams/gallons); the implications of this are still under study.

III.A. Maintenance Savings in the All Unleaded Case

III.A.1. Sources of Data

In assessing the effects of lead on vehicle maintenance requirements and the potential savings of switching to unleaded gasoline, we evaluated nine studies and some independent ancillary data. Most of these studies were conducted in the late 1960s and early mid-1970s (Cordera, 1965; Pahnke and Conte, 1969; Pahnke and Bettoney, 1971; Gallopoulos, 1971; Gray and Azhari, 1972; Wintringham et al., 1972; Pless, 1974; Gergel and Sheahan, 1976; Hickling Partners, 1981).

Concerning exhaust systems and spark plugs, we examined four on-road vehicle studies involving nine samples of light-duty vehicles in both commercial and personal use. One oil company has provided a theoretical calculation based on its experiences with the effects of gasoline quality on vehicles (reported in Pahnke and Bettoney, 1971). For this analysis, we also examined changes in automobile manufacturers' recommendations for vehicle maintenance periods and the reasons for the changes, and we quantified the portion attributable strictly to a switch from leaded to unleaded gasoline. We have summarized the findings of these studies for spark plug and exhaust systems in Table III-1; we scaled the reported rates to reflect equivalent mileages to facilitate the comparison of results.

There are fewer data concerning the effects of lead in gasoline on oil change intervals, and some discussion is only qualitative. In addition to drawing upon manufacturers' recommendations, we used four sources of information. One was research

TABLE III-1
SUMMARY OF STUDIES ON MAINTENANCE DIFFERENCES BETWEEN LEADED AND UNLEADED VEHICLES

STUDY	REPLACEMENT RATES PER 11,000 MILES (OR PER 1 YEAR) SPARK PLUGS UNLEADED	SPARK PLUGS LEADED	EXHAUST SYSTEMS UNLEADED	EXHAUST SYSTEMS LEADED	AVG. MPV/YR	ACCUMULATED AVG. MVP	TYPE OF SERVICE	# OF VEHICLES LENGTH OF TEST	LOCATION
Pahnke & Bettoney (DuPont, 1969)	.534	.726	.0033	.187	11,400	65,000	Personal Use	59 matched pairs/ 4.7 years	South New Jersey and Wilmington, Delaware
Humble (rpted. in Pahnke & Bettoney, 1971)	.330	.550	.220	.275	10,000	--	Theoretical	--	--
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
Changes in Manufacturers Recommendations	2.2 more replacements for leaded vehicles, net of other technology changes		(Not Applicable)		(Not Applicable)	(Not Applicable)	(Not Applicable)	(Not Applicable)	(Not Applicable)

conducted on four fleets of commercial vehicles under conditions that strain oil performance (Pless, 1974). Another study used engine tests on a road simulator to compare the use of leaded gasoline at standard oil change intervals with unleaded gasoline at extended intervals (Gergel and Sheahan, 1976). The third source was a detailed analysis of the potential lengthening of periods between oil changes by switching to unleaded gasoline (Gallopoulos, 1971). Finally, Cordera et al. (1965) related engine rust build-up to lead concentration in gasoline.

In addition, some studies found other categories in which unleaded vehicles experienced lower maintenance expenses -- notably fewer carburetor adjustments (Gary and Azhari, 1971; Wintringham et al., 1972) and fewer engine overhauls. We did not include them, however, for several reasons: some of these effects may not be related exclusively to differences between leaded and unleaded gasoline, several studies used data bases that were too small to support meaningful conclusions, and some were not considered reasonable to extrapolate to vehicles operating in 1988.

III.A.2. General Comments on the Method

In quantifying the consumer benefits of switching from leaded to unleaded gasoline, we considered changes in the observed maintenance behavior of vehicle owners. For matched pairs of vehicles and drivers, changes in observed maintenance reflect, and are used as a proxy for, underlying effects of gasoline quality on vehicle performance and durability. If most people maintain their vehicles at manufacturers' recommended schedules, and would continue to do so with a switch to unleaded gasoline, our method could overestimate

maintenance benefits. This would also be true if manufacturers' recommended schedules were based on the performance and durability of the worst cars, rather than average cars. In this case, scheduled maintenance may provide a large safety factor relative to the average car, and we may have overestimated maintenance savings.

On the other hand, manufacturers may develop maintenance schedules by balancing the extra maintenance expenses of the average or better vehicles against the expected avoided costs of the more problem-prone vehicles. In this case, our evaluation of changes in maintenance behavior probably does not overstate benefits.

In any case, the evidence -- from the fleet studies we cite here, consumer surveys, and conversations with auto specialists -- indicates that, in general, people substantially under-maintain their vehicles relative to recommendations. (See, for example, the 1984 AAA Potomac Division survey that found most of 2,600 cars suffering from maintenance problems.) In sum, we expect that observing owners' behavior correctly reflects the intervals at which they begin to notice performance degradation. (An exception to this is exhaust systems, which comprise half the estimated savings, because people repair these only when they fail.)

III.A.3. Fewer Replacements of Exhaust Systems

All of the studies found demonstrable differences in expected lifetimes (measured in miles) of exhaust systems between matched pairs of unleaded and leaded vehicles. The range of

estimated differences between leaded and unleaded replacement rates, however, was very broad, from only 20% fewer muffler changes (at equivalent mileage) for unleaded vehicles (but based only on a theoretical calculation) to, more commonly, virtually no replacements for unleaded vehicles in four of the nine distinguishable fleets. 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.

Unfortunately, however, 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 travelled another 10,000 to 20,000 miles. The reported findings, thus, may have overestimated the differences between unleaded and leaded vehicles.

It is useful to look most closely at the Ethyl Corporation (Wintringham et al., 1972) findings, since their vehicles had the greatest mileage, and there is a clear geographic distinction between the fleets. Their Baton Rouge fleet, after over 84,000 miles of travel per car (compared to a projected lifetime of 100,000 miles), had essentially zero exhaust system repairs for unleaded vehicles, but rates 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 repair per 46,000 miles, but rates of one 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. This was consistent with the Environment Canada findings (Hickling Partners, 1982) for two municipal fleets, which had 42% fewer exhaust system replacements (at equivalent mileage) for cars using unleaded fuel in cold climates.

On the other hand, the DuPont (Pahnke and Bettoney, 1971) and Amoco (Gray and Azhari, 1971) findings, conducted in the mid-Atlantic region, 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. (The average muffler replacement rates for leaded cars among the different studies also varied greatly, ranging from 1 per 20,500 miles to 1 per 154,900 miles.)

Weighting Ethyl's findings for Detroit and Baton Rouge according to the portion of registered cars in Sunbelt versus Snowbelt states in 1982 (43% and 57%, respectively, according to MVMA, 1983), mufflers nationally would last an average of three times longer on unleaded vehicles than on leaded ones. However, because of our concern that these limited duration studies may have underestimated muffler replacements over the lives of vehicles using unleaded fuel, we conservatively concluded that mufflers on vehicles using unleaded fuel would last twice as long (in miles) as those on vehicles using leaded fuel. Given the projected lifetime of a car (100,000 miles), this

meant about two exhaust system changes per leaded vehicle versus one per vehicle using unleaded.

We assumed mufflers on vehicles using leaded gasoline would last about 50,000 miles. In the studies we reviewed, the leaded fleets averaged about 40,000 to 60,000 miles between exhaust system replacements. Several automotive specialists independently confirmed the reasonableness of this assumption.*

We calculated exhaust system replacement savings as follows: for leaded exhaust systems replaced once every 50,000 miles,** each mile therefore accounts for .00002 of the system replacement; for unleaded vehicles replaced once every 100,000 miles (doubled exhaust lifetime), the system replacement figure is .00001/mile. The difference is .00001/mile. At $120 per repair (muffler, tailpipe, and exhaust pipe), this was 0.12 cents/mile, or 1.68

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* Passing references in literature and several reviewers of this document 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, this design improvement might affect performance so that 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 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.

** It can be argued that effects due to fuel use are best determined in terms of total gallons consumed rather than miles traveled. For a majority of the studies examined in this paper, fuel consumption data were unavailable. Thus, we used an assumed value of 14 miles per gallon. To the extent that this value is higher or lower than the actual fuel economy of the vehicles in the studies used here, our estimates will vary accordingly.

cents/gallon (at the average 14 miles per gallon achieved by cars in the late 1960s).* A savings of 1.68¢ per gallon times light-duty vehicle demand (22.8 billion gallons of leaded gasoline in 1988) yielded exhaust system savings of $383 million in 1988 for the all unleaded case (1983 dollars).

III.A.4. Better Performance or Less Frequent Spark Plug Changes

The second category of maintenance savings is better vehicle performance by avoiding the fouling and corrosion of spark plugs by lead deposits. The fleet studies results were more consistent in establishing spark plug effects than exhaust system effects.

Eight fleets in four studies (Pahnke and Bettoney, 1971; Gray and Azhari, 1971; Wintringham et al., 1971; and Hickling Partners, 1982) clearly showed that owners of vehicles using unleaded fuel increased mileage intervals between spark plug changes by 35% to

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* Changing to savings per gallon, then extrapolating to 1988 via changes in leaded gasoline demand, automatically adjusts for changes in fuel economy and changes in miles per year among different cohorts of vehicles. Vehicles traveling fewer miles would burn fewer gallons and, hence, acquire fewer savings. Likewise, vehicles with better fuel economy would achieve lower savings than average. It should be noted that our benefits estimation assumes that these savings are a function of fuel use. Given the current trend towards more fuel efficient cars, such an assumption may considerably underestimate actual benefits, as the age of the vehicle becomes an important variable in determining the life of a muffler. Implicit in our model is the assumption that an automobile that gets 28 mpg will need a new muffler every 200,000 miles, or at 42 mpg, 300,000 miles. To the extent that these muffler lifetimes are overestimated, benefits are underestimated. Unfortunately, we were constrained by the lack of data concerning the effects of time on muffler life.

300% over the intervals for leaded vehicles. The average of the studies was about a 60% increase in the distance traveled between spark plug changes on unleaded versus leaded vehicles. We used this change in replacement to compute the savings of lowering lead concentrations from the pre-phasedown level of 2.3 g/gal to zero lead.

By comparison, the Environment Canada/Hickling Partners study (1982) found over a 50% increase in spark plug life for unleaded cars in the municipal fleet studies. They also found almost a doubling of the intervals recommended by auto manufacturers for spark plug changes on unleaded vehicles compared to leaded ones, a function of several technological improvements (e.g., the addition of high energy ignition systems).

However, some evidence suggested that the 1982 lead phasedown rule making, which lowered average lead concentrations to 1.1 grams per gallon, has already achieved a portion of this 60% increase in spark plug life. (Other data suggested that the same is <u>not</u> true for exhaust systems and oil changes -- lowering lead to 1.1 g/gal may not have provided savings in these categories. These are discussed in greater detail in section III.B. of this chapter.) Therefore, we pro-rated the 60% savings according to the portion left to be gained by further restrictions of lead.

There are scant data on spark plug fouling at very low lead levels. In 1971, Toyota (Champion, 1971) reported finding that fouling of spark plugs occurred at equivalent rates with unleaded and low-lead gasoline of 0.20 g/gal (both maintaining ignition

performance for 30,000 miles). At the 1972 Champion Spark Plug Conference, Union Oil also reported that spark plug performance was similar for unleaded and low-lead gasoline of 0.5 g/gal. Both outlasted by over four times the spark plugs operating with leaded fuel of 3.0 g/gal. These findings suggested that there was some threshold for gasoline lead content above which the lead in gasoline degraded spark plug performance. For lack of other information, we assumed that this threshold was 0.5 g/gal,* and that the relationship between lead and spark plug fouling was linear from this threshold to higher lead levels.

Earlier, we noted that intervals between spark plug changes could increase 60% by reducing lead from 2.3 g/gal to zero (0.0 g/gal.) If the threshold by which all benefits have been achieved is 0.5 g/gal, then the 1982 lead phase-down rule (which lowered the lead content of gasoline to 1.1 g/gal) would have resulted in (2.3-1.1)/(2.3-0.5) times 60% = 40% (estimated increase in change intervals). Thus, we have achieved 40% of these benefits already. The remaining 20% (i.e., 60% - 40%) gain would be achieved by phasing down from 1.1 g/gal to 0.5 g/gal (or to 0.0 g/gal). We used this 20% rate of savings to calculate the benefits of fewer spark plug changes.

We assumed that drivers of vehicles using leaded gasoline began to experience significant performance degradation by about

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* The Toyota and Union Oil results could have been averaged for a threshold of 0.35 g/gal. Use of a lower threshold will result in benefits of 23% increased spark plug life in phasing down from 1.1 to 0.35 g/gal (Champion, 1971, 1972).

12,000 miles of spark plug life. This was a little longer than automobile manufacturers' recommendations would imply, but somewhat less than the actual change intervals for leaded vehicles observed in most of the fleet studies. The observed intervals averaged about every 15,000 to 16,000 miles (but ranged from 10,000 to 37,000 miles for leaded vehicles).

A 20% increase in spark plug life accompanying a switch to unleaded gasoline would provide savings of about 0.35 cents/gallon of gasoline.* This, multiplied by the projected 22.8 billion gallons of demand for leaded gasoline in 1988 in the base case, translated to savings from fewer spark plug changes in 1988 of about $80 million under an all unleaded policy.

Interestingly, the effects appeared smaller than the researchers had hypothesized. Apparently, owners tuned up their vehicles and changed the spark plugs more as a function of mileage (and habit) than performance. Using the difference in observed behavior between paired drivers of leaded and unleaded vehicles, as these studies did, may underestimate the performance degradation of leaded gasoline on spark plugs and engine timing.

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* Calculation: A 20% increase in the 12,000 miles between spark plug change experienced in cars using leaded gasoline translates to 14,400 miles between changes. The difference in the number of changes per mile is therefore 1/14,400 - 1/12,000 or .000014/ mile. Given a price of $18 per spark plug change, this becomes .025¢/mile, or .35¢/gallon (at 14 miles per gallon).

In fact, using the data from MVMA (1984) reveals an estimate of $328 million* -- over four times the value derived in our analysis.

III.A.5. 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% of the lead in gasoline ends up in the used oil, comprising up to 50% of the weight of engine oil sludge (Gallopoulos, 1971).

The particles that accumulate in the used oil cause substantial abrasive wear in the engine, while the internal engine rust may cause hydraulic valve lifter sticking (Cordera et al., 1965). 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. While rusting can occur even in the absence of the halogen acids derived from lead salts, engine oil tends to be the major cause of internal rusting under normal driving conditions.

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* MVMA estimated spark plug changes to occur every 30,000 miles for vehicles using unleaded gasoline, compared with every 15,000 miles for those using leaded gasoline. This yields 1/15,000 - 1/30,000 = .000033 spark plug changes/mile difference. At $18 per plug change this is .06¢/mile or 1.44¢/gallon using the MVMA's figure of 24 miles per gallon. Given an estimate of 22.8 billion gallons of leaded fuel in 1988, total benefits are just over $328 million.

The fleet studies investigating differences in maintenance costs between unleaded and leaded vehicles tended either not to consider effects on engine oil, or found very small savings. In general, these studies were not conducted in a manner to determine easily the effects on oil change intervals or engine wear from using leaded or unleaded gasoline. Possibly consumers were not aware of the potential decrease in oil change requirements when using unleaded gasoline, and/or did not tend to change their habitual maintenance behavior. Therefore, this analysis relies more heavily on experimental studies of engine wear with unleaded and leaded gasolines at varying oil change intervals than the fleet studies. The exception is Pless (1974) which was a fleet study specifically designed to examine oil change effects. Even if consumers did not realize the possible short-term cost savings of fewer oil changes, they would have benefited from better engine durability with unleaded gasoline. Since most evidence indicates that vehicle owners do not change oil often enough, this would be especially true.

Many of the experimental studies in the early 1970s on oil change requirements did not provide conclusive evidence on oil quality after extended intervals between changes. The results consistently did show that unleaded gasoline decreased rusting, corrosion, and sludge; low temperature piston varnish tended to increase, however. No significant difference was found for oil thickening, high temperature varnish, or adhesive wear or scuffing. It was not clear whether, overall, unleaded fuel would allow substantially longer intervals between oil changes.

In any case, manufacturers have changed their specifications for oil changes from every 3-5,000 miles to every 7-10,000 miles.

A study by Gallopoulos, of the General Motors Corporation (GM), was one of the earliest works that we examined. He concluded in 1971 that with unleaded gasoline it might be feasible to extend requirements from 2-3 yearly changes to only one annual oil change, but added that more investigation was needed.

Pless, also of GM, reported more conclusive results in 1974 from experiments on taxicabs in conditions that take an unusually severe toll on oil quality. In a group of twenty taxis (1970 model year), Pless found less piston varnish, ring wear, and used-oil insolubles for the unleaded vehicles after 16,000 miles of stop-and-go service. However, the unleaded vehicles experienced more oil filter plugging and higher used-oil viscosity. On a fleet of 1971 taxis, he found that doubling oil change intervals with unleaded gasoline (from 8,000 miles to 16,000 miles) significantly increased oil filter plugging and used-oil insolubles.

On a fleet of 1972 taxis, Pless 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.

He qualified this conclusion by stating that only unleaded gasoline and SE or better quality oils be used. (Currently, SF oils, which are better than SE, are the most widely used.) Subsequent to these findings, both GM and Chrysler recommended lengthened periods between oil changes. Both companies now only manufacture cars built to run on unleaded fuel.

In 1976, Gergel and Sheahan (Lubrizol Corp.) found results similar to those of Pless, but found no significant plugging of oil filters. Importantly, they concluded that engine wear was the limiting factor in extending oil change intervals suggesting a maximum of 12,000 miles between changes for leaded 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 (either through reduction in abrasive lead particles or rust), oil degradation, and general engine and engine part cleanliness (e.g., lack of deposits and sludge). For analytical purposes, we need to determine the

functional relationship between lead in gasoline and oil change intervals. The available direct evidence is from Pless who tested engine oil change intervals on unleaded gasoline and 2.70 grams/gallon leaded gasoline. However, the existing lead phase-down regulation limits the lead content to 1.1 grams/gal. Given this data, the issue is whether some of the benefits of reduced oil change intervals already occurred in going to 1.1 grams/gal, and how much remains to be obtained in decreasing from 1.1 grams/gal to 0.1 or 0 grams/gal.

Gallapoulous, in discussing future engine oil requirements for unleaded vehicles, noted a number of studies which examined lead or lead scavenger use in relation to engine or engine part rusting. He concluded that the use of unleaded gasoline would result in less internal rusting. The inference was that with less sludge, oil degradation, and deposit build-up, the overall task of engine oils is reduced. As a result, a switch to unleaded gasoline would produce a net increase in engine oil lifetime.

With this engineering data in mind, we examined the studies relating lead additives or lead scavengers to engine rust. While it may be argued that most of these studies were designed to identify lead scavenger effects, it is also true that such scavengers would not be used in the absence of lead in gasoline. Furthermore, a substantial portion of the corrosive elements in the engine are acids derived from the lead halide salts, a product of both lead and its scavengers. In fact, all of the studies looked at various lead concentrations as well as lead scavenger concentrations.

One notable study (Cordera et al., 1965) examined the relationship betweeen engine rust and lead scavenger concentrations, while varying lead content. Cordera et al. showed that in addition to a relationship between lead scavenger concentration and degree of internal rust, there also was a relationship between lead concentration and rust. These authors evaluated valve lifter rusting at 0, 0.53, and 3.2 grams of lead per gallon of gasoline. The level of rust decreased non-linearly with decreasing lead content. An examination of the data indicated that in going from roughly 2.3 grams (pre-phasedown) to 1.1 grams there was a 12.7% improvement. From 1.1 to 0.1 g/gal there was an additional 58.3% less rust, and from 0.1 to 0 there was an additional 29% improvement. Thus, going from a current gasoline lead level of 1.1 grams to 0.1 grams would yield 58.3% of the benefits of eliminating lead and its scavengers, whereas going from 1.1 to 0 gives 87.3%.

The preponderance of evidence indicated that using unleaded gasoline decreases oil contamination, engine wear, and rust, even with a doubled oil change interval. We believe a decrease from 2.3 grams/gallon to zero would yield dollar savings at least as great as those which would accrue by the doubling of oil change periods. (Vehicle manufacturers recommended such a doubling for their vehicles concurrently with the switch to unleaded gasoline.) It is important to note that even if owners did not change maintenance behavior, i.e., if they continued with their prior oil

change intervals, they would still achieve longer engine durability from the greatly preserved oil quality when using only unleaded gasoline, and therefore achieve long-term savings.

Manufacturers' specifications have changed from one oil change roughly every 3,000-5,000 miles, to about one every 7,000-10,000 miles. This translated to about one or two -- instead of two or three -- oil changes per year. We assumed an oil change required 4 quarts of oil at $1.50 each, that oil filters ($4 each) would be replaced every other oil change (so $2/change) and we assumed 15 minutes of labor. (We valued that labor at an hourly wage rate of $10.00, the average for manufacturing.) This calculation was for a "typical" owner changing his/her own vehicle's oil and would be substantially less than the prices people generally pay at service stations. This yielded $10.50 per avoidable oil change, or savings of about 1.47 cents per gallon of gasoline.* In 1988, this produced additional savings of $332 million for the all unleaded case. But note, in the study by Cordera et al. we found that only 87.3% of these benefits were achieved in going from 1.1 to 0 grams of lead per gallon. Thus, we have lowered this value by

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* For vehicles using leaded gas, one oil change every 5,000 miles was assumed versus one every 10,000 miles for the vehicles using unleaded gas. Therefore, 1 change/5,000 miles minus 1 change/10,000 = .001/ mile. At $10.50 per oil change and an average fuel economy of 14 miles per gallon, 1.47 cents/ gallon is the average savings. With 22.8 billion gallons projected consumption in 1988, the value is $332 million.

12.7% for a savings of $292 million. Again, this is less than the value of $547 million predicted from using the MVMA analysis.*

III.A.6. Improved Fuel Economy

There are three reasons why drivers could expect to get better fuel economy by switching from leaded to unleaded gasoline:

- Unleaded gasoline has more energy content per gallon. These small per-gallon savings would accrue to any consumer of unleaded, rather than leaded, gasoline (Exxon, 1978).
- Lead fouls spark plugs, which hurts fuel economy. The benefits of avoiding this would be counted mostly by our spark plug estimate.
- For vehicles built after 1980, misfueling with leaded gas affects oxygen sensors, which can adversely affect fuel metering.

Energy Content

An analysis by Exxon (1978) on the energy content of different kinds of gasoline showed that vehicles using unleaded gasoline should get better mileage because unleaded gasoline contains more aromatic compounds and is "denser" (i.e., has higher energy content per unit volume) than leaded gasoline. Also, engines that run on unleaded gasoline build up more deposits in

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* The MVMA assumed unleaded gasoline vehicles require an oil change every 7,500 miles versus 5,000 miles for leaded gas users. The difference is, therefore, .00007 change/mile. Using MVMA estimates of $15/oil change and an average of 24 miles per gallon, this produced a value of 2.4 cents/gallon, or $547 million in 1988.

the combustion chambers which tend to increase the compression ratio, thereby improving engine efficiency slightly. For these reasons, vehicles burning unleaded gasoline should be slightly more fuel efficient. The Exxon memo calculated that improved fuel economy might be about 1 to 1.5%. Using a Society of Automotive Engineers formula (SAE #J1082b) to adjust miles per gallon for differences in gasoline density, and using the Exxon density estimates, we computed about 0.8% fuel economy improvements from using regular unleaded gasoline. This produced savings of about $199 million in 1988.*

We are not sure what the difference in density may be between future grades of leaded and unleaded gasoline. Because of this uncertainty we did not include these savings in our tabulation of benefits.

Spark Plug Fouling

Spark plug fouling caused by leaded gasoline also reduces fuel economy. This loss necessarily occurs in the interim between spark plug cleanings and changes, not just if maintenance does not occur. High energy ignitions, used in most vehicles by the mid-1970s, help extend spark plug life by maintaining reliability and may have some impact on delaying fouling and adverse fuel economy impacts. We probably have included much of this fuel economy increase in the previous section on spark plug savings, so

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* $199 million = 22.8 billion gallons of light-duty vehicle demand for leaded gasoline in 1988 times $1.10 per gallon times 0.8% fuel economy improvement.

we did not include it again here. But as a check on our previous estimate of spark plug savings, we calculated the fuel economy loss if spark plugs were not changed frequently enough. The fuel economy penalty of extra spark plug fouling would have to be only 0.32%* to be comparable to the estimated spark plug savings of $79 million in Section III.A.4.

Oxygen Sensor Fouling

For some misfueled vehicles, lead deposits can also affect oxygen sensors causing engines to run richer and thereby reducing fuel economy. How much this occurs depends on the types of feedback and failure modes of specific electronic controls, as well as how particular oxygen sensors react to the introduction of lead.

EPA's Office of Mobile Sources has estimated that the impact of these factors on gasoline consumption could be from 0-15% of misfuelers' consumption. Arbitrarily taking a low estimate from their range -- a 3% loss** -- we estimated roughly what preserving fuel economy might be worth for would-be misfuelers in 1988. The 3% loss, times 10.3 billion gallons of

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* Given retail gasoline prices of $1.10, our estimate of $79 million from spark plug savings is equivalent to 71.8 million gallons of gasoline. This, divided by the 22.8 billion gallons of light-duty vehicle demand for leaded gasoline, is equivalent to a .32% increase in fuel consumption.

** Four Canadian studies have estimated that fuel economy may be up to 4% greater for vehicles using unleaded gasoline. However, the applicability of these findings to the U.S. situations is questionable, so we did not use them in this analysis.

misfuelers' demand, times $1.10 per gallon, equals $339 million in 1988. Currently, we have insufficient data to estimate this more precisely, or to include it in our tabulation of benefits.

III.B. Maintenance Savings for the Low-Lead Case

The previous sections estimated the maintenance benefits likely to result from an all unleaded policy. If leaded gasoline were unavailable, however, some vehicles might experience excess valve wear. This risk suggested that we evaluate an option lowering the concentration of lead in gasoline to a level that still would be sufficient to protect against valve recession. (Valve recession and alternatives to prevent it are discussed in the next section of this chapter.)

This section examines the relationship between lead concentrations and maintenance benefits at high, low, and no lead levels. We then discuss the savings likely from a low-lead case allowing 0.10 grams of lead per gallon of gasoline. To estimate these benefits we had to assess the shape of the effects function in order to interpolate between the relatively high lead levels at which most research has been conducted (about 2.3 to 3.0 g/gal) and zero lead. With that information, we calculated the portion of the "all unleaded option" savings that would be achieved by the low-lead option.

Data with which to interpolate the relationships, and thus, to estimate savings, were scant. We are confident that current lead concentrations of 1.1 g/gal are above the threshold where

lowering lead levels would result in savings related to exhaust systems or oil changes. However, it is likely that the 1982 lead phase-down regulations may have already achieved some of the potential spark plug savings of going from 2.3 g/gal to 0 lead.

III.B.1. Exhaust System Savings

Most of the studies we evaluated to estimate maintenance savings involved fleets of vehicles, half of which used commercially available leaded gasoline. In the late 1960s, when these studies were conducted, the weighted average lead content of gasoline (weighted by the portions of premium and regular grades) was about 2.3 grams of lead per gallon. Unfortunately, because the discussion at the time focussed on relatively high lead levels versus "zero" lead, there are extremely few data with which to define the relationship between low lead concentrations and exhaust system corrosion between 2.3 and 0 g/gal.

Gray and Azhari (1971) was the only study that examined exhaust system corrosion rates at lead levels as low as 0.5 g/gal. They found no difference between corrosion rates at 2.3 g/gal and 0.5 g/gal, with corrosion rates at both lead levels 10-20 times higher than those of vehicles using unleaded gasoline. This suggested that there was some threshold at or below 0.5 g/gal, below which lead levels must fall before any savings may be achieved by fewer muffler replacements. It also suggested that no savings were achieved from previous "lead phase-downs", since

the current lead concentration is 1.1 g/gal -- well over Gray and Azhari's upper bound threshold of 0.5 g/gal. With no information to the contrary, we assumed that the relationship between lead levels and exhaust corrosion was linear below this threshold.

To calculate the exhaust system savings at 0.1 g/gal, we distinguished between two categories of would-be users of leaded gasoline: misfuelers and those vehicles designed to use leaded gasoline. Because of the likely changes in price differentials, marketing strategies, and possible administrative controls on the distribution of leaded gasoline, we assumed that there would be no misfueling under the 0.1 g/gal low-lead case. For consumers who previously had misfueled, the savings would be the same under both the low-lead and no lead cases: 1.68 cents/gallon savings, or $173 million in 1988.*

For light-duty vehicles designed to use leaded gasoline, savings in the low-lead case would be (.5-.1)/.5 or 80% of the all unleaded savings for fewer exhaust system replacements. This would equal 1.34 cents/gallon, or $168 million in 1988.** Adding this to the savings for misfuelers, we estimated the exhaust system replacement savings under the low-lead option would be $341 million in 1988.

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* $173 million = 1.68 ¢/gal times 10.3 billion gallons of misfuelers' demand in 1988.

** $168 million = 1.34 ¢/gal times 12.5 billion gallons of legal (non-misfueling) light-duty vehicle demand for leaded gasoline.

III.B.2. Spark Plug Savings

As with exhaust system corrosion, we had little information about the form of the relationship between low lead concentrations and spark plug fouling. As we discussed in section III.A.4 of this chapter, two citations indicated that all spark plug savings are likely to be achieved by lowering lead concentrations to 0.5 g/gal (from the studies' beginning point of 2.3 g/gal). For the purposes of this analysis, further savings would be gained from less spark plug foulings by going from current levels of 1.1 g/gal only to 0.5 g/gal; no further savings would be achieved by reducing from 0.5 g/gal to zero lead.* Thus, given the state of our current knowledge, total savings would be identical for misfuelers and other leaded light-duty vehicles in both options under consideration. (But the uncertainty surrounding the correct threshold for additional engine fouling is substantial, and affected our estimates for both the low-lead and all unleaded cases.) As earlier, we estimated that spark plugs would last 20% longer under either policy, resulting in .35 cents/gallon savings, or about $79 million for both the low-lead and all unleaded cases in 1988.

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* As previously noted in section III.A.4 the threshold could be an average of the two available studies (Toyota and Union Oil), in which case the threshold would be 0.35 g/gal with 23% of the savings available in going from 1.1 to .35 g/gal versus the 20% value used in going from 1.1 to 0.5 g/gal. We have thus understated benefits by using a higher threshold.

III.B.3. Oil Change Savings

Our discussion of the savings to be achieved from fewer required oil changes was made in Section III.A.5. Principally, we relied on the work of Pless, who found decreased engine wear with unleaded gasoline and doubled oil change intervals compared with engines using gasoline containing 2.70 grams of lead per gallon and a standard oil change interval. We also relied on Gallopoulos and Cordera, who described the relationship between engine rust and lead additive variations. To interpolate oil change savings to our low-lead case of 0.1 g/gal, we used the same methodology as in the no lead case. In going from 1.1 to 0.1 grams of lead per gallon, 58.3% of the benefits are achieved. For legal leaded gasoline users this becomes a savings of $107 million. Since we assume that no misfueling would occur under the low-lead option, the savings achieved by misfuelers under this option are the same as the all unleaded option, or $132 million. Total savings are the savings for the legal leaded gasoline user plus the misfuelers' savings: $239 million.*

III.B.4. Sum of Maintenance Savings for the Low-Lead Case

As calculated in the previous three sections, we estimated $339 million savings from decreased exhaust system replacements, $79 million savings for less spark plug fouling, and $200 million savings with fewer required oil changes. In total, lowering lead

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* Calculation: Legal leaded users: 1.47¢/gallon times .583 = .857¢/gal savings. Thus, .857¢/gal times 12.49 billion gallons of legal leaded use equals $107 million. Misfuelers: 1.47¢/gal times .873 = 1.283¢/gal. Thus, 1.283¢/gal times 10.29 billion gallons equals $132 million.

concentrations to 0.1 g/gal would yield about $618 million in vehicle maintenance benefits.

III.C. Risk of Valve Recession

Balancing these savings is the fact that reducing the amount of lead in gasoline may increase wear on some engines requiring the lubrication that lead compounds now provide. In particular, some studies argued that severe exhaust valve recession could occur, resulting in leaking valves, loss of compression pressure, greatly increased hydrocarbon emissions, degraded vehicle performance, and reduced fuel economy. Reviewing the available data, it appeared that eliminating leaded gasoline could mean exhaust valve recession in some light-duty vehicles and other engines that were originally designed to run on leaded gasoline.

The following paragraphs describe:

- the process of valve recession,
- the conditions under which it is likely to occur,
- the concentration of lead in gasoline needed to prevent damage, and
- alternative mechanisms that might provide sufficient protection.

We also estimated the types and numbers of engines that might be at risk without leaded gasoline.

Exhaust valve recession appeared to result from both abrasion and adhesion on the valve seat when engines operated under high temperatures, loads, or engine speeds. (For detailed discussions of the mechanisms of valve wear, see Godfrey and Courtney, 1971; Giles, 1971; or Kent and Finnegan, 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, and Godfrey and Courtney were consistent in finding that recession rates appeared to be mostly a function of engine speeds. Giles, for example, found that valve recession increased almost linearly with higher engine speeds to a point (on the engine he tested, about 3700 rpm), and then rose as an exponential function of engine speed. The shape of this function apparently varied significantly by vehicle models and years.

We reviewed two types of research about the causes and rates of valve recession. The first type of study was engine tests on dynamometers, done either using unusually high engine loads to test valve durability, or using 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. An advantage of engine tests was their greater measurement precision and control over test conditions. The vehicle studies, on the other hand, may have been more likely to reflect "real world" effects.

Table III-2 summarizes the available studies of valve recession as a function of lead concentrations. It should be noted 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 two described later, 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 a vehicle traveling at 60 mph than vehicles traveling at 70 mph, despite going 22% to 280% more miles. The following conditions were used in engine studies finding serious valve wear with unleaded gasoline:

- Giles (1971) conducted tests with passenger car engines under varied conditions from steady-state wide-open throttle (WOT) to simulated road-load cycles. He found that the valve recession rates were about ten times higher without lead than with 2 to 3 grams of lead per gallon of gasoline. But since he does not report the magnitudes of recession, it was impossible to tell how serious the recession was under the various conditions. (Valve recession occurred at a slight rate even with lead additives.)

- Giles and Updike (1971) ran one engine for 50 hours at a steady-rate of 3500 rpm WOT (speed selected to maximize valve recession rates while minimizing the other engine durability problems of other components at higher engine speeds); another engine ran for 50 hours at 2600 rpm WOT. Finding: about three times the rate of recession with unleaded gasoline vs. leaded.

- Kent and Finnegan (1971) also found severe valve recession in tests simulating a 1970 V-8 pick-up truck hauling a camper at freeway speeds of approximately 65-70 mph, with some engine cycling, for a total running time of 80 hours. In contrast, they found very low exhaust valve wear when running engines at 2300 and 2400 rpm.

- Godfrey and Courtney (1971) found somewhat lower rates of recession than did other high load engine studies when they tested an engine at 4400 rpm WOT for 10 1/4 hours. They also found recessions of unreported magnitudes on six other engines running 9,000 to 11,000 miles at 70 mph under conditions designed to generate artificially high temperatures.

- Felt and Kerley (1971) used both dynamometer and road tests, mostly testing at 70 mph freeway schedules, and some on a cycled route of combined city and freeway driving. They found 2/3 less valve wear at 60 mph than at 70 mph.

- Pahnke and Bettoney (1971) found serious valve recession in three unleaded dynamometer tests after the equivalent of 8,000 miles at a steady speed of 70 mph.

All 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 valve recession under normal driving conditions.

Overall, it seemed that using unleaded gasoline exclusively in vehicles requiring lead's lubrication may risk premature valve failure under severe engine loads. These studies indicated that such severe recession is most likely to occur in vehicles traveling at high loads or speeds well above the legal speed limit of 55 mph for extended periods of time (tens or hundreds of hours, or for thousands of miles). Several related studies using fleets of drivers under more typical conditions found little or no incidence of valve problems with unleaded gasoline (Pahnke and Conte, 1969; Orrin et al., 1972; Gray and Azhari, 1971). Two studies cited more valve problems for unleaded than for leaded vehicles (Wintringham et al., 1971; Felt and Kerley, 1971).

TABLE III-2

SUMMARY OF FINDINGS: VALVE RECESSION AT VARIED LEAD CONCENTRATIONS

Paper	Test Type	g Pb/gal	Findings
Pahnke & Conte, 1969	Employee fleet, Personal use	2.8,0.1,0	No extra valve problems with unleaded
Gray & Azhari, 1971	a. Employee fleet b. Consumer survey	2.3,0 2.3,0	a. " b. "
Pless, 1974	Taxi fleets	0	No severe valve problems, but some valve stem wear in one fleet with unleaded
Orrin et al., 1972	Taxi fleets	2.8,0	No extra valve problems with unleaded
Giles, 1971	Varied engine loads	2.5,<0.03	Need more than 0.03 g/gal
Giles & Updike, 1971	Varied high loads	0	Recession rate inc. above 70 mph. Avg. driving should not pose problem.
Doelling, 1971	Engine tests	0.14,0.07, 0.04,0	Between 0.04 and 0.07g/gal is adequate
Kent & Finnegan, 1971	High load	3.0,0.5,0.2,0	0.20 g/gal is adequate
Pahnke & Bettoney, 1971	a. Consumer survey	2.3,0	a. No clear difference, but somewhat more valve problems with unleaded
	b. High load, engine tests	0.5,0	b. Severe recession after 8000 miles on unleaded; none at 0.5 g/gal.
Fuchs, 1971	Engine tests	0.5,0	0.5 g/gal virtually eliminates recession
Felt & Kerley, 1971	a. Employee fleet	0.5,0	a. More valve problems with unleaded
	b. High load & cycled	0	b. Recession rates accelerate with high speeds; 0.5 g/gal is adequate
Godfrey & Courtney, 1971	High load engine tests	0	High loads and speeds are major causes of recession
Crouse et al., 1971	a. Patrol fleet, very severe service	3.1,0	a. Recession after 10-15K miles or more in severe service
	b. 50K mile road test (.008 g/gal)	2.6,0	b. In matched pairs directionally less tip wear on unleaded; severe recession in one unmatched engine
Wintringham et al., 1972	Employee fleets	2.3,0	More expensive valve problems with unleaded

Other fleet studies were inconclusive concerning the relative incidence of valve problems for unleaded vehicles (Pahnke and Bettoney, 1971; Crouse et al., 1971; Pless, 1974). Finally, reported incidents of valve problems were rare among users of unleaded gasoline in the late 1960s (Wintringham et al., 1972).

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 (from 0.001 inch per hour (iph) initially, stabilizing at less than 0.0001 iph after 25 hours). Giles then 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). Recession then rose again to high rates. In sum, it may take 10-25 hours for lead deposits to build up sufficiently to mitigate valve wear; if leaded fuel is not used, these deposits will wear off in several hours (about 10), leaving the exhaust valve vulnerable again to wear.

III.C.1. How Much Lead is Required to Protect Valves

A critical question is: "How much lead or similar additive in gasoline is necessary to protect against severe valve recession?" Most studies were performed with the high lead concen-

trations in gasoline that were common in the late 1960s -- about 2.3 grams of lead per gallon of gasoline. Also present in that gasoline are traces of sulfur, which occurs naturally in petroleum, and small quantities of phosphorus, which is added with lead to modify the deposits in the cylinder.

Several studies concluded that 0.5 grams of lead per gallon of gasoline was a sufficient concentration to protect against valve recession (Kent and Finnegan, 1971; Pahnke and Bettoney, 1971; Felt and Kerley, 1971; Fuchs, 1971). Kent and Finnegan 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 2650 rpm at lead levels of 0.04, 0.07, and 0.14 g/gal, for 100 hours each. Focusing on maximum recession of any of the valves, Doelling found no recession at 0.07 or 0.14 g/gal, but found excess wear at 0.04 g/gal. He thus concluded that leaded gasoline would protect exhaust valves beginning at levels between 0.04 and 0.07 grams of lead per gallon.

III.C.2. Alternatives to Lead to Avoid Valve Recession

Other mechanisms besides lead in gasoline can mitigate significant valve recession. Among these, improved metallurgy of the valves and phosphorus additives to gasoline are of greatest interest for this analysis.

Since 1971, the automobile industry has used induction hardened valve seats, seat inserts, or chrome or nickel plating in light-duty vehicles to mitigate valve recession without lead. Essentially, these technologies either stop the oxidation of the iron valve seat or harden the valve seat (or face) to protect against the adhesive and abrasive processes. By 1971, General Motors Corporation was using this improved metallurgy on all its light-duty vehicles to compensate for the use of unleaded gasoline. Ford made these improvements on most of its light-duty vehicles by 1971 as well. After that date, other manufacturers also implemented these changes. By the 1975 model year, virtually all cars were "clear fuel tolerant," although the changes in light trucks may have been slower. The widespread use of these improvements since the early 1970s has greatly limited the number of vehicles that might be at risk of valve damage due to the unavailability of leaded gasoline.

In addition, other substances could feasibly provide vehicles with the lubrication they now receive from lead in gasoline. Most plausible among the alternatives are phosphorous compounds which are already added to gasoline along with lead (i.e., the alternative technology is already in use and has been found to be effective in reducing valve wear).

Several experiments suggested that phosphorus in unleaded gasoline could reduce or eliminate the threat of valve recession at high speeds. Specifically, at about 0.06 or 0.07 g phosphorous/gallon, valve wear proceeds at one-half to one-third the rate

occurring with no additives (Giles and Updike, 1971; Kent and Finnegan, 1971; Felt and Kerley, 1971; and Wagner, 1971). 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% 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) showed that neither altering the scavenger mix nor eliminating scavengers from the fuel curtailed exhaust valve life in the engines. They found the presence of phosphorus in the gasoline was critical to exhaust valve life durability. All of these results indicated that the addition of phosphorus to unleaded gasoline would substantially reduce the risk of valve recession for those vehicles at risk.

In addition to gasoline engines in light-duty vehicles, three other categories of engines might require leaded gasoline.

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* Giles (1971) said that the limit of tolerable recession was about 0.125 to 0.150 inches of change, at which point the hydraulic valve lifters had problems operating.

The first of these is a variety of classes of small engines: lawnmowers, snowmobiles, snowblowers, garden tillers, and others small equipment. We asked representatives of the three major manufacturers of small engines in the United States (Briggs, Tecumseh, and Kohler) what kind of gasoline they recommend for these engines. They said that their engines almost always could use either leaded or unleaded gasoline, or they should use unleaded gasoline. These representatives also believed that this had been true for their engines for at least a decade (and knew of no changes that would make this untrue for earlier engines). Importantly, the reason they cited for preferring unleaded gasoline for this equipment was that leaded gasoline caused harmful deposits and corrosion in the engines.

Second, we investigated the possibility that marine engines required leaded gasoline, but the responses from manufacturers of boat engines were not clear-cut. Some boat engines may require the lubrication they now get from lead, while others are supposed to use clear fuel. Two-stroke engines should not be affected by using unleaded gasoline. One complicating issue was the relative octanes of leaded and unleaded gasolines. With the limited information we had, it appeared that some, but not all, marine engines would require leaded gasoline.

Third, gasoline-powered heavy-duty trucks are designed to use leaded gasoline. (Such trucks account for roughly 4% of all gasoline demand.) Because heavy-duty trucks are more likely than passenger cars to travel under heavy loads for long durations,

this category may carry the highest risk of premature valve failure if fueled with only unleaded gasoline. However, the extra magnitude of risk was difficult to assess because we did not know what fraction of these trucks used a high portion of their potential power. Giles (1971) wrote that

> Heavy duty engines, however, usually have valve seat inserts, rotators, and heavy duty valves already included in the design package.... Valve face recession and seat wear both are observed with heavy duty engines running on leaded fuels today. Wear rates are low, however, and recession is noticed only because of the extended operating life of these engines. Some increase in wear rates might occur when these engines are switched to lead-free gasoline, but catastrophic wear is not expected. Limited dynamometer testing does indicate that wear is not increased significantly but each engine design and application should be weighed separately. (p.1483)

Nonetheless, manufacturers reportedly would recommend against allowing heavy-duty trucks to operate solely on unleaded gasoline. As a result, heavy trucks may be the single category significantly affected if leaded gasoline were not available.

We have estimated approximately how many engines might be "at risk" of severe valve recession if leaded gasoline were not available.* Using the method and assumptions described in detail in the Technical Appendix to Chapter IV, we estimated that about 2.2 million light-duty vehicles without improved valves would exist

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* For most of these vehicles at risk, the probability of severe valve recession due to lack of lubrication from lead appears to be well below 10% in any year. Because of limited data, we were unable to quantify this probability with any greater precision. The probability for any individual vehicle will depend very much on its particular design and the ways in which it is operated.

in 1988. An additional 12.2 million cars (model years 1971-80) were designed for leaded gasoline and some of these may be at risk. However, GM has indicated that since 1971 all of its cars used the improved valves. Because roughly 50% of the market was GM vehicles, we have reduced this number by half, to 6.1 million cars. Ford also used improved valves and we have reduced the value by Ford's market share of roughly 20%. This final value (3.7 million cars) is most likely too high also because other manufacturers probably used improved valves as well. Table III-3 shows a more disaggregated forecast and presents projections for the full range of engines. These 25.5 million "at risk" vehicles represent about 13.5% of the 188 million projected total fleet of highway vehicles.*

III.D. Summary

This chapter has presented national estimates of the vehicle maintenance benefits of a reduction of lead in gasoline. Two scenarios have been examined: a reduction of lead in gasoline from the current 1.10 grams/gallon to 0.10 grams/gallon, as well as the total elimination of leaded gasoline. These estimates are based on projections of gasoline demand and vehicle fleet characteristics in 1988, and are valued in 1983 dollars.

Three sources of vehicle maintenance benefits have been tabulated and are presented in Table III-4. Both policy options

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* Equal to 1.17 times the projected light-duty truck and light-duty vehicle fleet of 160 million, using Bureau of Census 1977 proportions.

Table III-3

NUMBER OF ENGINES AT RISK OF SEVERE VALVE RECESSION WITHOUT LEADED GASOLINE

Type of Vehicle	Thousands of Vehicles in 1988	
	High Risk*	Low Risk*
Cars Pre-'71	2,240	--
LDT† Pre-'71	1,146	--
Cars 1971-1980	--	3,700
LDT† 1971-1975	--	7,505
Heavy-Duty Trucks	10,865**	--
Boats	Not calculated	Not calculated
TOTAL "AT RISK" GROUP:	14,251	11,205

TOTAL ALL VEHICLES: 25,456,000

* The "high risk" group represents heavy-duty and those light-duty vehicles manufactured before 1971. While many vehicles manufactured between 1971-1980 were built to use leaded gasoline, most of these have newer more durable valve and valve seat materials and and thus form a "low risk" group.

** This is equal to 30% of our projection of light trucks in 1988 -- the same proportion as was found in the Bureau of the Census publication 1977 Census of Transportation (1980).

† Light-duty trucks.

are expected to result in savings in decreased exhaust system replacements, longer life for spark plugs, and increased time intervals between oil changes. The point estimate of vehicle maintenance benefits for the low-lead option is $618 million, while the no lead scenario yields an estimate of $741 million in benefits. Several other sources of potential vehicle maintenance benefits have also been discussed in this chapter, but no monetary estimate of their magnitude has been attempted.

Chapter III also presents estimates of the number and types of vehicles expected to be at increased risk of severe valve recession if leaded gasoline is completely eliminated. As shown in Table III-3, at most 25 million vehicles are potentially at increased risk of damage in 1988. However, monetary values for these damages have not been computed due to considerable uncertainty as to the number of vehicles likely to experience damage.

TABLE III-4

SUMMARY OF MAINTENANCE SAVINGS
(1983 dollars, in millions)

	Misfuelers	LDVs* Designed to Use Leaded	Total
Billions of gallons of leaded gasoline demand in 1988	10.29	12.49	22.78
ALL UNLEADED SAVINGS:			
Exhaust Systems @ $1.68¢/gal	$173	$210	$383
Spark Plugs @ 0.35¢/gal	$ 36	$ 44	$ 80
Oil Changes @ 1.47¢/gal	$132	$160	$292
		TOTAL:	$755 Million
LOW - LEAD SAVINGS:			
Exhaust Systems	$173[1]	$168[2]	$341
Spark Plugs	$ 36[1]	$ 44[3]	$ 80
Oil Changes	$132	$107	$239
		TOTAL:	$660 Million

1. Assumes that changes in price differentials, marketing strategies, and possible administrative controls on distribution of leaded gasoline would effectively eliminate misfueling in the 0.1 g/gal case.

2. Assumed threshold to achieve any savings is at 0.5 g/gal.

3. All savings achieved by 0.5 g/gal.

* Light-duty vehicles.

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CHAPTER IV

BENEFITS OF AVOIDING EXCESS HC, NO_x AND CO EMISSIONS

This chapter discusses the effects of increased emissions from poisoned catalysts of vehicles misfueled with leaded gasoline. We estimated the excess emissions of hydrocarbons (HC), nitrogen oxides (NO_x), and carbon monoxide (CO) that we could avoid by eliminating misfueling by 1988. We then examined three alternative methods to value avoiding this air pollution. We synthesized this information to generate a best estimate of the economic benefits of reducing misfueling.

Both the all unleaded and low-lead policy options are assumed to eliminate misfueling and its consequent excess emissions. "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 gasoline, some drivers deliberately misfuel their vehicles in an attempt to reduce expenses or to improve vehicle performance. Our low-lead option assumes a low-lead fuel (0.10 grams of lead per gallon of gasoline) for the few classes of vehicles that may require the valve lubrication lead provides, but with availability restrictions designed to eliminate misfueling as a practical problem.

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, 1983c), but this accounts for less than 1% of misfueling.

It is illegal for service stations or commercial fleet owners to misfuel or to allow misfueling of vehicles originally equipped with catalytic converters. However, federal law does not apply to individuals who misfuel their own vehicles. This limitation hurts EPA's ability to curb this harmful practice.

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 IV-1 shows the emissions increases. The excess HC and NO_x emissions also increase ozone concentrations.

TABLE IV-1

Increase in Emissions Due to Misfueling (grams/mile)

Light-Duty Vehicle Model Years	HC	CO	NO_x
1975 - 80	2.67	17.85	-
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.

According to a 1982 survey by EPA (U.S. EPA, 1983c) the current misfueling rate of light-duty vehicles designed to use unleaded gasoline is about 12%.* We assumed for our analysis that

*The unweighted average the survey found was 10.5%. Weighting the results by the portions of the light-duty fleet in areas with and without Inspection and Maintenance (I/M) programs, by the fractions of light-duty vehicles of each age (in 1982), and by the number of vehicle miles traveled by each model year, the weighted average is 12.2%. (About 17%-18% of the light-duty fleet was in I/M areas.) We believe this estimate may be an underestimate of actual misfueling rates for several reasons discussed later in this and other chapters.

this rate would stay constant to 1988. If this rate rises over time, as preliminary data from the 1983 survey imply, our emission estimates may be too low.

Misfueling rates apparently vary by the age of vehicle, by whether the vehicles are in localities that have Inspection and Maintenance (I/M) mobile source enforcement programs, by whether they are part of a commercial fleet, and other factors. Table IV-2 provides 1982 misfueling rates by model year of vehicle and by I/M status.

TABLE IV-2

1982 Misfueling Rates by Age of Vehicle and by I/M Status

Model Year	Overall Misfueling Rates	I/M Areas	Non-I/M Areas
1982	5.2%	4.4%	6.3%
1981	7.4%	4.3%	9.6%
1980	8.1%	5.7%	10.1%
1979	12.1%	4.9%	20.3%
1978	12.2%	5.9%	19.5%
1977	12.4%	9.9%	16.5%
1976	14.5%	9.6%	20.2%
1975	17.7%	6.3%	30.9%
Weighted Average:*	13.5%	6.2%	15.1%

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

The EPA surveys probably underestimated real misfueling rates by a significant margin. One of the main reasons for this was that vehicle inspections for misfueling were voluntary, which would bias the results downward. In some areas, the rates of drivers refusing inspections were very high. The refusal rates ranged from less than 1% to 8% in I/M areas, and from 3% to 44% in non-I/M areas.

Imperfect indicators of misfueling also provided a possible downward bias in these rates. EPA used three tests to check for misfueling: whether the fuel inlet restrictor was removed or damaged, whether the gasoline in the tank had more than 0.05 grams of lead per gallon, and whether traces of lead could be found inside the tailpipe (the plumbtesmo test). Each of these three tests is likely to miss a substantial portion of misfuelers, and the plumbtesmo test had a high rate of false negative findings when administered hastily in field tests. By using the three indicators together, EPA tried to minimize the likelihood of missing catalysts damaged by misfueling. The results suggested that excess emissions from misfueling in 1983 were significant. As explained in detail later in this chapter, misfueling accounted for roughly 2.48% of all HC emissions, 5.18% of all CO emissions, and 0.78% of all NO_x emissions, nationally.

In economic analysis, because 1988 dollars are not equal to 1983 dollars, future costs are discounted to arrive at a "present value." To make the benefits of avoiding excess emissions comparable to the estimate of costs that we presented in Chapter II, we discounted our emission figures by 3% (the standard rate used for long-term analysis). Table IV-3 shows the 1988 estimates of the discounted stream of future emissions avoided by implementing either of the policy options this paper is examining. The emissions estimates are from all cohorts of vehicles that would misfuel in 1988 in the absence of any change in policies or practices.

TABLE IV-3

Discounted Future Emissions Avoided
by Eliminating Misfueling in 1988
(thousands of metric tons)

HC	CO	NO_x	TOTAL
314	2,202	130	2,646

We estimated the tons of excess emissions that would be avoided in 1988 if EPA were to eliminate misfueling for all light-duty vehicles in 1988 (under either the low-lead or all unleaded option). We did not consider emissions that would occur in 1988 from misfuelings in previous years. Since the costs of eliminating misfueling are calculated for one year (1988), the benefits should include only the avoided emissions attributable to eliminating misfueling in that year, and none from other years. The technical appendix to this chapter provides a description of the fleet model and the discounting procedure for avoided emissions beyond 1988, and a discussion of uncertainties that may have biased our results.

There is no consensus on a good, simple way to value the benefits of eliminating habitual misfueling and its consequent excess emissions. As a result we have used three different approaches:

A. the value by the costs of alternative regulations;

B. the value of preserving catalytic converters; and

C. the value of avoiding damage to health, vegetation, and materials.

Table IV-8, which appears at the end of this chapter, summarizes the values we derived by each of these three methods. In the next three sections, we present our calculations by each method in more detail.

IV.A. Value by the Costs of "Next-Step" Regulations

Our first method computes the value of avoiding misfueling emissions by using the cost per ton of other HC, CO, and NO_x regulations that the Agency is considering promulgating (Table IV-4). The rationale for this method is that these "next-step" regulations reveal the low end of the range of values that EPA or Congress imputes for controlling further increments of these pollutants. (This does not imply that EPA would not promulgate such regulations if we were to adopt either the low-lead or all unleaded option.)

To value these emissions, we chose future regulatory options from among the least costly alternatives that could potentially control a similar amount of each pollutant. However, EPA has imposed much more costly regulations for these pollutants in the past. In many of the more expensive cases, the cost per ton of pollutants abated was not a good measure of what the Agency or Congress was valuing with that particular regulation. For instance, Congress frequently required EPA to choose the best technology available -- sometimes without regard to costs. In other cases, controlling certain sources was considered more valuable than abating an apparently similar quantity from other sources. This might have been because potential exposures to some sources were greater, or because the particular pollutants may have different toxicities. Thus, "cost per ton" may be a very crude measure of cost-effectiveness or the social value of controlling pollution.

Table IV-4 shows our estimates of the present value of emissions avoided by eliminating misfueling using the costs per metric ton of alternative regulations. The total benefit from avoiding these pollutants would be $121-452 million in 1988.

TABLE IV-4

Benefits Valued by "Next-Step" EPA Regulations
(1983 dollars)

	HC	CO	NO_x	Total
Total Value of Emissions	$81-217M	$27-140M	$13-95M	$121-452M
$/metric ton	$232-618*	$11-57**	$92-660†	N/A

* Cost of $232-$618/ton of HC removed by Stage II vapor recovery from gasoline marketing. Estimates were from Pacific Environmental Services, Inc., Update of the Gasoline Marketing Emissions Data Base, September 1983 (4th Quarter 1982 dollars). The "next-step" at petroleum refineries - another major source of HC - would be secondary seals on gasoline storage tanks, at $882 per ton of HC. This is from SCI, Impacts of Revising EPA Regulations Relating to Petroleum Refining and Petrochemical Production, June 1983. Using this cost per ton would value avoiding excess HC emissions from misfueling at $217 million in 1988 (1983 dollars).

** Cost of $11-$57/ton of CO removed by engine modification, catalysts, and inspection and maintenance on heavy trucks. Estimates were from Chapter 3, Regulatory Impact Analysis of the HC and CO Standards for Heavy Duty Trucks, U.S. EPA, forthcoming.

† Cost of $92-660/ton of NO_x removed by low excess air and staged combustion at utility and industrial coal boilers. Estimates for utility boilers were $84-251/short ton, from ICF, Inc., Analysis of a 10 Million Ton Reduction in Emissions from Existing Utility Powerplants, June 1982; industrial boiler estimates were $320-$600/short ton, calculated using emission factors from AP-42 and from the draft U.S. EPA, Background Information Document for the Industrial Boiler NSPS (1979), while costs came from Costs of Sulfur Dioxide, Particulate Matter, and Nitrogen Oxide Controls on Fossil Fuel Fired Industrial Boilers, EPA-450/3-82-021, August 1982.

IV.B. The Value of Preserving Pollution Control Equipment

Our second method of valuation used the cost per ton of emissions control by catalytic converters and other equipment disabled by misfueling. To estimate the benefits of eliminating misfueling, this method used EPA's implicit balancing of costs and benefits in selecting catalytic converters as the method for emissions control on mobile sources. We assumed that this cost per ton reflected the value that EPA and society placed on reducing these pollutants. In addition, this method of valuation most nearly approximated the loss of catalytic converters poisoned by misfueling. Each year, consumers purchase roughly 9.7 million catalytic converters on their new light-duty vehicles; over 12% of these are subsequently disabled by misfueling with leaded gasoline.

We estimated a cost of $283 per car for emission control equipment (U.S. EPA, 1981).* (About half of the cost of oxygen sensors and other equipment was allocated to fuel efficiency, not pollution control.) We counted the emissions controlled by that equipment over an average ten-year car life, beginning with 90% control efficiency in the first year, and leveling off by the fifth year to 50% efficiency. (EPA regulations require that manufacturers provide warranties on catalytic converters for only

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* The costs are "retail price equivalents," which are 30% to 50% of the manufacturers' suggested retail price of the components 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.

five years, but EPA data indicated that this equipment can be effective for the life of the vehicle (Faucett, 1983).) We may have understated the rate at which catalytic converter efficiencies deteriorate at low mileage. If so, our estimate would overstate the tons controlled and underestimate the value of avoiding emissions by eliminating misfueling.

We projected the tons of HC, CO, and NO_X controlled in each year of a ten-year catalytic converter life. We then discounted the future emissions at a 3% rate (as when calculating excess emissions and costs) to the first year, when the equipment costs would be incurred. This produced a cost per ton of $163 for avoiding HC, CO, and NO_X emissions.* We multiplied this by the 2.65 million tons of excess emissions avoided (from Table IV-3). This gave us a benefit estimate of $432 million for eliminating misfueling under either the low-lead or all unleaded policy options.

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* Our calculations used 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. This totaled 4.81 g/mi for all pollutants in each future year.

We divided 4.81 g/mi by (1 - catalytic converter efficiency in that year), multiplied by 10,000 mi/yr, and divided by 1000 b/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 1.733 tons controlled by each car's catalytic converter over a ten-year life.

A cost of control equipment of $283, divided by 1.733 tons controlled, equalled a cost per ton of $163 in 1983 dollars.

IV.C. Benefits Estimated Directly from Health and Welfare Improvements

Research has suggested that HC, NO_x, and CO emissions may directly affect human health and welfare. Our third method of valuing the adverse effects of misfueling provided direct estimates of the health, vegetation, material, and ecosystem benefits that would result from reducing these emissions. In addition, since HC and NO_x are precursors of ozone, we estimated the benefits that a reduction in ozone would have on agricultural yield, materials damage, and health.

Unfortunately, because of scientific uncertainty, lack of data or quantitative estimates, or inability to value certain effects monetarily, we have presented only a partial calculation of the total benefits of reducing misfueling. For example, we did not calculate the effects of ozone on ecosystems or the effects of chronic exposure of ozone on health, nor did we include a quantitative estimate of health benefits from reducing CO emissions. When possible, however, we have described these effects qualitatively.

A few of the studies used in this analysis are EPA contractor reports in progress or in completed draft. As such, they have not undergone full peer review and should be considered preliminary.

IV.C.1. Benefits of Reducing Ozone

To calculate the benefits of reducing ozone, we first had to determine the relationship between reducing HC and NO_x and the subsequent decrease in ozone. Once we determined the reduction

in ozone, we used both dose-response (bottom-up) approaches and proportionate share (top-down) approaches to determine the corresponding amount of economic benefits. In the bottom-up approach, we used disaggregated damage functions to estimate the impact of a given change in ambient levels. In the top-down approach, we interpolated aggregate damage numbers to obtain the impacts of a single pollutant or of a given change in ambient levels. Regardless of the approach, the benefit estimates contain a good deal of uncertainty and should be interpreted with caution.

We needed two general simplifying assumptions to use the top-down technique. First, unless noted otherwise, we assumed a constant elasticity between pollution reduction and the economic effect of concern. Second, for certain estimates, we assumed that the current base level for calculating changes in ambient air quality was roughly equivalent to levels existing in the mid-1970s. Given the overall uncertainty in the available information on benefits, changing the base year is within the "noise level" of the estimates.

The effects of ozone on human health, vegetation, materials, and ecosystems were summarized in the EPA Criteria Document for ozone (U.S. EPA, 1978), currently being revised. In addition, considerable new research has become available since the Criteria Document was published.

Ozone changes are influenced by the amount of solar radiation and changes in the concentrations of NO_x and HC. They are, therefore, very dependent on local conditions. To estimate the national change in ambient ozone, we assumed average U.S.

atmospheric and meteorologic conditions. This averaging will introduce additional uncertainty into the estimate because localized conditions are not fully represented. Using the estimates of avoided emissions in Table IV-3 and projections of total emissions in 1988, we calculated the reductions in the HC and NO_x emissions under either of the two policy options under consideration.* The reductions in HC and NO_x in 1988 would be approximately 2.48% and 0.8%, respectively.

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* Baseline projections for NO_x, HC, and CO for 1988 were calculated as follows:

For NO_x we used an EPA emission factor generated in the draft model of MOBILE III for on-road vehicles in 1988 of approximately 3.19 g/mi. We assumed 159.6 million on-road vehicles traveling an average 11,436 miles (see Appendix). Multiplying:

$$\frac{3.19 \text{ g/mi} \times 11{,}436 \text{ mi/vehicle} \times 159.6 \times 10^6 \text{ vehicles}}{1 \times 10^6 \text{ g/metric ton}}$$

$$= 5.82 \times 10^6 \text{ metric tons}$$

Assuming motor vehicles account for 35% of the NO_x emissions from transportation, stationary source fuel combustion, and industrial processes, total NO_x emissions in 1988 will be 16.634×10^6 tons (U.S. EPA, 1982b). Therefore, the 130,000 avoided tons of NO_x in 1988 (Table IV-3) is approximately a 0.78% reduction.

The emission factors for HC and CO generated by EPA's MOBILE III were reduced by .75 to adjust for that model's exclusion of localities with Inspection and Maintenance programs and the state of California which has its own, more stringent, emission controls.

For HC, a 2.5 g/mi emission factor was used, along with the assumption that motor vehicles account for 36% of the HC emission from transportation, stationary source fuel combustion, and industrial processes. Therefore, the HC reduction is $314{,}000/12.67 \times 10^6$, or roughly 2.48%.

For CO, a 20 g/mi emission factor was used (generating 36.5×10^6 tons of CO), with the assumption that motor vehicles emit 86% of all CO from transportation and residential fuel combustion. The reduction of 2,202,000 metric tons is 5.18% of the total ($5.18 = 2.2 \times .86/36.5$) in 1988.

Converting these changes in HC and NO_x to subsequent changes in ozone involves considerable uncertainty. Disagreement exists as to the ultimate magnitude of the effect. For example, research by General Motors suggested that because of scavenging effects of NO_x on ozone, decreases in NO_x (holding HC constant) may actually increase ozone levels in the area near the NO_x source; further downwind from the source, ozone levels would decrease (Glasson, 1981). To the extent that ozone is scavenged, however, nitrogen dioxide levels would increase and potentially contribute to health effects and materials damage. Other General Motors research suggested that decreases in both NO_x and HC will reduce subsequent ozone, but by less than that resulting from a reduction in HC alone (Chock et al., 1981). Because of the uncertainty in predicting changes in ozone, we considered three separate estimates to determine a reasonable point estimate for the change.

First, a preliminary report recently completed for EPA by ETA Engineering (1983) employed a method for relating HC emissions to ozone production using the Empirical Kinetic Modeling Approach (EKMA) recommended by EPA. ETA Engineering also evaluated the actual changes in HC and ozone in the Chicago metropolitan area. The analysis suggested a one-to-one relationship as an upper bound between the percent change in HC and the resulting percent change in ozone. Using this method, the decrease in ozone concentration would be 2.48%. Unfortunately, the ETA model did not explicitly incorporate the impact of changes in NO_x.

A second estimate of the change in ozone was provided by the work of Kinosian (1982). Using EKMA curves derived from the Los Angeles basin as data, he regressed ozone levels on HC and NO_x concentrations. He found the following functional form fit the data well for a wide range of HC/NO_x ratios:

$$Z = a + b\,(H \times N)^{0.5}$$

where: a and b are empirical constants that vary across locations

$$(\ .04 \leq a \leq .06;\quad .6 \leq b \leq .8\ )$$

and where:

Z = Ambient ozone levels

H = Ambient hydrocarbons

N = Ambient oxides of nitrogen.

To approximate the percent change in ozone due to percent changes in HC and NO_x, we set a = 0 and b = b_o. Taking the logarithm of this equation and the total derivative, we obtained:

$$d\log Z = .5\,(d\log H + d\log N)$$

Since the derivative of the log function is a percent change, the equation yielded:

$$\%\text{ change in } Z = .5\,(\%\text{ change in } H + \%\text{ change in } N)$$

$$= .5\,(2.48 + .78)$$
$$= 1.63$$

However because "a" is actually greater than zero, the change in Z according to this model would be less than 1.63%. With a nonzero "a", we obtained an approximation for the percent change in Z using a power series expansion. Specifically:

$$\log Z = \log(a + b\,(H \times N)^{.5}) \doteq \log T + \frac{2a}{2T + a}$$

where $T = b\ (H \times N)^{.5}$

Taking the derivative and collecting terms:

$$\frac{dZ}{Z} = \%\ \text{change in ozone} = 1.625[1-(2ab(HN)^{.5})/(2T + a)^2]$$

To determine the change in ozone, we assumed a = .05, b = .7, an HC to NO_x ratio of 10, and an average daily maximum ambient ozone level of .054 ppm. These point estimates were averaged from currently available data (Council on Environmental Quality, 1980). We were then able to solve directly for H and N using Kinosian's equation relating ozone levels to HC and NO_x. Substituting these values into the above equation, we obtained:

$$\%\ \text{change in ozone} = (1.625)(1 - .118) = 1.43$$

Therefore, this technique generates a point estimate of 1.43% for the actual change in ozone.

A third estimate of the change in ozone was determined using recent EPA data from 1982 ozone State Implementation Plans (SIPs) (U.S. EPA, 1984b). These SIPs estimated the percent of HC control that was required to obtain a given reduction in ozone. The data indicated that, as a best estimate, a 1.5% change in HC would change ozone, on average, by 1%. Extrapolating linearly, this suggested that a 2.48% reduction in HC would generate a 1.63% reduction in ozone.

These three techniques yielded potential changes in ozone of 2.48%, 1.43%, and 1.63%. Since the last two methods explicitly incorporate the impact of changes in NO_x on ambient levels, we have given them greater weight and used a point estimate of 1.5% as the predicted change in ozone.

Because of transport, oxidant pollution is a regional, rather than local, problem. Oxidant transport can occur over a range of several hundred miles or more. Given its regional nature and the nationwide distribution of the sources of ozone, we assumed a 1.5% reduction in ozone concentration for the nation. Since the benefits from ozone reduction will occur in both urban and rural areas, despite site-to-site variation,* this 1.5% change for a national estimate appeared reasonable.

We have estimated four benefits of reducing ozone levels: health, agriculture, non-agricultural vegetation, and materials. This is followed by estimates of the direct benefits of reduced HC, NO_X, and CO.

IV.C.1.a. Ozone Health Benefits

Studies of the effects of ozone on human health have investigated the relationships between changes in ozone concentrations 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).

Regarding the "sub-clinical" effects, for example, Hammer et al. (1974) found an association of increased oxidants with symptom rates of eye discomfort, cough, headache, and chest discomfort in young, healthy adults. He obtained the symptom

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*The ultimate change in ozone levels for rural areas is least certain.

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. Even low levels of exposure to photochemical oxidants were shown to provoke these respiratory symptoms for adults with predisposing factors, such as smoking or respiratory illness (Zagraniski et al., 1979). Evidence of decreased athletic performance and dysfunction of pulmonary systems was provided by Lippmann et al. (1983) and Lebowitz et al. (1974).

Unfortunately, it was not possible to estimate economic benefits from these studies of "sub-clinical" effects. Most of them focused on determining either a threshold level for health effects or whether there was a particular effect relating to ozone exposure. Thus, no exposure-response relationship was available from this literature.

Recent work by Portney and Mullahy (1983) at Resources for the Future (RFF)* and data reanalysis by Hasselblad and Svendsgaard (1975) were exceptions. The former study considered the effect of alternative levels of ozone on, among other measures, the number of minor restricted activity days (MRADs) over a two-week survey period. This health measure indicated how frequently a person curtailed normal activity without actually missing work or being bedridden. The second study was a statistical reanalysis of the Hammer et al. study cited above. It

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*The RFF study will undergo formal EPA peer review in April, 1984.

used logistic estimation to relate sub-clinical health effects to alternative levels of ozone. Our results from applying each of these studies follow.

The RFF analysis consisted of regressing MRADs on a number of independent variables, including socioeconomic and demographic factors, chronic health status, urban variables, ozone, and other pollutants. Because of the inherent uncertainty in the analysis, we used the 1.5-2.5 range for the estimated regression coefficient of ozone (measured as the average daily maximum 1-hour concentration during the two-week survey period) indicated in the RFF study. This resulted in an elasticity of 0.17-0.29 MRADs to ozone. Therefore, a 1.5% reduction in annual average ozone levels would reduce MRADs by 0.255% to 0.435%.

To calculate the health benefit from the reduction in ozone, we applied these elasticities to the entire U.S. population in 1988, projected to be 245 million. We used summary statistics from the RFF report that indicated an annual average of 10.32 MRADs per person. Using the low estimate of elasticity, the improvement in ozone would result in 6.4 million ($245 \times 10^6 \times 10.32 \times .00255$) fewer MRADs per year for the U.S. population. The higher elasticity generated an estimate of 11.0 million MRADs, assuming some risk at all levels of exposure.

To generate "low-low" and "high-high" estimates we placed, somewhat arbitrarily, two alternative values on an MRAD. As a lower bound, we assigned a value of $7 per episode, approximately 10% of the average daily wage, to indicate some minimum amount

a person would pay to avoid the minor restriction in activity. We then applied this value to the lower estimate of the total MRADs to yield an estimate of $45.1 million.

For the "high-high" estimate, we used $20 as the value of an MRAD which, applied to the 11.0 million MRADs, yielded an estimate of $220.0 million. This still may be a conservative estimate for several reasons. To obtain the health benefits of reducing air pollution, Freeman (1982) used a value of $20 (1978 dollars) for a restricted activity day (non-minor) and also added expected reductions in medical expenses. In addition, MRADs may affect work productivity. Our "low-low" and "high-high" estimates produced a range of $45 to $220 million for 1988.

An alternative estimate of the health benefits from reduced ozone concentrations was derived from the preliminary results of Gerking et al. (1983), which demonstrated that survey respondents were willing to pay to avoid suffering an increase in ozone concentrations. Specifically, the study estimated that a 10% reduction in ambient ozone concentrations would generate a per person "willingness to pay" of $1.55 to $1.92 per year. Assuming linearity, a 1.5% reduction would result in a benefit of $0.23 to $0.29 per person, or $57 to $71 million nationally. Unfortunately, potentially serious problems with data and methodology render this study only suggestive of the benefits of reducing ozone. Nonetheless, it lends credence to the monetary estimates suggested by applying the RFF model.

To check the plausibility of these results, separate estimates of symptoms were obtained using the Hasselblad and Svendsgaard (1975) results. The authors fit logistic curves to estimate the relationship between ozone concentration (measured as a daily maximum hourly concentration) and eye irritation, headache, cough, and chest discomfort. The probability of a response at an ozone level, X, measured in parts per hundred million (pphm), was given as:

$$p(X) = C + (1 - C)/[1 + \exp(-A - BX)]$$

The following parameter values (A, B, and C) were determined:

For eye irritation	:	A = -4.96, B = .0907, C = .0407
headache	:	A = -4.88, B = .0470, C = .0976
cough	:	A = -2.98, B = .0092, C = .0450
chest discomfort	:	A = -3.53, B = .0023, C = -.0166

To calculate the change in this probability due to a change in ozone, we differentiated the above with respect to X. The change in the probability (dp) of a given symptom due to a change in ozone (dx) was:

$$dp(X) = B(1 - C)[\exp(- A - BX)]\, dX/ [1 + \exp(- A - BX)]^2$$

The change in probability can be estimated given information on the existing daily maximum ozone levels. To use pollution measures commensurate with the original estimation, we obtained EPA's data from the entire Storage and Retrieval of Aerometric Data (SAROAD) network of ozone monitors. Because of the non-linearity in the equation representing the probability of a

health effect, the mean daily one-hour maximum of ozone was calculated for two separate six-month periods. One period included data from the second and third quarters, April through September (when higher ozone levels generally occur), while the other period used the first and fourth quarters of the year. For the two periods we obtained average ozone measures of 6.1 and 3.3 pphm, respectively, yielding an annual average of 4.7 pphm.

To reduce the chances of obtaining more than one symptom per person and thus double-counting people affected, we used chest discomfort to represent the symptoms reported as cough as well as those reported as chest discomfort. We calculated separately the number of persons with reduced eye irritation and headaches.

Substituting the appropriate values for A, B, C, X, and dX into the above equation for each six-month period, we obtained the number of reduced symptoms. For example, to calculate the expected number of cases of chest discomfort in the second and third quarters, we used the following values: A = -3.53, B = .0023, C = -.0166, X = 6.1, dX = (.015)(6.1) = .0915. Substituting, to obtain the probability of a chest discomfort symptom per person per day, yielded:

$$dp(X) = (.0915)(.0023)(1.0166)H/(1 + H)^2$$

$$\text{where } H = \exp(3.53 - (.0023)(6.1))$$

$$= 6.00 \times 10^{-6} \text{ per person per day}$$

Multiplying by 245 million people and 183 days, we obtained a six month total projection of 269,000 cases of chest discomfort in

1988. Using this equation, the total change in the number of symptom days expected in 1988 was:

Chest discomfort:	400,000
Eye irritation :	5,783,000
Headaches :	2,493,000
Total:	8,676,000

Although we were not able to determine the exact correspondence between MRADs and these reported symptoms, the projection for these symptoms fell within the 6.4 to 11.0 million range estimated for MRADs and supported the estimate.

Another important health effect of ozone, reported by Whittemore and Korn (1980) and Linn et al. (1981), was the exacerbation of asthma and nonspecific obstructive respiratory disease. To estimate the decrement in asthmatic attacks resulting from the reduction in misfueling, we used the analysis of Whittemore and Korn. They used a logistic curve to estimate the probability that an asthmatic would have one or more attacks on a given day. This probability was hypothesized to depend on air pollution levels, temperature and humidity, the day of the week, and the presence of an attack on the preceding day.

The results suggested that the probability of an attack was significantly related to exposure to ozone. Specifically, their results suggested the following:

$$\log (p/1-p) = 1.66\ z + b_i X_i$$

where p = the probability of an attack

z = ambient ozone (24-hour average concentration)

X_i = meteorologic and other control variables.

To determine the change in the probability of an attack (dp) due to a change in ozone (dz), we partially differentiated both sides of the above (i.e., dX = 0) and solved for dp:

$$dp = 1.66\ (p)\ (1-p)\ dz$$

To estimate the actual change in probability, we had to determine the ambient ozone level and the baseline probability of an attack, represented as p. Because of the inherent uncertainty in these numbers, we used the point estimate to determine economic benefits and then conducted a sensitivity analysis using alternative values for p and dz. An ambient ozone level was approximated using available data for 1979 through 1982 (U.S. EPA, 1982a; Evans et al., 1983). For this analysis, we used a point estimate of 0.040 ppm, but considered 0.035 ppm in the sensitivity analysis.

Data on the baseline probability of an asthmatic attack were difficult to obtain. These attacks vary widely in frequency, duration, and intensity.* For example, asthmatics with a condition characterized as "mild and intermittent" (roughly 60% of all asthmatics) may have two or three attacks a year. However, they may be ill-prepared to respond to severe attacks and may undertake significant medical expenses. "Moderate" asthmatics (25% of all asthmatics) may have one attack a month, requiring

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* Estimates of the frequency and severity of asthmatic attacks were based on personal communications with Jeff Cohen, U.S. EPA, Office of Air Quality Planning and Standards, based on his survey of experts.

some medical expense, lost work, or restrictions in activity. There may be some chronic respiratory impairment. "Severe" asthmatics (up to 15% of all asthmatics) may have several attacks a month. Evidence from daily diaries in Salt Lake City and New York City suggested 30-40 attacks per year. This group may be better prepared for the attack, but may be on continuous medication and/or be forced to undertake significant preventive actions.

To estimate the baseline probability of an attack, we used a weighted average of the expected number of attacks for each group. Multiplying the proportion of asthmatics in each classification by their average number of attacks per year, we obtained:

$$(.6)(3) + (.25)(12) + (.15)(36) = 10.2 \text{ attacks per year}$$

This generated a probability of 2.8% per day (10.2/365=2.8%). Other research suggested a daily probability of an attack ranging from 1.4-2.5% per day with a point estimate of 1.8%.* Therefore, as a point estimate we used 2.0% for the daily probability of an attack, indicating an average of 7.3 attacks per year. (This estimate obviously does not reflect the extreme variability among asthmatics.)

The change in the probability of an attack was calculated using a point estimate of .04 ppm for ozone exposure, a 1.5% change in ozone, and a value of .02 for the baseline probability

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* These estimates were also based on personal communications with Jeff Cohen, U.S. EPA, Office of Air Quality Planning and Standards.

of an attack. Substituting:

$$dp = (1.66)(.02)(.98)(.0006) = 1.95 \times 10^{-5}/\text{person/day}$$

To estimate the population at risk, we used estimates of the numbers of asthmatics and atopics (persons potentially sensitive to ozone) in the entire U.S. population (245 million). Currently, 4% of the population is considered asthmatic, with an additional 9% considered atopic. Thus, the population at risk is 13% of 245 million, or 31.85 million. The total reduction in the annual number of attacks would be:

$$227{,}000 = 1.95 \times 10^{-5} \times 31.85 \times 10^{6} \times 365$$

To determine the monetary benefit of these reduced attacks we had to assign a value per attack avoided. Ideally, this would equal the individual's (or society's) willingness to pay to prevent the occurrence of an attack. Unfortunately, no data exists on this willingness to pay. Likewise, we could not find any published estimates of the average medical costs incurred for an attack.

Based upon the existing evidence of the potential severity of an attack, we arbitrarily valued each attack at $70, the average daily wage, as a "ballpark" estimate. Because of their chronic condition, 7% of all asthmatics are consistently forced to limit their activities outside of their "major activity" such as working, keeping house, or going to school (U.S. DHEW, 1973a). Another 7% are forced to limit their amount or kind of major activity. Finally, an additional 1.5% are unable to pursue any major activity.

Data also existed on the frequency and degree of annoyance from an asthmatic condition (U.S. DHEW, 1973b). Of the asthmatics sampled, 52% reported that they were bothered by asthma "once in a while," 21% were bothered "often," and 14% were bothered "all the time." Regarding the degree of bother, 11% reported "very little," but 36% reported "some" and 43% reported a "great deal" of bother. Evidence on visits to physicians indicated that 40% of the asthmatics saw a doctor two or more times a year, while 20% saw a doctor five or more times a year. Finally, 51% of the asthmatics were taking medicine or following treatment recommended by a doctor. Thus, given the degree of bother and medical care involved, we used $70 per attack as a point estimate. However, it may well be that many asthmatics would be willing to pay more than $490 ($70/attack x 7 attacks per year) to prevent any asthma attacks from occurring in the year.

Multiplying the 227,000 expected attacks by $70, we estimated benefits of $15.9 million in 1988. Table IV-5 displays the sensitivity of this estimate to alternative values for the baseline probability, the ozone level, and the value of an attack. A reasonable range for the benefits of reduced asthma attacks is $10.5 to $28.2 million, with a point estimate of $15.9 million.

Table IV-5

Benefits of Reducing Asthmatic Attacks in 1988

	Ozone Level	Baseline Attack Rate Probability	Value of Attack	Benefits
High Estimate	.04 ppm	2.5%	$100	$28.2 million
Point Estimate	.04 ppm	2%	$ 70	$15.9 million
Low Estimate	.035 ppm	1.5%	$ 70	$10.5 million

To avoid the double counting of asthma attacks with the MRADs calculated above, we subtracted the estimated 227,000 attacks from the number of MRADs that were estimated to occur. We then added the point estimate of $15.9 million for reduced asthma attacks to the adjusted "low-low" and "high-high" estimates of MRADs to obtain the total acute health benefits. Consequently, these studies suggested benefits from the reduction in acute health effects in 1988, including both MRADs and asthma attacks, ranging from $59.3 million to $233.4 million, with a point estimate of $146 million.

These ozone health benefits reflect the likely acute effects generated by intense, short-term exposure to ozone. Long-term exposure to ozone may also 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 function, and lower pulmonary function.*

In addition to the sparse epidemiological evidence of the effects of long-term exposure to ozone, 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, 1983a). While work is now under way to extrapolate these animal data to human dose-response functions, this is not presently possible. Therefore, we could not quantify the chronic health effects attributable to ozone, but we believe that some of these effects may be significant at current ambient levels.

Using the studies cited above, the total health benefits in 1988 from the reduction in ozone due to reduced misfueling was $146 million from reduced MRADs and asthmatic attacks, plus potential reductions in chronic health conditions from decreased ozone levels. If we symbolize these non-monetized health benefits as OZ_C, the total health benefits are $146 million + OZ_C.

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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 NO_2 and SO_4 and there was some question about the adequacy of the measurement of ozone exposure and about the subject selection and the test measures. Although it is both reasonable and likely that long-term exposures are harmful to health, the failure to correct for the effects of other pollutants raises uncertainties about the specific findings.

IV.C.1.b. Ozone Agricultural Benefits

Research has shown that ozone alone, or in combination with sulfur dioxide and nitrogen dioxide, is responsible for most of the U.S. crop damage associated with air pollution. Ozone can affect the foliage of plants by biochemical and cellular alteration, thus inhibiting photosynthesis and reducing plant growth, yield, and quality.

To generate a top-down estimate of agricultural benefits, we used generalized relationships between ozone concentration, yield, and economic loss. The aggregate estimates of Adams (1983) and SRI (1981), as summarized by Freeman (1982), suggested that the average annual benefits associated with a 10% reduction in ozone concentrations were $200 to $500 million in 1983 dollars. To use the top-down approach, we assumed that this relationship held over a broad range of exposures. Thus, the 1.5% ozone reduction could produce a benefit of $30 to $75 million from increased crop yields (1.5%/10% times $200 to $500 million = $30 to $75 million).

As an alternate approach, we followed the bottom-up approach of Kopp (1983). He estimated the effects of ozone changes on soybeans, wheat, corn, peanuts, and cotton on a county-by-county basis. Because this analysis directly incorporated estimates of the demand and supply elasticities for these crops, it appeared to be the most precise assessment of benefits available. His estimates suggested that a 1.5% change in ozone would produce approximately $120 million in lost economic value (1983 dollars). These five crops accounted for roughly 76% of the total value of

commercial crop production in the United States. Therefore, we scaled Kopp's estimate of crop damage by assuming that ozone damages to all other crops occur in the same proportion as their relative values. The benefits of the 1.5% change in ozone grew to approximately $157.5 million (1983 dollars).

Another benefit estimate for reduced ozone, conducted on a crop and region-specific basis, was provided directly from the National Crop Loss Assessment Network (Heck et al., 1983). Their estimate of the effects on economic surplus (consumer and producer well-being) included only crops in the corn belt -- corn, soybeans, and wheat -- which are less than half of all expected crop losses from ozone. Their results suggested that a 3% reduction in ozone would increase economic surplus by $140 to $230 million. Assuming linearity, a 1.5% change would generate a surplus of $70 to $115 million. If these crops in the corn belt constitute 50% of all ozone-related damages (probably a high estimate), the total benefits of the 1.5% reduction would be $140 to $230 million.

Together, the benefit estimates from these studies ranged from $30 to $230 million per year. To determine a point estimate of the damage to agricultural crops, we weighted the last two analyses most heavily, because they contained the most precise estimates of changes in economic welfare. This suggested a point estimate for agricultural loss from ozone concentrations of approximately $160 million.

IV.C.1.c. Nonagricultural Vegetation Benefits of Reduced Ozone

The estimates presented above addressed only agricultural crops. They excluded damages to forests and ornamental plants, which may be substantial. For example, in a very small contingent valuation study, Crocker and Vaux (1983) found that the shift of an acre of timberland in the San Bernardino National Forest from either the severely or moderately harmed category into the unharmed category would generate additional annual recreational benefits of $21 to $68 per person. These findings are difficult to generalize for the rest of the nation because the San Bernardino area has very high ozone concentrations and because of other site attributes and socioeconomic characteristics. Nevertheless, they indicate that reduced damages to vegetation may produce significant benefits.

The preliminary draft of the Ozone Criteria Document (1983a) also provided additional qualitative evidence: "The influence of O_3 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 (1-hour maximum) ranging from 0.05 ppm (111 ug/m^3) to 0.40 ppm (785 ug/m^3)." For commercial timber purposes, however, damages are likely to be small, as most

commercial forests are in areas with low ozone concentrations. In areas with relatively high concentrations, trees resistant to ozone can be planted.

Finally, we present one quantitative estimate, noting that it was based on very sparse data and generated by making some significant abstractions from existing studies. Leighton et al. (1983) have estimated that the benefits associated with non-agricultural vegetation from a 10% reduction in ozone concentrations are $0.0 to $100 million. Assuming linearity, the benefits from a 1.5% reduction in ozone would be $0.0 to $15.0 million, with a point estimate of $7.5 million for 1988. We stress, however, that the existing evidence is uncertain.

IV.C.1.d. Ozone Materials Benefits

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 (purchasing of specially resistant material) and aesthetic losses. Only the direct costs were incorporated in this analysis, however.

In his survey of the literature, Freeman (1982) suggested that annual material damages from oxidants and NO_x amounted to approximately $1.1 billion (1978 dollars). Using the Consumer Price Index as well as census figures on projected population increases to update the figure, produced an estimated $1.88

billion for 1983. An ozone reduction of 1.5%, assuming linearity, suggested a benefit of $28.2 million annually.

We obtained an alternative estimate of the benefits from reduced materials damage by using dose-response information from the Ozone Criteria Document (U.S. EPA, 1978). The text supplied per capita economic damages for elastomers, textiles, industrial maintenance, and vinyl paint costs as a function of annual ozone levels. Using an annual 24-hour mean for ozone of .040 ppm, as reported above, and a population estimate of 245 million for 1988, we calculated a benefit range of $16.4 to $22.6 million in 1983 dollars (point estimate of $19.5 million), for a 1.5% reduction in ozone. Taking the arithmetic mean of the point estimate from the two different approaches for materials benefits yielded a point estimate of $24 million annually in 1988.

IV.C.2. Benefits of Reducing NO_x Emissions

NO_x emissions are believed to damage 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 relating to NO_x is sparse. As a consequence, only broad aggregate estimates were presented to approximate the effects of NO_x emissions on health and welfare.

Materials damage from NO_x are not included in this section since it was contained in the ozone benefits section. While there may be acid rain benefits as well, we have not included

them because of the uncertainties over the role of NO_x in acidic deposition. Therefore, we included only the benefits of reduced health effects and improved visibility, as benefits for reducing NO_x emissions in 1988.

Regarding NO_x health effects, EPA is currently reviewing its ambient air standard for nitrogen dioxide (NO_2).* The Clean Air Scientific Advisory Committee (CASAC) recommended that any NO_2 standard should protect against repeated short-term "peak" exposures and against long-term "chronic" exposures because of possible health effects.**

Repeated exposure to short-term peaks of NO_x has been associated with excess respiratory illness and symptoms in children, and with small (but statistically significant) reductions in lung function (U.S. EPA, 1982c). Because repeated episodes of respiratory tract irritation and illness in children may carry into adult life in the form of reduced lung function and chronic bronchitis, NO_x reductions may also reduce subsequent adult cases of chronic bronchitis. Long-term exposure to low level NO_2 may contribute to emphysema. Thus, significant benefits, although unquantified in this paper, may result from controlling NO_x.

Surveying several research efforts, including those linking NO_x to changes in property values (which may capture both health

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* NO_2 is an indicator pollutant for all nitrogen oxides.

** CASAC closure letter on OAQPS Staff Paper for NO_x, July 6, 1982.

and welfare effects), the National Academy of Sciences (1974) suggested a range of $1.0 to $8.0 billion, adjusted to 1983 dollars, for the annual effects other than materials damage. Assuming proportionality between the predicted .78% reduction of NO_x and reduced damages, the benefits would be $7.8 to $62.4 million annually. We used the midpoint of this range, $35 million, as the point estimate.

IV.C.3 Reducing Emissions of Hydrocarbons

The various chemicals constituting hydrocarbons from automobile emissions may affect health. Specifically, benzene, which is believed to cause leukemia, constitutes 4% of total tailpipe HC emissions (U.S. EPA, 1983b).

To estimate the number of benzene-linked leukemia deaths we might avoid by eliminating misfueling, we used the EPA Carcinogen Assessment Group (CAG) Risk Assessment for Benzene. This analysis predicted that human exposure to automobile benzene emissions* resulted in an estimated 50.89 leukemia deaths per year in 1976 (U.S. EPA, 1979). As displayed in Table IV-3, we estimated that misfueling in 1988 would produce 314,000 metric tons of HC emissions, or 4.99% of the 6.29 million tons of automobile HC emissions in 1976, the year of CAG's analysis (U.S. EPA, 1982b). This estimate, however, was based on a unit risk estimate for

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* CAG assesses risks as the amount of exposure (in parts per billion), times the population exposed, times duration of exposure. Their benzene analysis yielded 150 million ppb-person-years.

benzene (.024/ppm) which was revised by CAG in November 1981 (.022/ppm). Using this new unit risk estimate and CAG's analysis, automobile tailpipe-benzene emissions were predicted to result in an estimated 47.34 leukemia cases per year (U.S. EPA, 1974a). Therefore, assuming linearity, 4.99% of the 47.34 leukemia deaths, or 2.36 deaths, would be avoided by preventing misfueling in 1988. This assumed that benzene would be the same fraction of the reduced HC emissions as it was of total automotive HC emissions in 1976.

Economic studies (Brown, 1978; Thaler and Rosen, 1976) suggested that people are willing to pay $0.45 to $7.0 million to save a "statistical" life. Under this assumption, the health benefits of avoiding the HC emissions would be $1.06 to $16.52 million in 1988. We used the geometric mean of this range, $4.19 million, as our point estimate.

Hydrocarbons also 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 Systems Applications Incorporated (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 hydrocarbon and NO_x concentrations, and oxidant and sulfur dioxide levels) for which we have only limited data.

A recent modeling analysis by SAI (Seigneur et al., 1982)

indicated that a 50% reduction in HC would reduce sulfates in urban areas by 30% to 60%. However, because of the uncertainty surrounding this estimate at this time, and the uncertainty in interpolating this to a 2.4% change, we did not explicitly consider the reduction in sulfates in this analysis. Because the reduction in sulfates would generate significant economic benefits from improved health and visibility and reduced soiling, this omission may seriously underestimate the benefits.

IV.C.4. Reducing Emissions of Carbon Monoxide

Existing scientific knowledge concerning CO indicates that health impacts are the primary concern at or near ambient levels. Current information suggests that persons with cardiovascular disease are most sensitive to low levels of CO. Additional subgroups of the population also believed to be sensitive to CO exposure are people with chronic respiratory diseases, pregnant women, and the elderly. Unfortunately, clinical dose-response functions relating low level CO exposure to particular health effects, when estimated, have not been conclusive. Therefore, we have not estimated quantitatively the impact that reduced CO (through reduced misfueling) may have on health. However, we have described the impact that misfueling may have on the distribution of carboxyhemoglobin (COHb) levels for the U.S. population.

Probably the greatest concern about CO exposure is its effect on the cardiovascular system. The effect of CO thus far

measured at the lowest level of exposure is reduced exercise time until the onset of angina pectoris. This clinical phenomenon is a result of insufficient oxygen supply to the heart muscle and is characterized by spasmodic chest pain, usually precipitated by increased activity or stress, and relieved with 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 is not believed to be 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 the risk of myocardial infarction (the death of a portion of the heart muscle). However, epidemiological studies as yet have provided inconclusive 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 carboxyhemoglobin (COHb), and thereby reducing the oxygen-carrying capacity of the blood. The median concentrations of COHb in blood are about 0.7% for nonsmokers and about 4.0% for smokers. At 2.9% COHb, at least one clinical study (Anderson et al., 1973) associated reduced exercise time until the onset of pain in patients with angina pectoris. At 4.5% COHb, this same study reported an increased duration of angina attacks.

The potential health improvements from reduced CO may be great for two reasons. First, there are many people in the

population believed to be sensitive. EPA has estimated that 5.0% of the U.S. adult population -- roughly 9.5 million people -- have definite or suspected coronary heart disease. Of this group, 80% have suspected or definite angina pectoris (U.S. EPA, 1980). Second, the blood of many people shows concentrations of COHb above 2.9%, the lowest level of COHb where adverse effects are indicated. Data from the second National Health and Nutrition Examination Survey (NHANES II) indicated that for the U.S. population over twelve, 2% of those who have never smoked, 3% of former smokers, and 66% of current smokers exceeded 2.9% 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 (3% to 7%) in work time until exhaustion in healthy young men with COHb levels at 2.3% to 4.3% (Horvath et al., 1975; Drinkwater et al., 1974; Raven et al., 1974). At higher COHb levels (5% to 7.6% and above), investigators have reported impaired visual perception, manual dexterity, ability to learn, and performance of complex sensorimotor tasks in healthy subjects.

Finally, 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, that enhance oxygen need, or that elevate the sensitivity of the tissues to any oxygen imbalance. 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 (0.1-.245 million people);
- people drinking alcohol or taking certain medications (e.g., vasoconstrictors);
- the elderly;
- visitors to high altitudes; and
- fetuses and infants* (3.7 million total live births per year).

A comprehensive economic estimate of the benefits from reduced CO is not possible. The current medical literature does not provide a dose-response relationship between COHb levels and specific health effects that can be valued monetarily. However, analysis relating changes in CO emissions to the distribution of COHb levels in the U.S. is possible using NHANES II. Work still in progress indicates that a change in ambient CO levels may have significant impacts on the distribution of COHb.

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* Animal studies showed that pregnant females exposed to CO reported lower birth weights, increased newborn mortality, and lower behavioral levels in newborn animals, even when no effects on the mothers were detected. In addition, research has reported a possible association between elevated CO levels and Sudden Infant Death Syndrome (Hoppenbrouwers et al., 1981).

IV.D. Summary of Health and Welfare Benefits

Table IV-6 summarizes the direct estimates of the 1988 benefits of reducing HC and NO_x by pollutant and benefit subcategory. The range of $114 to $579 million in annual benefits incorporates the estimates of both the top-down and bottom-up approaches. The point estimate of $377 million was derived by aggregating the best estimates of each subcategory.

Table IV-6

1988 Benefits of Reducing HC, NO_x and CO Emissions by Direct Estimation
(millions of 1983 dollars)

Benefit Category	Range	Point Estimate
Ozone		
Acute Health	$59-233	$146
Agriculture	$30-225	$160
Vegetation	$ 0- 15	$7.5
Materials Damage	$16- 28	$ 24
Chronic Health	NA	OZ_C
NO_x		
Health and Visibility	$7.8-62	$35
Hydrocarbons		
Health	$1.06-16.52	$4.19
Sulfate Deposition (Health, Materials Damage, Visibility)	NA	NA
Carbon Monoxide		
Acute Health	NA	CM_A
TOTAL	$ 114-579	$ 377 +OZ_C +CM_A

NA = Quantitative estimates not available or attempted.

CM_A = Non-quantified benefit of reducing acute health effects from CO.

OZ_C = Non-quantified benefit of reducing chronic health effects from ozone.

IV.E. Summary of HC, CO, and NO_x Benefits

As we noted earlier, there is no consensus on a good, simple way to value the benefits of eliminating misfueling and its consequent excess emissions. As a result we have used three different approaches:

- the value using the costs of alternative regulations;
- the value of preserving catalysts; and
- the value of avoiding damage to health, vegetation, and materials.

Table IV-7 summarizes the values obtained by each of these three methods.

The first method computed the value of reduced emissions by using the cost of HC, CO, and NO_x regulations that EPA is considering promulgating. This revealed the low end of the range of values that EPA or Congress impute for controlling additional increments of these pollutants.

The second method of valuation used the cost of catalytic converters and other emission control equipment disabled by misfueling to approximate the benefits of eliminating misfueling.

Finally, the third method directly calculated some of the health and welfare benefits of reducing HC and NO_x emissions, by applying the results of research that related improvements in air quality to improvements in human health, or reductions in damages to materials and vegetation.

Our health and welfare estimates are probably low because they do not include all the potential health and ecological

effects of ozone and CO. This method of valuation is also less certain than the other methods. Conceptually, however, it is a reasonable (and probably the best) way to measure the social benefits of reducing emissions of HC, NO_x, and CO.

We used the mean of this direct estimate and the value of preserving catalytic converters as the best estimate of the benefits of reducing misfueling. We obtained a value of $405 million to represent the 1988 benefits of reducing HC, NO_x, and CO emissions through the elimination of misfueling. Note that the different methods yielded fairly similar estimates of the benefits.

Table IV-7

Summary of Benefits in 1988 of Reducing HC, CO, and NO_x Emissions
(millions of 1983 dollars)

Value by Next-Step Regulations	Value of Perserving Catalytic Converters	Value by Directly Estimating Health and Welfare
$121 - 452	$432	$114 - 579 (point estimate: $377)

TECHNICAL APPENDIX FOR CHAPTER IV

Accurately estimating the costs and benefits of reducing lead in gasoline required the use of disaggregated data, some of which was not readily available. For this reason, we developed a fairly simple "bottom-up" model to forecast light-duty fleet size and mix, numbers of misfuelers, and gasoline demands by various categories of vehicles. This technical appendix describes this model.

Overall Structure of Model and Summary of Estimates

In general terms, the fleet model can be broken into five major pieces:

- It ages the existing stock of cars (1982) and light trucks (1980) -- using data from Polk, 1983 -- and includes Data Resources, Inc. (DRI) projections of sales from 1983 to 1988, to estimate the size and composition of the light-duty fleet in 1988. Appendix Tables IV-1 and IV-2 show the projection of this fleet into 1988.
- Misfueling rates by age of vehicle are used to estimate both the number of misfueled vehicles and those that would misfuel for the first time in 1988 under current policies. The sources of misfueling data are surveys conducted by EPA's Office of Mobile Sources. Our analysis assumed that current misfueling rates would continue.
- The model estimates excess emissions due to new misfuelings in 1988 by aging (retiring) the new misfuelers over the subsequent 20 years (to 2007), calculating the expected

APPENDIX TABLE IV-1

LIGHT-DUTY VEHICLE PROJECTIONS
(thousands of vehicles)

MODEL YR	CARS IN OPERATION 1982 (Polk)	1983	1984	1985	1986	1987	1988
1988							11,800
1987						11,600	11,588
1986					11,200	11,189	11,155
1985				11,000	10,989	10,956	10,829
1984			10,500	10,490	10,458	10,337	10,094
1983		9,200	9,191	9,163	9,057	8,844	8,646
1982	8,000	7,992	7,968	7,876	7,690	7,518	7,337
1981	8,280	8,255	8,159	7,968	7,789	7,602	7,268
1980	8,825	8,723	8,518	8,327	8,127	7,770	7,268
1979	10,075	9,838	9,618	9,386	8,974	8,395	7,653
1978	10,155	9,927	9,689	9,263	8,665	7,900	6,964
1977	9,661	9,429	9,015	8,432	7,688	6,777	5,768
1976	8,471	8,099	7,576	6,907	6,088	5,182	4,300
1975	6,190	5,790	5,279	4,653	3,961	3,287	2,671
1974	7,498	6,836	6,026	5,129	4,256	3,459	2,798
1973	7,629	6,725	5,724	4,750	3,860	3,122	2,530
1972	5,989	5,098	4,230	3,438	2,781	2,253	1,829
1971	4,243	3,521	2,861	2,314	1,875	1,523	1,218
1970	3,581	2,910	2,354	1,907	1,549	1,239	979
1969	2,822	2,282	1,850	1,502	1,201	949	731
1968	2,208	1,789	1,453	1,162	918	707	530
1967	1,609	1,306	1,045	826	636	477	0
1966	5,220	4,176	3,299	2,540	1,905	0	0
	110,456	111,897	114,353	117,033	119,668	121,084	123,957

APPENDIX TABLE IV-2

LIGHT-DUTY TRUCK PROJECTIONS*
(thousands of vehicles)

MODEL YEAR	LDTs IN OPERATION 1980 (Polk*.87)	1981	1982	1983	1984	1985	1986	1987	1988
1988									3,450
1987								3,320	3,303
1986							3,120	3,104	3,079
1985						3,050	3,035	3,010	2,980
1984					2,970	2,955	2,931	2,902	2,863
1983				2,520	2,507	2,487	2,462	2,429	2,384
1982			1,748	1,739	1,725	1,708	1,685	1,654	1,613
1981		1,826	1,817	1,802	1,784	1,760	1,727	1,685	1,632
1980	1,936	1,926	1,911	1,891	1,866	1,831	1,787	1,731	1,661
1979	2,931	2,907	2,878	2,840	2,787	2,719	2,633	2,527	2,507
1978	2,844	2,815	2,778	2,726	2,660	2,576	2,472	2,452	2,204
1977	2,580	2,546	2,498	2,437	2,361	2,266	2,247	2,020	1,875
1976	2,139	2,099	2,048	1,984	1,904	1,888	1,697	1,575	1,444
1975	1,494	1,458	1,412	1,355	1,344	1,208	1,121	1,028	933
1974	1,976	1,914	1,837	1,822	1,638	1,520	1,394	1,265	1,137
1973	1,927	1,849	1,834	1,649	1,530	1,403	1,274	1,145	1,022
1972	1,608	1,595	1,434	1,331	1,220	1,108	995	888	785
1971	1,127	1,013	940	862	783	703	628	555	490
1970	1,022	949	870	790	709	633	560	494	434
1969	1,048	961	872	784	700	618	546	480	419
1968	769	698	627	560	495	437	384	335	293
1967	640	575	513	454	401	352	308	269	0
1966	556	496	439	387	340	297	260	0	0
	24,597	25,628	26,456	27,933	29,724	31,521	33,267	34,870	36,511

* Trucks 0-8500 lbs.

excess emissions of HC, CO, and NO_x in each year (based on both the extra grams of emissions per mile traveled and annual miles per vehicle by age). It then discounts these emissions (at 3% rate) back to the year of misfueling -- 1988.

° The model estimates gasoline demand in 1988 for four major categories of demand: those vehicles designed for and using leaded gasoline, those designed for leaded gasoline but switching to unleaded premium (for the octane), those vehicles designed for and using unleaded gasoline, and those misfueling with leaded gasoline. A fifth category is "special" uses for heavy trucks, agricultural equipment, boats, etc. We hold "special" use demand constant at 9.6% of total gasoline demand, the 1982 percentage.

Table IV-3 is a summary of the results.

APPENDIX TABLE IV-3

SUMMARY OF FLEET MODEL PARAMETERS

Total # of light-duty cars and trucks in 1988:	159,644,000	
Incremental # of vehicles assumed to misfuel in 1988:	2,524,000	
Total # of vehicles in 1988 misfueling in all years:	19,481,000	
Overall misfueling rate:	12.2%	
Average miles per gallon for cars and trucks:	20.4	
Average miles per year per cars and trucks:	11,436	
Total demand for gasoline (million gal/yr):	100,737	100%
Legal light duty demand for leaded (million gal/yr):	12,485	12.4%
Misfuelers' demand for leaded (million gal/yr):	10,290	10.2%
Demand for unleaded (million gal/yr):	68,290	67.8%
Other legal demand for leaded (million gal/yr):	9,671	9.6%

Sources of Data and Major Assumptions

We found it necessary to draw actual data from several different sources to estimate other important pieces of information. In general, we used the following hierarchy of sources; if a preferred source did not provide the data, or did not provide it in enough disaggregation, we turned to the next-preferred source.

HIERARCHY OF SOURCES OF DATA

R.L. Polk & Co. (mostly provided in MVMA Facts & Figures)
U.S. DOT/FHA: Highway Statistics 1982
U.S. EPA Office of Mobile Sources: MOBILE II Documentation
The Transportation Energy Book

These sources are all referenced in Chapters III or IV. In addition, we also derived certain estimates based on the data these sources presented.

Sensitivities of Our Projections to Alternative Assumptions

Our predictions of total gasoline demand in 1988 are sensitive to the average miles per year traveled by vehicles, to the projected sales of cars and light trucks in each year to 1988, and to the scrappage rates we used to retire portions of each cohort in each year. Roughly, changes in these parameters cause proportional changes in gasoline demand. Appendix Table IV-4 lists the basic age-related assumptions.

Data concerning average miles per vehicle per year* (MPV) came from EPA's MOBILE II documentation, representing about a

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* Wherever possible with data and method, we disaggregated by cars and light trucks (0-8500 lbs. GVW), and by age of vehicle.

APPENDIX TABLE IV-4

GENERAL FLEET ASSUMPTIONS

AGE	# OF LDVs+	% MIS-FUELING IN 1 YR++	CUMU-LATIVE % MIS-FUELING	VEHICLE SURVIVAL RATES *		AVG ANNUAL MILES PER VEHICLE **	
				CARS	LDT	CARS	LDT
1 OR <	15,250	5.5	5.5	0.999	0.995	14,400	15,676
2	14,892	1.7	7.7	0.996	0.987	14,275	15,276
3	14,235	1.6	8.8	0.988	0.977	13,775	13,692
4	13,809	1.6	10.4	0.976	0.964	13,250	12,223
5	12,957	1.6	12.0	0.978	0.946	13,250	11,001
6	11,030	1.6	13.6	0.976	0.923	12,675	9,992
7	8,951	1.7	15.3	0.956	0.894	12,175	9,238
8	8,900	1.6	16.9	0.935	0.858	11,650	8,488
9	8,929	1.6	18.5	0.912	0.851	11,075	7,913
10	10,160	1.6	20.1	0.881	0.765	10,575	7,413
11	9,168	0.0	20.1	0.851	0.710	10,050	6,929
12	7,643	0.0	20.1	0.830	0.651	9,475	6,510
13	5,745	0.0	20.1	0.813	0.591	8,975	6,163
14	3,604	0.0	20.1	0.809	0.531	8,450	5,829
15	3,934	0.0	20.1	0.810	0.474	7,875	5,425
16	3,552	0.0	20.1	0.812	0.419	7,375	5,160
17	2,615	0.0	20.1	0.800	0.370	6,850	4,954
18	1,708	0.0	20.1	0.790	0.325	6,275	4,625
19	1,413	0.0	20.1	0.770	0.284	5,775	4,400
20	1,150	0.0	20.1	0.750	0.248	5,000	4,400
	159644		13.5				

Note: Light-duty vehicles (LDVs) includes cars and light-duty trucks (LDTs)

* First time misfuelers

** Includes current and past misfuelers. (Source: U.S. EPA Office of Mobile Sources, 1983d.)

\+ Source: R.L. Polk & Co. in MVMA Facts & Figures, 1981

++ Source: U.S. EPA Office of Mobile Sources, 1983d.

1.1% annual growth from actual 1980 MPV figures (estimated by Polk in MVMA, 1982). In 1988, these figures are about 9% greater than 1980 figures. Consequently, if one were to use 1980 data, gasoline demand would be 8-9% lower.

Estimates of the initial number of cars from each model year came from Polk, as reported by MVMA. The data on light trucks were acquired by the EPA directly from Polk, but were adjusted downward by 13%, to transform the category from 0 to 10,000 pounds to 0 to 8500 pounds. This adjustment was derived by a comparison of several different sources of data and is used by EPA's Office of Air and Radiation.

We use transitional probabilities of survival in order to retire some portion of each cohort as it moved into the next age category. That is, 99.6% of one year old cars live to be two years old; 98.8% of two year olds live to be three, etc. For cars, we averaged the transitional probability of survival for each age group reported by Polk in MVMA for 1978-1982. For light trucks, sufficient Polk data were not available; instead, we used survival rates estimated in Kulp and Holcomb (1982). We did not use their estimates for cars because it was derived by a model with which we were not familiar and which used scrappage rates well above any observed in recent years. We used a 7.4% scrappage rate in the current analysis. Using Kulp and Holcomb's estimate of 10.5% would decrease gasoline demand from 100.7 billion gallons to 89 billion gallons in 1988. In addition, such a change in assumptions would increase the unleaded market share from 67.8% to 70.5%.

We used Data Resources, Inc. (DRI) projections for sales of cars and light trucks (TRENDLONG2008B), as reported in U.S. Long-Term Review (Fall, 1983). Miles per gallon per vehicle came from the road mileages reported in EPA's Passenger Car Fuel Economy and were adjusted for change in fuel economy by age.

There are several assumptions that do not influence total demand for gasoline but do determine the split between leaded and unleaded grades. Most important are misfueling rates by age of vehicle, and, in particular, the shape of this curve in the youngest model year cohorts (i.e., model years 1985-1988). This part of the fleet is particularly important because: there will be more of these vehicles in 1988 than older cohorts, these vehicles will be emitting farther into the future than older vehicles, and, because of discounting back to the year of misfueling, they are weighted most heavily. We used EPA's 1982 survey of vehicle tampering for raw data, which provided misfueling rates by age of vehicle. We used regression analysis to estimate the relationship between age and incremental misfueling, using several specifications of form. By far, the best fit was a tri-linear form, with a 5.5% increase in the first year of the cohort's existence, a 1.66% increase per year for ages 2 to 9, and with no incremental misfueling in subsequent years. (In 1982, the time of the survey, vehicles with catalytic converters had been sold for only seven years, so no data existed on misfueling beyond the seventh year.)

Listed below are the actual 1982 survey results and the regression estimates used in the analysis.

APPENDIX TABLE IV-5

MISFUELING RATES BY AGE
(as percentage of model year cohort)

AGE	EPA 1982 SURVEY	REGRESSION ESTIMATES
1 or less	5.2%	5.5
2	7.4%	7.2
3	8.1%	8.8
4	12.1%	10.4
5	12.2%	12.0
6	12.4%	13.6
7	14.5%	15.3
8	17.7%	16.9
9	NA	18.5
10	NA	20.1
11	NA	20.1

METHOD FOR ESTIMATING DISCOUNTED STREAM OF AVOIDED EMISSIONS

In estimating the discounted streams of avoided emissions, the following procedure was used:

1. We assumed 87.3 million cars and 31.9 million light-duty trucks designed to use unleaded fuel would be on the road in 1988. Using vehicle survival rates, we estimated that approximately 82% of the total light-duty vehicles (cars and trucks) would be equipped with catalytic converters in 1988.

2. We then estimated, from EPA survey data, the proportion of these vehicles expected to misfuel for the first time in 1988. These estimates are presented below:

Model Year	Total # of Vehicles* (thousands of vehicles)	First-Time** Misfueling Rates	Total # of First-Time Misfuelers
1988-89	15,250	.055	839
1987	14,892	.017	253
1986	14,235	.016	228
1985	13,809	.016	221
1984	12,957	.016	207
1983	11,030	.016	176
1982	8,951	.017	152
1981	8,900	.016	142
1980	8,924	.016	143
1979	10,160	.016	163

3. The projected number of misfueling vehicles was multiplied by an estimate of the number of miles driven per vehicle in 1988. The average annual mileage factors were specific both for class† and age of vehicle. These calculations were

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* Automobile data from MVMA Facts and Figures '83; light-duty truck data from R.L. Polk & Co.

**No first-time misfueling was assumed for vehicles older than model year 1979.

† Automobiles, light-duty trucks between 0 and 6000 lbs., and light-duty trucks between 6000 and 8500 lbs.

repeated for every year of the assumed 20-year life of the vehicle, with the fleet size being diminished annually according to contemporary scrappage rates. Annual mileage per vehicle was adjusted according to vehicle age. In this way, model year- and vehicle class-specific estimates for total miles driven after misfueling in 1988 were derived, with the final year investigated being 2007 (when the 1988 model year fleet was assumed to be retired). This forecast the mileage from each misfueled cohort in each future year.

4. Each future year's mileage etimates were discounted back to 1988 at a 3% discount rate. The total discounted miles driven (in billions of miles) are shown below:

Model Year	Automobiles	LDT_1*	LDT_2*
1979	3.45	1.00	0.45
1980	4.21	0.72	0.33
1981	4.54	0.82	0.38
1982	5.64	0.97	0.42
1983	7.34	1.52	0.75
1984	10.06	2.06	0.96
1985	12.48	2.43	1.14
1986	14.74	2.85	1.34
1987	18.44	3.68	1.73
1988-89	67.90	13.94	6.57

The 1988-89 numbers are large because of the 5.2% rate of misfueling in the first year and because 15 months of auto sales are included in the last category.

5. Discounted future mileage was multiplied by the excess emissions factor developed by EPA (1983d), measured in grams of pollutant per mile (see below). This yielded total discounted future emissions of conventional pollutants as a result of misfueling in 1988.

Model Year	CO	NO_x	HC
1981-1988	11.07g/mi	0.71g/mi	1.57g/mi
1979-1980	17.65g/mi	--	2.67g/mi

6. This result was divided by $1x10^6$ to calculate the total metric tons of discounted emissions shown in Table IV-3.

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* LDT_1 = Trucks between 0 and 6000 lbs.
  LDT_2 = Trucks between 6000 and 8500 lbs.

POSSIBLE BIASES IN AVOIDED EMISSIONS

A. Reasons our Emissions Estimates may be too Low

° 1982 misfueling rates, based on EPA surveys, may be too low for reasons explained on page IV-3ff and in the 1979 EPA survey. Most notable is that vehicle inspections for misfueling were voluntary and in some areas, the rates of drivers refusing inspections were very high (up to 44% in one non-I/M area).

° We held misfueling rates constant over time, but these rates may be increasing over time.

° Vehicles are lasting longer than previously; therefore, our vehicle survival rates may be too low. With longer lifetimes, older, dirtier, misfueled vehicles would be in operation longer, and the stream of excess emissions would extend farther into the future. Furthermore, we retired each cohort after its twentieth year of operation (with about 7% remaining in the twentieth year).

° If vehicles are not well-maintained, excess emissions factors for misfueling will be higher.

B. Reasons our Emissions Estimates may be too High

° We assumed that pollution control equipment would be effective past the five-year manufacturer's warranty, for the life of the vehicle. Some EPA data indicated that this was true if vehicles were not misfueled or tampered with.

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CHAPTER V

BENEFITS OF REDUCING LEAD: CHILDREN WITH HIGH BLOOD LEAD

Our analysis of the 1988 health benefits of reducing lead is presented in two parts. This chapter describes the benefits associated with reducing the number of children with blood lead levels above 30 ug/dl. Currently the Centers for Disease Control (CDC) considers this level the criterion for lead toxicity when combined with FEP levels of 50 ug/dl or more (CDC, 1978). Chapter VI addresses the benefits for children with blood lead below 30 ug/dl. We have focused our analysis on children. Although adults experience adverse effects from lead, these effects generally occur at higher lead levels than in children and, as described below, children have higher body lead burdens for the same exposure.

Blood lead levels above 30 ug/dl are associated with adverse cognitive effects, anemia, kidney damage, hypertension, and other pathophysiological consequences. Several of these effects have only been documented at blood lead levels well above 30 ug/dl. In the next chapter, we discuss the physiological and cognitive effects that occur below 30 ug/dl.

It should be noted that while our discussion of reducing lead emissions has focused on <u>airborne</u> lead, airborne lead is eventually deposited in the environment on land, water, buildings, etc. Children, as a class, are most at risk from all sources of lead -- inhaled or ingested. Small children who crawl and "mouth" objects and hands are especially likely to ingest lead. Fetuses and young children are more vulnerable than the population as a whole. The absorption and retention rates, and the partitioning of lead in hard and soft tissues all contribute to the fact that

children possess greater lead body burdens for a given exposure. Children have also been shown to display a greater sensitivity to lead toxicity, and their inability to recognize symptoms may make them especially vulnerable. In the late 1970s data indicated that well over 10% of black children had blood lead levels above 30 ug/dl (Mahaffey et al., 1982a).

The first section of this chapter presents the evidence supporting the relation of blood lead to gasoline lead. Next, two aspects of the effects of lead exposure that we have been able to monetize are discussed. First, we assessed the costs associated with medical treatment and follow-up care for the children who have elevated blood lead levels. Second, we considered the cognitive and behavioral impacts of high blood lead levels (above 40 ug/dl) in children. This chapter also presents the methodology by which we predicted the changes in the number of children above 30 ug/dl (and other thresholds) as a function of changes in the total amount of lead used in gasoline.

The monetized benefits of reducing the number of children with blood lead levels above 30 ug/dl in 1988 fall into two categories: 1) the avoided costs of testing for and monitoring children with elevated blood lead levels, and medically treating children with very elevated levels; and 2) the costs associated with the cognitive effects of lead exposure above 30 ug/dl.

The benefits computed in this chapter are a linear function of the reduction in the number of children above 30 ug/dl of blood lead. For each policy option, we estimated these reductions by

by using the techniques discussed in the statistical methodologies section (V.E.). The results are shown below in Table V-1.

TABLE V-1

Reduction in Number of Children above 30 ug/dl in 1988

Low-Lead Option	All Unleaded
43,000	45,000

V.A. The Relationship between Gasoline Lead and Blood Lead

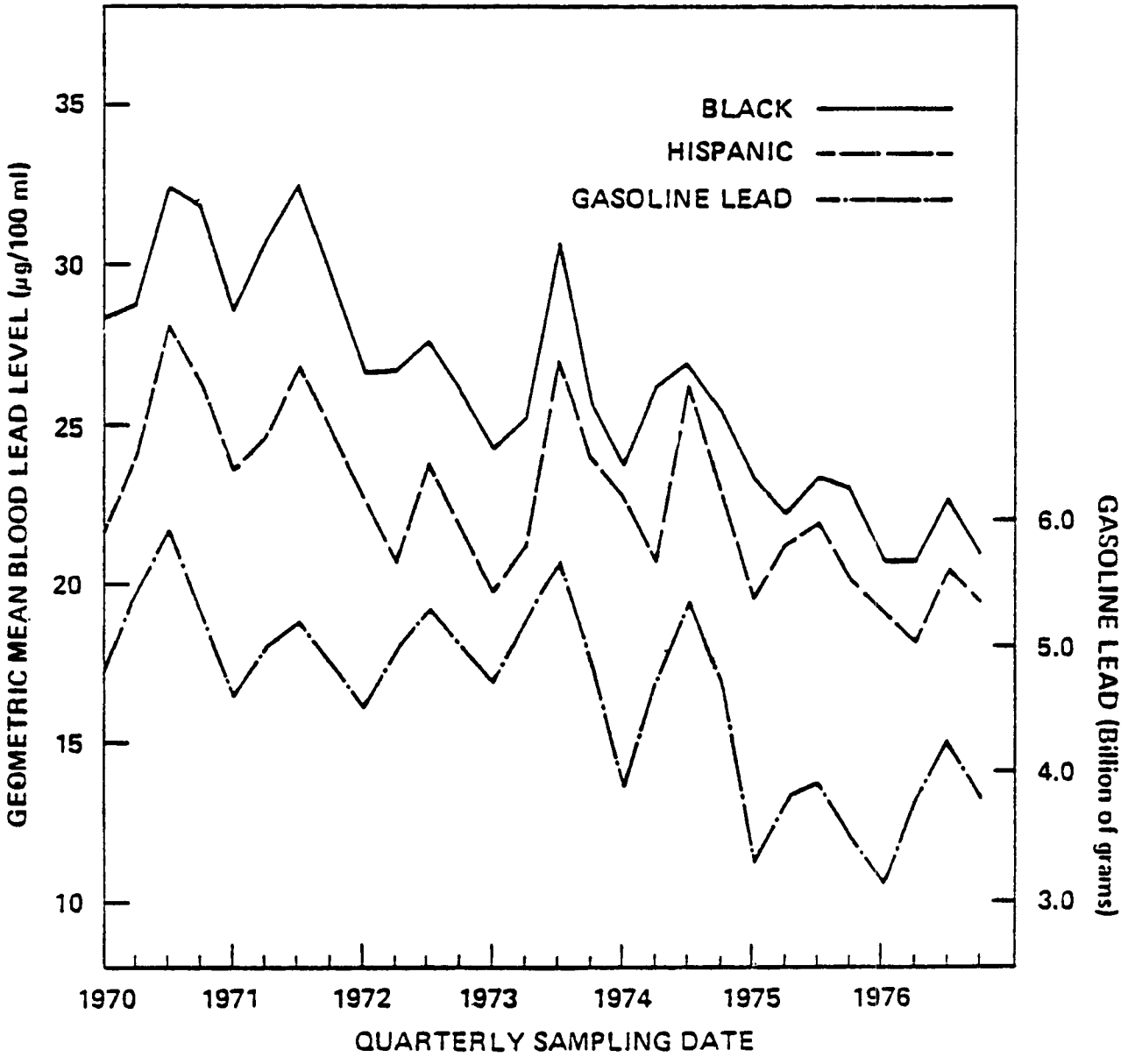
Several recent articles have shown persuasively that blood lead levels for a given age group will fall as gasoline lead content falls. The first important statistical studies were done by Billick et al. (1979), who showed a strong relationship between the blood lead levels of several hundred thousand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

In 1982, Billick 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 his earlier results.

A recent paper by EPA's Office of Policy Analysis (Schwartz, Janney, and Pitcher, 1984), presented the results of a study concerning the relationship between blood lead levels and gasoline lead. Three different data sets were employed for this analysis,

FIGURE V-1

CHILDREN'S BLOOD-LEAD LEVELS VARY
DIRECTLY WITH LEVELS OF LEAD IN GASOLINE



including the second National Health and Nutrition Examination Survey (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. External estimates of environmental lead from other sources clearly indicated that paint and other dietary lead were not the primary sources of the observed decline in blood lead levels. (An earlier paper on this subject was presented by Schwartz at the International Conference on Heavy Metals in the Environment (1983) in Heidelberg, West Germany.)

Critics questioned whether the association between gasoline lead and blood lead levels could have been due to the sequence in which the NHANES II survey team moved from one site to the next. Repeated tests of the results, using variables for each location, indicated that specific locations or geographic regions did not confound the relationship between blood lead and gasoline lead. Furthermore, performing separate regressions for urban areas, rural areas, adults, children, blacks, and whites indicated these factors could not be substituted for gas lead to explain the changes in blood lead levels.

A third study, by Annest et al. (1983) of the U.S. Public Health Service, also used data from the NHANES II, finding that the only reasonable explanation for the decline in blood lead levels was the decline in the amount of lead in gasoline.

Finally, the Draft Lead Criteria Document cited two studies (Fachetti and Geiss, 1982; and Manton, 1977) which, by introducing

tetraethyl lead with a different isotope ratio into gasoline, were able to directly measure the contribution to blood lead levels from gasoline. Both of these papers showed that gasoline accounted for about 5-10 ug/dl of blood lead.

V.B. Medical Benefits of Reducing High Blood Lead Levels

To estimate the benefits from reduced numbers of children with blood lead levels above 30 ug/dl, we assumed that all children whose lead levels were elevated above this limit would receive follow-up medical attention and/or immediate medical treatment. Unfortunately, however, many -- perhaps even most -- children with elevated lead levels are not detected, although their lives and health are adversely affected. It should be noted, therefore, that children with blood lead levels greater than 30 ug/dl who go untreated bear a burden which we valued equal to the cost of follow-up and/or treatment. Furthermore, the dollar estimate of average medical management costs (testing and monitoring) assumed a prototypical method of determining treatment; these costs were representative of the costs associated with treatment and follow-up techniques in general use, although the exact procedures may vary.

We have distinguished between three basic follow-up and treatment categories: children with blood lead levels over 30 ug/dl but with free erythrocyte protoporphyrin (FEP) levels below 50 ug/dl, children in the Centers for Disease Control's

(CDC) lead toxicity category II, and children in CDC categories III and IV.* Treatment and follow-up practices may differ for each.

For children with over 30 ug/dl of blood lead, given FEP levels below 50 ug/dl, we assumed one follow-up blood test and the associated overhead costs. From the regression presented by Piomelli et al. (1982) on the probability of elevated FEP versus blood lead, we estimated that 60% of the children over 30 ug/dl had FEP levels above 50 ug/dl. However, Mahaffey and coworkers (1982) cited data from the CDC screening program indicating that 75% of all screened children over 30 ug/dl of blood lead also had FEP levels above 50 ug/dl. Since the CDC sample was both larger and more representative of the entire nation than that used by Piomelli et al., we have placed slightly greater emphasis on this result and assumed 70% of the children above 30 ug/dl would be classified lead toxic by CDC. To further estimate the fraction of the most severely lead toxic children in categories III and IV, we examined the results of the CDC screening program for 1977-81. They showed a relatively constant 33% of all lead toxic children were in categories III or IV; the remaining 67%

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* CDC classifies children as "lead toxic" if they have blood lead levels above 30 ug/dl and FEP levels above 50 ug/dl. Children between 30-49 ug/dl blood lead and 50-109 ug/dl FEP are category II. Category III is either children > 50 ug/dl blood lead and <250 ug/dl FEP or > 110 ug/dl FEP and 30-50 ug/dl blood lead. Children >50 ug/dl blood lead and >250 ug/dl FEP or children >70 ug/dl blood lead are category IV.

must, therefore, have been in category II.* Therefore, for all children with blood lead levels above 30 ug/dl, 30% have FEP levels below 50 ug/dl, 47% are in category II, and 23% are in CDC's categories III and IV.

We assumed that children in category II would receive six regularly scheduled blood tests, and that about half of these children would also have a county sanitarian visit their homes to evaluate possible sources of lead exposure. (The CDC screening program data indicated that 65% of the homes of all lead toxic children were visited. Assuming all category III and IV children had home visits, this suggested a 50% rate for category II.)

We also assumed, per the CDC's recommendations, that detailed medical histories, physical examinations, and an assessment of nutritional status would be performed by a physician. For children in categories III and IV, we assumed a three-day hospital stay for testing, and that a county sanitarian would

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* The quarterly prevalence data for the percent of all lead toxic children who were category III or IV are:

Year	1st Quarter	2nd Quarter	3rd Quarter	4th Quarter
1977	32%	32%	34%	31%
1978	31%	32%	35%	31%
1979	30%	33%	37%	34%
1980	29%	34%	38%	35%
1981	30%	33%		

(source: Morbidity and Mortality Weekly Reports)

Approximately 7,000 children per quarter were found to be lead toxic. Note that the percent in categories III and IV was highest in the 3rd quarter (July, August and September) when gasoline lead emissions are highest.

inspect their homes. On the basis of CDC recommendations, it was assumed these children would have six monthly follow-up blood tests after discharge, and another six quarterly follow-ups. Finally, we assumed the children in these severely afflicted categories would receive a neurological examination, and that one-third of them would undergo provocative ethylenediaminetetraacetic acid (EDTA) testing and chelation therapy to remove lead from the body.

EPA has estimated the cost of blood tests to be $30. We assumed (1) a one-time administrative overhead charge of $50 for every child who entered the system, (2) a physician's cost of $50 per visit, and (3) a home inspection by a county sanitarian cost of $60, including overhead. We have used 1982 hospital costs per adjusted inpatient day from the Department of Health and Human Services publication Hospital Statistics (1983). Having regressed the trend in these costs since 1972 against the GNP deflator, we obtained an average rate of increase in real costs and projected costs per day in 1988 (including lab tests, etc.) to be $425 (in 1983 dollars). For each of the major hospitalization stages, physician's costs of $250 have been estimated, including a neurological work-up. Using these figures we estimated the average medical costs for children over 30 ug/dl to be $950 per child.

Table V-2 shows the medical cost savings in 1988 of reducing the number of children over 30 ug/dl. Because we have not estimated welfare losses (such as work time lost by parents), the adverse health effects of chelation (such as the removal of helpful minerals), or such non-quantifiables as the pain from the

treatment, our estimate of the benefits of reduced treatment is conservative. As mentioned above, we have taken these medical costs as a measure of avoidable damage for all the incremental cases of lead toxicity, whether detected or not.

TABLE V-2

Medical Cost Savings in 1988
(1983 dollars)

Low-Lead	All Unleaded
$41 million	$43 million

Our analysis has assumed 30 ug/dl as the criterion for defining when a child is at risk for undue lead exposure or toxicity and may require pediatric care. (This is the criterion now used by CDC, in conjunction with elevated FEP levels.) If that criterion is lowered, greater numbers of children would receive medical management, thereby increasing the medical expense savings from lowering blood lead levels. This is not an unlikely event, as the Draft Lead Criteria Document (1983) indicated:

> "If, for example, blood lead levels of 40-50 ug/dl in "asymptomatic" children are associated with chelatable lead burdens which overlap those encountered in frank pediatric plumbism, as documented in one series of lead exposed children, then there is no margin of safety at these blood levels for severe effects which are not at all a matter of controversy. Were it both logistically feasible to do so on a large scale and were the use of chelants free of health risk to the subjects, serial provocative chelation testing would appear to be the better indicator of exposure and risk. Failing this, the only prudent alternative is the use of a large safety factor applied to blood lead which would translate to an "acceptable" chelatable burden. It is likely that this blood lead value would lie well below the currently accepted upper limit of 30 ug/dl, since the safety factor would have to be large enough to protect against frank plumbism as well as more subtle health effects seen with non-overt lead intoxication." (Chapter 13, p. 15) (emphasis added)

For example, the estimated number of children whose blood lead levels would be expected to drop from above 25 ug/dl to below this figure as a result of an all unleaded gasoline scenario in 1988 is 150,000, over three times the figure of 45,000 used in the present analysis, derived from a criterion of 30 ug/dl blood lead.

V.C. Cognitive and Behavioral Effects

Many studies have noted neurological effects in children with elevated blood lead levels. De la Burde and Choate's results (1972, 1975) have been summarized by the Draft Lead Criteria Document as showing persisting neurobehavioral deficits at blood lead levels of 40-60 ug/dl. In the 1975 study, seven times as many high lead children were found to have repeated grades in school or were referred to school psychologists as low lead control children. The control children were drawn from the same clinic population and were matched for age, sex, race, parents' socioeconomic status, housing density, mother's IQ, number of children below six in the family, presence of father in the family, and mother working.

Although the children examined in the work of de la Burde and Choate included some with blood lead levels between 30 and 40 ug/dl, the issue of whether the cognitive deficits occurred at those levels was not clear from the results. Several additional studies cited in the Draft Criteria Document, as well as recent work by Odenbro et al. (1983), indicated a significant association between these blood lead levels and neurological/

cognitive effects in children (e.g., Needleman et al., 1979; McBride et al., 1982; Yule et al., 1981; Yule et al., 1983; Smith et al., 1983; Yule and Lansdown, 1983; Harvey et al., 1983; and Winneke et al., 1982). All of these studies generally support these results, even though individually the probability of a false positive was not always less than 5% and the possibility of uncontrolled covariates existed. Nevertheless and despite the difficulties with the specific studies, the combined weight of the evidence showed that cognitive deficits occurred at blood lead levels over 30 ug/dl, with the work of de la Burde and Choate indicating that the most serious damage may be associated with blood lead levels over 40 ug/dl. (A more detailed analysis of the studies is presented in Section VI.E.)

V.D. Estimating Avoided Costs of Compensatory Education

The evidence for cognitive effects of lead in children above 30 ug/dl is fairly strong, and the studies by de la Burde and Choate gave direct evidence of poorer classroom performance by children with higher lead levels, particularly those over 40 ug/dl. It also showed that the cognitive effects remained three years later.

To value avoiding such cognitive damage, we could posit that children involuntarily exposed to enough lead to make them seven times more likely to be forced to repeat a grade should be given enough supplementary educational assistance to bring their school performance back to what it otherwise would have been. Therefore, we could use the cost of such compensatory education as a proxy for the avoided cost.

Of course, it is probably impossible to completely restore these high lead children's performance. Therefore, lifetime work and production may be affected. However, tutoring, reading teachers, school psychologists, and the like can help improve their achievement in school.

Given the finding of at least a three year persistence in the cognitive effects of lead, we assumed that the cost of correcting these cognitive effects would be at least three years of compensatory education. We judged that de la Burde and Choate's exposed population corresponded to children in CDC's categories III and IV, as well as some category II children. From January 1977 until mid-1981, one-third of the children identified by the CDC screening program as being lead toxic (over 30 ug/dl blood lead and 50 ug/dl FEP) were in CDC categories III and IV, the more severe categories of lead toxicity. From this we estimated that one-third of the children above 30 ug/dl would fall in the category of those severely enough affected to need compensatory education to recover their previously expected performance levels. Children with lower internal lead/FEP levels were assumed not to need this education. Therefore, an average of one year of compensatory education would be required per child with blood levels over 30 ug/dl to compensate for the deficits.*

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* We assumed that the number of person-years of compensatory education divided by the number of children would be about one. In other words, if one-third of these children require three years of compensatory education, there is an average of one year of education for each of the children.

As a rough approximation of the expense of such compensation, we have used the cost of part time special education for children who remain in regular classrooms. The staff of the Department of Education's Office of Special Education Programs (OSEP) felt this level of effort was appropriate for these children. According to a report written for OSEP (Kakalik et al., 1981), a child needing this form of compensatory education incurred additional costs of $3,064 per year in 1978 dollars, or $4,290 in 1983 dollars (using the GNP deflator). This figure was quite close to Provenzano's (1980) estimate of the special education costs for non-retarded lead exposed children. We applied these costs to our estimate of the number of children who would fall below 30 ug/dl as a result of our two hypothetical policy options to calculate the benefits in 1988 of reducing their cognitive damage. These benefits are displayed below in Table V-3.*

TABLE V-3

Benefits in 1988 of Reduced Cognitive Losses
(1983 dollars)

Low-Lead	All Unleaded
$184 million	$193 million

* We have not assumed that all these children would be classified as having learning disabilities, but rather that they would all perform worse than they would have otherwise. Thus, compensatory education costs were used as a proxy for the cost of restoring their cognitive functioning.

V.E. Statistical Methodologies

In this section we present the regression results and forecast procedures that underlie the estimates of the changes in the number of children at risk of elevated blood lead levels used in this and the subsequent chapter. First, however, we review the evidence of a relationship between blood lead levels and the amount of lead in gasoline. Following this, we describe the data base used for our regression work and the regression results. Finally, there is a discussion of our forecasting procedures and a consideration of the implications of forecasting prevalence rather than incidence.

V.E.1. The NHANES II Data

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 selected according to 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 were 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 between seven through 74 years. The non-respondent rate for blood samples was 39% and did not correlate with race, sex, annual family income, or degree of urbanization.* A study of the potential non-response biases 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 twenty NHANES II blood samples. No temporal trend was evident in the blind quality control measurements.

The NHANES II data did, however, display a marked relationship between blood lead and gasoline lead, as is shown in Figures V-2 and V-3. A similar pattern existed between average blood lead levels for black children in Chicago and lead use in local Chicago gasoline during the same period. This is evident in Figure V-4.

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* Because children were less likely to respond, they were double sampled, and 51% of the children did not provide blood for lead determinations in the NHANES II data set. 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)

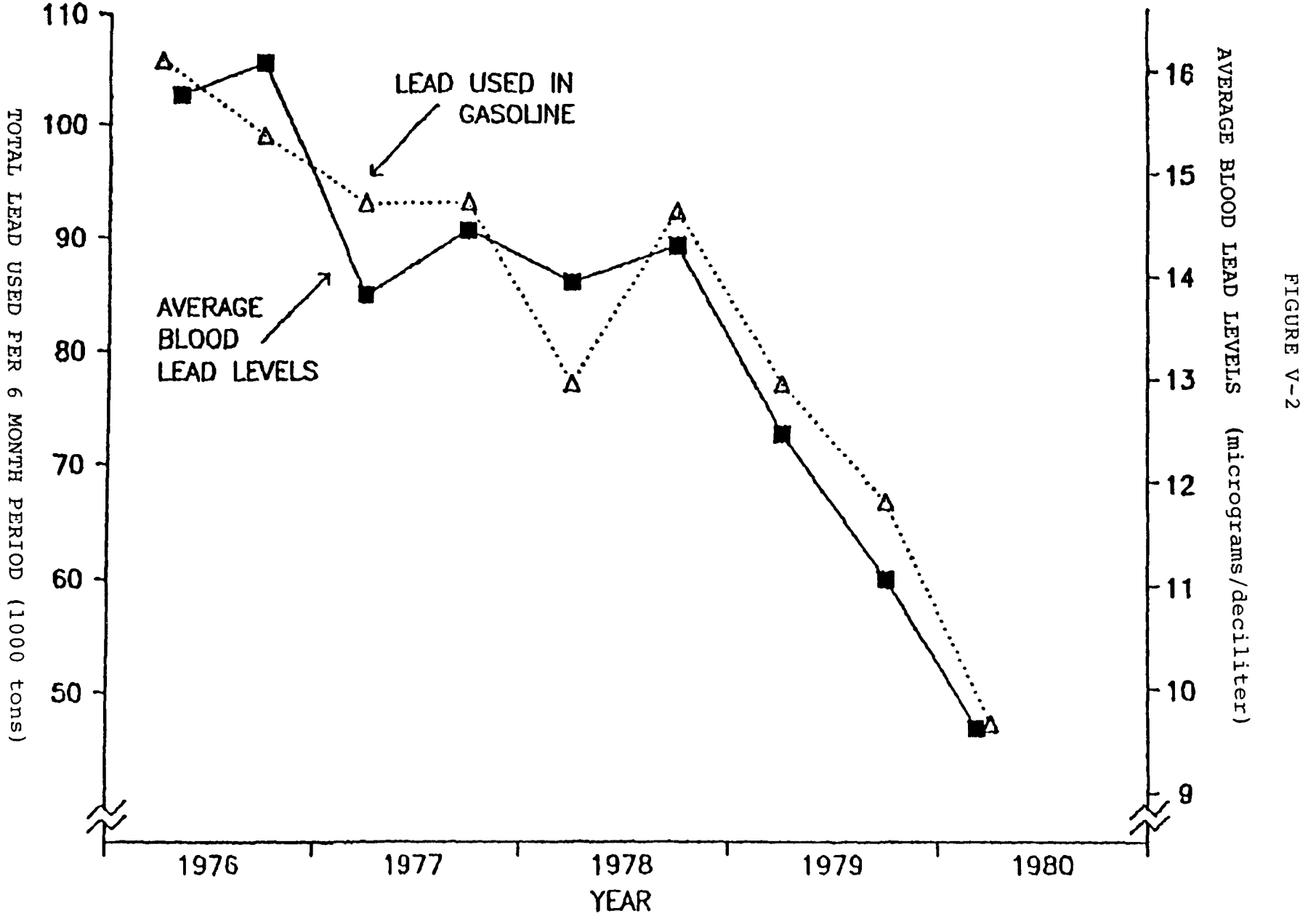


FIGURE V-2

AVERAGE NHANES II BLOOD LEAD LEVELS VS. LEAD USED IN GASOLINE PRODUCTION

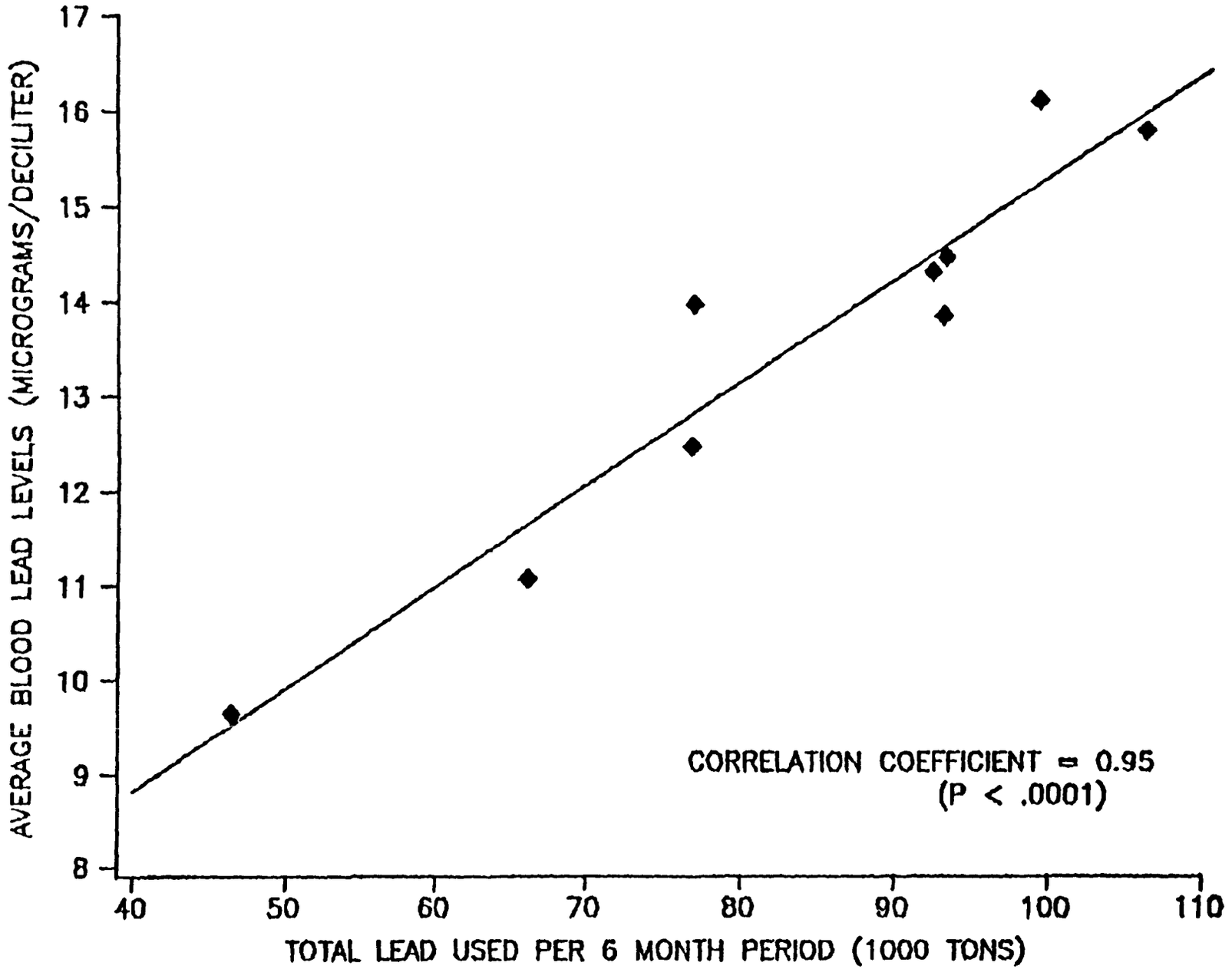
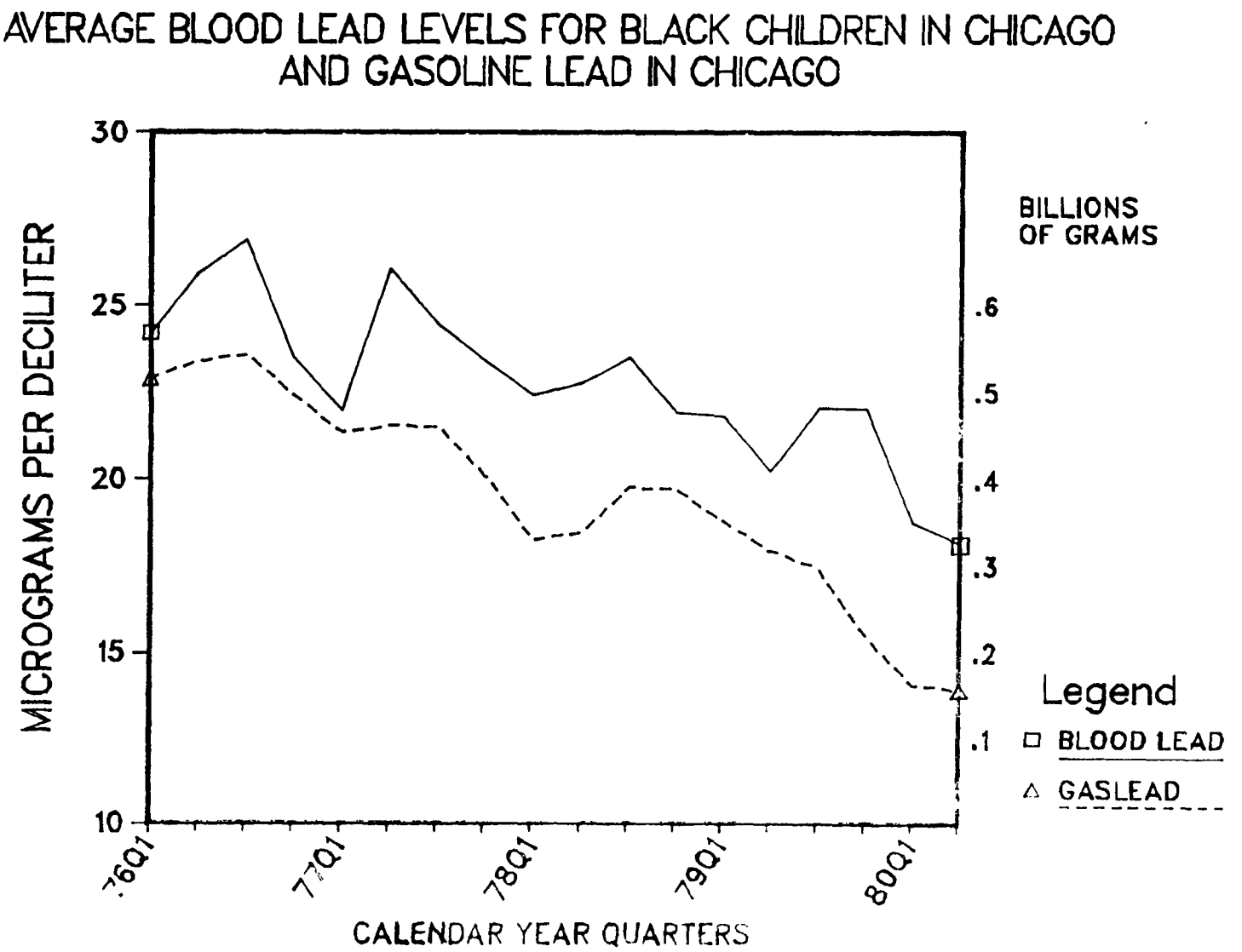


FIGURE V-3

FIGURE V-4


AVERAGE BLOOD LEAD LEVELS FOR BLACK CHILDREN IN CHICAGO AND GASOLINE LEAD IN CHICAGO
MICROGRAMS PER DECILITER
30
25
20
15
10
BILLIONS OF GRAMS
.6
.5
.4
.3
.2
.1
76Q1
77Q1
78Q1
79Q1
80Q1
CALENDAR YEAR QUARTERS
Legend
BLOOD LEAD
GASLEAD

V.E.2. Reduction in Number of Children Below Critical Thresholds

The NHANES II data was used to estimate both linear regressions relating blood lead to gas lead and the percentage of children who would be expected to have blood leads above various thresholds.

To estimate these percentages, logistic regressions were performed separately for white and black children to see how the odds of having blood lead levels above a 30 ug/dl threshold varied with gasoline lead. These regressions were performed on data from individual children. The dependent variable was the natural log of the odds of being above the threshold while the independent variables were various demographic factors* and gasoline lead. The original selection of demographic factors for consideration was based on linear regressions on individual blood lead levels, discussed in detail in the paper by Schwartz, Janney, and Pitcher (1983).**

To predict how the number of children above each threshold would change as the amount of lead in gasoline was reduced, a mechanism was needed to forecast the distribution of blood lead

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* The demographic variables were selected by backwards stepwise elimination. We also used a procedure that maximized R^2 for any given number of variables and a procedure that minimized the difference between C_p and the number of independent variables. They produced the same model as backwards elimination.

** The regressions were all performed on individual data using the SAS procedure SURREGR to estimate the coefficients. SURREGR is a special procedure designed to estimate the variances in regressions using clustered stratified samples. Demographic control variables were eliminated by backwards stepwise elimination until all the remaining variables were significant at the 95% confidence level. See Schwartz, Janney, and Pitcher (1983) for further detail.

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 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.

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

$$a = \exp (u + 1/2\ s^2) \text{ and}$$

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

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

$$e_g = \exp (u + v_g\ s)$$

We used the logistic regressions to estimate e_g in equation (2) and the SURREGR regressions to estimate a in equation (1) which yielded

$$(1) \quad a = \exp (u + 1/2\ s^2)$$

$$(2) \quad e_g = \exp (u + v_g\ s)$$

Solving these equations for u and s produced a quadratic equation

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

which had the solution $s = -v_g \pm (v_g^2 - 2(\ln(e) - \ln(a))^{.5}$. Then $u = \ln(a) - 1/2\ s^2$. Only the smaller root yielded sensible values for u and s. 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 chose to use a logistic 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 deviation, we would have predicted that removing lead from gasoline would have resulted in there being no children above 30 ug/dl. This seemed unreasonable since paint, food, and water are known alternate sources of lead, and are sometimes associated with high blood lead levels.

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 number of children above various thresholds represented the sum of distributions for each age category. The regression results are shown in the Appendix to this chapter.

For children from six months to seven years of age, we used logistic regressions for the percent above 30 ug/dl 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 work.

Because our figures included only children under age thirteen and no adults, these results significantly underestimated the benefits from reduced lead levels in the entire population. To make our predictions, we used our projections of lead used each year under the various scenarios. These are shown on Table V-4 below.

TABLE V-4

Estimated Lead Used for Gasoline in 1988*
(metric tons per day)

Base Case	Low-Lead	All Unleaded
97.6	6.1	0

*Computed using gasoline demand in Table II-1 and assuming 1.1 g/gal, 0.1 g/gal, and 0 g/gal.

From this, we could also predict changes in mean blood lead levels. These are shown in Table V-5 below.

TABLE V-5

Changes in Mean Blood Lead in 1988**
for Black and White Children aged 5 and under
(micrograms per deciliter)

	Base Case	Low-Lead		All Unleaded	
		Incremental Decline	Projected Level	Incremental Decline	Projected Level
White	7.93	1.93	6.00	2.13	5.80
Black	14.31	1.72	12.59	1.89	12.42

**Derived using gasoline lead values in Table V-4 and the regressions presented in the Appendix to this chapter.

To compute the number of children above the various thresholds in 1988, we needed estimates of the population at different ages. These were produced by linearly interpolating the Bureau of the Census population projections (mid-range forecast) for 1985 and 1990 and are shown in Table V-6.

TABLE V-6

1988 Population Projections*

Ages	Blacks	Whites
1/2 year - 7 years	4,573,000	23,259,000
8 years - 13 years	3,797,000	16,528,000

*Bureau of the Census, 1982

These results also tend to underestimate the extent of the problem because the NHANES II survey, upon which our model was based, omitted children under six months of age. This was especially significant because we are learning that the damage to this infant population from elevated blood lead levels may be more severe than that of older children.

V.E.3. Incidence Versus Prevalence

Our predicted decreases in the number of children above a given threshold were for a specific point in time; our costs were 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 raised the difficult epidemiological issue of prevalence versus incidence. Prevalence means 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 the integral of the incidence of cases times their duration, or prevalence is approximately incidence times average duration.

This issue became 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 wanted to estimate may 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) found fairly stable blood lead levels in individual children with high levels in Chicago. For these children, levels remained high for more than a few days, usually for months or years. However, if the average elevation of 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 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 remained 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 effects. To do this we looked at 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.

As our first source we looked at the CDC screening program. This program screened approximately 100-125,000 children per quarter of the year to detect lead toxicity. Approximately 6-7,000 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 previous cases.

We have also 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 were generally followed for several 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 number 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 was 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.

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, Janney, and Pitcher (1983) found that the lag structure of average blood lead levels' dependence on gasoline lead extended about 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 (450,000 children screened) over a ten 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 and coworkers analyzed this 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 number of children above 30 ug/dl during 1988 is low, as is our estimate of avoided medical expenses.

V.E.4. Assessing the Accuracy of our Forecasting Procedures

The NHANES II data we used to estimate the regressions in our forecasting model corresponded with a range of gasoline lead usage from 193 to 550 metric tons per day. The options we are considering have gasoline lead usage rates of 97.6, 6.1, and 0 metric tons per day,* values that are below the range associated with

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*See Table V-4.

the NHANES II data set. An obvious concern was the applicability of results gathered from NHANES II data to the policy options under consideration.

To examine the hypothesis that the gasoline lead coefficient changed at lower gasoline lead values, we regressed blood lead levels for white children for just the last two years of the NHANES II period. (During this time period both blood and gasoline lead levels were lowest.) The gasoline lead coefficient changed by 3%, which was not significantly different from that derived for the full period. For blacks, the small sample size did not allow separate estimates for different periods. While there is no reason to believe that the functional form of the dependence was different for blacks and whites, we used an alternate procedure that did not require a reduction in sample size to check the linearity of blood lead's dependence on gasoline lead for blacks.

The log of blood lead was regressed against age, income, sex, and degree of urbanization, and against the log of gasoline lead. This produced a model in which blood lead was a function of gas lead to some power B, where B was the coefficient of log (gaslead) in the regression. We performed this regression to estimate the power law of blood lead's relation to gasoline.

Had we just regressed log (blood lead) on log (gaslead), we would have artificially forced blood lead to be zero when gasoline lead was zero. While studies of the bones of ancient Nubians indicated that prehistoric lead levels were essentially

trivial, studies of remote populations today (e.g., in the Himalayas) suggested that general environmental contamination produced 3-5 ug/dl blood lead levels in the absence of any gasoline or local industrial emissions (Piomelli et al., 1980).

Since background levels in the United States were likely to be higher than those of remote populations, we tested models with intercepts ranging from 6 to 10 ug/dl. They yielded exponents ranging from 0.82 to 1.08 for the dependence of black children's blood lead levels on gasoline lead. The model with the highest R^2 had an intercept of 8 ug/dl and an exponent of 0.98. The fact that the exponent values that fit the data best were very close to unity implied that blood lead is equal to $(\text{gaslead})^1$ -- i.e., the relationship was linear.

Finally, we tested a model where blood lead was related to the square root of gasoline lead, and it did not fit as well as the linear model. We believe, therefore, that the assumption that blood lead levels in black children are a linear function of gasoline lead is reasonable.

V.F. Conclusion

We have monetized two health related effects of reducing the amount of lead in gasoline. The projected benefits in 1988 estimated from these two effects alone are presented in Table V-7.

TABLE V-7

Monetized Benefits of Reduced Numbers
of Children Above 30 ug/dl Blood Lead Level in 1988
(millions of 1983 dollars)

Low-Lead	All Unleaded
$225 million	$236 million

There are additional effects that we have not monetized which have also been associated with blood lead levels above 30 ug/dl.

- We have not estimated the value of adverse effects in adults or infants under six months. As we mentioned above, new data have indicated that fetuses and newborn infants may be most vulnerable to lead effects.
- Non-neurological effects such as kidney damage, anemia, and other medical problems have not been assessed.
- Behavioral problems have not been addressed. (These can adversely alter attention span or take more overt forms such as serious behavioral abnormalities, perhaps affecting the education of other children in the classroom.)
- Finally, we mentioned certain non-quantifiable problems earlier such as the pain associated with some medical procedures, lost work (and leisure) time by family members, and the potential long-term social costs from the lower employment

potential of individuals whose learning abilities have been impaired. As a result, the health benefits presented in Table V-7 are likely to be much less than the real cost to society.

TECHNICAL APPENDIX TO CHAPTER V

In addition to the regressions shown in Schwartz, Janney, and Pitcher (1983), we have used the regressions presented in this appendix for our forecasts. We used the following variables in these regressions:

Variable Name	Description
Gaslead	Lead used in gasoline, in hundreds of of tons/day, lagged one month
Poor	1 if Income 1 (see below); 0 otherwise
Age 1	1 if age $\geq$ 6 months and $<$ 2 years; 0 otherwise
Age 2	1 if age $\geq$ 2 years and $\leq$ 3 years; 0 otherwise
Age 3	1 if age $\geq$ 4 years and $\leq$ 5 years; 0 otherwise
Age 4	1 if age $\geq$ 6 years and $\leq$ 7 years; 0 otherwise
Age 5	1 if age $\geq$ 8 years and $\leq$ 9 years; 0 otherwise
Age 6	1 if age $\geq$ 10 years and $\leq$ 11 years; 0 otherwise
Age 7	1 if age $\geq$ 12 years and $\leq$ 13 years; 0 otherwise
Income 1	1 if family income $<$ \$6,000; 0 otherwise
Income 2	1 if family income $<$ \$15,000 and $>$ \$6,000; 0 otherwise
Teen	1 if age $\geq$ 14 years and $<$ 18 years; 0 otherwise
Male	1 if gender is male; 0 if female

Variable Name	Description
Teen Male	1 if gender is male and age $\geq$ 14 years; and < 18; 0 otherwise
Adult Male	1 if gender is male and age $\geq$ 19 years; 0 otherwise
Small City	1 if residence is in city with population $\leq$ 1,000,000; 0 otherwise
Rural	1 if residence is a rural area as defined by the Bureau of the Census; 0 otherwise
Drinker	1 if alcohol consumption is $\geq$ 1 drink/ week and $\leq$ 6 drinks/week; 0 otherwise
Heavy Drinker	1 if alcohol consumption is $\geq$ 1 drink/ day; 0 otherwise
Northeast, Midwest, South	Are regions of the country as defined by the Bureau of the Census.
Education	Is 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

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; 0 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

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*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 one if the individual is above the threshold, and zero otherwise.

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
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
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

SURREGR Regression Results

Whites: children 6 months to 7 years

Dependent variable: individual blood lead levels

Variable	Beta	Std. Error	F-Statistic	P
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

Whites: 6 months to 13 years

Dependent variable: individual blood lead levels

Variable	Beta	Std. Error	F-Statistic	P
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: 6 months to 7 years

Dependent variable: individual blood lead levels

Variable	Beta	Std. Error	F-Statistic	P
Intercept	4.8847	2.4116	--	--
Gaslead	1.9342	0.1432	26.12	0.0000
Income 1	1.1457	0.2593	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: 6 months to 13 years

Dependent variable: individual blood lead levels

Variable	Beta	Std. Error	F-Statistic	P
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

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CHAPTER VI

BENEFITS OF REDUCING LEAD: CHILDREN WITH MODERATE BLOOD LEAD

In this chapter, we discuss the known pathophysiological effects of lead that may occur in children below 30 ug/dl of blood lead. As noted in the introduction to Chapter V, we focused our analysis on children because, on the whole, they are more sensitive and vulnerable to lead than adults. We discuss the hematological and neurological effects in particular, as well as the expected change in the number of children at potential risk of those effects under our policy alternatives.

Our benefit estimates present only changes in the numbers of children at risk of these effects; we have not associated any dollar values with reducing these exposures. Although no monetary estimate of adverse effects is provided, the social costs (to the individuals affected and society as a whole) associated with even low blood lead levels is probably substantial.

The scientific literature presents evidence of a continuum of biological effects associated with lead across a broad range of exposure. Even at low exposure levels, the Draft Lead Criteria Document (EPA, 1983) found that:

> biochemical changes, e.g., disruption of certain enzymatic activities involved in heme biosynthesis and erythropoietic pyrimidine metabolism, are detectable. With increasing lead exposure, there are sequentially more pronounced effects on heme synthesis and a broadening of lead effects to additional biochemical and physiological mechanisms in various tissues, such that increasingly more severe disruption of the normal functioning of many different organ systems becomes apparent. In addition to impairment of heme biosynthesis, signs of disruption of normal functioning of the erythropoietic and nervous systems are among the earliest effects observed in response to increasing lead exposure. At increasingly higher exposure levels, more severe disruption of the erythropoietic

> and nervous systems occurs; and other organ systems are also affected so as to result in the manifestation of renal effects, disruption of reproductive functions, impairment of immunological functions, and many other biological effects. At sufficiently high levels of exposure, the damage to the nervous system and other effects can be severe enough to result in death or, in some cases of non-fatal lead poisoning, long-lasting sequelae such as permanent mental retardation.
>
> (Chapter 12, pages 1-2)

While the hematopoietic, nervous, and renal systems are generally considered to be the most sensitive to lead, lead has a significant impact on reproductive and developmental processes as well.

Table VI-1 presents blood lead levels from the second National Health and Nutrition Examination Survey (NHANES II).

TABLE VI-1

BLOOD LEAD LEVELS OF PERSONS
Aged 6 Months - 74 Years in the United States 1976-80*
(percent in each cell)

	<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%
18-74 years	21.2%	62.3%	14.3%	1.8%	0.3%
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%
18-74 years	21.9%	62.3%	13.7%	1.8%	0.4%
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%
18-74 years	2.3%	56.4%	34.9%	4.5%	1.8%

*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.

VI.A. Pathophysiological Effects

Pathophysiological effects are found at blood lead levels well below 30 ug/dl, particularly in children. There is evidence that blood lead levels under 30 ug/dl 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 (Angle et al., 1982). Hernberg and Nikkanen (1970) found 50% 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 interference 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 EEG patterns (Otto et al., 1981, 1982) which begin to occur at about 15 ug/dl, and which 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 from about 15 ug/dl on (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 (Draft Lead Criteria Document, p 13-34; 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 12-32).

6. Inhibition of vitamin D pathways, which has been detected as low as 10 to 15 ug/dl (Rosen et al., 1980, 1981; Mahaffey et al., 1982b). Further, as blood lead levels increased, the inhibition became increasingly severe, and the lead absorption rate was enhanced.

These levels approximate the lowest observed effect levels to date and do not necessarily represent the affirmative findings of a threshold.

The types of specific effects listed above as occurring at blood lead levels below 30 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 production, and (d) lead-induced perturbations in central and peripheral nervous system functioning. The medical significance of such effects is not yet fully understood. But current knowledge regarding the deleterious nature of such effects and the 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. Drawing on material in Chapter 12 of the Draft Lead Criteria Document, we discuss the potential consequences of these findings below.

Heme, in addition to being part of hemoglobin, is the obligatory prosthetic group 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., drug detoxification in the liver). Available data (on elevated ALA and FEP levels, inhibited ALA-D, etc.) show clear and significant inhibition in the heme biosynthetic pathway at low blood lead levels, with

statistically significant effects detectable at 10-15 ug/dl. This heme biosynthetic disturbance may result in the impairment of many normal physiological processes and/or the reduced reserve capacity of many cells or organs to deal with other types of stress (e.g., infectious diseases).

The best known effect of lead on erythrocytic pyrimidine metabolism is the pronounced inhibition of PY-5-N activity. This enzyme figures 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). As noted earlier, the disruption of this function by lead has been noted at levels of exposure beginning at 10 ug/dl. At blood lead levels of 30-40 ug/dl, this disturbance is sufficient to materially contribute to red blood cell lysis (destruction) and, possibly, decreased hemoglobin production contributing to anemia (Draft Lead Criteria Document, p 12-27f).

Another 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. Interference with vitamin D production disrupts calcium, zinc, and phosphorous homeostasis, partially resulting in the reduced absorption of these elements from the gastrointestinal 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. There is interference 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 create a feedback response further exacerbating the vitamin D production inhibition, reduced calcium availability, and consequently even greater lead absorption and greater vulnerability to increasingly more severe lead-induced health effects (Draft Lead Criteria Document, p 10-32f). These effects are especially dangerous for young (preschool age) children who are developing rapidly. These children, even in the absence of lead, generally are deficient in calcium 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.

The negative correlation between blood lead and serum 1,25-$(OH)_2D$, the active form of vitamin D, appears to be an 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 10-15 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. This indicated that lead may inhibit renal 1-hydroxylase, the enzyme that converts the precursor to vitamin D. Renal 1-hydroxylase is a mitochondrial enzyme system, which is mediated by the hemoprotein cytochrome P-450. This suggests that the damage to the mitochondrial systems detected at 15 ug/dl has uncompensated consequences.

If cytochrome P-450 is being inhibited at the low levels that the reduced renal 1-hydroxylase activity suggests, we must consider the possibility that other physiological functions related to cytochrome P-450 may also be disrupted. In particular, 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.

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

The elevation of ALA levels is another indication of lead's interference in mitochondrial functioning. In vitro studies have shown that ALA can interfere with several physiological processes involved in the GABA-ergic neurotransmitter system, including a possible role as a GABA-agonist. There appears to be no threshold concentration for ALA at the neuronal synapse below which presynaptic inhibition of GABA release ceases. We do not know at what blood lead level detectable interference with brain

functions by ALA begins in-vivo, nor the level at which the neural interference becomes "critical". However, 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). Lead in the brain is likely to enhance brain ALA concentrations because neurons are rich in mitochondria, the subcellular site of ALA production. Blood ALA elevations begin to be detectable at 15 ug/dl of blood lead. Since ALA is a neurotoxin, the potential implications for brain function are disturbing. The fact that EEG patterns also begin to change at this blood lead level is an additional source of concern.

In addition to the effects of lead on the brain and central nervous sysem, there is evidence that peripheral nerves are affected as well. Silbergeld and Adle (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. The Draft Criteria Document notes:

> ...[lead causes] a blockade of calcium binding to the synaptosomal membrane reducing calcium-dependent choline uptake and subsequent release of acetylcholine from the nerve terminal. Calcium efflux from neurons is mediated by the membrane (Na^+, K^+)-ATPase via an exchange process with sodium. Inhibition of the enzyme by lead, as also occurs with the erythroctye...., increases the concentration of calcium within nerve endings (Goddard and Robinson, 1976). As seen from the data of Pounds et al. (1982a), lead can also elicit retention of calcium in neural cells by easy entry into the cell and by directly affecting the deep calcium compartment within the cell, of which the mitochondrion is a major component. (Section 12.2.3)

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 conduction velocity in children in a smelter community. This effect may indicate advancing peripheral neuropathy.

VI.B. Hematological Effects of Lead

High levels of blood lead are known to produce anemia. Previously it was an unresolved question whether blood lead below 30 ug/dl increased the risk of anemia in children. We addressed this question in two ways. First, we examined the relationship between blood lead levels and various measures of anemia, and the inhibition of heme synthesis as evidenced by elevated free erythrocyte protoporphyrin (FEP) levels. Second, because FEP is a more stable indicator of a person's lead exposure over several months than a single blood lead determination, we also analyzed the relationship between elevated FEP levels and anemia. We found that blood lead and FEP levels were associated with increased risks of anemia in children, even below 30 ug/dl of blood lead.

For this analysis we again used data from the NHANES II survey. Among the hematological information collected was 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.

VI.B.1. Effects 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 those currently considered to be safe.

Linear regressions were performed of MCV and MCH on blood lead levels in children, controlling for race, age, income, and iron status (i.e., the level of iron in their blood). 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. 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.

The regressions for both MCV and MCH found blood lead to be a significant explanatory variable ($p < .0001$ and .0033, respectively) for the decreases in each.

Because small decreases in MCV and MCH are of unknown significance, we also analyzed the probability of children having abnormally low MCV or 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 both in mean cell volume being low (MCV < 80 femptoliters [fl], $p < .0001$), and in mean

cell volume being seriously low (MCV < 74 fl, $p < .0001$). Blood lead levels were also significantly associated ($p < .023$) with the percent of children having MCH less than 25 pico grams (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 abnormal MCV using only those children whose blood lead levels were less than 25 ug/dl. The regression coefficient for blood lead was unchanged and significant ($p < .014$). Thus, blood lead levels under 25 ug/dl were associated with increased risks of microcytic anemia.

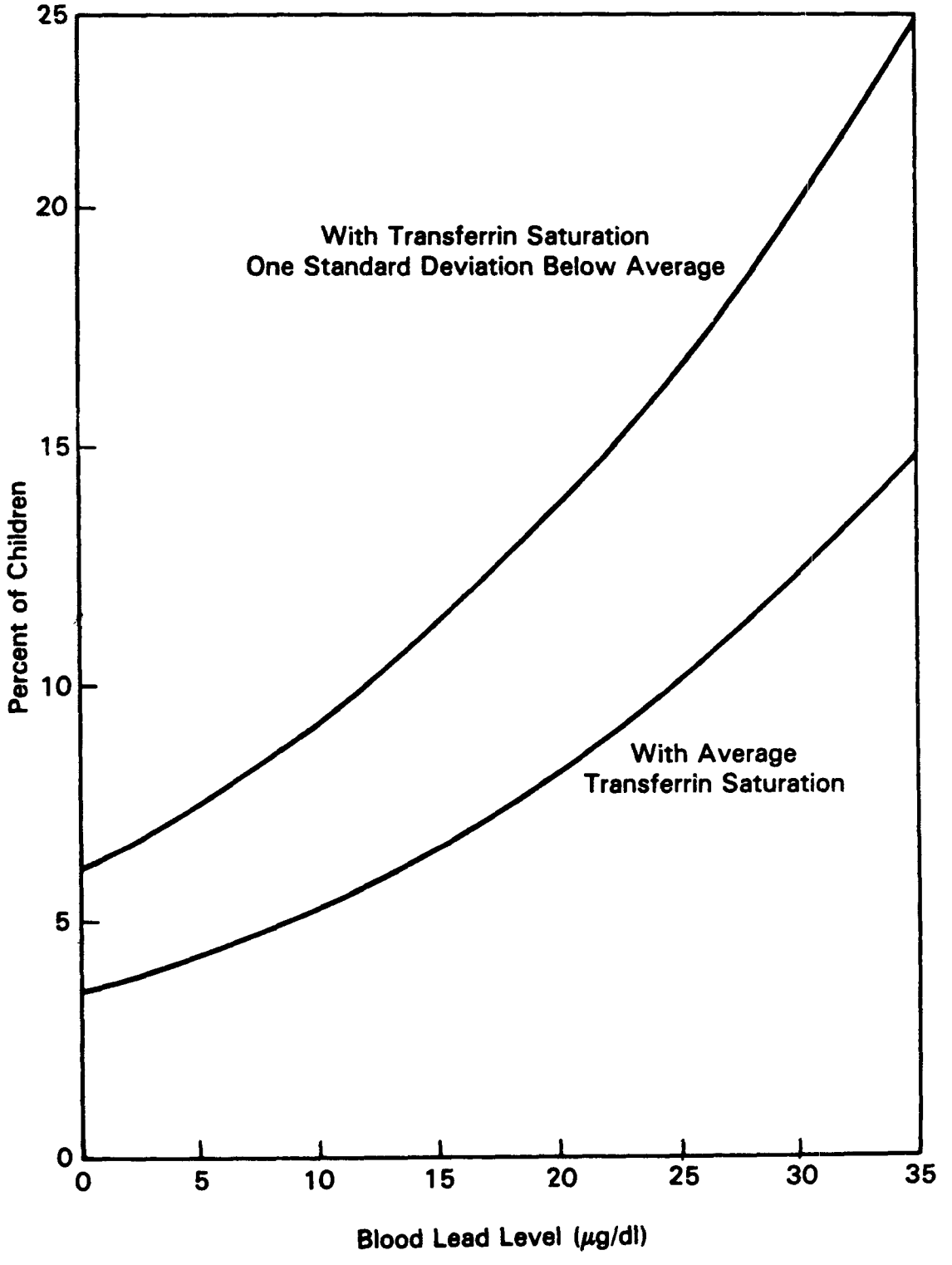
To further investigate the relationship between lead and abnormal hematological variables, we used our regression to predict the percentage of children with MCV < 74 fl as a function of blood lead for two cases: children with average transferrin saturation levels (22.4% saturated for children in the NHANES II survey) and children with transferrin saturation levels one standard deviation below average (13.6%). The results are shown in Figure VI-1. Note that at 25 ug/dl of blood lead almost 10% of the children with average iron levels and 17% 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% 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%). Since logistic regressions

Figure VI-1

PERCENT OF CHILDREN WITH MCV BELOW 74

(Age 6 Months to 8 Years)



gave the same results when we used only children with blood lead levels under 25 ug/dl, and since the 95% 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.

VI.B.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 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. (The authors have not yet published these findings.) 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; Roels et al., 1976). To test this relationship, we tested several alternative specifications. We considered a linear model, we examined 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 (Transferrin saturation)^B, and a model where FEP was proportional to

(Blood lead)^{B1} and (Transferrin saturation)^{B2}. The model that that fit best was exp(Blood lead) times (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 the relationship: FEP = 36.73 (Transferrin saturation) $^{-0.11684}$ exp(0.01183 Blood lead).*

While others have found sex differences in the response of FEP to blood lead, sex was not a significant variable in any of our models for children. This was probably a result of the fact that the sex difference in the response of FEP to blood lead is smaller in children. We also suspect that the differences in adults are due predominantly to sex differences in iron status, which we controlled for directly.

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, and obtaining a better fit with blood lead. The 50 ug/dl FEP level is considered to indicate severe enough interference with heme processes that medical attention is usually required even when not coupled with elevated blood lead levels.

Again, we checked to see whether the relationship between the risk of elevated FEP and blood lead held at lower blood lead levels,

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* Transferrin saturation is expressed in tenths of a percent.

repeating the regression only for children with blood lead levels under 30 ug/dl. Using maximum likelihood analysis, blood lead was again extremely significant ($p < .0001$). The coefficient of blood lead for the low group was .178 $\pm$ .04 compared to .175 $\pm$ .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 well below the currently defined safety level.

Piomelli and coworkers' studies have suggested a threshold for lead-induced increases in FEP levels of about 15 ug/dl. Taking 17.5 ug/dl of blood lead as our reference level, our regression predicted that the relative risk of FEP levels over 50 ug/dl was 1.55 (1.42-1.70 at 95%) at 20 ug/dl of blood lead, and was 3.73 (2.55-4.89 at 95%) at 25 ug/dl of blood lead. This was true across all transferrin saturation levels.

VI.B.3. The Relationship Between FEP Levels and Anemia

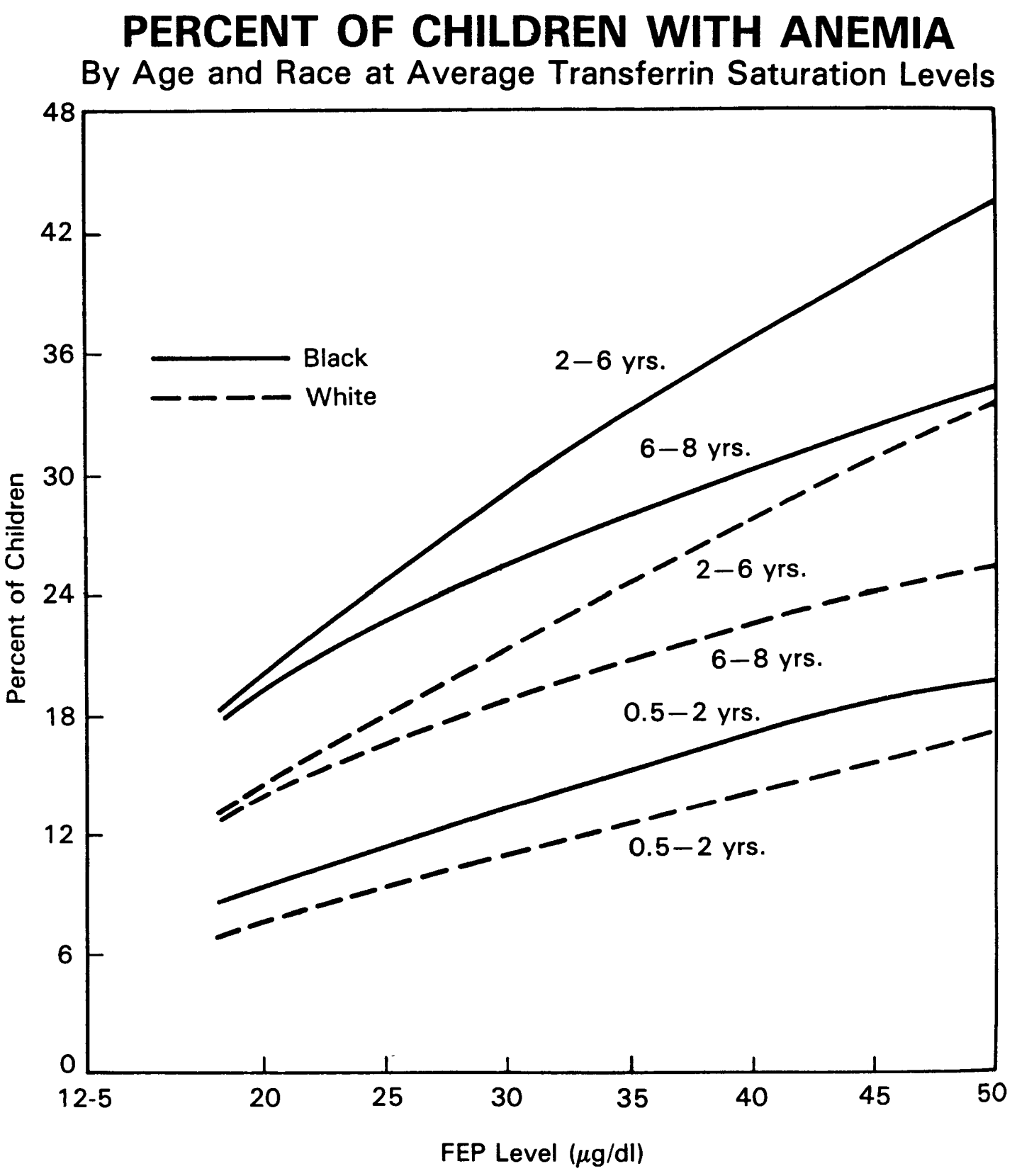
Since the average lifetime of erythrocytes is approximately 120 days, a single blood lead level measured concurrently with hematocrit levels, MCV, and MCH cannot adequately evaluate the role of lead in the impairment of red cell production. Such a single measurement is a poor proxy for the blood lead levels over the previous 120 days, as these levels may not have been constant. By contrast, FEP, once created, remains in red cells for their lifetime. While FEP levels are affected by iron status as well as blood lead, using iron status as an independent variable along with FEP restricts FEP to principally being a surrogate for

average blood lead levels when studying its association with anemia. Because FEP levels are exponentially associated with blood levels, log(FEP) was used as a proxy for lead exposure over the relevant period.

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 ($p < .0001$ 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 ($p < .0001$) of the probability of abnormally low levels of all three indicators. Again, we repeated our regressions using only children with FEP values of less than 33 ug/dl, and FEP was still very significant ($p < .0001$). 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.

FEP levels of less than 33 ug/dl are generally associated with blood lead levels under 30 ug/dl. Figure VI-2 shows the regression's prediction of the percent of children with anemia as a function of FEP levels at <u>normal</u> 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

Figure VI-2


PERCENT OF CHILDREN WITH ANEMIA
By Age and Race at Average Transferrin Saturation Levels
Percent of Children
48
42
36
30
24
18
12
6
0
Black
White
2—6 yrs.
6—8 yrs.
2—6 yrs.
6—8 yrs.
0.5—2 yrs.
0.5—2 yrs.
12-5
20
25
30
35
40
45
50
FEP Level (μg/dl)

hematocrit levels of less than 33% for ages 0.5-2, less than 34% for ages 2-6, and less than 35% for ages 6-8 -- the minimum normal range levels recommended by the Journal of Pediatrics (1977). These definitions are supported by the work of Yip et al. (1981).

Figure VI-2 shows that as FEP levels increase from 20 ug/dl to 50 ug/dl, an additional 20% 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 ud/dl. Since elevated FEP is a symptom of 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 VI-2 were for normal iron levels, the anemia appeared to be the outcome of the lead exposure underlying the FEP values.

In summary, blood lead levels below the currently defined "undue lead exposure" range of 30 ug/dl (and, indeed, even below 25 ug/dl) seem to be associated with increased incidence of anemia in children and increased interference with heme synthesis producing elevated levels of free erythrocyte protoporphyrin. This suggests that both the levels of blood lead and FEP used in the current Centers for Disease Control definition of undue lead exposure may be inadequate to protect children from the risk of anemia. In addition, the reduced mean cell volumes and the lower hematocrits again indicate that lead's effect on heme synthesis has uncompensated effects at levels below 30 ug/dl. This further

strengthens the case for considering elevated FEP levels, which mark lead's interference with normal body activity, as a patho-physiological effect.

VI.C. Fetal Effects

A growing concern in the public health community is that the most sensitive population for lead exposure is not children, but fetuses and newborn infants. This concern is supported by both animal studies and, recently, human data.

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 post-natal 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 (3-4 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, 1977; Bull et al., 1979) have shown that the prenatal exposure of rats to 0.2% lead chloride in the mother's drinking water markedly reduced the cytochrome C content in the cerebral cortex, and possibly produced 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).

Human data are scarcer. Needleman et al. (1984) have analyzed data from over 4,000 live births at Boston Women's Hospital and found an association between some congenital anomalies and umbilical cord blood lead levels. Holding other covariates constant, the relative risk of a child's demonstrating a minor malformation at birth increased by 50% as lead levels increased from 0.7 ug/dl to 6.3 ug/dl (the mean cord lead level). This increased an additional 50% 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 preliminary analysis by Needleman 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 cord lead levels in this study were very low.

Finally Erickson et al. (1983) found lung and bone lead levels in children who died from Sudden Infant Death Syndrome were statistically significantly higher than in children who died of other causes, after controlling for age.

VI.D. Neurological Effects

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 documented.

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 (Draft Lead Criteria Document, Section 12.3.4). 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 adenosine triphosphate (ATP) energy for the entire cell. Given the high energy demands of neurons, selective damage to the nervous system seems plausible.

Paralleling these cellular or biochemical effects were electrophysiological changes indicating the perturbation of peripheral and central nervous system functioning observed in children with blood lead levels of approximately 15 ug/dl (Otto et al., 1981, 1982; Benignus et al., 1981). These included slowed nerve conduction velocities, as well as persistent abnormal EEG patterns. Aberrant learning behavior has been

noted in rats with blood lead levels below 30 ug/dl (Draft Criteria Document, Section 12.4.3.1.3). This behavior evidenced both reduced performance on complex learning problems and signs of hyperactivity and excessive response to negative feedbacks (Winneke, 1977, 1982).

Finally, the cognitive effects of lead in children showed 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 a 4-5 point difference in mean IQ and reduced attention spans. Several studies discussed in more detail later in this chapter have found smaller effects at lower blood lead levels. The precise biological mechanisms connected with these effects are not yet clearly defined.

While some of these effects have only been observed at higher blood lead levels, in animals, or <u>in vitro</u>, they all showed 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 those larger scale studies with what has been discovered on the molecular and cellular levels.

VI.D.1. Cognitive and Behavioral Effects

Many studies have noted neurological effects in children with elevated blood lead levels. A brief discussion of these is presented in Section V.B. of this paper, concentrating on those examining the effects of blood lead levels above 30 ug/dl. In this section, we will examine the effects below 30 ug/dl.

VI.D.1.a. Assessment of the Relationship Between IQ or Cognitive Function and Low Blood Lead Levels

The answer to the question of whether the relationship between blood lead and cognitive performance extends to levels below 30 ug/dl is tremendously important. If IQ is affected at blood lead levels below 30 ug/dl, the benefit of reducing lead emissions is very large because of the many children who would be at risk.

The literature on cognitive effects at low lead levels is extensive. However, most of the studies have methodological flaws of varying importance and few display indisputable results concerning the relationship between IQ effects and changes in low lead levels. The Draft Lead Criteria Document 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.

The summary table in Chapter 12 of the Criteria Document (pp 55-58) indicated that virtually all of the 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%, 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.) However, because the reported sample sizes were small, it was not likely that small effects would have been detected. The consistent pattern in all the studies of high lead groups doing less well indicated that the combined evidence of a significant effect was stronger than the evaluations of the individual studies suggested.

In developing a better test for the existence of a specific effect, we limited the studies we examined for two reasons. First, because we were interested in low level exposure effects in the whole population, we used only the six general population studies. The smelter study by Winneke et al. (1982) was also included, as blood lead levels appeared to be in the same range as the general population studies. Second, because we were interested in general effects, we chose to look 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.

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

observed 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. Unfortunately, as indicated above, they were not reported where they were larger than 0.05, so we had to calculate several p-values from the data presented.

For each study we used the standard deviation of the IQ measure to compute the standard deviation for the difference in the mean IQs across groups. From the ratio of the IQ difference to this standard deviation, we could compute a p-value. We could directly apply this method to the study by Smith et al. (1983). In this study, one of the best methodologically, all of the IQ effects were reported as "not significant". However, when we computed the p-values, we found that the p-value was 0.051 when comparing high and low lead groups for the Full Scale IQ.* Similar computations for the Verbal and Performance IQs produced p-values of 0.068 and 0.105, respectively.**

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* W. Yule, in a personal communication at the the International Conference on Heavy Metals in the Environment (Heidelberg, September 1983), said that a recomputation paying more attention to round-off and computational errors found a one-tailed p-value of less than 0.05.

**The mean IQs for the low and high groups given in Smith et al. (1983) were quoted with 95% confidence intervals. For the sample size (145, 155) for these groups we can assume normality. [The sample size is taken from Table 13 of Smith et al. (1983).] Thus, for the low group, 2.0 IQ points = $-1.96 s_1$, and, for the high group, 1.9 IQ points = $1.96 s_2$, where s_1 and s_2 were the standard deviations for the low and the high groups, respectively. This implied values for s_1 of 150.98 and s_2 of 145.66. Combining these variances yielded an overall variance of 148.23. Weighting this by the sum of the inverses of the sample sizes gave the variance for the difference of the means, which was 1.978. Taking the square root of these yielded a standard deviation of 1.407. Dividing the difference between the high and low group (2.3) by 1.407 produced a normal statistic of 1.635, which has an associated p-value of 0.051 (Bryant, 1966).

For other studies, where we could not determine the standard deviation for the test procedure, we assumed it was equal to 15. This is the commonly cited standard deviation for IQ, although it varies slightly from test to test. Because this standard deviation was somewhat higher than the standard deviations in the studies that reported such values (the study groups were more homogeneous than the general population), our calculations probably produced p-values larger than the true p-values.

We used these p-values and the Fisher procedure to compute a joint probability for the observed results, presented in Table VI-2. The resulting probability of 0.014 indicates that it was very unlikely that we could get the observed pattern of results if there were really no effect. The overwhelming preponderance of the data (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 at least one case (Smith et al., 1983), 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 remaining IQ effects were regressed on lead in the next step. Many of these covariates (e.g., parental care, income, IQ) negatively correlate with lead exposure, and this procedure attributed all of the joint variation to the nonlead variable.

TABLE VI-2

Computation of Joint P-Value from Epidemiological Studies of Cognitive Effects from Low Level Lead Exposures in Children

Study[a]	Sample Sizes		Internal Lead Levels: Blood (ug/dl)		Internal Lead Levels: Teeth (ppm)		IQ Difference	P-Value	-2 ln p
	Control	Exposed	Control	Exposed	Control	Exposed			
McBride et al. (1982)	100	100	0.5-9	19-30	------	------	1[b]	.32	2.28
Yule et al. (1981)	20	21	7-10	17-32	------	------	7.6[c]	.029	7.08
Smith et al. (1983)	145	155	------	------	< 2.5	8	2.2[c]	.051	5.95
Yule and Lansdown (1983)	80	82	7-12	13-24	------	------	2[c]	.22	3.03
Harvey et al. (1983)	Total of 189		N/A	N/A	N/A	N/A	.7[d]	.34	2.15
Winneke et al. (1982a)	26	26	------	------	2.4	7	5[c]	.10	4.61
									25.10

Joint P-Value for Studies: $P(X^2_{12} > 25.10) = .014$

Needleman et al. (1979)	100	58			< 10	< 20	4[c]	.03	7.01
									32.11

Joint p-value including Needleman $P(X^2_{14} > 32.02) = .004$

[a] Citations refer to Draft Lead Criteria Document, October 1983
[b] Peabody Picture Vocabulary IQ Test
[c] Welchsler Intelligence Scale for Children-Revised
[d] British Ability Scales

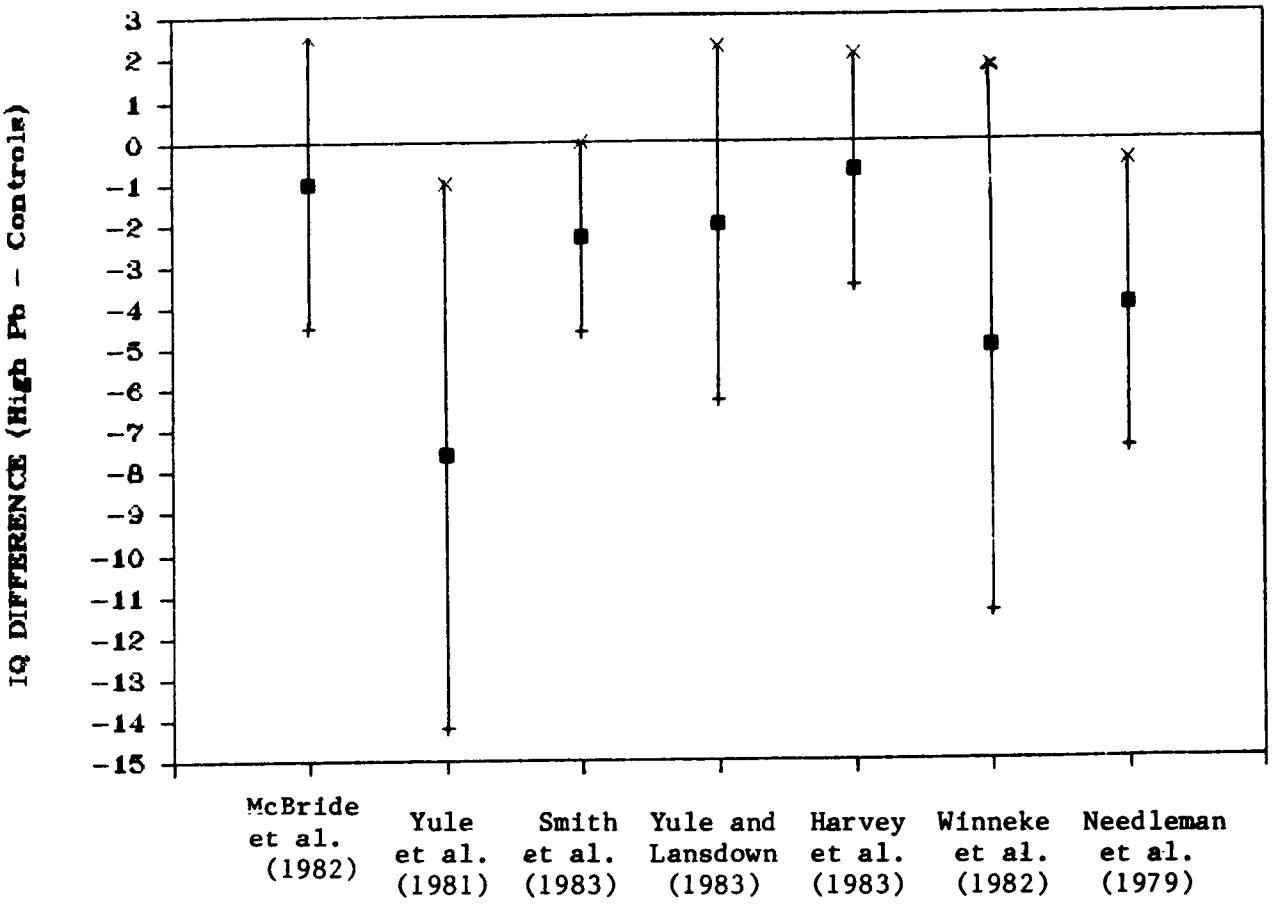
Another study (Harvey et al., 1983) had IQ measurements on 131 children but only 71 degrees of freedom in the t-test for lead. If this study included 59 covariates in the analysis, such an over specification clearly would bias downward the significance of lead as well.

We have treated one major study (Needleman et al., 1979) differently because a recent critique raised questions about the appropriateness of the p-value reported in the study (Draft Criteria Document, Appendix 12C). Pending the resolution of that issue, we have presented our results both with and without this study. Even when it was omitted, the p-value (.014) was clearly significant. Recent reanalysis by Needleman using the alternative specifications for his model suggested by the review committee still found a significant lead effect. Including this study would lower the joint p-value to .004.

Figure VI-3 presents an alternative method to evaluate the results from these studies. For each study the figure shows the 90% confidence interval for the full scale IQ difference between the high and low lead groups. The mean is represented by the square, the upper limit by an X, and the lower limit by a +. For two of the studies the confidence interval does not include zero, which is computationally equivalent to finding that a one-tailed hypothesis test would reject a null hypothesis of no effect at the 5% level. However, as the figure shows, <u>all</u> of the studies found that the high lead group had a lower mean IQ, and that the

FIGURE VI-3

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



■ = Mean

X= Upper Confidence Limit

+= Lower Confidence Limit

range of effects consistent with all of them was a loss of one to three and a half IQ points.

Therefore, we accepted the implication of the joint probability computation that there was an association between cognitive deficits and differences in lead exposure even at low levels. Ignoring such a risk without considering the potential cost of the error associated with that risk would have been inappropriate in determining the desirability of implementing a policy.

VI.D.1.b. Policy Implications of Significance Tests

In making policy decisions, we should be concerned with the cost of not implementing an appropriate policy (false negative) as well as with the cost of implementing an inappropriate policy. Policy makers must balance the risks of each type of error, weighted by its costs. Because of this, even if the p-value for the joint test had been slightly larger than the arbitrary 0.05 level,* we still would have considered the cognitive effect. For example, for the Smith et al. study, we computed that the probability of falsely asserting the null hypothesis was true

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* Over the years the significance level of 0.05 has become the basis for rejecting the scientific ("null") hypothesis. While adherence to the strict definition of "statistical significance" has been important in science, it must be remembered that this p-value is arbitrary and may not be the appropriate sole criterion for regulatory decision making.

was .587.* This false negative rate would be even higher in the other studies considered here owing to their smaller sample sizes.

The cost of not avoiding small cognitive effects for millions of children is high. If insisting on a p-value of less than 0.05 before accepting that a cognitive effect exists means a substantial risk of a false negative, then the potential cost of the wrong decision may be too large. In this case, given the relatively small sample sizes, the small cognitive deficits one might expect, and the standard deviation of the test procedure, the risk of a false negative is high. We believe that this association, coupled with the biochemical studies, animal studies, and high level effects discussed in the introduction to VI.D. suggests a causal relationship.

The existence of a large chance of a false negative for outcomes where costs are potentially high suggested the need for carefully considering the entire process by which the validity of the hypothesis was evaluated. In particular, where the choice of a null hypothesis gives credence to one point of view, which is not justified given the power of the test, a hypothesis test may not be appropriate. An alternative method is to look at confidence intervals around the estimated parameter

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* We computed the false positive from the Smith et al. data as follows. To estimate the p-value, we derived a standard deviation for the difference of the high and low lead groups of 1.407. At a 5% chance of rejecting the null hypothesis when it was true, the normal one-tailed statistic was 1.65. Therefore, we would reject the null hypothesis only for differences greater than (1.407) (1.65) = 2.32. If the difference in the groups were two IQ points, the probability of the difference being below 2.32 is given by $p\ (z < [2.32-2]/\ 1.407) = .587$.

values. As an example, consider the confidence intervals in Figure VI-3, which shows graphically that elevated lead levels have a negative effect on IQ.

This conclusion, plus the above joint probability estimate of p = 0.014 for the general population studies, led us to accept the existence of cognitive effects of low level lead exposure.

VI.D.2. Estimating Avoided IQ Loss Associated with Reduced Blood Lead Levels

We used two hypotheses to evaluate the extent of IQ loss. Both were based on the Smith et al. study which used tooth lead as the measure of lead intoxication, where particular attention was paid to measuring and controlling for covariates. Their "high lead group" had teeth with lead levels of 8.0 ug/g or more, a relatively low cutoff level.

Our first hypothesis, assuming a step function with a threshold, was that a group of children whose teeth lead levels were above 8 ug/g would have an average IQ 2.3 points lower than the average IQ of children in the control group, whose lead exposure resulted in tooth lead levels below 2.5 ug/g.

To convert tooth lead to blood lead, we used three methods. First, we followed Steenhout and Pourtois (1981) and Steenhout (1982), who used regression analysis to estimate the increase in tooth lead (t) concentration that would result from various blood lead (BL) levels over time. Her model was:

$$\text{Tooth Lead}(t') = \int_{t_0}^{t_1} q(t)BL(t)dt.$$

For adults, q = 0.045, a constant, and the model reduced to:

$$\text{Tooth lead}(t') = q\ \overline{BL}\ \Delta t.$$

At the International Conference on Heavy Metals and the Environment (September 1983), held in Heidleberg, West Germany, Steenhout presented additional results. For children, the rate of tooth lead accumulation per unit of blood lead was much higher than for adults and appeared to decline exponentially to the adult level with age. Steenhout's best fit of the data was:

$$\text{Tooth lead}(t') = \int_{t_0}^{t_1} [0.045 + 0.2\ \exp(-t/4.5)\text{ppm}]BL(t)dt$$

where t was measured from Steenhout's "midgrowth stage". Replacing BL(t) by $\overline{BL}$, we could solve for $\overline{BL}$. This analysis obtained a $\overline{BL}$ of 5.0 ug/dl or less for Smith's low exposure group, and 16 ug/dl or more for her high group.

Second, we used Winneke's data (Winneke, 1979) which showed mean blood lead levels equaled 2.5 times mean tooth lead levels. This gave blood lead levels of 6.25 ug/dl for the control group and 18 ug/dl for the high group, which was consistent with Steenhout's results.

Finally, we examined Smith's data on blood lead levels for a non-random sample of her survey population. These showed blood lead levels of 11.5 ug/dl for 20 low lead children and 15.1 ug/dl for her high group. While this yielded about the same results for the high group, it showed much higher levels for the control group. We are not sure what caused this discrepancy, although the number

of low lead children was very small. In any case, the three different procedures for imputing blood lead suggested a threshold for IQ loss at about 15 ug/dl.

The second hypothesis assumed that, instead of a step function with a threshold occurring at 15 ug/dl of blood lead, there was a linear function relating IQ loss to blood lead level. For our estimate of the effect, we also used the Smith et al. study and assumed the low tooth lead group had an average blood lead level of 3 ug/dl and the high group had an average blood lead level of 18 ug/dl. We used the estimated blood lead levels based on Steenhout's procedure and divided the difference in IQ by the difference in blood lead to yield a slope of 0.15 IQ/ug/dl of blood lead. Other studies, such as the 1981 study by Yule et al., had coefficients as high as 0.7 IQ points per ug/dl of blood lead. Using Smith's limited blood lead data would suggest a slope of 0.64. To be conservative, we have used the 0.15 slope.

We computed the total lost IQ points for several hypotheses, but did not attach monetary values to the lost IQ.

VI.D.3. Thresholds for Effects of Blood Lead on IQ and the Size of the Affected Population

In assessing the size of the population at risk, two alternative hypotheses were again possible. The first was a no threshold model. It assumed that the effect of lead on IQ was a continuous function, with increasing risk and effect as blood

lead levels rose. Under this assumption, adverse effects on either IQ or behavioral patterns, such as disruptive behavior or shortened attention span, occurred at lower lead levels and increased at higher lead levels, and despite individual differences, the extent of effect was related to the extent of exposure.

Alternatively, many people believe that cognitive deficits from lead exposure occur only above a specific threshold, i.e., that blood lead levels below some value will not affect either intelligence or behavior patterns that may reduce educational attainment. Several alternative threshold values are possible.

Because there is little dispute concerning cognitive effects above 30 ug/dl, selecting that blood lead level was one option. On the other hand, ALA levels are elevated at 15 ug/dl and EEG patterns also show persistent changes at that level. This evidence suggested that blood lead levels of 15 ug/dl may be a threshold. This is buttressed by the Smith et al. study, where we have determined that children whose exposure averaged above 16 ug/dl had lower IQ levels than children whose exposure averaged below 5 ug/dl. While the cognitive damage may have occurred at earlier ages when blood lead levels were higher, the work of Harvey, who surveyed two year old children in Birmingham, England, indicated that blood lead levels among two-year-olds averaged 15.6 ug/dl. This was only slightly higher than the average among Smith's older children. Furthermore, the study by Yule et al. (1981) indicated that children with blood leads of 7-10 ug/dl had higher IQs than those with blood leads of 17-32 ug/dl. Yule and

Landsdown (1983) can be taken as supporting this level or even indicating that the threshold may be somewhat lower.

Alternatively, the study by McBride et al. (1982) showed a small difference between children above 19 ug/dl and those below 10. This data suggested that the threshold may be around 20 ug/dl.

Because all three thresholds (15, 20, and 30 ug/dl) were possible, we have calculated the number of children potentially at risk in 1988 for each of the three. These estimates are shown in Table VI-3.

TABLE VI-3

Decrease in Number of Children in 1988
Above Thresholds for Cognitive Effects

Possible Threshold	Low-Lead Option	All Unleaded
15 ug/dl	1,475,000	1,552,000
20 ug/dl	476,000	500,000
30 ug/dl	43,000	45,000

As noted, accepting the hypothesis of a cognitive effect at a given threshold does not imply that all the children above the threshold are affected or that all below the threshold are free of the effect.

In addition to computing the number of children at risk, we estimated the total effect on intelligence in 1988, expressed as the number of children at risk times the mean change in IQ. Our estimate of the change in IQ was 2.2 IQ points.* We then computed

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* The 2.2 IQ figure is the difference between the average IQ of the Smith et al. middle group of children and the average IQ of that study's high lead group. We had found the average blood lead level of the children below 15 ug/dl was nearer that of the children in the Smith et al. middle group.

the change in person-IQ points (i.e., the number of people at risk times the average 2.2 IQ points lost) as a result of the policy options. For simplicity, we used the same 2.2 IQ point decrement for the other two thresholds. The results are shown on Table VI-4.

Finally, assuming there is no threshold, we converted the changes in mean blood lead levels to changes in IQ using an estimate of the rate of change of IQ per ug/dl. We assumed that the mean of the IQ change for any child was dependent on the mean change in blood lead levels. (As shown in Table VI-1, black and white children had different blood lead levels and this was considered in our calculation.) The estimated changes in IQ points in 1988 for children aged 6 months to 7 years are shown in Table VI-4. These were computed using the coefficient of 0.15 IQ/ug/dl derived earlier from the Smith et al. study, from changes in mean blood lead levels given in Table V-5, and from population figures in Table VI-6 for children aged 0 to 7.

TABLE VI-4

Possible Change in Person-IQ Points in 1988 as a Function of Threshold Levels for Children 6 Months to 7 Years

Threshold	Low-Lead	All Unleaded
15 ug/dl	2,867,000	3,018,000
20 ug/dl	986,000	1,035,000
30 ug/dl	92,000	97,000
No threshold	7,913,000	8,728,000

Because there are so many children at risk, any reasonable monetary value ascribed to avoiding the loss of one person's IQ

point would produce very large benefits for these changes in person-IQ points. For example, if parents were willing to pay $100 per IQ point to remove the possibility of such a loss, we would have estimated benefits for the all unleaded case ranging from $9.2 to $302 million for the thresholds listed above and up to $873 million if there were no threshold. Thus, even if only small changes in IQ are found to be associated with lead exposure, the large number of children affected would make the benefits of avoiding such effects extremely large.

VI.E. Estimating the Reduction in the Number of Children at Risk

Reducing or eliminating leaded gasoline will reduce the number of children at risk for the pathophysiological effects from elevated blood lead levels. Table VI-5 presents the decrease in the number of children above the "minimum observed effect level," or "apparent thresholds," for various health effects in 1988. In many cases, these apparent thresholds reflect the limitations of current experimental measurement techniques and not a finding that no effect exists at lower levels. Therefore, our estimates are likely to be conservative. Our estimates of the decreased number of children with abnormal physiological functioning are based on statistical methods described in section V.E.

TABLE VI-5

Decreased Number of Children (under 14 years old) Above Apparent Threshold Levels in 1988

Medical Effect	Apparent Threshold	Low-Lead	All Unleaded
Inhibition of PY-5-N	10 ug/dl	4,257,000	4,486,000
Inhibition of ALA-D	10 ug/dl		
Inhibition of vitamin D	10-15 ug/dl		
Elevated ZPP	15 ug/dl		
EEG changes	15 ug/dl	1,475,000	1,553,000
Elevated ALA levels	15 ug/dl		
Inhibition of globin synthesis	20 ug/dl	476,000	500,000

Even if we take the thresholds in Table VI-5 as true thresholds, it is very unlikely that all individuals with blood lead concentrations above a given threshold will suffer a particular effect, and it is unlikely that all those below the threshold are free from the effect. The specific blood lead level at which a particular effect begins to occur varies from person to person. In the general population, such variation generally produces an S-shaped curve of the percent of people with the effect as a function of blood lead level or other exposure index. In Table VI-5 we approximated the dose-response curve with a step function instead of a continuous curve; the numbers, therefore, only roughly estimate the true values.

We also used regressions to predict the distribution of blood lead levels in 1988. These values are given in Table VI-6. (Details of how these numbers were calculated are contained in Section E of Chapter V.)

TABLE VI-6

Estimated Distribution of Blood Lead Levels in 1988
(in thousands of children aged 13 and under)

Black

Blood lead	<10ug/dl	10-15 ug/dl	15-20 ug/dl	20-30 ug/dl	>30ug/dl
Base Case	3,386	2,588	1,191	490	36
Low-Lead	4,496	2,131	790	267	17
All Unleaded	4,559	2,096	766	256	16

Non-Black

Blood lead	<10ug/dl	10-15 ug/dl	15-20 ug/dl	20-30 ug/dl	>30ug/dl
Base Case	34,608	4,326	1,085	397	52
Low-Lead	37,764	2,001	486	186	29
All Unleaded	37,921	1,884	458	177	28

VI.E.1. Distributional Aspects of Lead Exposure

One feature often overlooked in analyzing the pathophysiological changes induced by lead is the close correlation between the occurrence of high lead levels and high levels of other stressors, which, like lead, both have direct adverse effects and reduce the reserve capacity of the body to deal with environmental insults. When two or more stressors act in concert, the severity of the adverse impacts increases and makes it much more likely that the reduced reserve capacity produced by lead will, in fact, produce adverse consequences.

People who have the highest blood lead levels tend to be children, in general; black children, in particular; and poor people. Children are often deficient in iron and calcium, the adverse effects of which are exacerbated by lead. Children's nervous systems are more sensitive to toxins, and they are just beginning their cognitive development. Blacks tend to have higher hypertension rates, which may also be associated with or exacerbated by lead (Beevers et al., 1976). Blacks also tend to have lower vitamin D levels which are further reduced by lead, and tend to be poor. Poor people usually have a lower level of vaccination, well baby care, and preventive medicine in general. Poor people are more likely to be sick and/or malnourished, have inadequate medical care, and be under greater stress, both physical (e.g., poor heating and sanitation) and psychological.

Poor people, on average, are less successful in school so even marginal central nervous system or cognitive effects of lead may have more serious implications for this group. Many of the people at high risk of lead exposure have a high risk of experiencing these other factors. For them lead effects that would be sub-clinical in the absence of these other factors may not be sub-clinical.

VI.F. Conclusion

We examined several different ways to value the benefits in 1988 of reduced lead exposure through reduced use of lead in gasoline. In Table VI-7 we present a summary of the estimated

benefits for children under age fourteen of reducing the adverse effects resulting from exposure to lead from gasoline.

TABLE VI-7

Summary of the 1988 Health Benefits of Reducing Low Level Lead Exposure

	Low-Lead	All Unleaded
Reduction in number of children (under 14 years of age) at risk of:		
At 10 ug/dl	4,257,000	4,486,000
Inhibition of PY-5-N Inhibition of ALA-D		
At 15 ug/dl	1,475,000	1,553,000
Inhibition of vitamin D Elevated ZPP EEG changes Elevated ALA levels		
At 20 ug/dl		
Inhibition of globin synthesis	476,000	500,000
Average loss of 2.2 IQ points	43,000 to 1,475,000	45,000 to 1,552,000
Percent change in children's mean blood lead levels:		
Whites	24%	27%
Blacks	12%	13%

The size of the populations potentially at risk for the low level effects preceding overt manifestations of clinical symptoms of lead poisoning is large. Although we have not attached any dollar values, the changes that would occur under our two policy

options suggest that reducing the pathophysiological effects of lead exposure would be a significant public health benefit of reducing lead in gasoline.

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