

THE ECONOMIC BENEFITS OF CONTROLLING VOLATILE ORGANIC
COMPOUNDS AND NITROGEN OXIDES: A GENERIC APPROACH

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TABLE OF CONTENTS

Introduction	Chapter 1.
The Benefits of a One Percent Reduction in Ozone	Chapter 2.
Linking NO _x and VOCs to Ozone	Chapter 3.
Evaluating the Benefits of VOC Control	Chapter 4.
Direct Benefits of NO _x Control	Chapter 5.
Conclusions	Chapter 6.

Chapter I

INTRODUCTION

In developing environmental regulations, EPA routinely estimates the costs of alternative levels of pollution control. It is difficult, however, for decision makers to interpret these marginal cost estimates without information on the benefits, either relative or absolute, of controlling different types of emissions from various source categories. For example, for every New Source Performance Standard that is promulgated, EPA must select from a number of control options, each with a different cost to society. Information on the economic benefits of each option could be useful in making these selections.

Although detailed benefit analyses for each regulatory decision are impracticable, we have developed rough estimates of the health and welfare benefits per controlled ton in 1984 of nitrogen oxides (NO_x) and volatile organic compounds (VOCs). These estimates could provide the starting point for performing more detailed analyses. For example, this paper estimates the benefits per ton of NO_x and VOC controlled for both rural and metropolitan areas. Even more detail is possible if the location of relevant sources is known.

These estimates are necessarily based on some crude assumptions. We have initiated several projects that

will allow us to relax some of these assumptions in the future. As these new results become available, we will incorporate them into revisions of this summary report. For now, three caveats are particularly important: (1) the analysis only partly reflects the specific geographical distribution of any regulation in question, (2) it does not reflect how relative benefits may change over time, and (3) some of the health and welfare benefits that may be generated from controlling VOCs and NO_x are not quantified.

Both VOCs and NO_x affect the formation of ozone which, in turn, damages vegetation, materials, and human health. Therefore, before discussing the direct effects of NO_x and VOCs, we first present the health and welfare benefits of a one percent reduction in ozone for rural and metropolitan areas. Next, in Chapter 3 we examine the relationship between VOCs, NO_x, and ozone, and convert the ozone benefits to the desired form of benefits per ton of reduced VOCs and NO_x. Chapters 4 and 5 present estimates of the direct effects from VOCs and NO_x respectively. Finally, Chapter 6 summarizes our finding.

EPA's Office of Research and Development is updating the criteria document for ozone. Nothing in this paper is intended to prejudge or supersede the outcome of that process. In addition, several of the studies relied upon are EPA contractor draft reports that may have not undergone full peer review.

Chapter 2

THE BENEFITS OF A ONE PERCENT REDUCTION IN OZONE

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

The effects of ozone on human health, vegetation, materials, and ecosystems were summarized in the EPA Air Quality Criteria for Ozone and Other Photochemical Oxidants (U.S. EPA, 1978). In addition, our estimates rely on the considerable amount of research that has become available since that document was finished. As part of EPA's periodic review of the ozone National Ambient Air Quality Standard, the Office of Research and Development currently is updating the Criteria Document. Nothing in this report is intended to prejudge or supercede the outcome of that process. In the sections that follow, we estimate the economic benefits of reduced ozone resulting from improvements in health, and reductions in agricultural crop loss, nonagricultural vegetation, and material damage.

2.1 HEALTH EFFECTS OF REDUCING OZONE

2.1.1 General Review

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).

Uncertainty remains about whether a threshold (i.e., no observable effects) level exists for ozone and, if so, at what level. For example, McDonnell et al. (1983) found a nonlinear relationship between health and ozone exposure that "flattened" at ozone levels below 0.18 parts per million (ppm) -- a level above the ambient concentrations in most metropolitan areas. If such a threshold exists, the health benefits of reducing ozone from its current levels would be minimal. Population studies by Zaganiski et al. (1979) and Lebowitz et al. (1984), however, suggest effects may be occurring at ambient levels as low as 0.08 ppm. Moreover, other studies do not support the existence of any threshold for health effects (Portney and Mullahy, 1983; Hasselblad and Svendsgaard, 1975).

Hammer et al. (1974) found associations between increased oxidants and respiratory symptoms (such as cough and chest discomfort) and other symptoms (such as eye irritation and headache) in young, healthy adults. They obtained the symptom rates from daily diaries and adjusted them by excluding days on which subjects reported

fevers. Makino and Mizoguchi (1975) found a correlation between oxidant levels and eye irritation and sore throats in Japanese school children. Lippmann et al. (1983) and Lebowitz et al. (1982, 1983, 1984) found evidence of decreased athletic performance, increased prevalence of acute symptoms, and dysfunction of pulmonary systems resulting from ozone exposure.

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

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

2.1.2 Estimates of Health Effects

Unfortunately, it is difficult to use these studies to estimate the potential health benefits from deductions in ozone, because they did not estimate dose-response functions. Most were designed to investigate potential thresholds, or simply to determine if any relationship existed between ozone and particular effects.

In addition, studies using lung function changes as the health endpoint fail to provide a measure that can be valued in economic terms.

Recent work by Portney and Mullahy (1983, 1985) at Resources for the Future (RFF) is an exception. They considered the effect of alternative levels of ozone on various health measures, combining individual health data from the Health Interview Survey (HIS) with data on pollution concentrations during the same period covered by the survey. The people interviewed in the HIS provided information on their health status during the two weeks preceding the survey. As their health measure, Portney and Mullahy focused on the number of days of restricted activity due to a respiratory condition (RADRESP). The RADRESP measure included days when the symptoms were relatively minor, as well as those when they were serious enough to confine individuals to bed or to make them miss work.

Portney and Mullahy regressed RADRESP on a dozen or more independent variables, including socioeconomic and demographic factors, chronic health status, urban variables, and ozone and other pollutants. As their ozone measure, they used the daily maximum one-hour concentration (measured in parts per million) averaged over the two-week period covered for the individual. They considered several different specifications and functional forms. In their ordinary least squares (OLS) regressions, they tried various forms of the ozone measure, including the square and the square root as well as the untransformed variable.

The first part of Table 2.1 summarizes the OLS results. In all three specifications, the ozone coefficient was positive, but not significant, at the 95 percent confidence level. For the specification using the linear ozone term, the coefficient on ozone represents the average change in RADRESPs per person per two weeks for a one ppm change in ozone. Thus, for example, that coefficient predicts that reducing the average daily maximum ozone concentration by 0.01 ppm for one year for a population of 1 million adults would decrease the number of RADRESPs by 316,800 ($= 0.01 \times 1.22 \times 1,000,000 \times 52/2$).

In subsequent analysis, Portney and Mullahy (1985) estimated the relationship using a Poisson model, which can be written as:

$$E(\text{RADRESP}) = \exp(\text{XB}),$$

where $E(\text{RADRESP})$ is the expected number of RADRESPs, and XB is the sum of the product of the independent variables and their coefficients. The second part of Table 2.1 summarizes the results of the Poisson model. Because the model is nonlinear, the ozone coefficient is slightly harder to use for extrapolation. However, the model appears to fit the data better; RADRESPs have a Poisson-like distribution.

In separate models estimating RADRESPs for children, Portney and Mullahy (1983) did not find any consistently significant effects. As a lower bound, therefore, we assumed no effect on RADRESPs for children in the general population. However, incomplete data for children and the reliance on parents to report child-related health effects may explain this result. A restriction in activity probably was less likely to be reported for a child.

TABLE 2.1 Regression Results for Portney and Mullahy Study

<u>Ozone Specification</u>	<u>Ozone Coefficient</u>	<u>t-statistic</u>	<u>F-statistic</u>
<u>OLS</u>			
Linear	1.2185	1.13	2.743
Square root	0.8076	1.66	2.867
Squared	0.4667	0.07	2.636
<u>Poisson</u>	6.8827	1.97	N.A.
(log-likelihood ratio = -1395.4)			

Public health scientists continue to debate whether children are as susceptible to ozone as adults. Older children, for example, may not be as susceptible because they typically have large excess lung capacity. On the other hand, damages to the lungs of a child may result in a chronic respiratory condition in adulthood. Therefore, to place a plausible upper bound on our estimates, we applied the adult coefficients to children as well.

To estimate the change in RADRESPs due to a one percent change in ozone, we simulated the change using the data on individual exposures and characteristics constructed by Portney and Mullahy. Their study matched the 1979 HIS with air quality data, weather stations, and other area-specific data. Using the estimated regressions, we rolled back the exposure of each person in the data base by one percent. Using data from the Census Bureau, we assumed a population of 230 million in 1984, with 70 percent of the total above age 17 and 67 percent of the total living in metropolitan areas.

The changes in total annual RADRESPs for adults predicted by the Poisson and linear models were quite similar: 2.1 and 2.4 million, respectively. Because the Poisson model provided a better fit of the data, we used it as the basis for our estimates. For our high and low estimates of adult effects, we used plus or minus one standard deviation of the ozone coefficient. For children, our low estimate was zero, and our high estimate was 0.90 million cases (based on the adult coefficient). For our medium or point estimate, we used the midpoint of those extremes, or 0.45 million cases.

The ranges of estimates for respiratory effects are displayed in Table 2.2 separately for metropolitan and nonmetropolitan areas. These results should be interpreted cautiously, because cross-sectional studies of this type can be extremely sensitive to model specification, functional form, omitted and confounding variables, and the ambient air monitors used.

Portney and Mullahy (1985) also used a multinomial logit model to estimate the marginal impact of ozone on the two types of RADRESPs--the more serious ones that result in a day of bed rest or of lost work, and the less serious ones that resulted in a more minor restriction of normal activity. That analysis suggested that a marginal change in ozone was three times more likely to cause a day of minor restricted activity than a day of bed rest or lost work.

To provide an alternative estimate of respiratory conditions and a separate estimate of nonrespiratory irritations, we used the results of a statistical reanalysis of the Hammer et al. (1974) study discussed earlier. In an unpublished paper, Hasselblad and Svendsgaard (1975) fit simple logistic curves to estimate the relationship between ozone concentration (measured as a daily maximum hourly concentration) and eye irritation, headache, coughing, and chest discomfort. The probability of a response at an ozone level, X , measured in parts per hundred million (pphm), was given as:

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

TABLE 2.2 Estimated Health Effects of a One percent Reduction
in Ozone
(millions of days per year)

	Low Estimate	High Estimate	Medium Estimate
<u>Metropolitan Areas</u>			
Respiratory Effects			
Bed Rest/Work Loss			
Adults	0.24	0.67	0.36
Children	<u>0.00</u>	<u>0.15</u>	<u>0.07</u>
Subtotal	0.24	0.82	0.43
Minor Restrictions			
Adults	0.72	1.96	1.05
Children	<u>0.00</u>	<u>0.45</u>	<u>0.23</u>
Subtotal	0.72	2.41	1.28
Nonrespiratory Effects			
Headaches	0.84	0.84	0.84
Eye Irritation	<u>2.09</u>	<u>2.09</u>	<u>2.09</u>
Subtotal	2.93	2.93	2.93
<u>NonMetropolitan Areas</u>			
Respiratory Effects			
Bed Rest/Work Loss			
Adults	0.12	0.33	0.17
Children	<u>0.00</u>	<u>0.08</u>	<u>0.04</u>
Subtotal	0.12	0.41	0.21
Minor Restrictions			
Adults	0.35	0.97	0.52
Children	<u>0.00</u>	<u>0.22</u>	<u>0.11</u>
Subtotal	0.35	1.19	0.63
Nonrespiratory Effects			
Headaches	0.41	0.41	0.41
Eye Irritation	<u>1.03</u>	<u>1.03</u>	<u>1.03</u>
Subtotal	1.44	1.44	1.44

Table 2.3 displays the estimates of the parameters for the four outcome measures. These coefficients must be interpreted cautiously, because Hasselblad and Svensgaard did not control for some possible confounding variables, in particular temperature and humidity. In a later published paper, Hasselblad (1981) fit multiple logistic regression models to these same data, but that paper does not report the regression coefficients we needed to make our estimates.

We used these estimated dose-response functions with the individual information in the Portney and Mullahy data set to simulate the effects of a one percent reduction in ozone. These results are presented in Table 2.4 for metropolitan and nonmetropolitan areas. A range is not provided for this health effect because standard errors were not presented by Hasselblad and Svensgaard. For cough and chest discomfort, the Hasselblad and Svensgaard coefficients yielded a total of 1.54 million adult cases per year for a one percent reduction in ozone, compared to the estimate of 2.10 million based on the Portney and Mullahy Poisson model.

These estimates are remarkably consistent, as the RADRESP measure used by Portney and Mullahy included other symptoms besides cough and chest discomfort. In addition, the Hammer et al. sample used by Hasselblad and Svensgaard consisted of student nurses, who were young and generally healthy, while the Portney and Mullahy sample was more representative of the general population. For those reasons, our estimates for respiratory conditions rely solely on Portney and Mullahy's results.

TABLE 2.3 Regression Coefficients from Hasselblad and Svensgaard Study

<u>Effects</u>	<u>A</u>	<u>B</u>	<u>C</u>
Respiratory			
Cough	-2.98	0.0092	0.0450
Chest discomfort	-3.53	0.0023	0.0166
Nonrespiratory			
Eye irritation	-4.96	0.0907	0.0407
Headache	-4.88	0.0470	0.0976

TABLE 2.4 Changes in Days With Headache, Eye Irritation, Cough
or Chest Discomfort for a One Percent Reduction in
Ozone, in all Ages
(in millions of days)

<u>Effects</u>	<u>Metropolitan Areas</u>	<u>Nonmetropolitan Areas</u>
Respiratory		
Cough	.898	.443
Chest Discomfort	.137	.067
Nonrespiratory		
Headache	.838	.413
Eye Irritation	<u>2.092</u>	<u>1.030</u>
TOTAL	3.965	1.953

The results of Hasselblad and Svensgaard also can be used to estimate the number of nonrespiratory conditions, such as eye irritation and headache, possibly related to exposure to ozone and other photochemical oxidants. There is evidence suggesting that these symptoms are not related to ozone per se, but rather to other oxidants, such as peroxyacetyl nitrate (PAN), whose production may be proportional to that of ozone.

To account for this possibility, we used the estimates based on Hasselblad and Svensgaard as point estimates for nonrespiratory irritations (headache and eye irritation). Unfortunately, these researchers did not report the standard errors, so a confidence interval could not be determined.

These estimates reflect the likely acute effects generated by intense, short-term exposure to ozone. Long-term exposure to ozone also may affect the health of some people, but the epidemiological evidence on chronic ozone effects is sparse. One of the available studies, Detels et al. (1979), compared the effects of prolonged exposure to different levels of photochemical oxidants on the pulmonary functions of both healthy individuals and individuals with chronic obstructive pulmonary disease. Persons exposed to an annual mean of 0.11 ppm of oxidant, compared to a control group exposed to 0.03 ppm of oxidant, showed statistically

significantly increased chest illness, impairments of respiratory functions, and lower pulmonary function.*

While the epidemiological evidence of the effects of long-term exposure to ozone is sparse, several animal experiments have demonstrated effects on lung elasticity, blood chemistry, the central nervous system, the body's ability to defend against infection, and the rate at which drugs are metabolized (U.S. EPA, 1983a). Unfortunately, it is not possible to extrapolate those results to humans. Therefore, we could not quantify the chronic health effects attributable to ozone, but we believe that some of these effects may be present at current ambient levels.

Table 2.5 summarizes our estimates of the economic benefits from health effects of a one percent reduction in ozone. The estimates of respiratory effects are based on the Portney and Mullahy results, while the nonrespiratory effects are derived from Hasselblad's and Svensgaard's analysis.

The ozone-related health effects have three subcategories: days of bed rest or lost work due to respiratory symptoms, days of more minor restrictions due to respiratory symptoms, and cases of minor nonrespiratory irritations (headache and eye irritation). We valued each of these three subcategories separately.

* 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 were some questions about the adequacy of the measurement of ozone exposure, about the subject selection, and about the test measures. Although it is both reasonable and likely that long-term exposures affect health, the failure to correct for the effects of other pollutants raises uncertainties about the specific findings.

For the more serious category of respiratory conditions (days of bed rest or lost work), we used the average daily wage (\$80) as a lower bound. This is the value of the lost output to society for an employed individual. Some of these days may include the exacerbation of a preexisting respiratory condition, such as asthma or bronchitis. Since these days may involve additional medical expenses, we used an upper bound of \$120, giving a mean value of \$100. The range of benefits for this category is derived by multiplying the low value of illness by the low estimate of effects. Similarly for the high estimate, the high value of illness is multiplied by the high estimate of effects.

To value a day of more minor restrictions in activity due to respiratory conditions, we relied upon Loehman et al. (1979), whose survey results suggested a willingness to pay of \$2.31 to prevent a day of minor coughing, \$4.90 to prevent minor shortness of breath, and \$8.17 to prevent minor head congestion. We converted these estimates from 1978 dollars to 1983 dollars to yield values of \$3.50 to \$12.50 for avoiding a minor restricted day, with a point estimate of \$8.00.

We were unable to find estimates in the literature for the value of avoiding headaches or eye irritation. These conditions, however, seemed less serious than the respiratory effects, so we used a value of \$3 per case, just below the lower end of the range from the Loehman et al. study.

TABLE 2.5 Value of RADRESPs Due to a One Percent Reduction
in Ozone
(millions of 1983 dollars)

	<u>Low Estimate</u>	<u>High Estimate</u>	<u>Medium Estimate</u>
<u>Metropolitan Areas</u>			
Respiratory Effects			
Bed Rest/Work Loss			
Adults	19.3	80.4	35.5
Children	<u>0.0</u>	<u>18.5</u>	<u>7.4</u>
Subtotal	19.3	98.9	42.9
Minor Restrictions			
Adults	2.5	24.5	8.4
Children	<u>0.0</u>	<u>5.6</u>	<u>1.8</u>
Subtotal	2.5	30.1	10.2
Nonrespiratory Effects	8.8	8.8	8.8
<hr/>			
TOTAL	30.6	137.8	61.9
 <u>Nonmetropolitan Areas</u>			
Respiratory Effects			
Bed Rest/Work Loss			
Adults	9.5	39.6	17.5
Children	<u>0.0</u>	<u>9.1</u>	<u>3.6</u>
Subtotal	9.5	48.7	21.1
Minor Restrictions			
Adults	1.2	12.1	4.1
Children	<u>0.0</u>	<u>2.8</u>	<u>.9</u>
Subtotal	1.2	14.9	5.0
Nonrespiratory Effects	4.3	4.3	4.3
<hr/>			
TOTAL	15.1	67.9	30.5

The results suggest that a one percent reduction in ozone generates total health benefits in metropolitan areas ranging from \$31 to \$138 million, with a midpoint of \$62 million. In nonmetropolitan areas the health benefits range from \$15 million to \$68 million with a midpoint of \$31 million.

2.2 AGRICULTURAL EFFECTS OF OZONE

Ozone, alone or in combination with sulfur dioxide and nitrogen dioxide, is responsible for most of the U.S. crop damage attributed to air pollution (Heck et al., 1983). Ozone affects the foliage of plants by biochemical and cellular alteration, thus inhibiting photosynthesis and reducing plant growth, yield, and quality.

Early studies of ozone-related damages used generalized relationships between ozone concentrations, yield, and economic loss. Insufficient information precluded the construction of an economic model with credible dose-yield data. Thus, for example, Freeman (1982), in a general survey of the literature, could only conclude that the total agricultural damages from ozone were \$1 - \$4 billion in 1978 dollars.

Recent work by the National Crop Loss Assessment Network (NCLAN) suggests that prior studies have underestimated ozone-related damages. NCLAN'S estimated dose-yield functions for soybeans, wheat, corn, peanuts, cotton, barley, and sorghum have provided more accurate information on ozone's effects on crops. Kopp (1983, 1984) and Adams et al. (1984) incorporated these functions

into models of agricultural production and demand to estimate the benefits of ozone reduction strategies.

Kopp constructed a detailed microeconomic model of farm behavior for over 200 producing regions in the United States. The NCLAN dose-yield functions are directly incorporated in Kopp's model of the supply side of each crop for each region. Because estimates of the demand and supply elasticities for these crops are used in the analysis, it gives a good indication of the actual change in economic welfare.

Kopp's simulations suggest that a one percent reduction in ozone would produce total benefits of roughly \$110 million (1983 dollars) per year for the seven major crops covered by NCLAN, as shown in Table 2.6. These seven crops accounted for only about 80 percent of the total value of U.S. crop production (USDA, 1982). If we increase Kopp's estimate by assuming that ozone damages to all other crops occur in the same proportion as their relative value, we conclude that the benefits of a one percent change in ozone are roughly \$137 million annually.

Adams et al. (1984) used a different approach. By incorporating the NCLAN dose-yield functions into an existing quadratic programming model, they calculated ozone benefits for six of the seven crops covered by Kopp (they did not include peanuts). They estimated that a 10 percent reduction in rural ozone would result in annual benefits of roughly \$674 million (1983 dollars). Assuming linearity and increasing the estimate to account for omitted crops, we estimated \$90 million in benefits for a one percent change

TABLE 2.6 Annual Agricultural Benefits of a One Percent Ozone Reduction
(millions of 1983 dollars)

<u>Crop</u>	<u>Estimate</u>
Soybeans	50.8
Corn	7.6
Wheat	21.1
Cotton	20.4
Peanuts	5.0
Sorghum	4.6
Barley	<u>0.2</u>
Total assessed	109.8

in ozone levels. Unfortunately, Adams et al. did not calculate the benefits on a crop-by-crop basis, so it is not possible to make a detailed comparison with Kopp's estimates. Finally, both the Kopp and Adams et al. studies have an important shortcoming. Neither study reflects the crop-subsidy programs at the state or federal levels. Because production is subsidized, the social value of the crop may be less than the existing price. Given some of the large subsidy programs for the crops discussed above, we expect that the true agricultural benefit estimates would be significantly less than the estimates presented here. However, at present, studies estimating the effects of current subsidy programs do not exist. Therefore, we were not able to correct the estimates of Kopp or Adams et al.

For our best estimates, we used Kopp (1983, 1984) and Adams et al. (1984), because they used the superior NCLAN data and based their estimates on economic measures of welfare loss. The two estimates are fairly close. We concluded that a one percent reduction in rural ozone would produce \$90 to \$140 million in annual agricultural benefits per year, with a point estimate of \$114 million. We qualify these numbers by noting that the estimates do not reflect unreported small "truck farm" sales or any averting activities that farmers may undertake, such as planting pollutant-resistant crops; however, we believe that these categories are likely to be small. On the other hand, the welfare measures do not reflect either the effects of crop-subsidy pro-

grams at the state and federal levels, or the effects of drought, both of which are likely to reduce the marginal welfare impacts of ozone. The effects of subsidies are likely to be particularly significant.

2.3 EFFECTS ON NONAGRICULTURAL VEGETATION

Forests and ornamental plants also may suffer substantial damages from exposure to ozone. The preliminary draft of the

Ozone Criteria Document discusses the issue:

The influence of 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 $\mu\text{g}/\text{m}^3$) to 0.40 ppm (785 $\mu\text{g}/\text{m}^3$). (U.S. EPA, 1983)

Additional evidence of significant damages from ozone associated with nonagricultural vegetation is provided by McLaughlin et al. (1984).

Unfortunately, no careful quantitative studies of the type done by NCLAN have been performed for nonagricultural vegetation. Heintz et al. (1976) have estimated losses to ornamental plants of \$100 million per year in 1973 dollars. Inflating to 1983 dollars using the Farm Products Index, and assuming linearity, yields estimated annual benefits of \$1.4 million for the reduction in damages to ornamentals from a one percent reduction in ozone. We assume that the damages occur in proportion to metropolitan versus nonmetropolitan population.

Damage to forests is potentially a much larger concern in terms of reduced production and decreased recreational and aesthetic values. In a very small contingent value study, Crocker and Vaux (1983) found that the shift of an acre of the current mix of severely, moderately, and unharmed timberland in the San Bernardino National Forest into the unharmed category would generate additional annual recreational benefits of between \$21 and \$68 per acre per year. These findings are difficult to generalize for the rest of the nation because ambient ozone levels are unusually high in the San Bernardino area (and the authors provide no dose-response function for extrapolating to areas with lower concentrations), and because other site attributes and visitors' socioeconomic characteristics have very large and significant effects on users' willingness to pay to reduce damages to forests.

We also lack data on the impact of ozone on commercial forests. Most commercial forests, however, are located in areas with low ozone concentrations, so damages may be small. Moreover, in areas with relatively high concentrations, damages can be reduced by planting trees that are resistant to ozone.

2.4 EFFECTS ON MATERIAL DAMAGE

Current research indicates that ozone may directly damage many types of nonbiological materials, including elastomers, paint, and textile fibers and dyes (U.S. EPA, 1978). The damages to elastomers are well documented. Ozone exposure can increase the rigidity of rubber and synthetic polymers, causing brittleness, cracking, and reduced elasticity. The evidence and reliability of

the damage estimates relating to textile fibers and dyes, and to paint are either poorly characterized or less well documented.

Ozone exposure also can generate other effects, such as avoidance costs (purchase of specially resistant materials) and aesthetic losses. Only the direct costs are incorporated in this analysis, however. Unfortunately, some of these costs are based on studies that may be over ten years old.

In his survey of the literature, Freeman (1982) suggested that annual material damages from oxidants and NO_x amount to approximately \$1.10 billion (1978 dollars). We updated that estimate using the U.S. government price indices for rubber and textile products and the index of personal consumption expenditures for durable goods; this yielded an estimate of \$2.25 billion for 1983. Assuming linearity, we found that a one percent ozone reduction generates a benefit of roughly \$22.50 million annually.

We obtained an alternative estimate of the benefits of reduced material damage by using dose-response information incorporated in the 1978 Criteria Document for ozone. The text contains per capita economic damage functions for elastomers, textiles, industrial maintenance, and vinyl paint. We used a population-weighted mean value of ozone of 0.03 ppm (60 ug/m^3) from the draft of the new Criteria Document (U.S. EPA, 1984), a population estimate of 230 million, and the indices cited above.

This method yielded annual benefits of \$15 million (in 1983 dollars) for a one percent reduction in ozone. Of this total,

approximately 40 percent results from damages to elastomers. Averaging these two estimates (with slightly more weight given to the lower estimate, which does not include any NO_x-related damages) yields a point estimate of \$18.0 million annually, with a range of \$15.0 to \$22.5 million. We assume that these damages are proportional to the population in metropolitan versus nonmetropolitan areas.

2.5 Summary of Benefits of a One Percent Change in Ozone

Table 2.7 summarizes our estimates of the effects of a one percent reduction in ozone. For metropolitan areas the estimated health benefits include roughly \$43.0 million from decreases in respiratory effects resulting in bed rest or work loss, \$10.2 million from reduced respiratory symptoms resulting in some minor restriction in activity, and \$8.8 million resulting from a lower rate of non-respiratory irritations (headaches and eye irritation) (1983 dollars). On the nonhealth side of the ledger, materials damage is \$12 million, while ornamental plants contribute \$1.0 million. The total ranges from \$42 million to \$154 million with a point estimate of \$75 million.

For nonmetropolitan areas, the benefits from reduced health effects are roughly \$30 million. Regarding welfare effects, increases in agricultural crop production dominate, with a total of \$114 million, with reduced material damages adding \$6 million in benefits. The total ranges from \$111 million to \$216 million, with a point estimate of \$150 million.

TABLE 2.7 Summary of Estimated Benefits of a One Percent
Reduction in Ozone
(millions of 1983 dollars)

<u>Type of Area</u>	<u>Low Estimate</u>	<u>High Estimate</u>	<u>Medium Estimate</u>
<u>Metropolitan Areas</u>			
<u>Health</u>			
Respiratory Symptoms			
Bed rest/Work loss	19.3	98.9	42.9
Minor restrictions	2.5	30.1	10.2
Nonrespiratory Symptoms	8.8	8.8	8.8
<u>Welfare</u>			
Agricultural crops	0	0	0
Ornamental plants	0.9	0.9	0.9
Material damage	10.0	15.1	12.0
	<hr/>	<hr/>	<hr/>
TOTAL	41.5	153.8	74.8
<u>Nonmetropolitan Areas</u>			
<u>Health</u>			
Respiratory Symptoms			
Bed rest/Work loss	9.5	48.7	21.1
Minor restrictions	1.2	14.9	4.7
Nonrespiratory Symptoms	4.3	4.3	4.3
<u>Welfare</u>			
Agricultural crops	90.0	140.0	114.0
Ornamental plants	0.5	0.5	0.5
Material damage	5.0	7.4	6.0
	<hr/>	<hr/>	<hr/>
TOTAL	110.6	216.3	150.5

In all cases, the estimates are subject to considerable uncertainty; the "high" and "low" estimates provide only a partial indication of that uncertainty, as they reflect only the ranges in available estimates or statistical uncertainty in the parameter estimates from particular studies. A more complete accounting for uncertainty -- which would include different functional forms for the doseresponse functions, omitted categories, etc. -- would yield substantially broader ranges.

CHAPTER 3

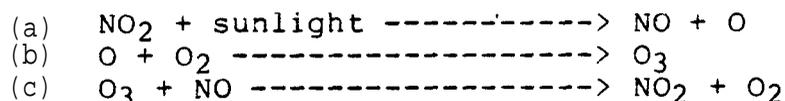
LINKING NO_x AND VOCs TO OZONE

The amount of VOC and NO_x emissions in the atmosphere directly affects ozone formation. This chapter first looks at this relationship, and it then quantifies how changes in VOCs and NO_x will change urban and rural ozone levels. Finally, we calculate the ozone-related benefits that will result for every ton of VOC and NO_x that is controlled.

Before beginning, we present a preliminary discussion of the ozone formation process to permit a deeper understanding of the control problem.

3.1 THE OZONE-VOC- NO_x RELATIONSHIP

The general process by which ozone (O_3) is formed is illustrated by the NO_2 -NO- O_3 cycle. These reaction equations can be written as:



Ozone is not emitted in any measurable quantity. In fact, reactions (a) and (b) represent its only significant source. However, these reactions are fully reversed by reaction (c). Since NO comprises roughly 90 percent of man-made NO_x emissions, reaction (c) suggests that additional NO_x emissions would reduce ozone concentrations by scavenging the ozone for one of the oxygen atoms, thereby producing O_2 and NO_2 . Therefore, increases

EPA has required VOC control to reach the ozone standard. This discussion suggests that as the VOC/NO_x ratio becomes smaller, VOC control will be more and more effective. Thus, decreasing NO_x is likely to lower the effectiveness of the VOC control program in reducing ozone.

Some empirical studies conclude that NO_x reduction is already counterproductive in controlling ozone. Glasson (1981) and Innes (1981) used smog chamber tests to simulate NO_x-O₃ relationships. Glasson concluded: "The results of the experimental simulation suggest that hydrocarbon reductions reduce O₃ in urban as well as downwind areas, while NO_x reduction increases O₃ in the urban area and has little effect on O₃ in downwind areas" (p. 1169). Innes produced similar results.

Relevant modeling results were generated by the Office of Air Quality Planning and Standards, Systems Applications, Inc. (1984), (1985), and others. In the St. Louis Ozone Modeling Project (U.S. EPA, 1983), simultaneous changes in hydrocarbons and NO_x were modeled by a state-of-the-art photochemical grid model. In every simulation, NO_x reductions were counterproductive in reducing ozone, while increases in NO_x increased the effectiveness of hydrocarbon control. "The results of these sensitivity tests suggest that controls on oxides of nitrogen emissions are counterproductive with regard to reducing ozone and that allowing oxides of nitrogen to increase will enhance the benefits achieved by lowering hydrocarbon emissions" (pp. 73-74).

OAQPS has also developed three sets of regional Empirical Kinetic Modeling Approach (EKMA) isopleths for use in the ozone NAAQS review. These models indicate that at all ratios typical of urban centers, controlling NO_x will be counterproductive to controlling ozone in urban areas.

Layland and Cole (1983) have used state-of-the-art modeling for St. Louis, Tulsa, Los Angeles, and Denver. They also conclude that higher NO_x emissions will decrease urban ozone. However, they speculate that NO_x may increase rural ozone because of the relatively high VOC/ NO_2 ratios in those areas. In addition to the information on NO_2 , they present ozone response curves showing the percentage reduction in ozone for various percentage reductions in VOCs.

3.2 QUANTIFYING THE EFFECTS OF VOCs AND NO_x ON OZONE

From these models, we generated estimates of the "average" impact of changes in VOCs and NO_x on urban and rural peak ozone concentrations (which would be relevant for health effects and materials damage) and on rural average ozone concentrations (which is more relevant for agricultural effects). (Recently, the National Crop Loss Assessment Network has concluded that the daily maximum 7 to 12 hour average is the most relevant ozone measure for agricultural damages.)

Given this preliminary background, we now present the following quantitative estimates:

1. How metropolitan VOCs affect peak metropolitan ozone, peak rural ozone and average rural ozone,

2. How rural VOCs affect peak and average rural ozone,
3. How metropolitan NO_x emissions affect peak metropolitan ozone, peak rural ozone, and average rural ozone,
4. How rural NO_x emissions affect peak and average rural ozone.

3.2.1 Metropolitan VOCs

The ozone-SIP data base presents estimates of how urban VOCs affect peak urban ozone for every city in nonattainment of the ozone standard. Analyses of these data by the Office of Air Quality, Planning, and Standards (U.S. EPA, 1984b) suggests that, on average, a one percent reduction in hydrocarbon emissions will reduce metropolitan ozone by 0.6 percent. Since this estimate is based on a number of EKMA urban simulations throughout the United States, it represents the best estimate of the percent reduction in urban ozone from a one percent reduction in VOCs.

Estimates of how urban VOCs affect rural average and peak ozone levels are presented in SAI (1984). SAI simulated regional air quality under four basic scenarios: winter and summer, mornings and afternoons. The two summer scenarios (relevant for estimating agricultural benefits) indicate that average ozone decreases by .13 percent for every 1 percent reduction in urban VOCs during the agricultural growing season. Peak rural ozone decreased by much less: roughly .05 percent for every one percent reduction in urban VOCs.

3.2.2 RURAL VOCs

Previous drafts of this report used the three sets of regional EKMA isopleths developed by OAQPS to estimate how rural VOCs affect ozone. By noting data points at VOC/NO_x ratios typical of rural areas we estimated that, on average, a one percent reduction in rural VOCs reduces peak rural ozone by .36 percent. However, reviewers of these previous drafts have since demonstrated that EKMA models are not suitable for estimating changes in rural ozone. Rural ozone is influenced by many factors that EKMA models do not capture. Therefore, we initiated a study of how rural emission changes affect rural ozone (SAI, 1985). SAI's regional oxidant model simulated changes in rural VOCs for low and high emission densities, winter and summer, mornings and afternoons. Changes in peak and maximum seven hour ozone were estimated. The results suggest that a one percent change in rural VOCs on average will change rural peak ozone by 0.1 percent and average ozone by 0.07 percent. The range of the simulation results as well as the point estimates are presented in Table 3.1.

3.2.3 URBAN NO_x

To estimate the effects of NO_x reductions on urban and rural ozone, we used SAI (1984) and the EKMA isopleths developed by OAQPS. SAI estimates that urban ozone will increase by 0.27 percent when NO_x emissions are reduced by 1 percent.

An alternative estimate is available by taking an average of data points from the isopleths developed by OAQPS. While individual observations may vary, we calculate that, on average, a one percent reduction in metropolitan NO_x will increase ozone by 0.1 percent. For our point estimate, we use the estimate from the EKMA isopleths of 0.1 percent.

SAI also estimated how rural ozone is affected by changes in NO_x emissions. A 1 percent reduction in urban NO_x would increase average rural ozone by 0.09 percent. Peak rural ozone would probably not change as much, since the urban plume is widely dispersed when it reaches the rural areas. For our estimate we assume that a 1 percent reduction in urban NO_x will reduce peak rural ozone by 0.04 percent. The SAI analysis showed average rural ozone increasing by a small amount in some situations, however, on average, a small decrease occurs.

3.2.4 RURAL NO_x

Again, this latest version of our report no longer must rely on using the EKMA isopleths to estimate how rural NO_x emissions affect peak and average rural ozone. Instead, we use the simulation results of a regional oxidant model, a more appropriate model to estimate rural ozone changes. (See SAI, 1985.) The average of these results indicate that a 1 percent reduction in rural NO_x will reduce peak rural ozone by 0.15 percent and average rural ozone by .11 percent. These point estimates, as well as the range indicated by the simulations, are presented in Table 3.1.

3.2.5 SUMMARY OF RESULTS

On the basis of these studies, we summarize ozone-VOC-NO_x relationships in Table 3.1. We qualify further the results with three caveats:

1. Many of the results are based on averages of various simulation results.
2. Our estimates best represent how ozone is affected on average across the U.S. They should not be used for estimating ozone changes for specific areas.
3. Although we used the best predictive models available, simulating secondary air pollutant changes is difficult. Over a hundred chemical reactions are involved. Therefore, we caution against attributing too much certainty with these estimates.

Because of these qualifiers, we are reluctant to associate any single point estimate with a high degree of certainty. For changes in VOCs we are more confident. However, there is more uncertainty about how rural ozone is affected by changes in NO_x. For these estimates we interpolated from a number of modeling results to arrive at our best estimate.

3.3 CALCULATING THE BENEFITS PER TON OF VOC AND NO_x CONTROL

Our final step was to calculate the ozone-related benefits per ton of VOC and NO_x controlled. Because we have separate estimates for rural and urban benefits from ozone reductions, we were able to calculate separate benefit estimates for rural and metropolitan VOC and NO_x controls.

TABLE 3.1

Estimated Ozone Reductions from One Percent Reductions
in Rural and Metropolitan VOCs and NO_x

<u>Pollutants</u>	<u>Percent Reduction in Urban Ozone</u>	<u>Percent Reduction in Peak Rural Ozone</u>	<u>Percent Reduction in Average Rural Ozone</u>
<u>VOCs</u>			
One Percent Reduction in Urban VOCs	.60	.05	.13
One Percent Reduction in Rural VOCs	0	.05 to .2 ^a (.01)	.04 to .13 ^a (.07)
<u>NO_x</u>			
One Percent Reduction in Urban NO _x	-.05 to -.3 ^a (-.1)	-.1 to -.1 ^b (.04)	-.09
One Percent Reduction in Rural NO _x	0	.05 to .24 ^a (.15)	.05 - .2 ^a (.11)

a | Estimates varied by a fairly wide range. Point estimates appear in parenthesis.

b | Sparse evidence does not permit estimation of a single point estimate with a reasonable degree of certainty. In many areas (the Northeast Corridor) wind trajectories would probably transport metropolitan ozone to other urban centers or over the ocean. In these cases, rural ozone would not change. In other areas, however, the increased ozone from urban NO_x reductions would travel to rural areas. But, lower rural NO₂ levels would reduce ozone. Therefore, the overall effect could be positive or negative. We assume that a one percent uniform reduction in metropolitan NO_x reduces rural ozone by .04 percent.

In 1979, roughly 21.9 million tons of VOCs and 21.3 million tons of NO_x were emitted. We use 1979 emissions because our two largest benefit categories (health effects and agricultural damages) are based on 1979 and 1978 air quality data. We allocate total VOC emissions of 21.9 million tons per year and NO_x emissions of 21.3 million tons per year on the basis of regional urban/rural emission ratios estimated by Systems Applications and by the National Research Council (1975). Their estimates of the possible range of metropolitan and rural emissions are summarized in Table 3.2. Note that when urban emissions are assumed to be higher than our point estimate, rural emissions must decrease (from the point estimate) by a corresponding amount.

We can combine our emission point estimates, dispersion modeling, and ozone benefits to calculate benefit per ton estimates for both rural and urban emission controls. These calculations involve several steps. For example, consider the total benefits associated with a one percent reduction in urban VOCs. Based on the coefficients in Table 3.1, that would reduce peak urban ozone by 0.60 percent, peak rural ozone by .05 percent and average rural ozone by .13 percent. Multiplying the nonagricultural benefits associated with 1 percent changes in urban and rural peak ozone respectively (from Table 2.8) by these coefficients yields benefits of $0.60 (74.9) + .05 (36.9) = \46.8 million. To calculate agricultural benefits for the same one percent reduction in urban VOCs, the change in average rural ozone is relevant. Therefore, multiplying the agricultural

TABLE 3.2

Estimates of VOC and NO_x Emissions
 Metropolitan and Rural Areas
 (millions of tons in 1979)

	<u>VOCs</u>			<u>NO_x</u>		
	<u>High Urban</u>	<u>Low Urban</u>	<u>Point Estimate</u>	<u>High Urban</u>	<u>Low Urban</u>	<u>Point Estimate</u>
Metropolitan Emissions	19.3	14.5	16.9	17.0	15.3	16.0
Rural Emissions	<u>2.6</u>	<u>7.4</u>	<u>5.0</u>	<u>4.3</u>	<u>6.0</u>	<u>5.3</u>
Total	21.9	21.9	21.9	21.3	21.3	21.3

benefits of a one percent reduction in ozone by 0.13 percent, or $\$114 \times 0.13$, yields \$14.8 million. Adding the urban and rural nonagricultural benefits to the agricultural benefits gives a total of $\$46.8 + \$14.8 = \$61.6$ million for a one percent reduction in urban VOCs. Using the Table 3.2 point estimate of 16.9 million tons of urban VOC emissions, we find the estimated benefit per ton is $\$61.6 \text{ million} / (0.169 \text{ million tons}) = \364 per ton. Calculations for individual benefit categories and for other types of emissions proceed in a similar fashion. These calculations assume implicitly that the benefits per ton are constant over the relevant ranges. Our benefit per ton estimates are summarized in Tables 3.3A-3.3B. These estimates are calculated based on our point estimates of urban and rural emissions in Table 3.2. In addition, only the point estimates for the VOC-NO_x-ozone relationships are used.

Weighting the metropolitan and rural estimates by their relative emissions yields averages of \$325 per ton of VOC controlled and \$8 per ton of NO_x controlled.

Finally, we have a number of qualifications and observations about our results in Tables 3.3A - 3.3B:

- (1) We were surprised at the value of the agricultural benefits. The number is large, especially for VOC control. Although work is ongoing, the best available NCLAN dose yield estimates were incorporated into two different economic models, which produced similar results. We again note, however, that the welfare

TABLE 3.3A

Benefits per Ton of VOC Controlled in
Metropolitan and Non-metropolitan Areas
(1983 dollars per ton)^a

<u>Category</u>	<u>Metropolitan</u>		<u>Non-metropolitan</u>	
	<u>Range</u>	<u>Point Estimate</u>	<u>Range</u>	<u>Point Estimate</u>
Health				
Value of Reduced Bed/Work Loss Days (Reduced cases per ton)	71 - 365 (.89) - (3.)	159 (1.6)	6 - 29 (.07) - (.25)	12 (.12)
Value of Reduced Minor Days (Reduced cases per ton)	9.3 - 111 (2.6) (8.9)	38 (4.7)	1 - 9 (.2) (.7)	3 (.4)
Value of Reduced non- respiratory cases (Reduced cases per ton)	32 - 32 (10.8) (10.8)	32 (10.8)	2.6 - 2.6 (.9) (.9)	2.6 (.9)
Agriculture	80 - 94	88	126 - 196	160
Nonagricultural Vegetation Ornamentals	4 - 4	4	1 - 1	1
Material Damage	43 - 49	45	10 - 15	12
Total Ozone-Related Benefits per Ton of VOCs.	241 - 656	365	145 - 251	190

a/ Totals may not agree due to rounding.

TABLE 3.3B

Benefits per Ton of NO_x Controlled in
Metropolitan and Nonmetropolitan Areas
(1983 dollars per ton)^a

<u>Category</u>	<u>Metropolitan</u>		<u>Nonmetropolitan</u>	
	<u>Range</u>	<u>Point Estimate</u>	<u>Range</u>	<u>Point Estimate</u>
Health				
Value- of Reduced Bed/Work Loss Days (Reduced Cases per ton)	-58 to - 4 (-.5) (-.5)	- 22 (- .2)	27 to 136 (.3) (1.1)	59 (.59)
Value of Reduced Minor Days (Reduced cases per ton)	-18 to - 1 (-1.4) (-.15)	-5 (-.6)	4 - 41 (1) (3.3)	14 (1.8)
Value of Reduced Non- respiratory Cases (Reduced cases per ton)	-4 -4 (-1.5) (-1.5)	-4 (-1.5)	12 12 (4) (4)	12 (4)
Agriculture	-79 to -51	-64	187 - 290	237
Nonagricultural Vegetation Ornamentals	- .5 to - .5	- .5	1.3 - 1.3	1.3
Material Damage	- 8 to - 4	- 6	14 - 21	17
Total Ozone-Related Benefits per Ton of NO _x , Controlled	-168 to -64	-102	244 - 502	340

^a/ Totals may not agree due to rounding

estimates do not account for any of the subsidy programs for crops or the antagonistic reactions to drought; accounting for these would reduce the benefit estimate.

- (2) Changes in our estimates of the distribution of total emissions between rural and metropolitan areas do not change the average benefit per ton estimates significantly. Table 3.4A-B presents benefit per ton estimates based on alternative assumptions about relative rural/metropolitan emissions.
- (3) These numbers only represent average benefits. Control of NO_x or VOCs in a particular area may produce lower or higher benefits per ton. If the location of new sources could be forecasted, more specific benefit estimates could be estimated using the framework developed here.
- (4) Changes in our estimates of the NO_x - or VOC-Ozone relationship (Table 3.1) affect our range of benefit estimates. In particular, our benefit per ton of VOCs ranges from \$305 to \$495 and the NO_x benefit per ton estimate ranges from \$-135 to \$127 when we use our range of VOC/ NO_x -Ozone relationships (presented in Table 3.1) to calculate benefits.

Table 3.4A
 Ozone - Benefits per ton of VOC
 Controlled Under Alternative Assumption About
 Relative Metropolitan/Rural Emissions

<u>Total VOC Emissions</u>	<u>Total Metropolitan VOC Emission</u>	<u>Total Rural VOC Emission</u>	<u>Benefit per ton of Metropolitan VOC</u>	<u>Benefit Per ton of Rural VOC</u>	<u>Weighted Average Benefit Per ton of VOC</u>
21.9	19.3	2.6	\$319	\$347	\$322
21.9	16.9	5.0	\$365	\$190	\$325
21.9	14.5	7.4	\$424	\$136	\$328

Table 3.4B
 Ozone - Benefits per ton of NO_x
 Controlled Under Alternative Assumption About
 Relative Metropolitan/Rural Emissions

<u>Total NO_x Emissions</u>	<u>Total Metropolitan NO_x Emissions</u>	<u>Total Rural NO_x Emissions</u>	<u>Benefit per ton of Metropolitan NO_x Controlled</u>	<u>Benefit Per ton of Rural NO_x Controlled</u>	<u>Weighted Average Benefit Per ton of NO_x Controlled</u>
21.3	17.0	4.3	\$ - 96	\$418	\$7.8
21.3	16.0	5.3	\$ -102	\$340	\$8.1
21.3	15.3	6.0	\$ -106	\$300	\$8.4

CHAPTER 4

EVALUATING THE BENEFITS OF VOC CONTROL (OTHER THAN OZONE)

Some VOCs pose no apparent direct threat to health, while others are relatively potent carcinogens, mutagens, or teratogens. Some plants emitting VOCs are located in densely populated cities, while others are far from population centers. Thus, the direct benefits of VOC control will depend on the location and potency of the specific VOCs.

Since this analysis is generic and not applicable to any particular NSPS, a precise estimate of the direct benefits of VOC control is not possible. However, we do provide a rough idea of the likely magnitude of the carcinogenic benefits, by presenting results of previous analyses of VOC control options. Some VOCs may also have teratogenic or other mutagenic benefits. Calculations for these other possible benefits are not presented.

4.1 VALUING THE BENEFITS OF REDUCED CANCER

Table 4.1 presents the estimated cancer cases avoided per ton of VOC controlled for a number of control options on a variety of source categories. These numbers were derived using the EPA Cancer Assessment Group's (CAG's) unit risk numbers and data on exposure per ton emitted. The CAG normally reports risk estimates in the form of cancer risk per lifetime exposure to one $\mu\text{g}/\text{m}^3$.

The incidence per ton is calculated by the following formula:

$$\frac{\text{Cancer cases}}{\text{ton}} = \frac{\text{Lifetime risk}}{\text{ug/m}^3/\text{person}} \times \frac{1 \text{ lifetime}}{70 \text{ years}} \times \frac{(\text{ug/m}^3\text{-person-year})}{\text{ton}}$$

In short, incidence per ton is estimated by multiplying exposure per ton by the risk per unit of exposure. If relevant, other chronic health effects would be calculated in the same manner.

For the VOCs listed in Table 4.1, cancer cases avoided per ton controlled range from a high of 9.9×10^{-5} cases per ton to a low of 1.6×10^{-6} cases per ton. However, many VOCs pose no cancer risk. Therefore, the cancer cases avoided per-ton controlled could be zero for many VOCs.

Monetizing the range of reductions in cancer cases per ton of controlled substance will allow us to add these estimates to the ozone-related benefits calculated in Chapter 2. However, valuing reductions in cancer risk is difficult and controversial.

Estimates in the literature of the value of saving a "statistical life" range from \$400,000 to \$7.0 million per fatality avoided. However, with carcinogens, there is likely to be a substantial lag between control expenditures and the receipt of benefits. Perhaps more important, cancer is primarily a disease of the elderly, so that each death averted saves relatively few years of life in comparison, say, to accident prevention. Moreover, many cases of cancer are not fatal. For these reasons we chose a value of \$1.0 million per cancer case avoided.

TABLE 4.1

INCIDENCE DATA FOR CARCINOGENIC SOURCES

<u>Substance/Sources</u>	<u>Emissions Reduced (Mgs)</u>	<u>Incidence per Mg</u>	<u>Benefits (\$ per ton)^a</u>
Coke By-product Plants	125,000	1.6×10^{-5}	16
Benzene Fugitive	5,450	5.7×10^{-5}	57
Benzene Storage	890	9.9×10^{-5}	99
Ethyl Benzene Styrene	142	3.6×10^{-5}	36
Maleic Anhydride	840	1.6×10^{-6}	2
Linear Alkyl Benzene	246	8.5×10^{-5}	85
Nitrobenzene	214	6.1×10^{-5}	61
Chloro-Benzene	104	1.7×10^{-5}	17
Ethylene	280	1.0×10^{-5}	10
Gas Marketing			
Stage I	217,000	5.1×10^{-6}	5
Stage II	94,000- 346,000	8.3×10^{-5} - 4.1×10^{-5}	83 41

^a | Assumes a value of \$1 million per case avoided.

Source: Pollutant Assessment Branch, Office of Air Quality,
Planning and Standards.

Using the value of \$1.0 million per cancer case and the range of cancer per ton estimates presented in Table 4.1, we calculate a range of benefits per ton of (1.0 million x $1.6 \cdot 10^{-6}$ cases per ton =) \$1.60 to (1.0 million x $9.9 \cdot 10^{-5}$ cases per ton =) \$99. However, these estimates are extremely uncertain, and the the unit risk estimates are upper confidence levels, leading to an overestimate of the benefits. Also, the incidence per ton associated with a particular NSPS will cover a huge range, depending upon plant locations, population densities, and the specific VOCs involved. Therefore, we are only able to present evidence suggesting a typical range for the direct benefits of VOC control.

4.3 Other Indirect Benefits of VOC Control

Through chemical reactions in the atmosphere, VOCs may affect the concentrations of sulfates and nitrates which in turn lead to acidic deposition. However, a recent article in Science (Seigneur et. al, 1984) suggests that even an enormous reduction in VOCs of 50 percent would lead only to a 2 percent reduction in sulfates and a 1 percent reduction in nitrates. Further, a linear relationship is not indicated, so that smaller changes in VOCs would not change sulfates or nitrates to any significant degree.

4.4 Summary

Because the health effects of VOCs differ, we are not able to calculate specific direct benefits per ton. However, even with

relatively potent carcinogens, we calculate a benefit per ton of \$99, roughly one fourth of the total direct + ozone-related benefits per ton of VOCs.

Chapter 5

DIRECT BENEFITS OF NO_x CONTROL

Although estimates vary widely, nitrogen oxides (NO_x) emitted from fossil fuel combustion and other human activities account for roughly 70 to 90 percent of the total NO_x emissions in the United States. The remainder comes from aquatic and terrestrial sources, primarily denitrification of acidic soils and waters. (See Annual Report-1983 of the National Acid Precipitation Assessment Program.)

NO_x represents the composite formula for NO (nitric oxide) and NO₂ (nitrogen dioxide). NO is the dominant oxide released initially; however, atmospheric interactions convert NO to NO₂. After considering the results of smog chamber tests and modeling experiments, Trijonis (1978, 1979) concluded that maximum and average NO₂ concentrations tend to be proportional to initial NO_x concentrations. In urban areas, almost all of the NO_x in the atmosphere comes from anthropogenic sources. For rural areas, however, a higher percentage of total NO_x comes from natural sources. As an average, we assume that a one percent reduction in anthropogenic NO_x results in a 0.9 percent reduction in NO₂.

The adverse effects associated with NO_x include:

1. changes in ozone concentrations, which in turn damage health, materials, and agricultural and other vegetation;
2. increases in NO₂, which affect visibility, materials, vegetation, and health; and

3. increases in nitric acidic deposition, damaging fisheries, forests, agriculture, and materials (however, decreases in NO_x also lead to increases in sulfates, thereby offsetting benefits of reduced nitric acidic deposition).

All three groups of effects depend on the geographic distribution of sources of NO_x -- for example, on population densities, climatic conditions, and types of crops. Because of limited information, however, we have not tailored our NO_2 benefit estimates to reflect location as we did for VOCs and ozone.

Following are our estimates for the benefits of controlling NO_x related to reduced NO_2 . Finally, we also present some qualitative results about the corresponding acid rain benefits from reduced nitrates resulting from NO_x control.

5.1 INCREASED VISIBILITY

NO_2 is a reddish-brown gas that reduces visibility by absorbing and discoloring light. In contrast, particulate matter scatters light to reduce visual range. While particulate matter accounts for almost all of the damage to visibility in the East, for some western regions NO_2 may play a significant role in determining visual range.

To bound the value of improved visibility per ton of NO_x reduced, we used the results of Brookshire et al. (1976). Their contingent valuation study showed that recreators were willing to pay \$1.2 million per year (annualized) to avoid visibility reductions that would result from a planned Kaiparowits power plant in southern Utah. Similar studies, by Randall et al. (1974) and by Blank et al. (1977), produced comparable results

for the value of visibility benefits in the Four Corners region. However, these estimates apply only to this particular region, known for its scenic vistas.

In the Kaiparowits study, climatic conditions, emission controls, and other factors allowed the investigators to assume that the major visibility-related impact would be the coloration of the sky by NO₂. Given the projected power plant emissions of 80,000 tons of NO_x per year, the estimate of \$1.2 million of potential damage translates into an upper bound of \$15 per ton (1976 dollars) if damages are allocated entirely to NO_x.

We stress that this figure is probably an upper bound, even for sensitive regions. Although the Kaiparowits area is not densely populated, a large number of recreators use the site. They typically place a high value on protecting clean areas. Further, it is doubtful that NO₂ has a noticeable impact on eastern visibility, where range is limited by buildings and largely influenced by particulate matter (based on a conversation with Shep Burton of Systems Applications, Inc.). To inflate the \$0 - \$15 per ton range to 1983 dollars, we use the Consumer Price Index to yield a range of approximately \$0 - \$26 per ton.

5.2 IMPROVED HEALTH

This section summarizes the studies reported in Air Quality Criteria for Oxides of Nitrogen (1982), the OAQPS staff paper on the NAAQS for nitrogen oxides, the Clean Air Science Advisory Committee's (CASAC's cover letter, the NO_x Regulatory Impact Analysis, and the published literature.

To present these studies, we placed research investigating the health effects of NO₂ into one of four classes:

1. Animal Toxicology Studies. Animals are exposed to controlled levels of NO₂. Researchers have the option of using invasive techniques to investigate the effects of NO₂.
2. Controlled Human Exposure Studies. These are human clinical studies in which humans are exposed to NO₂ in enclosed chambers. They are limited typically to examining the effects of a single, short-term (acute) exposure.
3. Outdoor Epidemiological Studies. Health indicators of cross-sectional groups are statistically related to real-world outdoor ambient concentrations. This class of studies is generally most appropriate to assess the benefits of controlling outdoor air pollution, since health effects are related directly to the control variable of interest.
4. Indoor Epidemiological Studies. Health measures of cross-sectional groups are statistically related to indicators of indoor pollutant concentrations. For example, the "gas stove" studies investigate the effect of indoor air pollution on individuals living in homes with gas stoves (a significant source of NO₂), compared with people living in homes with electric stoves.

5.2.1 Animal Toxicology Studies

Most animal studies involving NO₂ emphasize peak exposure, having been conducted at high concentrations (2 ppm to 20 ppm), roughly 40 to 400 times the annual average ambient NO₂ standard of 0.053 ppm.

A major limitation of these studies is that currently there is no generally accepted method for extrapolating results from animal studies to humans. However, the seriousness of these effects, the biological similarities between humans and test animals, and the relative scarcity of studies showing that

these effects do not occur at exposures near ambient levels suggest that there is some risk to humans from exposure to NO₂. Animal studies are also of value because of the difficulty of observing the cellular processes that lead to changes in pulmonary function without using invasive techniques. Animal experiments represent the best available information on these processes at this time.

Animal studies have clearly indicated that mild and reversible changes in pulmonary function can occur following short-term exposures to NO₂. At least one study (Port et al., 1977) found that repeated peaks of 1 ppm for two hrs/day with a 0.1 ppm background for six months generated emphysema-like changes in mice. Other studies have indicated that NO₂ impairs the respiratory defense mechanism. As such, they support human studies suggesting that NO₂ may be a factor in increased prevalence of respiratory illness among young children living in homes with gas stoves. Another study (Iqbal, 1980) found that nitrosamines formed in the lung, while others (Thomas et al., 1967) have demonstrated cellular changes as a result of NO₂ exposures.

Given the uncertainties inherent in these studies, CASAC concluded that animal studies should only be used as supporting evidence, and then only if the data base for a particular effect is adequate. Otherwise, the study should play no role in standard setting.

Based on the summaries provided by ORD, OAQPS, and CASAC, animal studies can lend support to some of the human-subject studies discussed below. However, because of the relatively high

exposures and the need to extrapolate to humans, these studies are of little help in quantifying health effects at the present time.

5.2.2 Controlled Human Experiments

The majority of these studies have examined the effects of NO₂ on healthy adults by exposing them to single, short-term concentrations in enclosed chambers. A very limited number of clinical studies have also examined potentially sensitive populations (asthmatics and chronic bronchitics), although others (children) have yet to be tested. Table 5.1 summarizes the group of studies reported in the OAQPS staff paper.

In general, these studies indicate that healthy adults are not affected by concentrations of 1 ppm or less. (Kerr et al., 1979; Hackney et al., 1978; Folinsbee et al., 1978; Van Nieding et al., 1973; Posin et al., 1978). Hackney et al., (1978) did report that 5 of 16 healthy adults reported an increase in symptomatic effects (cough, chest tightness, and nasal discharge) after exposure to 1 ppm for 2 hours. However, the difference was not significant over the control group's symptom scores. In a later analysis, Hackney found that neither the normal healthy population nor the asthmatics showed statistically significant increases in specific airway resistance (Shaw) attributable to NO₂ exposure, even after heavy exercise at concentrations of 4 ppm. "Symptoms, diastolic blood pressure, heart rate, skin conductance, and the anxiety inventory showed no meaningful changes attributed to NO₂ exposure" (Hackney 1984, p. 3).

TABLE 5.1

COMPILATION OF EFFECTS REPORTED IN SELECTED HUMAN STUDIES EXAMINING NITROGEN DIOXIDE EXPOSURES

NO ₂ Concentration (ppm)	Exposure Durations	Study Population	Reported Effects	References
0.1 - .2 ppm	1 hr	Asthmatics	Specific airway resistance increased and effect of bronchoconstriction enhanced in 13 of 20 subjects after exposure to NO ₂ . Neither effect observed in 7 of 20 subjects. A bronchoconstrictor (carbachol) was used. One study found no effects.	Crehek 1976 Ahmed et al. (1982) Kleinman et al. (1983) Ahmed et al. (1984)
0.5	2 hr	10 healthy adults 7 chronic bronchitics 13 asthmatics	1 healthy and 1 bronchitic subject reported slight nasal discharge. 7 asthmatics reported mild symptomatic effects. Bronchitics and asthmatics showed no statistically significant changes for all pulmonary functions tested when analyzed as separate groups; however, small but statistically significant changes in quasi-static compliance were found when analyzed as a single group.	Kerr et al. (1979)
0.5 to 5.0	15 min	13 healthy adults 88 chronic bronchitics	Significant decrement in blood gas parameters for both healthy adults and bronchitics. No changes observed below 2.0 ppm.	Von Nieding et al. (1973)
0.5 to 5.0	approx. 3 min	63 chronic bronchitics	Significant increase in airway resistance at or above 1.6 ppm	Von Nieding et al. (1971)
0.6	2 hr	15 healthy exercising adults	No physiologically significant changes in cardiovascular, metabolic, or pulmonary function after 15, 30, or 60 minutes of exercise during the 2-hr exposure	Folinsbee et al. (1978)

COMPILATION OF EFFECTS REPORTED IN SELECTED HUMAN STUDIES EXAMINING NITROGEN DIOXIDE EXPSOURES

NO ₂ Concentration (ppm)	Exposure Durations	Study Population	Reported Effects	References
0.7 to 2.0	10 min	10 healthy adults	Increased inspiratory and expiratory flow resistance of approximately 50% and 10% of control values measured 10 mins. after exposure.	Suzuki and Ishikawa (1965)
1.0	2 hr	16 healthy adults	No statistically significant changes in pulmonary function tests with exception of small changes in forced vital capacity (1.5% mean decrease; p <0.05). Respiratory systems slightly increased after exposure to NO ₂ , but change not statistically significant compared to controls.	Hackney (1978)
1.0 and 2.5	2 hr	8 healthy adults	Increase in airway resistance at 2.5 ppm but not at 1.0 ppm.	Beil and Ulmer (1976)
1.0 and 2.0	2-1/2 hr	10 healthy adults	Alternating exercise and rest produced statistically significant decrease in hemoglobin, hematocrit, and erythrocyte acetylcholinesterase.	Posin et al. (1978)
0.3	1/2 hr	6 asthmatics	After exercising asthmatics showed reductions in lung function.	Bauer et al. (1984)
0.1	1 hr	Asthmatics and healthy adults	Bronchial reactivity unaffected by NO ₂ .	Hazacho (1983)

Some stronger clinical evidence suggests that NO₂ may affect individuals with chronic lung disease (e.g., asthmatics). Kerr et al. (1979) found that 1 of 7 bronchitics and 7 of 13 asthmatics reported various symptoms resulting from exposure to 0.5 ppm for 2 hours with 15 minutes of exercise during exposure. The authors indicated that all the symptoms reported were mild and reversible, including slight headache, nasal discharge, and chest tightness.

One study (Orchek et al., 1976) showed asthmatics experienced increased sensitivity to a bronchoconstrictive agent after one hour of exposure to 0.1 ppm of NO₂. However, the California Air Resources Board (1984) has pointed out that a similar study (Ahmed et al., 1984) did not detect an effect, and that another recent study (Kleinman et al., 1983) at 0.2 ppm "did show indications of such an effect, although the author's conclusion was equivocal" (California Air Resources Board, 1984, p. 3). Because all of these studies used an artificial agent to test bronchial sensitivity, the significance of these findings is controversial. Finally, Bauer et al., showed that exercising asthmatics after exposure to 0.3 ppm and cold air provocation experienced reductions in lung function. However, the sample size (six) was very small.

Kagawa and Tsuru (1979) found that while adult males reported symptoms when exposed to ozone or to ozone and NO₂, no one in their sample had any symptoms during exposure to 0.15 ppm of NO₂ for two hours. Further, the symptoms were not intensified

by combined exposure to ozone and NO₂. There was a very small, but not perceived, decrease in pulmonary function for some of the subjects following exposure to NO₂.

In short, studies involving exposures in the range of 0.5 to 1.5 ppm of NO₂ have reported little or no change in pulmonary function for healthy adults. However, exposure of more sensitive individuals to that same range can reduce pulmonary function. Also, some studies have shown increased sensitivity to agents inducing bronchoconstriction at 0.1 to 0.2 ppm levels of NO₂.

Even if pulmonary function were affected at ambient levels, the changes measured in these studies would not be perceived by normal adults. A small portion of the population with respiratory problems (e.g., asthmatics), however, may be operating near the limit of their lung function when engaged in exercise. For these people, decreases in lung function may affect their ability to perform certain tasks.

Finally, several investigators have examined the effects of multiple pollutants and failed to find any other effects due to NO₂ beyond those found for ozone alone. For example, Hackney et al. (1975) found NO₂ caused little or no change in pulmonary function in healthy subjects exposed to NO₂ and other pollutants concurrently.

5.2.3 Outdoor Epidemiological Studies

Successful studies of this class are scarce, because investigators must separate many confounding effects and health hazards. Further, some of the studies used in setting the existing

annual standard of 0.053 ppm (100 ug/m³) have been criticized because of NO₂ measurement problems.

Table 5.2 summarizes four major studies that found no differences in adults living in areas with relatively high concentrations of NO₂ (a maximum of 0.5 ppm) compared with others in areas with low levels of NO₂. It also presents the results of Kagawa and Toyama (1975), who found changes in lung function due to simultaneous exposure to SO₂, total suspended particulates, and NO₂.

Even though adults were shown not to be sensitive to NO₂, one of the "Chattanooga" studies (Shy et al., 1970) reported a small but significant decrease in children's lung function and a higher incidence of respiratory disease. This study was later criticized because NO₂ concentrations were measured incorrectly. Shy and Love's (1979) follow-up study failed to find any pulmonary function deficits in children during the 1971-72 school year, but NO₂ levels were lower in 1971-72 due to the shutdown of the large NO_x point source in Chattanooga.

Using the Chattanooga data, Perlman et al. (1971) found that of the several respiratory disease indicators assessed, only some bronchitis rates in children were reported higher in the area of maximum NO₂ concentrations. While Portney et al. (1983) found NO₂ had no effect on adults, they did find that peak hourly NO₂ had a significant effect on lost school days for children. Restricted-activity or bed-disability days were also sensitive to NO₂, but not at the 90 percent confidence level.

TABLE 5.2
EFFECTS OF EXPOSURE TO NO₂ ON PULMONARY FUNCTION IN
COMMUNITY EPIDEMIOLOGY STUDIES

Exposure Concentrations (ppm)	Study Population	Reported Effects	References
Median hourly 0.07 NO ₂ Median hourly 0.15 O _x Median hourly 0.35 NO ₂ Median hourly 0.02 O _x	205 office workers in L.A. 439 office workers in San Francisco	No differences in most tests. Smokers in both cities showed greater changes in pulmonary function than non-smokers.	Linn et al., 1976
High exposure area 24 hr high 0.055 NO ₂ .035 SO ₂ 1-hr mean High exposure area 0.14 NO ₂ to 0.30 NO ₂ Low exposure area 0.06 NO ₂ to 0.09 NO ₂	128 traffic policemen in urban Boston and 140 patrol officers in nearby suburbs	No difference in various pulmonary function tests	Speizer and Ferris, 1973; Burgess et al. 1973
High exposure group: Estimated 1-hr max 0.25 to 0.51 NO ₂ Annual mean 24-hr 0.051 NO ₂ Low Exposure group: Estimated 1 hr max 0.12 to 0.23 NO ₂ Annual mean 24 hr 0.01 NO ₂	Non-smokers in L.A. (adult)	No differences found in several ventilatory measurements including spirometry and flow volume curves.	Cohen et al. 1972

EFFECTS OF EXPOSURE TO NO₂ ON PULMONARY FUNCTION IN
COMMUNITY EPIDEMIOLOGY STUDIES

Exposure Concentrations (ppm)	Study Population	Reported Effects	References
1 hr conc. at time 0.02 of testing (1:00 p.m.) to 0.19 NO ₂	20 school age children 11 years of age	During warmer part of year, NO ₂ , SO ₂ , and TSP signi- ficantly correlated with V _{max} at 25% and 50% FVC specific airway con- ductance. Significant correlation between each of four pollutants (NO ₂ , NO, SO ₂ and TSP) and V _{max} at 25% and 50% FVC; but no clear delineation of specific pollutant con- centrations at which effects occur.	Kagawa and Toyama, 1975
Average of 2 week daily 1 hr peaks 0 to .23 ppm	Adults and children Health Interview Survey (HIS) data	No effect for adults. No significant effect on chil- dren's minor restricted activity day or on bed dis- ability day. Significant effect on school-loss days, but other pollutants had the wrong sign.	Portney et al. 1983

Finally, although Kagawa and Toyama (1975) found significant health effects from exposure to a number of pollutants, many have been very critical of the Kagawa and Toyama (1975) study because of the failure to correct for simultaneous exposure to other pollutants and temperature. The Criteria Document (EPA 1982) concluded that the study could not provide proof that NO₂ induced any health changes.

5.2.4 Indoor Epidemiological Studies

Table 5.3 presents the principal studies examining the effects of gas-stove cooking (and corresponding higher levels of NO₂). In short, children living in gas-stove homes may have small, but statistically significant, decreased pulmonary function and more coughs, colds, or other respiratory disease symptoms. Comstock et al. (1981) found health effects from gas-stove use for nonsmoking adult males but also found that gas-stove cooking appears to decrease the relative risk of chest illness and breathlessness in women (i.e., women living in gas-stove homes were less likely to incur breathlessness and chest illness). Heising et al. (1982) duplicated the results for nonsmoking adult white males using the same data base, but did not use the observations for females. Note that Table 5.3 also indicates that several studies, including EPA's own study, found no effects in children or adults.

TABLE 5.3
 COMPILATION OF REPORTED EFFECTS ASSOCIATED WITH EXPOSURE TO NO₂ IN THE HOME IN COMMUNITY STUDIES INVOLVING GAS STOVES^a

NO ₂ Concentration (ppm)	Study Population	Reported Effects
95th percentile of 24 hr avg in activity room 0.02 - 0.06 (gas) 0.01 - 0.05 (elec.) Frequent peaks in 1 home of 0.4-0.6 (gas). Maximum peak 1.0 (gas).	8,120 children, ages 6-10, 6 different cities, data also collected on history of illness: before age 2.	Significant association between history of serious respiratory illness before age 2 and use of gas stoves ($p < .01$). Also, small but statistically significant decreases in pulmonary function (FEV ₁ and FVC) in children from gas-stove homes. Speizer et al., 1980
NO ₂ concentrations not measured at time of study.	2,554 children from homes using gas to cook compared to 3,204 children from homes using electricity, ages 6-11.	Proportion of children with one or more respiratory symptoms or disease (bronchitis, day or night cough, morning cough, cold going to chest, wheeze, asthma) increased in homes with gas stoves v. electric stove homes (for girls $p \leq 0.10$; boys not sig.) after controlling for confounding factors. Melia et al., 1977
NO ₂ concentrations not measured in some homes studied for health effects.	4,827 children, ages 5-10.	Higher incidence of respiratory symptoms and disease associated with gas stoves (for boys $p = 0.02$; girls $p \leq 0.15$) for residences in urban but not rural areas, after controlling for confounding factors. Melia et al., 1979
Kitchens (weekly avg.): 0.005-0.317 (gas) 0.006-0.188 (elec.) Bedrooms (weekly avg.): 0.004-0.169 (gas) 0.003-0.0.37 (elec.)	808 children, ages 6-7.	Higher incidence of respiratory illness in gas-stove homes ($p \leq 0.10$). Prevalence not related to kitchen NO ₂ levels, but increased with NO ₂ levels in bedrooms of children in gas-stove homes. Lung function not related to NO ₂ levels in kitchen or bedroom. Floria et al., 1979 and Goldstein et al., 1979. Both are companion papers to Melia et al. 1979
Sample of households 24 hr, avg: 0.005-0.11 (gas) 0-0.06 (elec.) 0.015-0.05 (outdoors)	128 children, ages 0-5 346 children, ages 6-10 421 children, ages 11-15	No significant difference in reported respiratory illness between homes with gas and electric stoves in children from birth to 12 years. Mitchell et al., 1974. See also Keller et al., 1979

COMPILATION OF REPORTED EFFECTS ASSOCIATED WITH EXPOSURE TO NO₂ IN THE HOME IN COMMUNITY STUDIES INVOLVING GAS STOVES^a

NO ₂ Concentration (ppm)	Study Population	Reported Effects
Sample of household same as reported in Mitchell et al.	174 children under 12	No evidence that cooking mode is associated with the incidence of acute respiratory illness. Keller et al., 1979
See above for monitoring.	Housewives cooking with gas stoves, compared to those cooking with electric stoves. 146 households.	No evidence that cooking with gas is associated with an increase in respiratory disease. Keller et al., 1979
See above for monitoring.	Members of 441 households.	No significant difference in reported respiratory illness among adults in gas v. electric cooking homes. Mitchell et al., 1974
Preliminary measurements peak hourly .25-0.50, max. 1.0.	Housewives cooking with gas stoves, compared to those cooking with electric stoves.	No increased respiratory illness associated with gas stove usage. U.S. EPA, 1976
NO ₂ concentrations not measured.	1900 adults.	Increased frequency of respiratory symptoms and impaired ventilatory functions among men but more than off-setting decrease in respiratory symptoms in women. Comstock et al., 1981
NO ₂ concentrations not measured.	708 adults.	Used same data set as Comstak et al., 1981. Increased increase of respiratory symptoms in non-smoking adults living in homes with gas stove Heilsing et al., 1982
NO ₂ concentrations not measured.	229 subjects (117 families)	Asthmatics had significantly high wheezing when living in homes with gas stoves. Others had high inflammation of nasal mucas membranes (Rhmitys). Did not correct for smoking, other pollutants, sex, pollen or other confounding factors. Lebowitz et al., 1982

COMPILATION OF REPORTED EFFECTS ASSOCIATED WITH EXPOSURE TO NO₂ IN THE HOME IN COMMUNITY STUDIES INVOLVING GAS STOVES^a

NO ₂ Concentration (ppm)	Study Population	Reported Effects
95th percentile of 24 hr avg in activity room 0.02 - 0.06 (gas) 0.01 - 0.05 (elec.) Frequent peaks in 1 home of 0.4-0.6 (gas). Maximum peak 1.0 (gas).	8,120 children, ages 6-10, 6 different cities, data also collected on history of illness before age 2.	Reanalysis of Speizer et al., 1980, reversed all the significant findings. When controlling for parental education reductions in pulmonary function and history of respiratory illness before age 2 were no longer significantly related to gas-stove homes. Ware et al., 1983

^a Exposures in gas-stove homes were to NO₂ plus other gas combustion products.

5.2.5 Estimation of Health Benefits

Based on the evidence reviewed here and elsewhere, asthmatics, chronic bronchitics, children, and individuals with other chronic respiratory diseases are considered groups susceptible to NO₂ exposure. Table 5.4 summarizes this evidence, indicating that NO₂ concentrations may pose health risks to these individuals.

The studies demonstrating effects on children and asthmatics are largely the "gas-stove" investigations. Based on these results, OAQPS has concluded that "repeated peaks in the range of 0.15 to 0.30 ppm may be of concern for children" (p. 2 of the executive summary of the OAQPS staff paper). Much higher chamber exposures are needed to produce health effects in the other sensitive populations. The minimum exposure inducing health effects for chronic bronchitics was 0.50 ppm, and for all others, roughly 1.60 ppm.

Information on outdoor peak exposures is needed to estimate possible benefits from NO₂ control. Currently, only the Los Angeles area is not meeting the annual NO₂ standard of 0.053 ppm. According to the OAQPS staff paper, areas attaining the standard would experience at most two to three days where NO₂ peaked at levels greater than 0.30 ppm. Figure 5 in the OAQPS staff paper indicates that 0.50 ppm probably would never occur: "... the available monitoring data indicate 1-hour peaks of 0.30 ppm are rarely seen and hourly peak levels never have been reported at or above 0.50 ppm" (OAQPS staff paper, p. 53).

TABLE 5.4

SUMMARY OF STUDIES INDICATING HEALTH EFFECTS OF NO₂

Sensitive Groups	Health Effects/Symptoms	Reference for Supporting Evidence	Exposure	Increase in Prevalence of Health Effects
Children (53 million)	-Increased prevalence of respiratory illness	Melia et al. (1979)	.15 - .3 ^a repeated peaks	Maximum of 4 percent
	-Decreased pulmonary function of .7 percent and increased prevalence of respiratory illness prior to age two	Speizer et al. (1980, 1983)	.15 - .3 ^a repeated peaks	3 - 3.5 percent
Asthmatics (6 million)	-Mild and reversible headache, chest tightness, or nasal discharge	Kerr et al. (1979)	.5 ppm for 2 hours	7 of 13 asthmatics were affected
	-Reductions in Lung function	Baurer et al. (1984)	.3 ppm for 112 hour	5 of 6 asthmatics were affected
Chronic Bronchitics and Emphysematics ^b (7 million)	-Increased airway resistance	Von Nieding et al. (1971)	1.6 ppm for 3 minutes	14 percent of bronchitics experienced increased airway resistance

a/ Studies used a gas-stove variable to approximate exposure. This is an estimate based on the OAQPS staff report and Spengler et al. (1979).

b/ Emphysematics are assumed sensitive even though no study has shown any effects. would never occur: "... the available monitoring data indicate

We have presented evidence suggesting that significant health effects occurring at ambient levels of NO_2 have not been demonstrated. (Also see the OAQPS staff paper and the accompanying CASAC cover letter, which conclude that current ambient levels provide a significant margin of safety, even for sensitive individuals.) But some studies have shown that very high indoor levels of NO_2 may induce health effects. Thus, we have estimated the health benefits that may result from reduced indoor NO_2 generated by reduced ambient NO_x .

Interpolating from the gas-stove studies in Table 3.5 that indicate significant health effects, we find that the benefits from reducing ambient NO_2 are very small. And if we use the latest studies (Ware et al., 1983) showing no health effects from ambient NO_2 levels, health benefits would not be measurable.

Melia (1979) and Speizer et al. (1980, 1983) found that, relative to children living in homes with electric cooking, an additional 3-4 percent of all children (32.4 per 1,000) living in homes with gas stoves had increased respiratory illness or symptoms. To estimate the upper bound of benefits, we assume:

- (a) repeated peak exposures in gas-stove homes average 0.18 ppm, and
- (b) repeated peak exposures in electric-stove homes average 0.06 ppm.

These assumptions lead to high benefit estimates. Spengler et al. (1979), reporting on monitoring results of gas- and electric-stove homes, stated that the indoor difference typically "ranges from

3 to 7 times larger for gas" (p. 1,279). To estimate an upper-bound estimate, we use a mean repeated peak exposure of 0.18 ppm for houses with gas stoves and a repeated peak exposure of 0.06 for houses with electric stoves, the lowest differential suggested by Spengler et al.

With these two assumptions, we can conclude that an increase in repeated peak exposures of 0.12 ppm (0.18 - 0.06) leads to an increase in childhood symptoms for 4 percent of the total primary-school-age population -- the high range suggested by the indoor studies. Summary statistics presented in Speizer et al. (1980) indicate that roughly 22 percent of all children experience respiratory illnesses before age 2. Melia et al. (1979) indicated that roughly 28 percent of school-age population experience at least one respiratory illness or symptom per year.

Summary statistics presented in Portney et al. (1983) show that the two-week average daily maximum **NO₂** concentration that metropolitan children are exposed to is roughly .0589 ppm. To relate changes in indoor **NO₂** concentrations to changes in ambient concentrations, we use the analysis of Sexton et al. (1983). They combined ambient and indoor data from monitoring programs in six U.S. cities to relate indoor and outdoor **NO₂** levels. Their regression analysis indicates that every .01 ppm change in outdoor **NO₂** changes indoor **NO₂** by .006 ppm.

Based on these results, we use the following steps to quantify possible health benefits due to **NO₂** reductions. We stress, however, that these results are extremely preliminary.

Until more research is completed, the existence of any significant health benefits from NO_2 reductions at current ambient levels remains an open question.

1. On average, a 10 percent reduction in NO_x (roughly 2,150,000 tons) would reduce our population-weighted measure of NO_2 exposure by 0.0053 ppm ($0.1 \times 0.9 \times 0.0589$ ppm). (Recall that, on average, a 1.0 percent reduction in man-made emissions would reduce ambient concentrations by 0.9 percent).
2. A 0.0053 ppm reduction in ambient concentrations would, on average, reduce indoor repeated peak concentrations by 0.00318 ppm (0.6×0.0053 ppm). (On average, a 1.0 percent reduction in outdoor NO_2 would reduce indoor NO_2 by 0.6 percent.)
3. Based on the increased incidence indicated by the gas-stove studies (an increase in NO_2 exposure of 0.12 ppm leads to increase respiratory symptoms in 4.0 percent of the children), and assuming linearity, we calculate that a reduction of .00318 ppm in indoor concentrations would reduce the number of children with respiratory symptoms by 0.106 percent ($(0.00318/0.12) \times 4.0$). That is, roughly one out of every thousand children would experience reduced respiratory symptoms.
4. Although only half of the homes in the United States have gas stoves, we will assume that all children benefit as much as the children in gas-stove homes. With a relevant childhood population of roughly 50 million, this translates into 53,000 children with decreased respiratory incidence. Dividing that figure by our number of tons of NO_x reduced (2,150,000) yields 0.02465 children experiencing diminished respiratory symptoms per ton of NO_x reduced.

The lack of credible data does not permit us to categorize the decreased respiratory illnesses/symptoms into bed and minor restricted activity days similar to the ozone analysis. However, since these symptoms are reversible -- typically mild headache, cough, chest tightness, etc., -- we place a range of \$50 to \$70 per child that avoids a respiratory illness. This translates into a value of \$ 1.23 per ton of reduced NO_x .

This estimate is probably too high for the following reasons:

1. The research on **NO₂-induced** health effects generally agrees that fairly high levels of **NO₂** are necessary to generate health effects. We assumed that children living in homes with electric stoves (and low levels of **NO₂**) would benefit as much as children in gas-stove homes.
2. The estimate of \$50-\$70 as the average household's willingness to pay for a child to avoid increased wheezing or coughing is probably high.
3. We attributed all of the differences in health effects found in the gas-stove studies to **NO₂** differences. Other pollutants are also emitted by gas stoves, and increased water vapor also occurs in gas-stove homes.

For the other sensitive populations, we rely on the OAQPS staff paper, which suggests that at current ambient levels these groups are not at risk. In discussing the Kerr et al. (1979) study, the only one to find health effects at 0.5 ppm, OAQPS wrote: "This study did not demonstrate measurable impairment of pulmonary function in healthy adults or asthmatics and chronic bronchitics exposed for short periods (2 hours) to **NO₂** concentrations at 0.5 ppm or below." Thus, although mild symptoms were reported for some asthmatics at 0.5 ppm, that level of **NO₂** probably would never be experienced in the United States. CASAC also agrees that based on the latest data, the current **NO₂** standard will "ensure an adequate margin of safety of protection against both long-term and short-term health effects" (CASAC letter, p. 6). We therefore conclude that any

health benefits would be generated from the reduction of the indoor peak exposures by children and possibly other sensitive populations.

We have presented calculations to show that our estimate of health benefits is relatively small, at least for NO_2 acute health benefits. We have not quantified the benefits of reduced exposure to asthmatics living in homes with gas stoves. The literature has not presented clear enough evidence on this matter.

We also have not quantified any reduction in chronic health effects. Because repeated episodes of respiratory-tract irritation and illness in children may carry into adult life in the form of decreased lung function and chronic bronchitis, NO_x reductions may also reduce adult cases of chronic bronchitis. High and long-term exposures have also been shown to induce emphysema-like changes in animals. Thus, there may be long-term chronic health effects that we were not able to quantify.

Finally, we do note that despite considerable efforts, researchers have yet to estimate dose-response relationships for chronic or acute health effects. This suggests that the effects from NO_x exposure may be very small, since a clear statistical relationship has not been shown to exist, especially for the long-term health effects. Because of the lack of adequate dose-response information, we also caution against any strict interpretations of our estimates of reduced damage to health. Rather, these calculations were made to place these possible health benefits in perspective with the other benefit categories.

5.3 REDUCED MATERIAL DAMAGE

Field studies and laboratory research have demonstrated that nitrogen oxides can significantly fade textile dyes. Table 5.5 presents damage estimates by Barrett and Waddell (1973), inflated to 1983 dollars using the Textile Products and Apparel Index.

The basis for the estimates included not only the reduced wear life of textiles of moderate fastness to NO_x but also the costs of research and quality control. The major share of the costs is the extra expense involved in using dyes of higher NO_x resistance and in using inhibitors. Additional costs also are incurred in dye application and in increased labor expenditures. The factors relating higher costs in the textile industry to NO_x are discussed in Chapter 8 of NAS (1976).

If we assume linearity (incremental damages are equal to average damages per ton) we calculate a \$13 per-ton estimate of the benefits of reducing NO_x emissions by dividing total damages (\$280 million) by the total tons of man-made NO_x emissions per year (21,400,000).

5.4 ENHANCED PLANT GROWTH

Data concerning the effects of NO_2 on plant growth and yield are limited. Nevertheless, it is reasonable to assume that **NO_2 -induced** reductions in the assimilative capacity of plants through altered metabolism, leaf injury, or abscission also affect plant growth.

TABLE 5.5

TOTAL COSTS OF DYE FADING
IN TEXTILES DUE
TO NO_x
(millions of 1983 dollars)

<u>Effect</u>	<u>Costs</u>
Fading on acetate and triacetate	166
Fading on viscose rayon	50
Fading on cotton	50
Yellowing of white acetate-nylon-spandex	<u>14</u>
Total	280

NOTE: All costs are rounded to nearest million and inflated to 1983 dollars.

Source: Barrett and Waddell (1973).

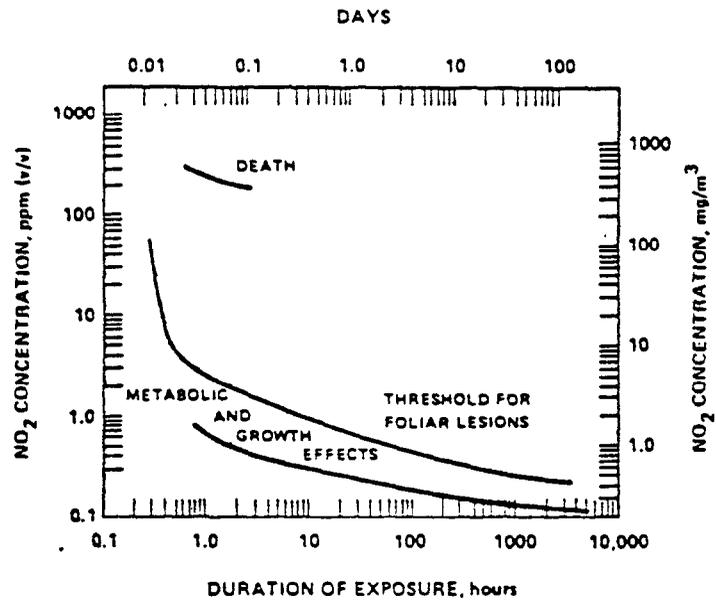


Figure 5.1 Threshold curves for the death of plants, foliar lesions, and metabolic or growth effects as related to the nitrogen dioxide concentration and the duration of exposure (McLean, 1975).

Three threshold curves depicting levels of peak and long-term exposure that plants can tolerate before death, foliar lesions, and growth effects are presented in Figure 5.1. These curves, taken from EPA (1982), are based on 14 different studies. The lower curve represents a lower-bound concentration that injures plants; no measurable effects have been reported for NO₂ concentrations below these doses.

Using these curves, MacLean (1975) concluded that average NO₂ concentrations are well below the threshold curve for damage to growth. In fact, the maximum NO₂ concentrations recorded in Los Angeles for 1966 would just begin to damage growth. Even in Los Angeles, however, average NO₂ concentrations are below the threshold for longer averaging periods.

Although NO₂ by itself is unlikely to damage plants at existing outdoor levels, several studies have demonstrated synergistic and antagonistic effects. The Criteria Document (EPA, 1982) concluded that "concentrations of NO₂ between 0.1 ppm and 0.25 ppm can cause direct effects on vegetation in combination with certain other pollutants" (pp. 12-44). But these data indicate that rural areas are still not at risk, since concentrations will not in general rise above 0.1 ppm for a sufficient time to allow damages to occur. Conversations with investigators at the National Crop Loss Assessment Network indicate that some very large point sources would have to be next to the fields for any significant crop damage to occur.

Therefore, we limit our estimate of benefits to account for urban-related vegetation damages. The plants most in danger would tend to be ornamental vegetation. Even these damages are likely to be small. Leighton et al. (1983) conclude from their review that "ozone appears to account for more than 90 percent of total vegetation damages" (p. 60; see also Heck et al., 1982, and Page et al., 1982). If NO₂ accounted for the other 10 percent of the total ornamental vegetation damage, as an upper bound we can use the high estimate of ozone ornamental vegetation damages of \$140 million to impute total NO₂ damages of roughly \$16 million. Assuming linearity, each ton of NO₂ emissions could cause \$.75 in damages.

5.5 ACID DEPOSITION BENEFITS OF REDUCING NO_x

In addition to Ozone and NO₂ effects, the other major benefit category from NO_x control is reduced acidic deposition. Acid deposition occurs when NO_x and SO₂ emissions are chemically altered into acids in the atmosphere and transported over long distances, or when the precursor emissions are acidified after being deposited in dry form on plant, soil, or building surfaces. Both wet and dry forms of acidic deposition are harmful to aquatic, terrestrial, and material resources. While these damages are potentially important in some regions, basic scientific understanding of the effects of acid deposition on resources and transport processes is quite limited.

The computation of damages in dollar-per-ton units requires several crucial pieces of information. Unfortunately, many of these data are so uncertain that confidence intervals of damage estimates are exceptionally wide. Dollar estimates of damage require concentration-response functions and a tabulation of the resources at risk. Reliable concentration-response data are lacking in nearly all resource areas, especially forestry and materials, and inventory data are unavailable for most material resources. Finally, translating damage measured in dollars to dollars per ton requires information on the relative contribution of nitrates and sulfates to total damages as well as the relationship between tonnage of NO_x emissions and nitrate deposition. All of these are the subjects of intensive ongoing research. Until more of this research program is completed, quantitative damage estimates will not be accurate enough to be useful in a policy context.

Although quantitative estimates are not possible at this time, we do have a qualitative sense of the seriousness of acidic damage. Nitrates account for roughly 30 percent of total acidic loadings, and the damage information available indicates that the proportion of nitrates to total damages is less than that implied by the relative emittant loadings. Also, there is less evidence of nitrate relative to sulfate damage, and nitrates are actually beneficial as nutrients in nitrogen-poor soils.

Finally, Seigneur et. al. (Science 1984) show that a 50 percent reduction in eastern NO_x will decrease nitrates by roughly 45 percent. However, this same NO_x reduction will

increase sulfates by 8 percent. Thus, at least some of the reduced nitrate deposition would be offset by increases in sulfate deposition.

5.6 SUMMARY

The benefits per ton of NO_x controlled related to NO_2 are presented in Table 5.6. Given the current status of reliable dose-response information, we were not able to separate the benefits to rural and metropolitan areas or estimate a range of benefits. Also, because of insufficient data, some benefit categories could not be quantified, while others could only be estimated with some very rough calculations. Therefore, we only present one estimate for direct NO_2 benefits: \$42 per ton of NO_x controlled. It is clear that additional research on NO_x benefits would allow us to be more certain of our estimates.

CHAPTER 6

SUMMARY OF BENEFITS OF VOC AND NO_x CONTROL

Tables 6.1 and 6.2 summarize the estimated benefits per ton, in both monetized and nonmonetized terms. Although we were not able to account fully for spatial and temporal characteristics, most of the estimates reflect the basic differences between metropolitan and rural areas. Thus, we were able to generate two separate estimates for each pollutant--the benefit per ton in metropolitan areas and the benefit per ton in rural areas.

Perhaps the most surprising result is the relatively small contribution that direct health benefits make to the totals. The range for both pollutants is substantial. For VOCs, a precise estimate of the health benefits is not possible, since the potency of VOCs varies. We did, however, present evidence suggesting that it is very unlikely that the direct health benefits per ton of VOCs reduced would be outside the range of \$0 to \$100 per ton. For NO_x, sufficient evidence on NO_x health benefits does not exist to allow as much certainty.

Our range of benefits is also wide. For example, on average our range of metropolitan benefits goes from \$241 to \$656 per ton for VOC control and from \$(-168) to \$(-64) per ton for NO_x control. Even that wide range, however, does not reflect the full degree of uncertainty. Most of these uncertainties are scientific and technical, rather than economic. They include

(1) the link between ozone and crop losses, material damages, and health effects; (2) the link between reductions in VOCs-NO_x and ozone concentrations; and (3) the potencies and exposure factors for specific VOCs.

In addition to these uncertainties, the quantitative benefit estimates suffer from the omission of several benefit categories, including possible chronic health effects from NO_x and ozone. Also, some VOCs may have noncarcinogenic toxic effects, which we were not able to quantify.

We should note that some of the analysis has incorporated the implicit assumption of a linear relationship between damages and pollutant concentrations. (This was not true in the case of ozone health effects.) To the extent that damage functions are nonlinear, additional uncertainty is introduced. It is not clear, however, whether the assumption of linearity is likely to lead to an under-or overestimate of the benefits (except in the case of the direct cancer-related benefits, where the assumption of linearity is likely to lead to an overestimate).

For these reasons, we are reluctant to draw firm conclusions. However, we believe that this work represents our best estimates of the benefits of VOC and NO_x control. For example, this latest report used results from regional oxidant models to generate estimates of second-day rural ozone effects, helping us refine further our estimates of the rural ozone-VOC-NO_x relationship. Other drafts of this analyses did not have these results available.

TABLE 6.1

Benefits per Ton of VOC Controlled in
Metropolitan and Non-metropolitan Areas
(1983 dollars per ton)

<u>Ozone-Related Benefits</u>	<u>Metropolitan</u>		<u>Non-metropolitan</u>	
	<u>Range</u>	<u>Point Estimate</u>	Range	Point Estimate
Health				
Value of Reduced Bed/Work Loss Days (Reduced cases per ton)	71 - 365 (.89) - (3.)	159 (1.6)	6 - 29 (.07) - (.25)	12 (.12)
Value of Reduced Minor Days (Reduced cases per ton)	9.3 - 111 (2.6) (8.9)	38 (4.7)	1 - 9 (.2) (.7)	3 (.4)
Value of Reduced non- respiratory cases (Reduced cases per ton)	32 - 32 (10.8) (10.8)	32 (10.8)	3 - 3 (.9) (.9)	3 (.9)
Agriculture	80 - 94	88	126 - 196	160
Nonagricultural Vegetation Ornamentals	4 - 4	4	1 - 1	1
Material Damage	43 - 49	45	10 - 15	12
Total Ozone-Related Benefits per Ton of VOCs.	241 - 656	365	145 - 251	190

Direct VOC Benefits

Cancer Cases Avoided Evidence suggest a range of \$0 - \$100. Point estimate depends on specific VOCs and exposure patterns.

Noncarcinogenic Toxic Effects Unquantified

Final Weighted Average of Metro. and Rural Benefits per Ton \$325 + direct health effects of VOC

TABLE 6.2

Benefits per Ton of NO_x Controlled in
Metropolitan and Non-metropolitan Areas
(1983 dollars per ton)

<u>Ozone-Related Benefits</u>	<u>Metropolitan</u>		<u>Non-metropolitan</u>	
	<u>Range</u>	<u>Point Estimate</u>	<u>Range</u>	<u>Point Estimate</u>
Health				
Value of Reduced Bed/Work Loss Days (Reduced cases per ton)	-58 to -4 (-.5) - (-.05)	-22 (.2)	27 - 136 (.3) - (1.1)	59 (.59)
Value of Reduced Minor Days (Reduced cases per ton)	-18 to -1 (-1.4) (-.15)	- 5 (-.6)	4 - 41 (1.)- (3.3)	14 (1.8)
Value of Reduced Non- respiratory Cases (Reduced cases per ton)	-4 -4 (-1.5) (-1.5)	-4 (-1.5)	12 - 12 (4) - (4)	12 (4)
Agriculture	-79 to -51	-64	187 - 290	237
Nonagricultural Vegetation Ornamentals	-.5 - .5	-.5	1.3 - 1.3	1.3
Material Damage	-8 to -4	-6	14 - 21	17
Total Ozone-Related Benefits per Ton of NO _x Controlled	-168 to -64	-102	244 - 502	340

(continued)

TABLE 6.2 (continued)

	<u>Point</u>
<u>Direct Benefits of NO₂</u> (Not broken down by metropolitan/rural)	
Visibility	26
Health	2
Material Damage	13
Vegetation ^b	<u>1</u>
Total NO ₂ Benefits	42

Final weighted average of Metro. and rural Ozone Benefits per Ton and Direct NO₂ Benefits per Ton = \$50.

Unquantified Benefits

- Reduced Chronic Health Effects of Ozone and NO₂^a
- Reduced Acidic Deposition Related to NO_x

a/
To the extent that chronic health effects exist for both ozone and NO₂, they will offset each other, since in heavily populated areas NO_x tends to reduce ozone.

b/
Rounded to the nearest dollar.

References

- Adams, R., and McCarl, B., "Assessing the Benefits of Alternative Oxidant Standards on Agriculture: The Role of Response Information," completed for U.S. EPA, September 1983.
- Adams, M. et al., "Economic Effects of Ozone on Agriculture." Final Report to U.S. EPA.
- Anderson, G. et al., "Development and Application of Methods for Estimating Effects of Industrial Emission Controls on Air Quality Impact of Reactive Pollutants." U.S. EPA July, 1984.
- Barrett, Larry B. and Waddell, Thomas. The Cost of Air Pollution Damages: A Status Report. Research Triangle Park, U.S. EPA 1973.
- Bates, D. and Sizto, R., "Relationship Between Air Pollution Levels and Hospital Admissions in Southern Ontario." Canadian J. of Pub. Health, Vol. 79, March/April, 1983.
- Blank, F.M., et al., 1977. Valuation of aesthetic Preferences: "A Case Study of the Economic Value of Visibility." Draft report to the Electric Power Research Institute, Palo Alto, Calif.
- Brookshire, D.S., et al., 1976. "The Valuation of Aesthetic Preferences." J. Environ. Econ. Manag. 3:325-346.
- Brown, C., "Equalizing Differences in the Labor Market," Quarterly Journal of Economics, Vol. 94, 1980.
- California Air Resources Board, Letter and Comments on Proposed Reaffirmation of the NAAQS for Nitrogen Dioxide sent to William D. Ruckelshaus; May 21, 1984.
- Chock, D., et al., "Effect of NO_x Emission Rates on Smog Formation in the California South Coast Air Basin", Environmental Science and Technology, Vol. 15, No. 8, August 1981.
- Clean Air Sciences Advisory Committee's cover letter to the OAQPA Staff Paper; "Review of the NAAQS for Nitrogen Oxides: Assessment of Scientific and Technical Information.
- Cohen, C., et al., "Respiratory Symptoms, Spirometry, and Oxidant Air Pollution in Nonsmoking Adults." Amer. Rev. Resp. Disease 105:251-261, 1972.
- Council on Environmental Quality, Environmental Quality - 1980, Government Printing Office, December 1980.
- Crocker, T., and Vaux, H., "Some Economic Consequences of Ambient Oxidant Impacts on a National Forest," completed for U.S. EPA, Office of Policy Analysis, August 1983.

Detels, R., et al., "The UCLA Population Studies of Chronic Obstructive Respiratory Disease," American Journal of Epidemiology, Vol. 109, 1979.

Durand, D., Stable Chaos, Morristown, N.J., General Learning Corporation, 1971.

Evans, et al., "Ozone Measurement from a Network of Remote Sites," Journal of the Air Pollution Control Association, Vol. 32, No. 4, April 1983.

Faucett Associates, Draft Report: Review and Critique of Previous OMSAPC Cost-Effectiveness Analysis, March 1983.

Ferris, B., Jr., "Health Effects of Exposure to Low Levels of Regulated Air Pollutants," Journal of the Air Pollution Control Association, Vol. 28, No. 5, May 1978.

Flore, C. du V. et al., 1979. "The Relation Between Respiratory Illness in Primary Schoolchildren and the Use of Gas for Cooking. III." Nitrogen dioxide, respiratory illness and lung infection. Int. J. Epidemiol. 8:347-353

Folinsbee, L. J. et al., 1978. "Effect of 0.62 ppm NO₂ on Cardiopulmonary Function in Young Male Non-Smokers." Environmental Research 15:199-205.

Freas, W., "A Digital Solution to City-Specific Ekma Isopleth Diagrams in Ozone Regulatory Analysis." U.S. Environmental Protection Agency, Research Triangle Park, NC. July 1983

Freeman, A.M., III, Air and Water Pollution Control: A Benefit-Cost Assessment, New York: John Wiley and Sons, 1982.

Friedlander, Sheldon K., Chairman, Clean Air Scientific Advisory Committee (CASAC), 1982. Memorandum to EPA Administrator. Subject: CASAC Review and Closure of the OAQPS Staff Paper for Nitrogen Oxides. July 6, 1982.

Gerking, S.; Stanley, L.; and Weirick, W., "An Economic Analysis of Air Pollution and Health: The Case of St. Louis," report to U.S. EPA, Office of Policy Analysis, July 1983.

Glasson, W., "Effect of Hydrocarbons and NO_x on Photochemical Smog Formation Under Simulated Transport Conditions," Journal of the Air Pollution Control Association, Vol. 31, No. 11, November 1981.

Glasson, W. A., and Tuesday, C. S. Environ. Sci. Technol. 1970, 4, 37.

Goldstein, E., moderator, Photochemical Air Pollution, inter-departmental conference sponsored by the Department of Medicine, University of California, School of Medicine, Davis, California, 1982.

Goldstein, E. et al., 1977. "Fate and Distribution of Inhaled Nitrogen dioxide in Rhesus Monkeys." Am. Rev. Respir. Dis. 115:403-412

Goldstein, B. D. 1979. "The Relation Between Respiratory Illness in Schoolchildren and the Use of Gas for Cooking. II." Factors affecting nitrogen dioxide levels in the home. Int. J. Epidemiol. 8:339-345.

Green, A. E. S. et al., "An Interdisciplinary Study of the Health, Social, and Environmental Economics of the Sulfur Oxide Pollution in Florida." Interdisciplinary Center for Aeronomy and (other) Atmospheric Sciences, Univ. of Florida, Gainesville, FL. 1978.

Hackney, J. D. et al., 1975a. "Experimental Studies on Human Health Effects on Air Pollutants. II." Four-hour exposure to ozone alone and in combination with other pollutants. Arch. Environ. Health 33:379-384.

Hackney, J. D. et al., 1975b. "Experimental Studies on Human Health Effects on Air Pollutants. III." Two hour exposure to ozone alone and in combination with other gases. Arch. Environ. Health 30:176-181.

Hackney, J. D., 1976. "Effects of Atmospheric Pollutants on Human Physiologic Function." Final report, U.S. EPA.

Hackney, J. D. et al., 1978. "Experimental Studies on Human Health Effects of Air Pollutants." IV. Short-term Physiological and clinical effects of nitrogen dioxide exposure. Arch. Environ. Health 33:176-181.

Hammer, D., et al., "Los Angeles Student Nurse Study. Daily Symptom Reporting and Photochemical Oxidants," Archives of Environmental Health, Vol. 28, 1974.

Hasselblad, v. "Modeling Dose Response Relations for Health Effects Data", Environmetrics 81: selected papers, SIAM, Philadelphia, 1981.

Hasselblad, V., and Svendsgaard, D., "Reanalysis of the Los Angeles Student Nurse Study," U.S. EPA, Health Effects Research Lab, Research Triangle Park, NC. August 1975.

Heck, W., et al., "A Reassessment of Crop Loss from Ozone," Environmental Science and Technology, Vol. 17, No. 12, 1983.

Heck, W. et al., "Nitrogen, Dioxide: Time-Concentration Model to Predict Acute Foliar Injury." U.S. EPA, Corvallis, Oregon. Pub. No. 600/3-79-057. 1979.

Heintz et al., "National Damages of Air and Water Pollution: A report submitted to U.S. EPA, 1976.

- Horvath, S. M. and L. J. Folinsbee. 1979. "Effects of Pollutants on Cariopulmonary function." Report to U.S. EPA
- Innes, W. B., Environmental Science and Technology. Vol. 15, 933, 1981.
- Kagawa, J. et al., 1975. "Photochemical Air Pollution: Its Effects on Respiratory Function of Elementary School Children." Arch. Environ. Health 30:117-122.
- Keller, M. D. et al., 1979. "Respiratory illness in household using gas and electricity for cooking. II. Symptoms and objective findings." Environ. Res. 19:504-515.
- Kerr, H. D. et al., 1978. "Effects of Nitrogen Dioxide on Pulmonary Function in Human Subjects: Environmental Chamber Study." U.S. EPA, Research Triangle Park, N.C.
- Kerr, J. C. S. et al., 1979. "Effects of Nitrogen Dioxide on Pulmonary Function in Human Subjects: Environmental Chamber Study." Environ. Res. 19:392-404.
- Killus, J. P., et al., 1983. "Application of a Regional Oxidant Model to the Northeast United States." "Presented at the International Conference on Long Range Transport Models for Photochemical Oxidants and Their Precursor," U.S. EPA, Research Triangle Park, NC. April 12-14.
- Kinosian, J., "Ozone Precursor Relationships from EKMA Diagrams," Environmental Science and Technology, Vol. 16, No. 12, 1982.
- Kopp, R., and Vaughan, W., "Agricultural Benefits Analysis: Alternative Ozone and Photochemical Oxidant Standards," Resources for the Future, June 30, 1983; and discussion with the authors.
- Larson, J. of Weyerhaeuser. Personal Communication, 1984.
- Lave, L., and Seskin, E., Air Pollution and Human Health, Baltimore, Johns Hopkins University Press, 1977.
- Layland, D., and Cole, H., "A Review of Recent Applications of the SAI Urban Airshed Model." U.S. EPA, Research Triangle Park, NC. December, 1983.
- Lebowitz, M., et al., "The Effect of Air Pollution and Weather on Lung Function in Exercising Children and Adolescents," American Review of Respiratory Diseases, Vol. 109, 1974.
- Leighton, J.; Shehadi, A.; and Wolcott, R., "The Aggregate Benefits of Air Pollution Control," prepared for U.S. EPA, Office of Policy Analysis, by Public Interest Economics Foundation, Washington, D.C., June 1983.

- Goldstein, E. et al., 1977. "Fate and Distribution of Inhaled Nitrogen dioxide in Rhesus Monkeys." Am. Rev. Respir. Dis. 115:403-412
- Goldstein, B. D. 1979. "The Relation Between Respiratory Illness in Schoolchildren and the Use of Gas for Cooking. II." Factors affecting nitrogen dioxide levels in the home. Int. J. Epidemiol. 8:339-345.
- Green, A. E. S. et al., "An Interdisciplinary Study of the Health, Social, and Environmental Economics of the Sulfur Oxide Pollution in Florida." Interdisciplinary Center for Aeronomy and (other) Atmospheric Sciences, Univ. of Florida, Gainesville, FL. 1978.
- Hackney, J. D. et al., 1975a. "Experimental Studies on Human Health Effects on Air Pollutants. II." Four-hour exposure to ozone alone and in combination with other pollutants. Arch. Environ. Health 33:379-384.
- Hackney, J. D. et al., 1975b. "Experimental Studies on Human Health Effects on Air Pollutants. III." Two hour exposure to ozone alone and in combination with other gases. Arch. Environ. Health 30:176-181.
- Hackney, J. D., 1976. "Effects of Atmospheric Pollutants on Human Physiologic Function." Final report, U.S. EPA.
- Hackney, J. D. et al., 1978. "Experimental Studies on Human Health Effects of Air Pollutants." IV. Short-term Physiological and clinical effects of nitrogen dioxide exposure. Arch. Environ. Health 33:176-181.
- Hammer, D., et al., "Los Angeles Student Nurse Study. Daily Symptom Reporting and Photochemical Oxidants," Archives of Environmental Health, Vol. 28, 1974.
- Hasselblad, v. "Modeling Dose Response Relations for Health Effects Data", Environmetrics 81: selected papers, SIAM, Philadelphia, 1981.
- Hasselblad, V., and Svendsgaard, D., "Reanalysis of the Los Angeles Student Nurse Study," U.S. EPA, Health Effects Research Lab, Research Triangle Park, NC. August 1975.
- Heck, W., et al., "A Reassessment of Crop Loss from Ozone," Environmental Science and Technology, Vol. 17, No. 12, 1983.
- Heck, W. et al., "Nitrogen Dioxide: Time-Concentration Model to Predict Acute Foliar Injury." U.S. EPA, Corvallis, Oregon. Pub. No. 600/3-79-057. 1979.
- Heintz et al., "National Damages of Air and Water Pollution: A report submitted to U.S. EPA, 1976.

Horvath, S. M. and L. J. Folinsbee. 1979. "Effects of Pollutants on Cariopulmonary function." Report to U.S. EPA

Innes, W. B., Environmental Science and Technology. Vol. 15, 933, 1981.

Kagawa, J. et al., 1975. "Photochemical Air Pollution: Its Effects on Respiratory Function of Elementary School Children." Arch. Environ. Health 30:117-122.

Keller, M. D. et al., 1979. "Respiratory Illness in Household Using Gas and Electricity for Cooking. II. Symptoms and Objective Findings." Environ. Res. 19:504-515.

Kerr, H. D. et al., 1978. "Effects of Nitrogen Dioxide on Pulmonary Function in Human Subjects: Environmental Chamber Study." U.S. EPA, Research Triangle Park, N.C.

Kerr, J. C. S. et al., 1979. "Effects of Nitrogen Dioxide on Pulmonary Function in Human Subjects: Environmental Chamber Study." Environ. Res. 19:392-404.

Killus, J. P., et al., 1983. "Application of a Regional Oxidant Model to the Northeast United States." "Presented at the International Conference on Long Range Transport Models for Photochemical Oxidants and Their Precursor," U.S. EPA, Research Triangle Park, NC. April 12-14.

Kinosian, J., "Ozone Precursor Relationships from EKMA Diagrams," Environmental Science and Technology, Vol. 16, No. 12, 1982.

Kopp, R., and Vaughan, W., "Agricultural Benefits Analysis: Alternative Ozone and Photochemical Oxidant Standards," Resources for the Future, June 30, 1983; and discussion with the authors.

Larson, J. of Weyerhauser. Personal Communication, 1984.

Lave, L., and Seskin, E., Air Pollution and Human Health, Baltimore, Johns Hopkins University Press, 1977.

Layland, D., and Cole, H., "A Review of Recent Applications of the SAI Urban Airshed Model." U.S. EPA, Research Triangle Park, NC. December, 1983.

Lebowitz, M., et al., "The Effect of Air Pollution and Weather on Lung Function in Exercising Children and Adolescents," American Review of Respiratory Diseases, Vol. 109, 1974.

Leighton, J.; Shehadi, A.; and Wolcott, R., "The Aggregate Benefits of Air Pollution Control," prepared for U.S. EPA, Office of Policy Analysis, by Public Interest Economics Foundation, Washington, D.C., June 1983.

- Loehman, E. et al. "Distributional Analysis of Regional Benefits and Costs of Air Quality Control," J. Environ. Econ. Manag., Vol. 6, No. 3, 1979.
- Linn, W., et al., "Human Respiratory Effects of Heavy Exercise in Oxidant-Polluted Ambient Air," American Review of Respiratory Disease, Vol. 123, No. 4, 1981.
- Linn, W. S. et al., "Respiratory Function and Symptoms in Urban Office Workers in Relation to Oxidant Air Pollution Exposure." Amer. Rev. Resp. Disease, 114:477-483, 1976.
- Lippmann, M., et al., "Effects of Ozone on the Pulmonary Function of Children," in: Lee, S., et al., eds, The Biomedical Effects of Ozone and Related Photochemical Oxidants, Princeton Scientific Publishers, Inc.: Princeton, N.J.: Advances in Modern Environmental Toxicology, V: 423-46: 1983.
- Love, G. J. et al., "Acute Respiratory Illness in Families Exposed to Nitrogen Dioxide Ambient Air Pollution in Chattanooga, TN." Arch. Environ. Health 37:75-80
- Makino, K., and Mizoguchi, I., "Symptoms Caused by Photochemical Smog," Japan Journal of Public Health, Vol. 22, No. 8, 1975.
- McLaughlin, S., et al., "Measuring Effects of Air Pollution Stress on Forest Productivity," Tappi Journal, Vol. 67, No. 1984 p. 74.
- Melia, R. J. W. et al., 1977. "Association Between Gas Cooking and Respiratory Disease in Children." Br. Med. J. 2:149-152.
- Melia, R. J. W. et al., 1978. "Differences in NO₂ Levels in Kitchens with Gas or Electric Cookers." Atm. Environ. 12:149-152
- Melia, R. J. W. et al., 1979. "The Relation Between Respiratory Illness in Primary Schoolchildren and the Use of Gas for Cooking." I - Results from a national survey. Int. J. Epid. 8:333.
- Menzel, D. B. 1976. "The Role of Free Radicals in the Toxicity of Air Pollutants (Nitrogen Oxides and Ozone). In Free Radicals in Biology, vol. III, ed. W. A. Bryor, pp. 181-202. New York: Academic.
- National Academy of Sciences 1977. Nitrogen Oxides. National Academy of Sciences, Washington, D.C. p. 197-214.
- National Academy of Sciences, 1976. Nitrogen Oxides: Medical and Biologic Effects of Environmental Pollutants. National Research Council, Washington, D.C.
- National Academy of Sciences, Air Quality and Automobile Emission Control, Vol. 4; prepared for The Committee on Public Works, U.S. Senate, U.S. Government Printing Office, 1974.

National acid Precipitation Assessment Program, Annual Report, 1983.

National Research Council, 1983. Acid Deposition: Atmospheric Processes in Eastern North America. NRC, National Academy Press, Washington, D.C.

National Research Council, 1977. Nitrogen Oxides: Medical and Biological Effects of Environmental Pollutants. Washington, D.C., National Academy of Sciences.

National Research Council, 1978. Nitrates. An Environmental Assessment. Washington, D.C., National Academy of Sciences.

National Research Council, 1974. Air Quality and Automobile Emission Control. Vol. 4: "The Costs and Benefits of Automobile Emission Control." A report by the Coordinating Committee on Air Quality Studies of the National Academy of Sciences and the National Academy of Engineering, prepared for the Committee on Public Works, United States Senate, 93rd Congress, 2nd Session. Washington, D.C.: U.S. Government Printing Office.

Nisbet, I.C.T. 1975. "Sulfates and Acidity in Precipitation: Their Relationship to Emissions and Regional Transport of Sulfur Oxides." pp. 276-312, "Air Quality and Stationary source Emission Control, a report by the Commission on Natural Resources of the National Research Council, prepared for the Committee on Public Works, U.S. Senate, 94th Congress, 1st Session. Washington, D.C.: U.S. Government Printing Office.

Orehek, J. et al., 1976. "Effect of Short-Term, Low-Level Nitrogen Dioxide Exposure on Bronchial Sensitivity of Asthmatic Patients." J. Clin. Invest. 57:301-307

Page, W. P. et al., "Estimation of Economic Losses to the Agricultural Sector from Air Borne Residuals in the Ohio River Basin." JAPCA 32: 151-154.

Pitts, J. N. Jr., et al., 1983. Comment on "Effect of Nitrogen Oxide Emissions on Ozone Levels in Metropolitan Regions," "Effects of NOx Emission Rates on Smog Formation in the California South Coast Air Basin," and "Effects of Hydrocarbon and NOx on Photochemical Smog Formation Under Simulated Transport Conditions." Environ. Sci. Technol. 17:54-57.

Port, C. D. 1977. "A Comparative Study of Experimental and Spontaneous Emphysema." J. Toxicol. Environ. Health 2:589-604

Portney, P and Mullahy, J., "Ambient Ozone and Human Health: An epidemiological analysis" prepared for U.S. EPA, OAQPS, 1983 September.

Portney, P., and Mullahy, J., "A Multinomial Model of the Air Pollution Respiratory Health Relationship." Resources for the Future, 1985.

Portney, P. and Mullahy, J., "Urban Air Quality and Acute Respiratory Illness," J. of Urban Economics, (forthcoming).

Posin, C. et al., 1978. "Nitrogen Dioxide and Human Blood Biochemistry." Arch. Environ. Health. (Nov/Dec), 318-324.

Randall, A., et al., 1974. "Bidding Games for Valuation of Aesthetic Environmental Improvements." J. Environ. Econ. Manag. 1:132-149.

Rowe, R. and Chestnut, L., 1984. "Ozone and Asthmatics in Los Angeles: A Benefits Analysis." Report completed for Office of Policy Analysis by Energy and Resouce Consultants, Forthcoming 1984.

Seigneur, C. et al., "Computer Simulation of the Atmospheric Chemistry of Sulfate and Nitrate Formation," Science. Sept 7, 1984.

Seigneur, C. et al., 1984. "Modeling Studies of Sulfate and Nitrate Chemistry: The Effect Changes in Sulfur Dioxide, Nitrogen Oxide, and Reactive Hydrocarbon Levels," final report.

Seigneur, C.; Saxena, P.; and Roth, P., "Preliminary Results of Acid Rain Modeling," submitted at a Specialty Conference on Atmospheric Deposition sponsored by the Air Pollution Control Association, November 7-10, 1982, Detroit, Michigan.

Sherwin, R. P., et al., 1977. "Sequestration of Exogenous Peroxidase in the lungs of Animals Exposed to Continuous 0.5 ppm Nitrogen Oxide." Fed. Proc. 36:1091.

Shy, C. M. et al., "The Chattanooga School Children Study: "Effects of Community Exposure of Nitrogen Dioxide. I." Methods, description of pollutant exposure and results of ventilatory function testing. J. Air Pollut. Control Assoc. 20 8:539-545, 1970.

Shy, C. M. and G. J. Love, 1979. "Recent Evidence on the Human Health Effects of Nitrogen Dioxide." Proceedings of the Symposium on Nitrogen Oxides, Honolulu. Hawaii, April 4-5.

Speizer, F. E. et al., 1980. "Respiratory Disease Rates and Pulmonary Function in Children Associated with No2 Exposure." Am. Rev. Resp. Dis. 121:3-10.

Spengler, J. D. 1983. "Nitrogen Dioxide Inside and Outside 137 Homes and Implications for Ambient Air Quality Standards and Health Effects Research." Environ. Sci. Technol. 17:164-168.

Spengler, J. D. et al., 1979. "Sulfur Dioxide and Nitrogen Dioxide Levels Inside and Outside Homes and the Implications on Health Effects Research." Environ. Sci. Techn. 13:1276-1271.

SRI International, "An Estimate of the Nonhealth Benefits of Meeting the Secondary National Ambient Air Quality Standards," a final report to the National Commission on Air Quality, 1981.

Systems Applications Inc., "Simulation of the Regional Air Quality Impacts of Industrial Emission Controls." Report submitted to U.S. EPA, 1984.

Systems Applications Inc., "Simulations of the Impact of Anthropogenic Rural Emission Controls on Rural Ozone Concentrations." Report submitted to U.S. EPA, 1985.

Thaler, R., and Rosen, S., "The Value of Saving a Life: Evidence from the Labor Market," in Household Production and Consumption, ed. N. E. Terleckyj, New York, Columbia University Press, 1976.

Thomas, H. V. et al., 1968. "Lipoperoxidation of Lung Lipids in Rats Exposed to Nitrogen Dioxide." Science 159:532-534.

Thompson, C. R. et al., 1970. "Effects of Continuous Exposure of Navel Oranges to NO₂." Atm. Environ. 4:349-355.

Trijonis, J. C. et al., "The Relationship of Ambient NO₂ to Hydrocarbon and NO_x Emissions." Draft report from Technology Service Corporation to EPA under Contract NO_x 68-02-2299. U.S. EPA, Office of Research and Development, Research Triangle Park, NC., 1979.

Trijonis, J. C. "Empirical Relationships Between Atmospheric Nitrogen Dioxide and Its Precursors." Environmental Sciences Research Laboratory, Office of Research and Development U.S. EPA, Research Triangle Park, NC. February, 1978. U.S. Department of Agriculture, Agricultural Statistics, 1982, Washington, D.C., U.S. Government Printing Office, 1982.

U.S. Department of Health and Human Services, Public Health Service, "Blood Carbon Monoxide Levels in Persons 3-74 Years of Age: United States, 1976-80," Advance Data, No. 76, March 17, 1982.

U.S. Department of Health, Education, and Welfare; Public Health Service, Vital and Health Statistics Series 10, No. 96, "Limitations of Activity and Mobility Due to Chronic Conditions", 1973a.

U.S. Department of Health, Education, and Welfare; Public Health Service, Vital and Health Statistics Series 10, No. 84, "Prevalence of Selected Chronic Respiratory Conditions: United States - 1970," 1973b.

U.S. EPA, "Ozone SIP Data Base and Summary Report." Office of Air Quality Planning and Standards, Research Triangle Park, NC.

SRI International, "An Estimate of the Nonhealth Benefits of Meeting the Secondary National Ambient Air Quality Standards," a final report to the National Commission on Air Quality, 1981.

Systems Applications Inc., "Simulation of the Regional Air Quality Impacts of Industrial Emission Controls." Report submitted to U.S. EPA, 1984.

Systems Applications Inc., "Simulations of the Impact of Anthropogenic Rural Emission Controls on Rural Ozone Concentrations." Report submitted to U.S. EPA, 1985.

Thaler, R., and Rosen, S., "The Value of Saving a Life: Evidence from the Labor Market," in Household Production and Consumption, ed. N. E. Terleckyj, New York, Columbia University Press, 1976.

Thomas, H. V. et al., 1968. "Lipoperoxidation of Lung Lipids in Rats Exposed to Nitrogen Dioxide." Science 159:532-534.

Thompson, C. R. et al., 1970. "Effects of Continuous Exposure of Navel Oranges to NO₂." Atm. Environ. 4:349-355.

Trijonis, J. C. et al., "The Relationship of Ambient NO₂ to Hydrocarbon and NO_x Emissions." Draft report from Technology Service Corporation to EPA under Contract NOx 68-02-2299. U.S. EPA, Office of Research and Development, Research Triangle Park, NC., 1979.

Trijonis, J. C. "Empirical Relationships Between Atmospheric Nitrogen Dioxide and Its Precursors." Environmental Sciences Research Laboratory, Office of Research and Development U.S. EPA, Research Triangle Park, NC. February, 1978. U.S. Department of Agriculture, Agricultural Statistics, 1982, Washington, D.C., U.S. Government Printing Office, 1982.

U.S. Department of Health and Human Services, Public Health Service, "Blood Carbon Monoxide Levels in Persons 3-74 Years of Age: United States, 1976-80," Advance Data, No. 76, March 17, 1982.

U.S. Department of Health, Education, and Welfare; Public Health Service, Vital and Health Statistics Series 10, No. 96, "Limitations of Activity and Mobility Due to Chronic Conditions", 1973a.

U.S. Department of Health, Education, and Welfare; Public Health Service, Vital and Health Statistics Series 10, No. 84, "Prevalance of Selected Chronic Respiratory Conditions: United States - 1970," 1973b.

U.S. EPA, "Ozone SIP Data Base and Summary Report." Office of Air Quality Planning and Standards, Research Triangle Park, NC.

U.S. EPA, "VOC/Ozone Relationships from EKMA," memo from Warren Freas (Air Management Technology Branch) to Alan McGartland (Benefits Branch), January 27, 1984b.

U.S. EPA, Draft Revised Air Quality Criteria for Ozone and Other Photochemical Oxidants, Office of Research and Development, 1983a.

U.S. EPA, "Review of the National Ambient Air Quality Standards for Nitrogen Oxides: Assessment of Scientific and Technical Information - OAQPS Staff Paper. EPA - 450/5-82-002 August 1982.

U.S. EPA, "The API Study and Its Possible Human Health Implications, memo from Al Lorang (Chief, Technical Support Staff) to Charles Gray, Jr. (Director, Emission Control Technology Division), May 16, 1983b.

U.S. EPA, 1982c. "Cost and Economic Assessment of Regulatory Alternatives for No₂ NAAQS (DRAFT). Research Triangle Park, NC.

U.S. EPA, 1982b. "Air Quality Criteria for Oxides of Nitrogen (Criteria Document). Research Triangle Park, NC.

U.S. EPA 1982d. "NAAQS Environmental Impact Statement for No₂. Research Triangle Park, NC.

U.S. EPA, 1982 NCLAN Annual Report, Environmental Research Lab, Corvallis, Oregon, 1982a.

U.S. EPA, National Air Pollutant Emissions Estimates, 1940 - 1980, Monitoring and Data Analysis Division, January 1982b.

U.S. EPA, Air Quality Criteria for Nitrogen Oxides, Office of Research and Development, 1982c.

U.S. EPA, Carcinogen Assessment Group, "The Carcinogen Assessment Group's Final Report on Population Risk to Ambient Benzene Exposure January 10, 1979.

U.S. EPA, Air Quality Criteria for Ozone and Other Photo-chemical Oxidants, Office of Research and Development, April 1978.

Von Nieding, G. et al., "Minimum Concentration of NO₂ Causing Acute Effects on the Respiratory gas Exchange and Airway Resistance in Patients with Chronic Bronchitis." Int. Arch. Arbeitsmed. 27: 338-348, 1971. U.S. EPA, Research Triangle Park, NC.

Von Nieding, G. et al., "Studies of the Acute Effect of NO₂ on Lung Function: Influence on Diffusion, Perfusion and Ventilation in the Lungs." Int. Arch. Arbeitsmed. 31:61-72, 1973.

Von Nieding, G. et al., 1977. "Acute Effects of Ozone on Lung Function of Men." VDI-Ber. 270:123-129.

Wade, W. A., III, et al., "A Study of Indoor Air Quality." J. Air Pollut. Control Assoc. 25:93-939 1975.

Ware et al., "Passive Smoking, Gas Cooking and Respiratory Health of Children Living in Six Cities." Amer. Rev. Respir. Dis 1984; 129: 366-374.

Whittemore, A., and Korn, E. L., "Asthma and Air Pollution in the Los Angeles Area," American Journal of Public Health, Vol. 70, 1980.

Zagraniski, R.; Leaderer, B.; and Stolwuk, J., "Ambient Sulfates, Photochemical Oxidants and Acute Adverse Health Effects: An Epidemiologic Study," Environmental Research, Vol. 19, 1979.