

ENERGY AND RESOURCE CONSULTANTS, INC.

DRAFT REPORT

ECONOMIC BENEFITS OF **NO_x** CONTROL:
DESIGN AND APPLICATION
FOR THE EASTERN U.S.

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WASHINGTON, D.C.

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August 10, 1987

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ACKNOWLEDGMENTS

We thank Stan Dawson of the California Air Resources Board for supplying literature and Bart Ostro, Joel Schwartz, Mark Thayer, Alan Krupnick, Allen **Basala**, Dave **McLamb**, John Mullahy, Brian Morton, and Joel Scheraga for information and suggestions. The conclusions are those of the author and do not reflect official policy of the U.S. Environmental Protection Agency.

At Energy and Resource **Consultants, Inc.**, Don Peterson drafted background material on forest damage, Lauraine Chestnut consulted on health and visibility impacts, Julie Sueker and **Tina** Neithercut assisted with air quality data and selected analyses, and Jo Whittenberg produced the report. Their assistance is greatly appreciated.

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ECONOMIC BENEFITS OF NO_x CONTROL:
DESIGN AND APPLICATION FOR THE EASTERN U.S.

EXECUTIVE SUMMARY

In April of 1985, EPA released a report entitled "The Economic Benefits of Controlling Volatile Organic Compounds and Nitrogen Oxides: A Generic Approach" by McGartland and Ostro (1985). McGartland and Ostro examined the benefits of NO_x control for human health and welfare indirectly through its combined effect with volatile organic compounds (VOC) upon ozone formation, and through the direct effects of NO₂.

The usefulness of such an analysis is in providing a preliminary assessment of the benefits of possible regulatory control options without conducting a separate detailed analysis for each control option.

The work reported upon herein:

1. Describes possible refinements and extensions to a McGartland and Ostro type of generic analysis for NO_x control benefits, and
2. Applies the refined approach to a case study of alternative NO_x controls on coal-fired power plants in the eastern U.S.

Benefits Calculation Framework

The NO_x control benefits framework reviews the existing literature and selects study results for point estimates and upper and lower bound estimates of physical and economic impacts due to changes in NO₂, ozone, nitrates and acid deposition. A summary of the selected health and welfare endpoints and studies upon which calculations are based is found in Table E-1.

Case Study of NO_x Control at Eastern Coal-Fired Power Plants

EPA provided four basic scenarios for analysis of NO_x controls on coal-fired power plants nationwide corresponding to the four data points in Table E-2.

Table E-1
summary Of Study **Selection**¹

Effect Category	Physical Effects Study Recommended	Economic Valuation Studies Recommended
I. <u>NO, Effects</u>		
Human Health		
- Eye Irritation	Schwartz et al. (1987) adults only	Krupnick et al. (1986a)
- Others	Possible further evaluation of Mullahy (1987) for MRADs and Lebowitz (1985) for asthmatics	
Visibility		
- Haze Use Values (values per household)	Tri jonis (1987)	Chestnut et al. (1986)
- Plume Use Values in West (per household)	?	Chestnut and Rowe (1983)
Materials	---	Barrett and Waddell (1973)
Agriculture, Vegetation and Forests	No significant effect	
II. <u>Nitrate Effects</u> (using nitrate or TSP or PM studies)		
Human Health		
- Mortality	Evans et al. (1984) Schwartz et al. (1986)	Violette et al. (1986)
- Emerg. Room Visits	Samet et al. (1981)	Chestnut et al. (1987)
- Rest. Act. Days	Ostro (1987)	See Text
Visibility		
- Haze Use Values (Values per household)	Trijonis (1987)	Chestnut et al. (1986)
Materials (per household)	---	Manual et al. (1982)
Agriculture, Vegetation and Forests	No significant effect	

Table E-1 (Continued)
Summary Of Study Selection'

Effect Category	Physical Effects Study Recommended	Economic Valuation Studies Recommended
III. <u>Ozone Effects</u>		
Health		
- Resp. Rest. Act. Days (for adults only in excess of asthma attacks for asthmatics)	Portney & Mullahy (1986)	Krupnick et al. (1986), Dickie et al. (1986)
- Eye irritation and cough symptom days in excess of RRAD (for adults only)	Schwartz et al. (1987)	Loehman et al. (1979), Tolley et al. (1986), Krupnick et al. (1986a)
- Asthma at tacks (for asthmatics)	Whittemore and Korn (1980), Holguin (1985)	Krupnick et al. (1986a) Rowe & Chestnut (1985)
- Symptoms Aggregate	Krupnick et al. (1986b)	
Materials		
		McCarthy (1983) Barrett and Waddell (1973)
Agriculture		
		Kopp et al. (1984, 1986), Adams et al. (1985), Howitt et al. (1984), Rowe & Chesrnu: (1985)
Ornaments		
		Heintz (1976)
Forests ²		
- Yield Effects	Wilhour (1986), Miller (1977)	Callaway et al. (1986) Botkin & Devine (1986)
- Aesthetics	Miller (1977), Crocker (1985)	
IV. <u>Acid Deposition Effects</u>		
Materials		
-Galvanized Steel		Horst et al. (1986)

Table E-1 (Concluded)
Summary Of Study Selection'

Effect Category	Physical Effects Study Recommended	Economic Valuation Studies Recommended
V. <u>Speculative Quantified Effects</u> ³		
NO₂ and nitrate non- use visibility values at national parks		
- Haze	Trijonis (1987)	Chestnut and Rowe (1983)
- Plumes	?	Chestnut and Rowe (1983)
Ozone chronic illness (for adults only)	Higgins et al. (1982) Mullahy (1987)	See Text
Acid deposition materials effects other than galvanized steel		Horst et al. (1986)
Acid deposit ions agriculture effects		Adams et al. (1986)

Notes:

1. See **text** for application discussion, especially with regard to best, lower and upper bound determinations and potentially overlapping categories.
2. Insufficient time has prevented thorough review and selection of all studies. Studies **identified** are studies of probable usefulness.
3. Speculative quantified effects are generally only suggested as upper bounds, or as otherwise indicated in the text.

Table E-2
 Summary of National **NO_x** Emissions Under Alternative Assumptions
 (1,000s of short tons/year)

	3.995	2000
Base Case	7,595.2	8,252.6
Controls on existing plants with .5 lb NO_x/MMBtu NSPS limit beginning in 1995	5,892.7 (77.6% of Base)	6,384.6 (77.4% of Base)
Controls on existing plants with .4 lb NO_x/MMBtu NSPS limit beginning in 1995	5,786.9 (76.2% of Base)	6,171.9 (74.8% of Base)

Source: EPA provided assumptions

For this analysis, we considered only changes in emitted **NO_x** tons for 31 eastern states and Washington D.C. For each scenario EPA provided baseline tons and new tons with new controls for urban and rural areas in each state.

To implement the benefits analysis a computer model was developed. The model allows calculation of changes in air quality conditions and calculation of economic measures of change in health and welfare status by rural and urban region by state by scenario. The geographic detail of the model allows improvement over previous similar generic assessments. This is because baseline conditions, population and changes in tons of **NO_x** emissions can be better matched. The increased level of detail herein accounts for some of the differences between the results reported here and those from previous assessments.

The full capability of the state by state, region by region analysis is not captured due to limited baseline air quality data, the use of consistent relationships between emitted and ambient pollutant concentrations across all states, and other interim air quality relationship assumptions. These limitations are the result of the quick response nature of the analysis; however, the model is designed to incorporate additional region specific data when they become available. The importance of these limitations is that the state by state analysis is likely to improve the accuracy of the total benefit calculations, but limited accuracy should be given to the exact numbers generated for each state.

EPA also provided several key air quality assumptions used in the analysis including:

- o Changes in **NO_x** emissions would have unknown effect upon nitrate concentrations, which was translated into a no change assumption. This eliminated the nitrate visibility benefits analysis and all PM benefit analyses.
- o Changes in **NO_x** emissions would have unknown effects upon acid deposition, which was translated into a no change assumption. This

eliminated the acid deposition benefits analysis, most of which was considered as speculative.

- o The **precent** change in **NO_x** emissions by state and region would equal the percent change in ambient **NO₂** by state and region, as used in McGartland and Ostro (1985).
- o The effect of changes in **NO_x** emissions on ambient ozone conditions would be typified by the relationships specified in McGartland and Ostro (1985).

The McGartland and Ostro assumptions are based upon a synthesis of atmospheric chemistry and model results relating changes in **NO_x** and **VOCs** to ambient concentration of **NO₂** and ozone in urban and rural areas. Their point estimates suggest:

- o Decreases in urban **NO_x** decrease **NO₂** but increase ozone in urban areas.
- o Decreases in urban **NO_x** decreases peak ozone levels in mean by rural areas, but increase average ozone levels in nearby rural areas.
- o Decreases in rural **NO_x** decrease both **NO₂** and ozone in rural areas.

The model also allows relatively easy calculation of alternative **NO_x** control scenarios and allows identification and analysis of critical assumptions. The model was run with nine alternative scenarios as listed in Table E-3. The first four correspond to the alternative **NO_x** controls provided by EPA. The remaining five are discussed below.

- o CASE 5: DAILY PEAKS ADJUSTMENT. Many of the health analyses utilize an ozone measure of the annual average of the daily high hour reading. However, the McGartland and Ostro assumption on changes in **NO_x** to changes in ambient ozone are not clear how they apply to the average of daily high hour. For the **basecase** analyses, the percentage change in the annual average of all hours was used for the percentage change in

Table E-3
Scenario Key

BASECASE #1:

1995 .5 lb limits with basic assumptions (with upper and lower physical impacts and values). Table 7-4, 7-5, A-1 to A-15

BASECASE #2:

1995 .4 LB = **Basecase #1** augmented only with 1995 .4 lb limits. Tables A16, A17

BASECASE #3:

2000 .5 LB = **Basecase #1** augmented only with 2000 .5 lb limits. Tables A18, A19

BASECASE #4:

2000 .4 LB = **Basecase #1** augmented only with 2000 .4 lb limits. Tables A20, A21

CASE 5:

DAILY PEARS ADJUSTMENT = **Basecase #1** augmented only with alternative assumption that % change in daily peak (and summer daytime O3) equals $(1-C1)*(\% \text{ change in annual peak}) + C1*(\% \text{ change in annual average})$. $C1 = .66$. Tables A22, A23

CASE 6:

NOx TO O3 UPPER LIMITS = **Basecase #1** augmented only with **use of** upper range assumptions on relationships between **NOx** to ozone as reported in M&O. Tables A24, A25

CASE 7:

NOx TO O3 LOWER LIMITS = **Basecase #1** augmented only with use of lower range assumptions on relationships between **NOx** to ozone as reported in M&O. Tables A26, A27

CASE 8:

MRADS ADJUSTMENT = **Basecase #1** augmented only by assuming all RRADS = MRADS for valuation. Tables A28, A29

CASE 9:

NO2 - EYE IRRITATION = **Basecase #1** augmented only by assuming the rate of NO2 eye irritation is $C2*$ (rate in Schwartz et al.). $C2 = .5$. Tables A30, A31

* All Scenarios for 31 eastern states plus Washington, D.C.

the annual average of the daily peak hours. An alternative assumption that the percentage change in the daily high hour equals **1/3rd** the percentage change in the peak hour and **2/3rd** the percentage change in the average hour is examined.

- o CASE 6: **NO_x** TO OZONE UPPER LIMITS. Examines the effect of using the upper estimates of **NO_x** to ozone formation reported in **Table E-4**.
7
- o CASE 7: **NO_x** TO OZONE LOWER LIMITS. Examines the effect of using the lower estimates of **NO_x** to ozone formation reported in **Table E-4**.
7
- o CASE 8: MRADS ADJUSTMENT. Respiratory restricted activity days (RRADs) play a significant role in the benefit calculations. The point estimate calculations assume RRADs are a mix of minor restricted activity days (**MRADs**) and bed disability days (**BDDs**) or work loss days. This alternative assumes all RRADs are **MRADs**. Another issue concerns whether the RRADs due to all pollutants estimated in different studies exceed available RRADs. This case can be used to examine the issue by assuming the RRAD estimate is overstated by a factor of two.
- o CASE 9: **NO₂** - EYE IRRITATION. **NO₂** eye irritation calculations are another major component of the economic value calculations. While the calculations attempt to adjust the relationships in the underlying study (Schwartz et al.) to eastern conditions, threshold conditions were difficult to account for. This analysis reduces the point and upper bound estimate by one-half to examine the sensitivity of the analysis to assumptions in these calculations.

The estimated dollar per ton **NO_x** controlled by effect category and in total for **BASECASE #1** are presented in Table E-4. Table E-5 provides the total dollar estimates for the four basecases. Direct damages due to **NO₂** are most affected by visibility and eye irritation, both of which exceed the values in **McGartland** and **Ostro**, with values per ton largest in urban areas due to the larger populations affected by each ton reduced.

Table E-4
SUMMARY \$/TON VALUES - \$1986

BASECASE #1: . 5 LB NOx NSPS LIMITS IN 1995

CATEGORY	ESTIMATE	RURAL	URBAN	TOTAL
QUANTIFIED EFFECTS				
VISIBILITY	BEST	22. 71	128. 99	76. 66
	UPPER	34. 07	193. 49	114. 98
	LOWER	11. 36	64. 50	38. 33
NO2-EYE IRR	BEST	35. 74	198. 00	118. 09
	UPPER	85. 77	475. 20	283. 43
	LOWER	0. 00	0. 00	0. 00
NO2-MATERIALS	BEST	19. 50	50. 76	35. 36
	UPPER	19. 50	50. 76	35. 36
	LOWER	9. 75	25. 38	17. 68
TOTAL NO2 EFFECTS				
	POINT	77. 95	377. 75	230. 11
	LOWER	21. 10	89. 88	56. 01
	UPPER	139. 34	719. 45	3 9 8 . 4 1
MATERIALS+ORNAMENTALS				
	BEST	0. 09	0. 08	0. 09
	UPPER	0. 09	0. 08	0. 09
	LOWER	0. 06	0. 06	0. 06
AGRICULTURE	BEST	- 10. 72	0. 00	- 5. 28
	UPPER	- 14. 26	0. 00	- 7. 02
	LOWER	- 7. 08	0. 00	- 3 . 4 8
03-RRADS	BEST	3. 53	- 183. 57	- 91. 43
	UPPER	5. 22	- 271. 68	- 135. 32
	LOWER	0. 71	- 36. 71	- 18. 29
03-ASTHMA	BEST	0. 23	- 11. 98	- 5. 97
	UPPER	0. 60	- 31. 16	- 15. 52
	LOWER	0. 03	- 1. 80	- 0. 90
03-EYE IRR	BEST	1. 85	- 96. 25	- 47. 94
	UPPER	4. 81	- 250. 25	- 124. 65
	LOWER	1. 70	- 88. 55	- 44. 11
03-COUGH	BEST	0. 41	- 21. 46	- 10. 69
	UPPER	0. 82	- 42. 93	- 21. 38
	LOWER	0. 00	0. 00	0. 00
MINOR SYMPTOMS	DAYS LOWER	1. 11	- 57. 85	- 28. 82
TOTAL NO2+OZONE				
	POINT	73. 33	64. 57	68. 89
	LOWER	15. 94	- 6. 43	8. 07
	UPPER	136. 62	123. 52	94. 61

Table E-4 - (Continued)

SPECULATIVE CATEGORIES

FORESTS	BEST	0.00	0.00	0.00
	UPPER	0.00	0.00	0.00
	LOWER	0.00	0.00	0.00
CHRONIC ILLNESS	UPPER ONLY	0.22	- 11.45	- 5.70
TOTAL QUANTIFIED				
	POINT	73.33	64.57	68.89
	LOWER	15.94	- 6.43	8.07
	UPPER	136.84	112.07	88.91

Table E-5

Summary of Total Benefits for the Four **Basecase** Scenarios: Point Estimate
 For the Eastern U.S.
 (\$1986 in millions)

Scenario	Rural	Urban	Total
BASECASE #1: .5 lb NO _x NSPS limits in 1995	49.9	45.3	95.2
BASECASE #2: .4 lb NO _x NSPS limits in 1995	116.6 85.6	65.1 45.5	91.5 131.5
BASECASE #3: .5 lb NO _x NSPS limits in 2000	78.4 54.4	62.7 45.8	70.7 wf.9
BASECASE #4: .4 lb NO _x NSPS limits in 2000	138.0 120.4	63.4 45.9	104.1 166.3

The most significant components of indirect damages due to **NO_x** effects through ozone are **RRADs** and eye irritation, both of which are slightly positive for rural areas, but significantly negative for urban, due to increases in ozone, areas resulting in total \$/ton across all areas being negative for changes in ozone induced effects. Of interest is that agriculture values are negative in rural areas while health values are positive. This is the result of different locations of crops and people versus the location of the changes in **NO_x** emissions in the scenarios.

The lower and upper estimates in the basecases reflect changes in assumptions on health and welfare impacts and values. Due to conservative assumptions, the upper bound is on the order of only 150 percent of the point estimate while the lower bound is about 10 percent of the point estimate.

Table E-6 compares the \$/ton results across the scenarios. The adjustment for daily peaks (**CASE #5**) has a very significant effect on results. The new point estimate now exceeds the **BASECASE #1** upper bound estimate by a factor of two.

The adjustments for upper and lower bound relationships between **NO_x** and ozone are also significant. The upper bound **NO_x** to ozone relationships drive the point estimate of total benefits to be negative and over 3.5 times the magnitude of the **BASECASE #1** upper bound as increased urban ozone concentrations dominates the analysis. **Similarly**, the lower bound **NO_x** ozone relationships drive the benefits to become much larger, about 3 times the **BASECASE #1** upper bound, as reduced rates of increase in ozone reduce ozone damages.

The **MRADs** (**CASE #8**) and **NO₂** eye irritation (**CASE #9**) adjustments are by far the most significant of all of the physical and economic relationships quantified in the analysis. The MRAD adjustment drives the point estimate up to the **BASECASE #1** upper bound, which occurs because the negative impacts due to ozone formation are reduced. On the other hand the **NO₂** eye irritation adjustment drives the benefits down to the **BASECASE #1** lower bound.

A scenario not run was to consider altering the relationship between the percent change in **NO_x** and **NO₂** formation. Any such change would approximately alter the

Table E-6
Summary of \$/Ton Point Estimates by Sensitivity Test Scenario

Scenario*	Estimate	Total \$/Ton Point Estimate (Urban & Rral)					
		NO2 only			NO2 + Ozone		
		Rural	Urban	Total	Rural	Urban	Total
1. Baseline 1995 .5 lb	point	78	378	230	73	65	69
	Lower	21	90	56	16	-6	8
	Upper	139	719	398	137	123	95
2. 1995, .4 lb	point	80	379	226	117	65	71
3. 2000, .5 lb	point	72	366	216	78	63	71
4. 2000, .4 lb	point	73	369	207	138	63	104
5. Daily Peak Adjustment, C1=.66	point	78	378	230	329	65	195
6. NO_x to 03 Upper Limits	point	78	378	230	-146	-563	-358
7. NO_x to 03 Lower Limits	point	78	378	230	403	22	311
8. MEADS Adjustment	point	78	378	230	72	139	106
9. NO2 - Eye Irritation	point	60	279	171	55	-34	10

* For comparison, the upper and lower bounds are also provided for the Baseline #1 case. All results for 31 eastern states plus Washington, D.C.

NO₂ direct benefits by a comparable adjustment. For example, assuming a 1 percent change in **NO_x** only changes **NO₂** by .75 percent would reduce **NO₂** benefits by about 25 percent and drive the point estimate of total benefits in BASELINE #1 to the lower bound. Alternatively, assuming an elasticity of 1.25 (rather than 1 or .75) would increase total benefits to one-third over the BASELINE #1 upper bound.

Many of the assumptions have an important impact on the results. However, changes in one assumption can often be offset by changes in another assumption elsewhere. Overwhelmingly the most important of the assumptions is the relationships between **NO_x** emission changes and ambient air pollution measures of interest to the physical and economic assessment. Variations in these relationships can easily overwhelm even the most significant assumption concerning changes in the physical and economic analysis. The most significant changes in the physical and economic analysis concern the change in the rate of, and value for, **RRADs** and eye irritation. Assumptions concerning other physical impacts and economic values are, relatively speaking, of much less consequence to the aggregate estimates. This, however, does not negate the importance of the impacts to those individuals who experience them.

To a large extent the potential significant biases have been addressed through the upper and lower bounds, speculative estimates and sensitivity analyses. One bias not addressed is that the model currently uses 1985 population estimates. Updating to 1995 and 2000 population estimates would increase benefit estimates by about 9 and 13 percent respectively using the U.S. Census middle series population estimates.

1.0 INTRODUCTION

In April of 1985, EPA released a report entitled "**The** Economic Benefits of Controlling Volatile Organic Compounds and Nitrogen Oxides: A Generic Approach" by McGartland and Ostro (1985).¹ McGartland and Ostro examined the benefits of **NO_x** control:

- o indirectly through its combined effect with volatile organic compounds (VOC) upon ozone formation and the resultant health and welfare effects, and
- o through the direct effects of **NO₂** on human health and welfare.

The purpose and design of the McGartland and Ostro analysis was stated as:

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. (Page 1)

1. This analysis was also included in the lead phasedown benefit cost analysis (Schwartz et al., 1985). Subsequent EPA analysis and review was also conducted and is used in this plan as discussed in Sections 3 through 5.

The work in this report describes possible refinements and extensions to how a **McGartland** and Ostro type of generic benefits analysis for **NO_x** control benefits should be conducted and proceeds to apply the procedures to a case study of **NO_x** emission limits in the eastern U.S. The work proceeds in two phases, Phase 1: Design of Procedures and Phase 2: Application.

Phase 1: Design of Procedures

The work in this phase is reported in Chapters 2 through 6.

- o Reviews and recommends selection of new technical literature available in the intervening two years since M&O,
- o Consideration of how to best measure direct physical and economic effects due to nitrate formation,
- o Consideration of how to best measure indirect physical and economic effects due to acid deposition formation, and
- o Alternative analysis and presentation strategies.

It is important to note that this revised plan is constrained:

- o to provide a framework that can be easily and quickly implemented in a generic application. Therefore elaborate approaches incorporating detailed location specific data, such as might be found in a regulatory impact analysis benefits analysis for a criteria pollutant, could not always be undertaken.
- o to use existing analyses, literature, or results that can be readily made available; and
- o by focusing only upon the identification appropriate studies and approaches and not upon the actual calculations that ultimately need to be made. These calculations will be undertaken in the next research

step. Further, by being conducted as a quick response task, some substantive issues, problems and reviews of new work are unaddressed.

In places, the Phase 1 plan is incomplete due to the above limitations and can be extended during the implementation phase as available data become known or as new research results become available. Even with new literature, the analysis will require substantial use of assumption and approximation, and numerous benefit categories will remain unquantified. This report only examines the selection and application of physical and economic studies assuming **NO_x** controls translate into NO₂, nitrates, ozone and acid deposition.

- o Section 2 overviews the general benefits analysis approach and briefly identifies issues that underlie such an analysis.
- o Sections 3 through 5 discuss the selection of physical and economic studies.
- o Section 6 summarizes the studies selected by health and welfare effect category and identifies air quality relationships and variables that will have to be addressed as inputs to the analysis. Alternatively, approximations based upon previous economic studies could **be determined** and employed to negate the need for some of the listed air quality relationships.

Phase 1 was completed July 10, 1987.

Phase 2: Application to **NO_x** Emission Links in the Eastern U.S.

For Phase 2, U.S. EPA provided four scenarios of **NO_x** control limits in 31 eastern U.S. states and Washington, D.C. The four scenarios are:

- o Controls on existing plants with .5 pound per MMBtu NSPS limit beginning in 1995 evaluated with conditions in 1995.
- o Controls on existing plants with .4 pound per MMBtu NSPS limit beginning in 1995 evaluated with conditions in 1995.

- o Controls on existing plants with **.5** pound per **MMBtu** NSPS limit beginning in 1995 evaluated with conditions in 2000.

- o Controls on existing plants with **.4** pound per **MMBtu** NSPS limit beginning in 1995 evaluated with conditions in 2000.

To implement the analysis, a computer model was developed to calculate point, upper bound and lower bound estimates. Sensitivity tests of key assumptions were also conducted. These detailed procedures and calculations are reported in Chapter 7 and Appendix A. Phase 2 was completed August 10, 1987.

2 .0 DESIGN PLAN AND ISSUES

This section briefly discusses the design and presentation strategies of the benefits analysis, issues inherent in conducting such an analysis, and the general criteria for study selection. The purpose is to put the discussions of Chapters three through five into perspective.

2.1 BENEFIT ANALYSIS DESIGN AND ISSUES

For the best possible accuracy, a benefit analysis should follow a multiple step process including many of the following steps:

1. Quantify emission changes in terms of rate, location and timing.
2. Translate emission changes to changes in ambient air pollution conditions at different locations and time periods, and for all air pollution measures of interest, such as annual average, daily high readings, etc. This requires consideration of whether emission changes will affect ambient outdoor and indoor pollution levels on all hours equally, affect only certain hours, etc.
3. Translate air pollution conditions to exposure.
4. Translate exposure to changes in human health and welfare accounting for conditions by location, time period of relevance, thresholds in response functions, non-linearities in response **functions**, and other relevant factors. Ideally, this would cover all potentially adverse impacts. The analysis would consider air pollution induced changes in behavior that mitigate adverse impacts.
5. Value changes in predicted health, velfare and behavioral impacts.

In some economic analyses, the procedures employed allow the direct comparison of different ambient conditions and economic values without steps 3 and 4.

Some of the issues in the above process are of considerable importance. For example, many damage functions apply only above certain thresholds or exhibit strong changes in functional form below the threshold. Further, changes in how one presumes emissions will affect ambient concentrations such as through changes in all hours, changes in all hours above a threshold, or changes in only peak hours during certain periods, can dramatically affect results. Work by Krupnick et al. (1986a) and Chestnut et al. (1987) indicate that assumptions about thresholds and how changes in ambient conditions are manifested can alter the benefit analysis by up to a factor of 10.

Implementing a comprehensive benefit assessment process may be quite costly and more elaborate than is needed, particularly when it is apparent that the benefits and costs of control are greatly different. A generic analysis of the type discussed herein can often be used to obtain a good understanding of the likely range of benefits, especially for marginal changes in ambient conditions, and to analyze which assumptions the analysis are most sensitive for future refined analyses. In these cases many of the design issues discussed still **will** be of importance, but can either be totally or partially controlled for, or acknowledged, in the analysis.

Threshold assumptions are still of concern and should be addressed to the degree possible. One must attempt to control for the percent of the affected population that is below the threshold so as not to calculate changes in damage where none exist. Some studies have calculated estimates of total economic damage for an effect category for a pollutant without explicitly addressing the threshold question. Without recognizing thresholds, one might presume the damage for a one percent change in ambient pollution concentration levels would be one percent of the total damage. If there is a threshold, however, true damage from a one percent change in ambient pollution levels would be greater than one percent of the total damage estimate.

The design of this benefit analysis considers impacts of reductions in NO_x emissions through changes in ambient levels of NO_2 , nitrates, ozone formation and acid deposition. The atmospheric chemistry aspects of the analysis are not considered. General benefit categories include: human health, visibility,

materials, forests, agriculture and other vegetation. Other possible categories exist but are not quantified due to limited empirical physical injury and economic damage studies. Within the categories, only some potential benefits are addressed due to limited available studies upon which to base estimates. This is particularly true for acid deposition impacts.

2.2 BENEFIT ANALYSIS PRESENTATION

It is proposed that the benefit analysis presentation be divided into three categories:

1. Presentation of quantified benefits in terms of probable and speculative benefits. Probable quantified benefits are those for which there is some consensus that the effect exists and physical injury and economic damage functions are potentially reasonable. However, best estimates, and reasonable lower and upper bound estimates should all be provided to 'recognize controversy, uncertainty and inaccuracy.

Speculative quantified benefits are those for which there is evidence that the effect exists at ambient pollution levels (and certainly no overwhelming evidence that the effect may not exist) for which physical and economic functions exist, but where there is considerable controversy over the accuracy of the evidence or the quality of the physical injury or economic damage functions.

2. Listing of probable but unquantified damages. Many studies suggest effects that may lead to damage, but the amount of damage cannot be quantified or yet translated to value measures. For example, **temporary** changes in lung function are often difficult to translate to quantified benefits. We do not suggest listing speculative effects categories that use results on one endpoint to infer similar results for another endpoint or population. We suggest their deletion because there is a potential unending list of speculative inferences with little or no evidence to currently support their inclusion. These inferences may

sometimes be correct and lead to new **productive** research. Omitting these endpoints may result in understatement of impacts.

3. Listing of significant omissions and biases. Omissions are tied to both unquantified effects and the manner in which results are applied. Most important in this section is to identify the direction of omissions and biases, and where possible identify how significant the bias might be relative to the included impacts. Again, there is the potential for an unending list of omissions and biases, and the interpretation of importance is strictly a **judgment of** the researcher. Therefore, we recommend the listing of only the most significant omissions and biases based on the analyst's judgment.

To the degree possible, it is desirable to tailor the analysis to urban/rural and east/west categories.

2.3 STUDY SELECTION

Studies selected for use in quantified benefit estimation are based upon several criteria. First, the study needs to provide endpoints useful in a benefits assessment. For example, many health measures are difficult to translate to economic benefits. Second, epidemiology studies are preferred for estimating effects on human health because they tend to have much larger and geographically dispersed sample populations in real life conditions upon which to base inferences. However, these studies may understate impacts (and therefore welfare losses) due to unmeasured mitigating behavior (Rowe et al. 1985b). Third, the technical approach and adequacy of the studies are considered. This includes the air pollution measures included controls for other pollutants and significant variables such as exposure to smoking, quality of statistical **analysis**, and reporting of results.

3.0 DIRECT NO₂ AND NITRATES EFFECTS

3.1 NO₂ AND ' HUMAN HEALTH EFFECTS

The McGartland and Ostro Review

The national NO₂ standard is 100 $\mu\text{g}/\text{m}^3$, or .053 ppm annual average. California has an additional 1-hour standard of .25 ppm, or 470 $\mu\text{g}/\text{m}^3$, and a 24-hour standard of .15 ppm used to trigger a stage 1 pollution alert. NO₂ standards are seldom exceeded. Even in the South Coast Air Basin of California, the California 1-hour standard is seldom exceeded and then only on a few hours per year.

McGartland and Ostro (M&O) review the literature through 1984 and conclude there are possible effects at NO₂ levels that exceed, or far exceed the standard, but there is little in the way of effects due to outdoor exposure of NO₂ at ambient levels. Some of the controlled human exposure studies do suggest mild reversible effects on individuals with chronic lung disease when exercising and exposed to .3 to .5 ppm NO₂ and other added stresses, but the study population sample sizes are quite small (Kerr et al. 1979 and Bauer et al. 1986).

M&O note the results from outdoor epidemiological studies results are mixed, with limited evidence of NO₂ affecting lost school days for children and potentially affecting restricted activity and bed disability days for children (Portney and Mullahy 1983) based upon examination of the "Chattanooga" (or CHES) studies.

Turning to indoor epidemiology studies, results suggest potential effects upon children, non-smoking adults and asthmatics in gas-stove homes in terms of reduced pulmonary function, more coughs, colds and other symptoms. However, the results are quite mixed with Comstock et al. (1981), Heising et al. (1982) and Lebowitz et al. (1982) all finding significant relationships, while Ware, et al. (1983), Keller et al. (1979) and others find no significant relationships. M&O conclude that outdoor levels of NO₂ affect indoor levels resulting in minor

symptoms to children, which are valued at \$50 to \$70 per symptom episode (as an admittedly high value) to result in \$1.23 per ton of NO_2 controlled.

New Studies

New animal studies show effects to mice brain serotonin (Sherwin et al. 1986) and short-term effects to rate alveolar epithelium with no difference between adults and juveniles (Chang et al. 1985). These studies make contributions to long term research, but provide little to augment a benefits analysis. Bauer et. al. (1986a,b) report on recent controlled human exposure studies. Asthmatics exposed for 30 minutes to NO_2 at .30 ppm while at rest showed no affects. Exposed asthmatics who were exercising, or who were exercising and exposed to cold air, showed reduced pulmonary function. However, one hour after exposure and exercise, pulmonary function had returned to baseline values. In a similar study of 20 chronic obstructive lung disease patients, a 4 hour exposure to .30 ppm NO_2 while intermittently exercising also showed reduced pulmonary function. Bylin et al. (1985) exposed 8 normals and 8 asthmatics to controlled doses of NO_2 at 0, 230, 460 and 910 $\mu\text{g}/\text{m}^3$ NO_2 for 20 minutes. In the non-asthmatic group airway resistance increased significantly after 20 minutes exposure to 460 $\mu\text{g}/\text{m}^3$, but decreased significantly after the same exposure to 910 $\mu\text{g}/\text{m}^3$. The same trend was found in asthmatics, but it was not statistically significant. This counterintuitive result suggests that low levels currently experienced are increasingly adverse, but that very high levels are increasingly beneficial.

Other recent studies by Frampton et al. (1986) and Kulle et al. (1986) also show that NO_2 may potentially enhance infectivity, but at levels higher than experienced.

The usefulness of these recent controlled human studies in benefit analysis is limited as the exposure levels are generally higher than experienced outside, sample sizes are small, the number of asthmatics or COPD patients exercising during the few high NO_2 episodes is likely to be very small as they often aware of air pollution conditions (Rowe and Chestnut 1985a) and the effects are potentially very minor and transitory. These effects could, however, be listed as probable but not quantified based upon these studies.

Several additional epidemiology studies have also been conducted. Harrington and Krupnick (1985) exhaustively reexamined the CHES data and found a consistent statistically significant (although not for all subgroups) and confusing relationship between ambient NO_2 and acute respiratory disease in children. The study used as its NO_2 measure the two-week average of the daily high hour. The relationship indicates declining rates of illness between 27 $\mu\text{g}/\text{m}^3$ (the lowest level in the study) and about 100 $\mu\text{g}/\text{m}^3$, and increasing rates of illness to 400 $\mu\text{g}/\text{m}^3$ (the highest level in the study), but at 400 $\mu\text{g}/\text{m}^3$ level, the rate of illness had not returned to that estimated at 27 $\mu\text{g}/\text{m}^3$. These results may be valid, but are not consistent with chamber evidence (Bylin et al. 1985), and, as suggested by the authors, may be the result of other factors. Until more supportive evidence of this V shaped relationship can be found, these results should be discarded for benefit analysis.

Berkey et al. (1986) studied 7,834 children exposed to sidestream cigarette smoke or gas stove emissions for changes in pulmonary function (PF) and growth rate changes. This report further updates the longitudinal Six Cities Study, which had shown no consistent pattern of increased risk for children from homes with gas stoves (Ware et al. 1984). The earlier study had shown that 24-hour NO_2 levels were four to seven times higher in homes with gas stoves than in homes with electric stoves, but generally below 0.05 ppm (Speizer et al. 1980). In the updated results, children whose mothers smoked showed statistically significant decreases in both PF and growth. Children in gas stove homes showed statistically significant PF changes, but no growth changes. However, the PF changes associated with gas stoves were less than .1 percent per year and substantially less than the effects of sidestream smoke.

Lebowitz et al. (1985) examined daily symptoms of 117 households, including 23 asthmatics, using a diary. The study found daily prevalence rates of productive cough in asthmatics were correlated with carbon monoxide, outdoor NO_2 and gas stove usage. In allergic subjects, outdoor NO_2 was associated with increased eye irritation in moderate to high temperatures. Lebowitz et al. also found that PF decreased with increased outdoor NO_2 only for those with electric stoves. Those with gas stoves had significantly lower PF than those with electric stoves when outdoor NO_2 was low. Moreover, while gas stoves were used

as a proxy measure for indoor **NO₂**, indoor carbon monoxide was found to be significantly correlated with gas stove usage.

New work by Schwartz et al. (1987) has reexamined the Hammer et al. (1974) Los Angeles student nurse data and found a significant correlation between **NO₂**, oxidants and eye irritation. These results may be reflecting the effects of both **pollutants**, or could be reflecting PAN which can be correlated with **NO₂**. The **NO₂** coefficient from this study is suggested for use in estimating eye irritation for adults. This work is further discussed in Section 4.1.

Ogston et al. (1985) studied the relationship between the use of gas for cooking and respiratory illness in 1565 infants in **Dundee**, Scotland. Again, a strong statistically significant relationship was found with parental cigarette smoking,, while the use of gas stoves resulted-in higher, but not statistically significant, rates of respiratory illness and hospital admissions. Remijn et al. (1985) and Fisher et al. (1985) examined pulmonary function of women in Dutch homes where gas was used for cooking and water heating. The weighted mean values of personal exposure to **NO₂** were estimated to be **.20** ppm. No measure of **NO₂** exposure showed a significant relationship with any pulmonary function decline.

Summary

The Berkey et al. **and Lebowitz** et al. studies continue to support the implication that gas stoves may injure health. However, the Lebowitz results indicate the health effects may also be related to other pollutants as well as **NO₂**. **Moreover**, changes in indoor **NO₂** levels are small and not so well related to **changes** in outdoor **NO₂** levels as to yield a consistent relationship between outdoor **NO₂** and health effects even for those with gas stoves. As such, and combined with valuation overstatements in the M&O analysis (see Section **4.1**), it appears the value of outdoor **NO₂** reductions on indoor exposures is, based upon current evidence, much smaller than the **M&O** estimated **\$1.23/ton NO_x** and should simply be listed as a possible health consequence with no value quantification. The Lebowitz et al. study could potentially be used to measure health effects of outdoor **NO₂** if the results, data or full study report can be obtained and analyzed in more detail. However, the relevant population (asthmatics and

allergy sufferers) is relatively small. Therefore, the Schwartz et al. (1987) analysis relating eye irritation to outdoor NO_2 for adults is recommended for the basis of the best, upper bound and lower bound estimates. Other effects for adults, and -effects for children, are possible, but there is no current evidence to merit their quantification. New results relating NO_2 to health status have recently been found by Mullahy (1987), but are unreviewed. These should be considered once they are released.

3.2 NITRATES AND **HUMAN HEALTH** EFFECTS

Changes in NO_x emissions will result in changes in ambient nitrate concentrations (predominately ammonium nitrate) and the formation of potentially carcinogenic and toxic nitrogenous compounds such as PAN, nitroarenes and nitrosamines (CARB 1985). The effects of nitrates on human health, although not extensively studied, can be considered through their contribution to ambient concentrations of particulate matter (PM), which has been extensively studied using measures of total suspended particulates (TSP), particulate matter less than 10 microns diameter (**PM10**) and fine particulates less than 2.5 microns diameter (FP). Because the relative potency of nitrates vis-a-vis other constituents of PM is unknown, some assumption will be required. To implement a benefit analysis will also require estimates of the relative share of nitrates in average PM. As M&O did not discuss effects due to nitrates, we present detailed discussion of recommended analyses for best, upper bound and lower bound estimates.

Mortality

There are two types of epidemiological studies that have found an association between ambient **PM/TSP** levels and human mortality rates. One type of study (time-series) has looked at changes in mortality rates from day to day in an area as PM levels fluctuate, and the other type of study (cross-sectional) has looked **at** differences in mortality rates across cities with different PM levels. In both cases a significant association between PM and mortality rates have been found by several different authors. Questions continue to arise about whether these studies have adequately controlled for potential confounding factors and

whether the results can be interpreted as evidence of a causal relationship. For this reason, we recommend selecting zero as a plausible lower bound estimate of the number of deaths associated with exposures to PM.

Lave and Seskin (1977) conducted an analysis of the relationship between PM and mortality rates for 100 U.S. **SMSAs** in 1960 and 1969. They found a significant positive relationship for both TSP and sulfates, but the work has been criticized because control variables such as smoking and diet were not included. **Chappie** and Lave (1982) used 1974 data for 104 U.S. **SMSAs** adding diet, smoking and occupational variables to those used by Lave and Seskin, and found combined effects of TSP and sulfates of a comparable magnitude.

Evans et al. (1984) conducted a re-analysis with the original Lave and Seskin data, adding more potential confounding variables and correcting for some coding errors in the original data. Their results, shown below, indicate a significant relationship between mortality rates and TSP and/or sulfates that is about half the magnitude of the relationship estimated by Lave and Seskin. These results are recommended for use in this analysis as the upper range estimate.

$$\text{Change in annual deaths/100,000} = .338 * \text{change in TSP}$$

where :

$$\text{TSP} = \text{annual arithmetic mean in } \mu\text{g/m}^3$$

The best estimate of deaths associated with PM can be based on analysis of data from London for 14 winters, **1958/59** through 1971/72. A significant relationship was found between daily deaths and daily levels of British Smoke (BS), a measure of PM that is somewhat different than TSP. These data have been analyzed by Mazumdar et al. (**1982**), Ostro (1984, **1985**), and Schwartz (1986). In its simplified form, the relationship estimated by Schwartz is as follows:

$$\text{Change in daily deaths in London} = .0881 * \text{change in daily BS.}$$

To convert this to TSP will require an adjustment. The California Air Resources Board staff paper on PM (CARB, **1982**), for example, reports that BS is

approximately 55 percent of TSP. Alternatively, recent EPA staff work using data from London estimates a ratio of BS to TSP of 71 percent. As converting from London BS in the early 1960's to U.S. TSP in the 1980's is a difficult leap, we suggest using the conservative 55 percent figure in the best estimate, while recognizing that the 71 percent figure would push the best estimate up by nearly 30 percent, or to nearly equal the upper bound estimate.

The approximate population in London during this period was eight million. The previous equation must also be multiplied by 365 to convert it to an annual estimate of deaths. Thus, the London data suggest the following estimates of annual deaths:

$$\begin{aligned} \text{Change in annual deaths/100,000} &= .0881 * (100,000/8,000,000) \\ & * .55 * 365 * \text{change in TSP} \\ & = .221 * \text{change in TSP} \end{aligned}$$

where :

$$\text{TSP} = \text{annual arithmetic mean in } \mu\text{g/m}^3.$$

The mortality results from Evans et al. and others are presented in terms of annual deaths per 100,000 people attributable to **PM** exposures, but it should be noted that everyone in the exposed population could not be expected to face the same risk of premature death. The earlier work by Lave and Seskin indicated that the majority of the deaths would be expected to occur for individuals aged 65 and over. Those with chronic illnesses would also be expected to be at greater risk than average.

Emergency Room Visits (ERV)

Samet et al. (1981) analyzed the relationship between emergency room visits (ERV) and TSP levels in Steubenville, Ohio. Daily ERV at the larger and more urban of the two hospitals in the area were matched with daily levels of TSP, SO₂ and **NO₂** for March, April, October, and November of 1974-1977. Visits were regressed on maximum temperature and each of the pollutants in separate estimations. The TSP and **SO₂** coefficients were statistically significant in

separate equations, but the high correlation between these two pollutants (.69) means that their effects cannot be separated. To the extent that SO₂ may also be affecting ERV, applying the Samet et al. findings nationally may overstate expected ERV-because the SO₂ levels in Steubenville during the study period are relatively high. There is, however, no strong evidence in the literature to suggest that SO₂ has an independent effect on ERV.

The estimated coefficient for daily TSP in a linear regression of daily ERV on TSP and temperature was .011. The authors do not report a standard error but report that the coefficient was significant at the 95 percent level, but not at the 99 percent level. This means that the t-statistic is between 1.960 and 2.576. Since the coefficient divided by the standard error equals the t-statistic, the standard error must be between .0043 and .0056. The 95 percent confidence interval for the estimated TSP coefficient (+ 1.960 * the standard error) is therefore between + .008 and + .011. For this analysis, + .010 was suggested for calculating the upper and lower ranges.

The coefficient must be divided by the Steubenville population to obtain an estimate of per capita ERV that can be applied. The authors report a population of 31,000 at the time of the study. Because the estimated relationship between ERV and TSP is linear, the annual change in ERV is 365 times the daily change. The following calculations can be made to estimate the change in annual ERV:

$$\begin{aligned} \text{Lower change in annual per capita ERV} &= (.001/31,000) * 365 * \\ \text{change in TSP} &= .000012 * \text{change in TSP} \end{aligned}$$

$$\begin{aligned} \text{Best change in annual per capita ERV} &= (.011/31,000) * 365 * \\ \text{change in TSP} &= .00013 * \text{change in TSP} \end{aligned}$$

$$\begin{aligned} \text{Upper change in annual per capita ERV} &= (.021/31,000) * 365 * \\ \text{change in TSP} &= .00025 * \text{change in TSP} \end{aligned}$$

Where:

$$\text{TSP} = \text{annual arithmetic mean in } \mu\text{g}/\text{m}^3.$$

Because the Samet et al. study **was** conducted in an area where the components of TSP may be significantly different than national averages (there is considerable steel industry activity in Steubenville), the error involved in applying these results to changes in TSP nationally is potentially greater than may be indicated by the upper and lower bound range.

Restricted Activity Days

Estimates of an association between restricted activity days (PAD) and PM can be based on Ostro (1987). **RAD** include days spent in bed (bed disability days: **BDD**), days missed from work (work loss days: WLD), as well as days when activities are restricted to a minor degree due to a health condition (minor restricted activity days: MEAD). Ostro estimated a relationship between RAD in a two-week period and fine particles (FP, diameter less than 2.5 microns) in the same two-week period in the U.S. Separate regression estimates were obtained for six years, 1976-1981. The RAD data were from the Health Interview Survey conducted annually by the National Center for Health Statistics. The FP data were estimated from visual range data available for airports in each area. Fine particles have a more significant impact on visual range than do larger suspended particles, so there is a direct relationship between visual range and the amount of FP in the air. A statistically significant result was -found in each year. This study follows and supports the findings of an earlier study (Ostro, 1983) where a significant relationship was found between **RAD** and TSP measured at stationary monitors in each area.

The form of the estimated relationship was such that the coefficient for FP gives the percentage change in RAD associated with a unit change in FP. For any two-week period *i*, the change in HAD would be:

$$\text{Change in } \mathbf{RAD}_i = b * \mathbf{RAD}_i * \text{change in } \mathbf{FP}_i$$

Where :

b = estimated coefficient.

The equation **above** can be converted to give an annual estimate of changes in **RAD** if one accepts the following assumptions.

(1) $RAD_i = RADA/26$, where **RADa** is the annual average number of RAD. This assumption is required due to data availability because **RAD** data for every two-week period of the year are not available.

(2) $(\sum_{i=1}^{26} FPI)/26 = FPa$, where **FPa** is the change in the annual average FP.

This assumption is valid if the number of observations in each two-week period is equal.

Thus, the annual change in RAD can be estimated as follows:

$$\text{Change in RADA} = b * RADA * \text{change in FPa.}$$

Data from the National Center for Health Statistics indicate that the average annual number of RAD per person in the U.S. is about 19 days. Equation (2-8) can thus be written:

$$\text{Change in RADA/person} = b * 19 * (FP/TSP) * \text{change in TSP}$$

Where:

$$TSP = \text{annual arithmetic mean in } \mu\text{g/m}^3.$$

The estimated coefficient (b) for FP ranged from **.003** to **.009** over the six years for which it was estimated. The estimated coefficients fell into two groups: **.003** to **.004** and **.006** to **.009**. The upper range estimate for this analysis could be selected as the mean of the higher group and the lower range estimate as the mean of the lower group. The best estimate is recommended as the mean of all six estimates.

Selected Coefficients

Upper	.0076
Best	.0048
Lower	.0034

For consistency, the estimated number of ERV **needs to** be subtracted from the estimates of RAD since these would also be considered restricted activity days.

Economic Values

Mortality effects are difficult to value. However, a recent review by Violette, Chestnut and Fisher (1986) suggests a lower bound of \$1.5 million. Recent work by Klema (1987) indicates the federal government appears, on average, to be using \$2 million in regulatory decisions. \$1.5 million may be a reasonable lower bound for this analysis, as risk values appear to increase as the involuntary nature of the risk increases, as may be the case with air pollution. Upper bound estimates, based upon the Violette et al. review, are recommended as \$8 million and a mid-point estimate of \$4.5 as approximately the average of the upper and lower bounds.

Values for **RADs** are recommended at \$44, which is the average of **MRADs** and **WLD/BDDs**, reflecting that **RADs** are often less severe than **WLDs** and **BDDs** (see ozone values). The value of an ERV can be based upon value of time, average visit costs and discomfort. Chestnut et al. (1987) suggest \$265 (\$1986) based upon California data. Eye irritation values are discussed in Section 4.1.

3.3 **NO₂**, NITRATE AND VISIBILITY EFFECTS

Physical Relationships

Nitrogen oxide emissions can affect visibility through both gaseous **NO₂** and fine particle nitrates (Trijonis 1987). **NO₂** absorbs and discolors light while fine particles scatter light, each of which increases light extinction and reduces visual range. Three studies are of primary importance in estimating the value

of NO_x reductions: Trijonis (1987), which examines the component of light extinction due to NO_x , and the work of Chestnut and co-authors (1986a,b), which summarizes the economics value literature. Because each of these works are new since the M&O report, they are given extended discussion.

Trijonis provides a national study of the relative share of visibility reduction due to NO_x . Trijonis' analysis is based upon the review of numerous studies in urban and rural locations nationwide. As a result, the results are based not upon any one study, but on the consensus of evidence. Upon review of all available studies on NO_2 and nitrate concentrations and visibility, their average contribution to extinction is calculated according to east/west and urban/rural categories. The results are summarized in Table 3-1. The underlying data suggest the contribution of nitrates is approximately double that for NO_2 in the metropolitan west and the rural east, approximately 50% larger in the rural west, and about the same as NO_2 in the metropolitan east. The Trijonis estimates provide a population weighted national estimate of 12% \pm (2.5-3.75%). East/west and urban/rural assessments could also be conducted with results reported by Trijonis. To calibrate the model, Trijonis assumes NO_2 and fine particle nitrate concentrations are directly proportional to NO_x emissions. While this assumption does not exactly hold for nitrates, it is perceived as a reasonable first approximation.

Another consideration is that the model pertains to regional haze, or at best to layered haze. NO_x emissions are also associated with coherent plumes that may be of particular importance in western regions. This problem is not addressed in the Trijonis work.

Economic Valuation

Economic studies of visibility aesthetics have typically focused upon changes in visual range associated with regional haze. In most of these studies a whitish haze is considered. However, work by Middleton et al. (1985), Brookshire et al. (1979) and Schulze et al. (1981) also suggest that discoloration and coherent plumes also cause potential reductions in welfare. Visibility studies are typically considered in two settings: residential and recreational.

Table 3-1

a. Contribution of **NO₂** and Fine Nitrates to Light Extinction*
(Trijonis 1987)

	Metropolitan	Rural	Population-Weighted Metro/Urban Total
West	23% ± 4.5%	10% ± 3%	20% ± 4.5%*
East	10% ± 2.5%	7% ± 2.5%	9% ± 2.5%"
Population Weighted East/West Total	13% ± 3%	8% ± 2.5%	12% ± 2.5%

b. Contribution of **NO₂** only to Light Extinction
(Trijonis 1987)

	Metropolitan	Rural
West	8% ± 1.5%	4% ± 2%
East	5% ± 1.5%	2% ± 1.5%

* According to Trijonis, the propagated uncertainties in the composite results were inflated by a factor of 1.3 to account for non-independence of the individual uncertainties. In using these results as a national NOx/visibility model, the uncertainties should be increased even more, perhaps by another factor of 1.5, to account for potential errors in the assumptions of such a model.

Turning to valuing visibility changes in residential settings, Chestnut et al. (1986) review the available studies and provide consensus valuation functions covering a broad range of baseline conditions and visibility changes. These functions are estimated across data from cities in the east and west using data from studies by Tolley et al. (1984), Rowe et al. (1980), Rae et al. (1979), Brookshire et al. (1979) and Loehman et al. (1981). As a result, the functions are best considered as national relationships. The consensus functions for residential visibility values are preferred to individual studies as:

- o Each study has individual strengths and limitations making the selection of one study as "best" difficult.
- o Some studies cover only one region with substantially different baseline and proposed changes in visibility conditions. The consensus approach is able to utilize this information to provide functions over a broad range of conditions rather than be constrained to "best" estimates only applicable to one region or set of visibility conditions. In addition, the functions overcome the misleading information sometimes presented when radically different \$/mile figures are compared across different studies. This finding is primarily the result of differences in the baseline and change in visibility conditions used in the studies; these factors are handled in the consensus functions.
- o The consensus estimates are fairly robust to the inclusion or exclusion of individual studies. The estimates are somewhat sensitive to functional form, although less so for small changes in visual range of the type that might be associated with NO_x control.

Two primary functions are reported: a quadratic function, and a semi-log function. The semi-log function is preferred for this analysis because of superior statistical fit and because the function is immediately compatible with the Trijonis NO_x visibility model. The semi-log function is:

$$\text{Value for Visibility Change} = 205 \cdot \text{LN}(\text{VR2}/\text{VR1}) + Z$$

where :

Value = annual household value in \$1986
LN = natural log
VR1 = initial visual range level (miles)
VR2 = new visual range level (miles)
Z = other control variables generally statistically insignificant

The Trijonis and Chestnut et al. models can be used to estimate either regional or national NO_x residential visibility value estimates as follows:

- o Calculate the percent change in NO_x emissions (for example 1%) and assume this is proportional to changes in NO_2 and nitrates (here we assume the percent change is equal).
- o Multiply this value by **.12** to obtain the population weighted percent change in the extinction coefficient (for example, **.12 * 1% = .12%**).
- o Convert percent change in extinction coefficient to percent changes in visual range for use in economic analysis (for example, **[100/(1-.0012)] - 100 = .12%**).
- o Input the percent change in visibility to the value model (for example, **205 * LN(1.0012) = \$.246** per year per household).
- o Multiply by households and divide by one percent of total NO_x tons to arrive at \$/ton.

Upper and lower bounds of **.5** and 1.5 of the best estimate are suggested to account for variation across economic studies, methods and functional forms and to account for uncertainties in the Trijonis model.

Chestnut and Rowe (1986) summarize and develop a consensus visibility value function for use in value studies at park and recreation areas outside of major urban areas. These data have been reanalyzed into a semi-log function as:

Value for change in visibility. = $5.136 * \ln(VR2/VR1) + Z$ per recreator party per day

where the variables are defined as above.

Total recreational use value estimates are likely to be small compared to total residential values. Again, use of .5 and 1.5 of the best estimates captures the variation across most studies for upper and lower bound estimates.

To estimate use values related to plumes, Chestnut and Rowe (1983) used the results of several studies conducted primarily in 'the southwest. The results suggest average values by park users of \$2-\$5 per plume day at the park. M&O's analysis using similar results for the Kaiparowits study by Brookshire et al. (1976) translate these values to about **\$15/ton** of **NO_x** in 1976 dollars. If plume days in southwest parks and plume tons could be provided, plume use values could be separately estimated.

The two above recreational use values ignore potential non-use values for visibility conditions at national parks. Using the results of **Schulze et al. (1981)**, Chestnut and Rowe (1983) calculated total non-use value for haze reduction at southwest national parks at 11 to 200 (200 for the Grand. Canyon) times use value totals, and non-use value totals for plumes at 21 times use value totals . Further calculations estimated the total value of preventing a plume day in a southwest national park of about \$2.3 million (1986) per plume day. Because these estimates are based upon preliminary analyses of preservation values that are considered less reliable, these estimates can only be categorized and quantified as speculative upper bound estimates.

Presentation of uncertainties and omitted value components in visibility value research can be presented based upon Chestnut and Rowe (**1986**), and identification of discoloration impacts literature.

3.4 **NO₂** AND **MATERIALS** EFFECTS

As identified by M&O, **NO₂** is associated with textile dye fading resulting in reduced useful life and increased mitigation costs (NAS **1976a**, 1977). M&O use inflation escalated estimates from Barrett and **Waddell** (BW) (1973,) to calculate per ton **NO₂** damages of \$14.2 (\$1986). We have found no new evidence upon which to base estimates of textile dye fading. However, two consideration are in order for interpreting the BW estimates not considered by M&O.

- o Substantial price effective mitigation may have occurred since the BW 1970 estimates resulting in reductions in damages per ton of **NO_x**. For example, current estimates of elastomer damage due to ozone are about 40 percent of the original BW estimates. Some of this may be due to changing ozone conditions, but most is due to more cost effective mitigation. It is plausible the BW **NO₂** damages are now also overstated, perhaps by a factor of two as well.
- o M&O calculated per ton damage in 1983 based upon the 1983 tons of **NO_x**, but 1970 population. The current calculation of damage should be based upon 1970 tons of **NO_x**, or total damages should be escalated by the ratio of **1986/1970** population. Assuming both population and tons have increased since 1970, the benefits per ton estimate would increase with this correction.

Based upon the above considerations, use of the original BW numbers, corrected for population and prices, serves as a plausible point estimate, with a lower bound estimate at **.5** of the point estimate and with an upper bound estimate set equal to the point estimate.

Little other evidence is available regarding **NO₂** materials damage. Kucera (1983) does suggest the importance of **NO₂** to metallic corrosion appears to be quite small and Johansson reports that **NO₂** causes synergistic corrosion effects to carbon steel, zinc and copper in the presence of **SO₂**. **NO₂** materials damage is an area where substantial improvements in estimates are warranted to account for new materials and to identify thresholds or non-linearities in the damage functions.

3.5 NITRATES AND MATERIALS EFFECTS

NO_x emissions result in nitrates, which can be considered as a component of TSP. Damage due to TSP is related to increased soiling and discoloration of materials. Materials damage estimates from TSP can be addressed through use of the economic analysis in the PM10 RIA based upon the work of Manuel et al. (1982). The limiting factor is the need to obtain an estimate of the share of nitrates in TSP, and the individual relative importance of nitrates to soiling and other materials damage compared to other components of TSP. To bridge the second problem, we suggest assuming nitrate damage and soiling rates equal to their share of total TSP damage and soiling rates. Improved assumptions are desired. Review of the Manuel et al. work in Chestnut et al. (1987) suggests the following rough estimates based upon the upper and lower bounds in that study.

$$DY = 15.00 * DTSP2 \text{ (upper bound)}$$

$$DY = 7.75 * DTSP2 \text{ (point estimate)}$$

$$DY = .50 * DTSP2 \text{ (lower bound)}$$

where :

DY = changes in annual household damages (\$1986)

DTSP2 = changes in annual second high 24 hour reading in $\mu\text{g}/\text{m}^3$ for TSP

The Manuel et al. estimates provide bounds for the estimates from other previous studies, and the best estimate, taken as the mid-point of the upper and lower estimates, is roughly consistent with reanalyses of earlier studies by Cummings et al. (1981). These damage estimates are based upon cleaning efforts and expenditures by households. As such, values associated with aesthetic and psychological impacts of soiling or increased cleaning activities may be omitted. Also omitted are cleaning damages incurred at commercial and industrial buildings. As a result, the estimates may be biased downward.

To implement this analysis **requires converting** changes in tons of **NO_x** to changes in nitrates, and the resultant impact upon the annual second high 24 hour reading of TSP. Note also that a one percent change in the geometric average TSP will not likely equal a one percent change in the second high 24 hour reading.

3.6 **NO₂**, NITRATES AND AGRICULTURAL EFFECTS

NO₂ and particulate matter at elevated concentrations are suspected of causing crop damage. However, review of this literature suggests damages would only occur very near to point sources in agricultural regions. Both regionally and nationally, crop loss due to **NO₂** and particulate matter is thought to be substantially less than **SO₂**, and **SO₂-induced** impacts substantially less than ozone impacts. For example, in Rowe and Chestnut (1985b) the estimated dollar value impact of **SO₂** was about 2 percent of the ozone impacts in the San Joaquin Valley. As there is no literature to suggest economic measures of value, and because **NO₂** and particulate matter effects are likely less than 1 percent of ozone damages, such damages should be incorporated with a zero value.

4.0 INDIRECT EFFECTS DUE TO OZONE FORMATION

4.1 OZONE AND HUMAN HEALTH EFFECTS

There is general agreement that short-term exposure to ozone at ambient levels affects pulmonary function and increases the probability of experiencing various respiratory symptoms and other minor symptoms such as coughing and eye irritation. For a detailed review see EPA (1986a) and Krupnick et al. (1986a). The analysis herein relies heavily upon the work in Krupnick where extended discussions and analyses are provided. This report focuses only upon those health endpoints useful in benefits analysis and which have been found to be statistically significantly related to ozone in empirical work. Other health endpoints may also be affected by ozone, but without supportive research can only be considered as unquantified and speculative and listed as such in the subsequent analysis.

Acute Respiratory Effects - RRADS

EPA (1986b) identifies work that suggests ozone causes decreased pulmonary function in controlled human studies. New findings by Liroy et al. (1985) and Avol et al. (1987) continue to confirm this relationship in adolescents as well. Unfortunately, the pulmonary function measures typically used are not conducive to benefits analysis. **Similarly**, the results of animal studies are of limited use in benefit studies. However, several studies have addressed other health endpoints of use in benefit analysis.

McGartland and Ostro (M&O) identify the work by Portney and Mullahy (1983, 1985, hereafter referred to as P&M) as defensible and useful for benefits analysis. P&M used the Health Interview Survey for 1979 to examine the relationship between restricted activity days and ozone levels. To the nationwide health data, the authors added pollution and weather data for each respondent's residential location. A significant positive relationship was found for adults between restricted activity days due to respiratory illness (RRAD) and ozone levels. These data were for two-week periods. The ozone measure was the

two-week average of the daily maximum hour for each location (Additional detailed discussion can be found in Krupnick et al. 1986).

Recent results by **P&M** (1986a) tested 5 different specifications of the relationship between ozone and RRAD for adults. In 4 of the 5 specifications, the estimated elasticity of changes in RRAD for adults with respect to ozone ranged from **.3** to **.6**, with a mean for all 5 elasticity estimates of **.42**. This means that a 1 percent change in ozone is associated with a **.42** percent change in RRAD. The **P&M** (1986a) results are recommended for use in the analysis with the results corresponding to **.3** and **.5** as the lower and upper bounds, and with a "best" estimate as the midpoint of the upper and lower estimates (See Chestnut et al. 1987 and Krupnick et al. 1986 for specifications). Because the baseline RRAD estimate has been reduced to 4.21, or by about one-half of numbers earlier reported (due to earlier reporting error), the total change in **RRADs** due to ozone estimates may significant decrease from some of the earlier reports. Further work should also consider controls for **PM10** and consistency with MRADS in the Ostro (1987) work.

P&M (1985) used a multinomial **logit** model to separate RRADS for adults into approximately 75 percent minor restricted activity days (MRADS) and 25 percent bed disability days (BDD) or work loss days (WLD), and can be used in the valuation analysis.

P&M (1983) also examined **RRADs** for children but did not find any consistently significant effects, but did find nearly significant results for children between ozone and school loss days (SLD) and bed disability days (BDD) due to all causes. New work recently completed by Hullahy (1987, but not reviewed or released by EPA in time for this review; these comments are based upon discussion with author) specifically reexamined the children issue. To focus upon possible ozone effects, only SLD and BDD linked to acute respiratory illness are considered. In this reanalysis, no statistically significant (even with low significant levels) relationship between ozone and illness in children is found.

Some have suggested that lack of relationships in the **P&M** data for children may be the result of parental reporting problems in the survey. Recent work

continues to suggest that children are sensitive to ozone, but no more sensitive than adults (Avol et al. 1987, p. 161). On the other hand, arguments are also posited that due to more excess lung capacity for children, temporary small lung function changes due to ozone in children just do not translate into changes in acute respiratory illness. h&o incorporate children using the adult coefficients. Until further clinical or epidemiological evidence can support acute illness effects in children, these speculative damages should not be quantified, but rather listed as possible.

Other Symptoms

Ozone has been associated with a variety of other symptoms ranging from coughing to eye irritation. Hammer et al. (1974) examined self reported respiratory and other symptoms in a sample of 112 student nurses in Los Angeles on days with higher ozone levels over a 3 year period from 1961 to 1964. They found statistically significant associations between ozone and symptoms of coughing, eye irritation, headache, and chest discomfort at different ozone thresholds. Evidence reported by U.S. EPA (1983) indicates that eye irritation is not caused by ozone, but with another component of photochemical smog that commonly occurs in association with ozone and is related to **NO_x** emissions--PAN.

Hasselblad and Svendsgaard (1975) reanalyzed the Hammer et al. results with logistic curves to relate daily maximum ozone to the probability of headache, eye irritation, cough, and chest discomfort on the same day. While specific functions were reported, the confidence intervals were not reported and the significance of the functions is uncertain. The estimated models for Hasselblad and Svendsgaard are reported in Krupnick et al. (1986a).

The Hammer et al. data have been further reanalyzed by Schwartz et al. (1987) with logistic analyses that allows for the time series nature of the data and which incorporates other pollutants, significantly improving the analysis of this data base. Of the pollution variables, Schwartz et al. find only carbon monoxide significantly correlated with headaches, **NO₂** and oxidants significantly correlated with eye discomfort, oxidants correlated with a cough and only **SO₂** correlated with chest discomfort. The results of the Schwartz et al. reanalysis, which is the most comprehensive, are recommended for use in the

analysis, leaving only symptoms of eye discomfort and coughing related to ozone. Further, the results apply only to adults over 18 as children were not included in the Hammer et al. sample.

A recent study by Krupnick et al. (1986b) examined the relationships between symptoms and nine air pollutants for 75,000 daily observations with individuals in the Glendora-Covina-Azuza communities of the south coast air basin. The study was unique in the great number of control variables considered. The authors defined two health effects measures: **ARD1**, where a severe medical indicator occurred on a day, such as a RAD medical advise sought or fever; and **ARD2**, where any one or more of **ARD1** or la respiratory symptoms occurred or eye irritation or headache occurred. Sample sizes were small and statistical significance limited when conducting the **ARD1** analysis so most results focused upon **ARD2**. Ozone was never significant for children but was significant for 'adults resulting in an elasticity of about **.11** between changes in daily symptoms and changes in outdoor ozone. The elasticity is about 50 percent higher when considering changes in symptoms versus estimated ozone exposure outdoors and indoors.

The large sample size of in the Krupnick et al. (1986b) study suggests it may ultimately be more defensible in benefit analysis than the results of the series of studies based on the Hammer et al. student nurse panel. Initially the Krupnick et al. results suggest lower response rates than in the Schwartz et al. reanalysis of the **Hammer** data. However, **ARD2** may for some individuals on some days represent multiple symptoms. Further breakdown of **ARD2**, in terms of regression analysis and in terms of correlation of symptoms, would be useful. Presently, we suggest using the Krupnick et al. results as a lower bound for all symptoms combined and treating **ARD2** as an **MRAD**.

Exercise appears to be a factor that increases adverse response to ozone (Herman, 1972, Colucci 1983, **Avol** et al. 1983, McDonnell et al. **1983**), which is believed to be primarily due to increased effective dose. However, this effect is not always found at low ozone levels. For example, Hackney et al. (1983) found no effect of **.12** ppm ozone on patients with chronic obstructive lung disease exposed during **mild to** intermittent exercise.

Most studies involving exercise measure only lung function and are of limited use in benefits analysis. However, McDonnell et al. (1983) examined lung function and symptom scores for 135 males exposed to "air" and ozone levels ranging from .12 to .40 for 2.5 hours while intermittently exercising. Lung function was affected for ozone levels as low as .12 ppm. Symptoms examined included cough, pain upon deep aspiration and shortness of breath. Significant differences in cough scores were found for .12 ppm and above, and for shortness of breath and pain upon deep breathing at .24 ppm. Additional analysis of this study is found in Krupnick et al. (1986a).

The choice and application of symptom functions is entwined with the calculation of **RRADS**. This is because days coughing and chest discomfort may be the symptoms that resulted in a reported **RRAD**. This potential double counting should be avoided. Therefore, for adults (\geq 18 years of age), days with coughing should be calculated with Schwartz et al. (1987), and included only for days in excess of calculated **RRADS** in the upper bound estimates. Earlier analyses suggested that the Hasselblad and Svendsgaard cough and chest discomfort estimates were only somewhat less than the **RRAD** estimates for adults, resulting in deleting these symptom calculations (as reported in Chestnut et al. 1987, and Krupnick et al. 1986), but with the baseline **RRAD** reduced there will likely be excess symptoms to be added in. The Schwartz et al. estimates for eye irritation are also recommended for use for adults.

The McDonnell et al. (1983) results, while potentially useful for benefit analysis, will add little and can be omitted. This is because the estimates overlap with those of Schwartz et al. for several symptoms; they apply only to individuals exercising for a 2-hour period, who comprise only 1.5 percent of the population according to some estimates; and, for the non-overlapping symptoms with the Schwartz et al. symptoms and **RRADS**, require that exercise occur at times of the day when, and locations where, ozone levels are over .24 ppm. This population is quite small. Given the above considerations, the net additional symptoms for benefit analysis will be very small.

It should be noted the symptom studies are all conducted with adults and their applicability to children is unknown. Given the recent **Mullahy** (1987) and Krupnick et al. (1986b) findings of no **MRADs**, **SLDs**, **BDD**, or symptoms for

children related to ozone, there is an argument that symptoms should not be calculated for children. On the other hand, pulmonary function studies do find significant relationships for children. Therefore, until more research is completed, we recommend including symptoms of children only as an unquantified possible health effect.

Asthma Attacks

Asthma attacks may be included in the Portney and Mullahy RRAD analysis, but should be considered separately as the number of asthma attacks may exceed RRAD for asthmatics. Two studies can be used to address incidental rate of asthma attack due to ozone exposure. Whittemore and Korn (1980 hereafter referred to as W&K) studied 16 panels of asthmatics (59% children and 41% adults and a total of 443 subject-years) who lived in the Los Angeles area during 1972-1975. Subjects made daily reports of asthma attack occurrence for a 35-week period each year. Using **logit** analysis, asthma attacks were found to be significantly related to the daily maximum **ozone** hour measured at nearby community monitors. Holguin et al. (1985) used the W&K basic approach in Houston to estimate the probability of an asthma attack as a function of exposure to ozone (both indoor and outdoor), **NO₂** and weather variables. The panel consisted of 51 individuals, with 75 percent being between 10 and 19 years of age. Holguin et al. obtain an attack rate approximately four times the W&K rate for the same change in ozone.

There are potentially significant differences in the two asthma attack studies that bear upon decisions about how they should be applied. Unlike W&K, Holguin et al. decided when a subject experienced an attack based upon analysis of each person's symptom occurrence rather than on respondent self-reported attacks. While the Holguin et al. response rate is greater, the statistical consistency of the results is weaker. A substantial portion of the individuals had the wrong sign on their **O₃** coefficients and the **O₃** coefficient is insignificant in all but 3 of 42 regressions. In addition, the younger Holguin sample may be naturally more responsive (Krupnick et al. 1986) and Holguin et al. measured both indoor and outdoor ozone exposure, which could account for the higher attack rate due to ozone finding.

Another recent study by Gong et al. (1985) examined asthmatics in **Glendora** California. For all respondents as a group, the analysis found no significant relationships between asthma symptoms and ozone. But when examined individually, a subgroup of about 75 percent of the sample showed a statistically significant relationship between ozone and asthma attack. However, the authors assert there is little clinical significance to the statistical results as even a **30+** pphm change in **O₃** would not cause more than a five percent change in a selected lung function measure (PEFR) or change symptom scores by one unit on a seven unit scale. The full reporting of results has yet to be released or reviewed to allow evaluation of the credibility of the findings for this benefits analysis.

One has a diversity of results to choose from concerning ozone and asthma. Based upon considerations identified above, we recommend use of the average of the W&K and Holguin estimates as the "best" estimates. For a lower bound, we recommend using the W&K central results. For an upper bound, we recommend the Holguin et al. central estimate.

Equations relating ozone to asthma attacks and supporting data on baseline attack rates and populations can be found in Krupnick et al. (1986) and Schwartz et al. (1987). One issue of importance is the selection of baseline attack rates. W&K, Holguin et al. and Gong et al. (in Rowe and Chestnut 1985b) all report sample average attack rates much higher than reported in other sources, even when adjusted for study dropouts. This may be due to over representation of active asthmatics in the studies, or differing definitions of an asthma attack. For example, Holguin et al. may be interpreting symptom aggravation, a valid adverse impact, as an asthma attack, **which** might be defined as more severe episodes in other data sources. Therefore, for these estimates, we recommend using each study's adjusted mean attack rate based upon the original study population, rather than impartial national averages, as the definition of attacks may differ.

It is possible that asthma attacks may be included in RRADS. To avoid double counting, asthma attacks should be valued with asthma values, and only attacks in excess of **RRADS** valued and added for the asthmatic population.

Increased Incidence of Chronic Illness

Chronic health effects of ozone have been reported in animal studies, but interpretation of these results for credible benefit analysis is limited. Chestnut and Burchfiel (1986) reviewed the epidemiology literature to examine lung function as a predictor of morbidity and mortality. They find the literature generally supports linkages between lung function and chronic disease, but not necessarily causality. One study (Higgins et al. 1982) does suggest that lung function in healthy individuals was a significant predictor of risks of subsequently developing obstructive airways disease (**OAD**) such that the odds of developing OAD increases three-fold for every 10 percent decrease in forced expiratory volume in one second (**FEV1**). Equations to estimate probabilities of developing OAD within ten years for males and females were also developed. Krupnick et al. (1986) has shown how this study can be linked to changes in ozone based on ozone effects upon lung function. The review by Chestnut and Burchfiel also found a summary relationship between **FEV1** and subsequent mortality rates. This is not completely unexpected as increases in chronic diseases are tied to increased rates of mortality. However, this evidence needs further review before inclusion in a benefits analysis.

Few human studies have directly addressed the relationship between ozone and chronic health incidence. Earlier Portney and Mullahy (1985) analyses did find that among individuals who do not smoke, ozone is positively and significantly associated with the likelihood of chronic respiratory disease (CRD). However, newer results (Mullahy, 1987, as yet unreleased) do not show significant effects for the most expected **CRDs** categories, but do show significant results for some CRD illnesses such as sinusitis.

Given the state of the literature, increased incidence of chronic morbidity should be included in the analysis, but because causality between ozone and chronic illness incidence is not well founded or understood, these damages should be listed as speculative and only as upper bounds. While short-term exposures to high ozone levels do temporarily affect lung function, their effect on long term lung function is unknown, but it is long-term lung function changes that have been generally linked to subsequent OAD and CRD changes. Moreover, there may be some double counting of increased cases of OAD, RRADS and other

symptoms if the RRAD, symptom and **chronic** illness **cases** are **all** added. This is because the **RRAD** equations may be picking up some increases in the number of persons experiencing **RRADs** as well as increases in the per person rate of RRAD.

A speculative upper bound could be based upon an average of calculations using Higgens et al. (1982, see Krupnick et al. 1986b for a procedure) and Mullahy (1987, see Fraas 1986 for a procedure).

Valuation of Health Effects

Values for the adverse health effects considered here have been summarized in Krupnick et al. (1986). The selection of economic values requires **interpreta-**tion of study results and judgement. Generally, the Krupnick summary values are recommended for use herein with a few modifications. A summary of values are reported in Table 4-1. The value sources are discussed below.

Asthma attack values are based upon Rowe and Chestnut (1985a) and work reported in Krupnick. We add further interpretation that the \$9 is from Rowe and Chestnut to avoid experiencing any symptoms by individuals who consider any symptoms a bad asthma day. Because experiencing mild symptoms may not constitute an asthma attack as defined in Whittemore and Korn, this value appears to be a clear lower bound. Rowe and Chestnut also find the value of \$30 per day of at least moderate symptoms by those who considered mild symptoms to be a good asthma day, while Krupnick estimates \$26 using a resource cost approach resulting in a "**best**" estimate of \$26.

The symptom day values were generally derived from willingness to pay studies by Loehman et al. (1979) and Tolley et al. (1986). Value for several symptoms are discussed even though only those with *'s in the table are included in the current analysis. A recent study by Dickie et al. (1987) is also used for symptom values, but only in a limited manner. The Dickie et al. work surveys respondents about the value of symptoms experienced. On an individual **symptom** basis, values were up to an order of magnitude higher than reported in Table 4-1. In part, this is because no extreme bids were deleted. Deleting extreme bids results in values **similar** to or slightly larger than the values in Table 4-1. Next, Dickie et al. totaled the value of all symptoms in a month and **asked**

Table 4-1
Unit Values Of Health End-Points*

End-Point	Estimate		
	Low	Medium	High
*Asthma Attack	\$9.00	\$26.00	\$43.00
Symptom Day			
*Mild Cough	\$4.00	\$ 4.00	\$ 8.00
Mild Short Breath	\$8.00	\$ 8.00	\$19.00
*Mild Chest Congestion	\$6.00	\$ 6.00	\$ 9.00
*Mild Eye Irritation	\$5.00	\$ 5.00	\$12.00
Non-severe Headache	\$5.00	\$10.00	\$22.00
*MRAD	\$12.00	\$24.00	\$36.00
*Bed Rest Day/Work Loss Day		\$84.00	

¹ Estimates updated to \$1986. Estimates from Krupnick et al. (1986) as updated in text. Estimates rounded to the nearest dollar. Symptoms with * included in current analysis.

for rebids. The new average value per symptom dropped to significantly less than those reported in Table 4-1, which the authors attribute to the accounting for all effects. There are many potential concerns with this revised bid analysis and implication, including limited follow-up with respondents on their revised bids. Further, bidding to reduce all symptoms in a month is, in fact, a different circumstance than reducing a few symptoms in a month, which may be more relevant for marginal changes in ambient pollution conditions.

The low and best estimates for mild cough, mild shortness of breath and mild chest congestion are all based upon the Loehman median estimates. The best estimates for mild eye irritation and mild headache are based upon trimmed means of the Tolley estimates. The best estimate for headaches is approximately one-half the Tolley trimmed mean, and the lower bound estimate for headache is arbitrarily set at one-fourth the trimmed mean, as reported in Krupnick. We have increased the best estimate for headaches from what is found in Krupnick (\$5) based also upon the trimmed mean estimate in Dickie et al. (1987).

The upper bound estimates are all (except for shortness of breath) equal to the trimmed mean in the Tolley study as reported in Krupnick et al. These values were selected as upper bounds as the Tolley symptoms descriptions were generally more severe than mild symptoms. The upper bound for short breath was based upon the severe shortness of breath median estimate in Loehman et al.

The low MRAD estimate is based upon a severe median symptom from Loehman et al. The upper bound is based upon results from Dickie et al. (1986) examining the implied MRAD value from data on **MRADs** avoided by air conditioning and costs of air conditioning, which reported an estimated value of \$36. Because other benefits are also gained from air conditioning, this value may be overstated. On the other hand, because other defensive actions may also be undertaken to help avoid the same MRAD, the value may be understated. By comparison, Tolley et al. trimmed mean estimate of a symptom combination, which might be argued as likely to lead to an MRAD, is \$32. The best estimate is taken as the midpoint of the upper and lower estimates.

Work loss days and bed disability days were not provided by Krupnick et al. To estimate these we suggest the approximate average daily wage of \$84. This may

overstate value due to some BDD days not falling on work days, but \$84 may also substantially understate value by ignoring medical costs, discomfort and lost of activities.

Increased incidences of chronic illness presents a difficult valuation problem. One approach would be to compute incremental adverse health effects such as MRAD, TRAD etc., per case, based upon national average symptom rates and values discussed above and in Krupnick et al. **(1986)**, then discount from future incidences to present values. An alternative approach would be to compute average annual cost of illness (medical costs and work loss)-for representative chronic respiratory illnesses from Health and Human Services data, add in values for risk of death, account for pain and suffering, and discount to present values.

The estimates of incidence and valuation of health effects of ozone may have a number of omissions and biases. These should be identified in terms of direction and potential relative magnitude where possible. One such bias is averting behavior. To the extent that individuals avert, or mitigate adverse health impacts, the incidence number times value per incidence calculations will understate total value by missing the costs of the averting actions.

4.2 **OZONE** AND MATERIALS EFFECTS

Ozone is believed to cause materials damage to elastomers, primarily rubber in tires; textiles in terms of dye fading and fiber damage; and potential damage to paints, artwork and a variety of other materials (NAS 1977).

Damage to elastomers is by far the most important damage category. Early estimates of elastomer damage had been quite substantial including Mueller and Stickney (1970) at \$1.2 billion **(\$1983)**, Barrett and **Waddell** (1973) at \$523 million (\$1983) and NAS (1977b) at \$1.4 billion (\$1983). Recent work by McCarthy **(1983)**, EPA (1983) and Stankunas (1983) suggest total damages to elastomers at only \$220 million (\$1986) based upon the most recent evidence. Applying this research with recent costs in California, Rowe et al. (1986) found per capita damage for a 1 pphm change in annual average ozone of about **\$.09**

(\$1986) per year. These estimates provide the basis for the "best" economic estimate for this damage category.

Evidence of ozone's effects on fabrics, paints and other materials is mixed, but in general suggests limited damage at or near ambient concentrations. Earlier estimates by Salmon, Salvin and NAS put fabrics/dye damages at \$220 to \$400 million/year (1986). Based upon the more recent evidence, upon the rate of reduction in the elastomer damage estimates, and assuming that mitigation of fabric and dyes has been about as effective as for elastomers, it is likely damage to fabrics and dyes are no more than **\$30-\$100** million-(\$1986) annually. Damage to artwork may also occur based upon work by Shaver (1982) and the typical long life of artwork, but no work has been completed to assess the possible value of such damages. Potential damage to fabrics and artwork could be incorporated in the estimates by adding (a somewhat speculative amount based upon reducing early estimates by 1/3 to 1/6) \$100 million/year in the "best" and "high" estimates only. This amounts to about **\$.04** per person per 1 pphm change in ozone.

4.3 OZONE AND **AGRICULTURAL** EFFECTS

Two major national studies provide the basis for ozone agricultural damage estimates: Adams et al. (1985, 1986) and Kopp et al. (1984). Both models examine losses tied to major field crops. These models, reviewed in detail in Krupnick et al. (1986), both use NCLAN crop loss data, similar farm data and ozone data. The models differ in mathematical specifications, degree of disaggregation, model assumptions and baseline time periods. Adams et al. use 1982-1983 and Kopp et al. use 1978.

The model predictions for a 10% decrease in the appropriate average ambient ozone measure are quite similar given the differences in their design. Adams et al. predict **\$.99** billion (updated to \$1986, including adjustment for Hay, using Aggregation II), and Kopp et al. \$1.18 billion (\$1986). While there are arguments for choosing one or the other estimates, the results are similar enough and the arguments not so compelling as to require a choice, but rather the use of the average of the two estimates is recommended as the foundation of the upper point estimate.

Arguments have been raised (**McGartland** 1986) that the Adams et al. and Kopp et al. estimates are overstated by ignoring the effects of government intervention through crop-subsidy programs for soybeans, wheat, cotton and corn. The **McGartland** arguments suggest that welfare benefits of ozone reduction should be reduced by as much as 50 percent. Reanalysis of the Kopp model (Kopp 1986) for the northeast found consideration of these effects reduced the overall welfare estimates by up to 50 percent. Unfortunately, these estimates are limited by the 1978 time period used for analysis, technical assumptions in the model, and recent changes in farm policy due to the Food Security Act **of** 1985 (Fraas 1986). Further, USDA projects indicate that by 1990 agricultural markets will return to a competitive market without substantial government intervention. As a result, it may be questionable to incorporate potentially temporary farm policies in the economic damage estimates to be used to address long run air pollution control policy.

The Adams et al. and Kopp et al. estimates cover only major field crops. Fruits, vegetables, grasses and other minor crops are omitted, which account for about 20 percent of agricultural production nationwide. Studies by Howitt et al. (1984) and Rowe and Chestnut (**1985b**, and Rowe et al. 1985) can be used to provide estimates for several of these crops based upon California conditions versus national conditions. Preliminarily, it appears the added benefits from fruits and other crops that can be incorporated through these studies will be less than 10 percent of the Adams et al. and Kopp et al. estimates. Other regional studies either do not address a significant variety of non-field crops, or do not use defensible **dose** response or economic methods to be relied upon to provide substantive additional useful information.

In summary, the average of the Kopp et al. and Adams et al. estimates, augmented with estimates for other crops based upon Howitt et al. and Rowe et al., serves as the suggested upper estimate. A central estimate of **.75** of the upper estimate could be used to incorporate uncertainties in the estimates and farm policy. A lower bound of **.5** of the upper estimate is suggested to fully incorporate potential farm policy impacts.

EPA is currently investigating alternative dose response functions using the NCLAN data to investigate the choice of functional form and specification of the ozone variable. If these results are available, they should be evaluated to determine whether they might significantly affect the economic analysis.

4.4 OZONE IMPACTS ON **ORNAMENTAL** VEGETATION

There is very limited generalized quantitative evidence regarding ornamentals. M&O identify Heintz et al. (1976) as suggesting losses to ornamental plants of \$172 million per year (increased to 1986 prices and population), which should be spatially allocated according to population. The Heintz et al. estimate is probably of low accuracy due to poor understanding of ozone effects on vegetation at the time. On the other hand, if the ornamental total damage estimate, as adjusted, is correct, dividing by 100 to get the value of a one percent change in ozone will likely understate damages as most vegetation research suggests thresholds below which little or no damage occurs.

4.5 OZONE, NITRATES AND FOREST EFFECTS

Surveys conducted in the United States have identified decline symptoms in numerous managed and unmanaged forest ecosystems exposed to elevated levels of ozone and nitrates (see Table 4-2). Ambient ozone and nitrogen deposition may be responsible for the injury or decline in some or all of the survey's referenced in Table 4-2. Because air pollutants represent a stress that is superimposed on complex interacting natural stresses to which managed and unmanaged forest ecosystems are exposed, evaluating air pollution involvement in forest damage is very complex and uncertain. Among the 15 forest declines listed by the EPA Science Advisory Review Board (1985b), air pollution has been listed as an "important factor" in only 4 cases: the declines of Jeffrey and ponderosa pine in the San Bernardino Mountains in California; the decline of eastern white pine in the eastern United States; the decline of softwood and hardwood species in Europe; and the decline of red spruce and balsam fir in the Appalachian Mountains. Air pollution may affect foliar injury and yields, and result in effects to the entire ecosystem integrity. Only limited effects,

Table 4-2
Summary Of Case Studies Of Forest **Decline**

Authors	Species	Location	Findings/Evaluation
1. Adams, et al. (1985)	Red Spruce, Fir	Virginia, West Virginia	<ul style="list-style-type: none"> ○ annual ring width declined post-1965 ○ growth rates 1930-1965 show correlation with drought events ○ no conclusions on cause for decline post-1965 presented
2. Bruck (1984)	Red Spruce, Fir	Southern Appalachians	<ul style="list-style-type: none"> ○ mortality and defoliation of both species increase with elevation. ○ no drought observed ○ metal toxicity to soil microorganisms suggested ○ basal area not reported, declining cannot be quantified ○ reported metals appear too low for toxicity, based on laboratory studies
3. Cook (1985)	Red Spruce	New York	<ul style="list-style-type: none"> ○ compared annual tree ring growth to climate factors for period 1750 to 1976 ○ concluded that climate may be a contributing, but not sole, cause of observed decline in ring width post-1967 ○ much of ring width variation pre-1968 explained by climate factors
4. Friedland, et al. (1984)	Red Spruce	Vermont, New York	<ul style="list-style-type: none"> ○ red spruce decline evident in 1980s ○ present hypothesis that winter damage, compounded by "over-stimulation" with nitrogen deposition may be a causitive factor ○ hypothesis requires further testing
5. Johnson, et al. (1981)	Pine	New Jersey	<ul style="list-style-type: none"> ○ 2/3 of trees show some growth decline post-1965 ○ no causative factors confirmed

Table 4-2 (cont.)

Authors	Species	Location	Findings/Evaluation
6. Johnson & Siccama (1983)	Red Spruce	Appalachians	<ul style="list-style-type: none"> o decreased growth post-1965 o authors concluded that decline is "stress-related" o several hypotheses presented; none proved
7. McClenahan & Dochinger (1985)	White Oak	Ohio	<ul style="list-style-type: none"> o tree growth during period 1900-1978 correlated with climate and air pollution factors o climate explains less of the growth variance post-1930 o authors suggest that air pollution may be contributing; but insufficient data presented for hypothesis testing
8. McLaughlin, et al. (1985)	Sugar Maple	Ontario	<ul style="list-style-type: none"> o decline in annual growth from 1956-1976 o drought, pathogens and caterpillar infestation suggested as contributor
9. Miller, et al. (1983) McBride (1985)	Ponderosa Pine, Jeffrey Pine, White Fir, etc.	San Bernardino National Forest, California	<ul style="list-style-type: none"> o decreased growth in sensitive pines o increased mortality in sensitive pines o injury increased along increasing air pollution gradient o paper demonstrates ozone injury is occurring at ambient concentrations
10. Puckett (1982)	Pine, others	New York	<ul style="list-style-type: none"> o relationship between tree growth and climate had changed from 1900 to 1973 o author suggests acid rain and/or air pollution as contributing factor, but no data on pollution used in the analysis
11. Raynal, et al. (1980)	Red Spruce	New York	<ul style="list-style-type: none"> o seedling mortality observed o reduced tree ring growth post-1965 o limited measurements, so hypotheses for decline and seedling mortality cannot be tested

Table 4-2 (cont.)

Authors	Species	Location	Findings/Evaluation
12. Reich & Amundson (1985)	Poplar, Eastern White Pine, Sugar Maple, Red Oak	Ithaca, N.Y.	<ul style="list-style-type: none"> o net photosynthesis decreased linearly with increasing ozone concentrations o compared to controls photosynthesis was depressed between 10 and 40%
13. Scott, et al. (1985)	Red Spruce, Fir, Birch	New York	<ul style="list-style-type: none"> o basal area decline in red spruce and balsam fir above 900 m from 1964 to 1982 o stand size not reported o no hypothesis for cause of decline presented
14. Sheffield, et al. (1985)	Southern Commercial Pines	Florida, Georgia, North Carolina, South Carolina, Virginia	<ul style="list-style-type: none"> o reported radial growth declines in yellow pines < 16" between 30-50% in Piedmont and Mountains of Southeast o authors offered no final explanation for observed decline
15. Siccama, et al. (1982)	Red Spruce	Vermont	<ul style="list-style-type: none"> o resampled red spruce plots on Camels Hump exhibit decline in basal area from 1965 to 1979 o variability not reported, so statistical significance cannot be assessed
16. Skelly, et al. (1983)	8 Eastern Tree Species	Blue Ridge Mountains, Virginia	<ul style="list-style-type: none"> o tulip poplar, black locust, table mtn. pine, green ash, and Virginia pine had increased growth in filtered chambers compared with ambient air
17. Stephenson & Adams (1984)	Red Spruce	Virginia	<ul style="list-style-type: none"> o no decline on Mt. Rodgers reported from 1954 to 1983 o basal area of larger trees increased
18. Vogelmann, et al. (1985)	Maple, Fir, Beech	Vermont	<ul style="list-style-type: none"> o declines in basal area, biomass and density reported for maple and beech o insects and fungal diseases eliminated as possible contributing factors o no definitive conclusions drawn on role of air pollution or drought

Table 4-2 (cont.)

Authors	Species	Location	Findings/Evaluation
19. Wang, et al. (1986)	Trembling Aspen	New York State	<ul style="list-style-type: none"> o measured significant $p < 0.05$ differences in growth between trembling aspen grown in ambient vs. filtered chambers o ozone was the major gaseous pollutant at the site
20. Westman (1985)	10 Chaparral Shrub Species	Santa Monica Mountains, California	<ul style="list-style-type: none"> o reproduced field foliar injury symptoms in ozone fumigation chambers o 20 types of foliar injury observed in field o unable to relate symptom intensity in field to presumed air pollution pattern

primarily yield, have been studied to a degree useful in benefits analysis at this time.

Ozone has been strongly implicated as the cause of pine decline in the first two cases. In these cases, the biological mechanism for ozone effects is known (EPA 1986b); geographical gradients and observed damage are correlated with gradients in ozone concentrations (Miller 1983, Skelly et al. 1983); and genetic variation in susceptibility and resistance to damage in the field is correlated with genetic variation and susceptibility in controlled experiments (Benoit et al. 1983 and McLaughlin et al. 1982).

Evidence for ozone/nitrate involvement in the balsam fir and red spruce decline has been presented by numerous investigators as well (Table 4-2). However, the evidence for air pollution involvement in the decline is less certain and rests principally on the fact that the amount of damage is greater at higher elevations than at lower elevations. This is consistent with altitudinal gradients in concentrations of **both** ozone and nitrate, the time of exposure to nutrient rich cloud water and fog, and the accumulation of lead in forest tree tissue and soils (indicating increased levels of regional air pollution)..

Ozone and nitrogen deposition have been linked to forest decline through four hypotheses:

1. Ozone exposure alters enzyme function and disrupts cell membranes causing a reduction in carbon fixation.
2. Ozone, combined with acid deposition or fog, stimulates nutrient leaching and reduced biomass (Prinz et al. 1982, Chevone and Yang 1985).
3. Nitrogen, deposited in excess, causes increased nitrogen availability and uptake, reallocation of carbon, and the alteration of foliar biochemistry resulting in increased susceptibility to drought stress. Research by Shafer (1984) and Heier **and Bruck** (1984) indicates that **simulated acid** rain treatments consisting of sulfuric and nitric acid had a detrimental effect on the incidence and vigor of ectomycorrhizal

short roots of loblolly pine. In addition, preliminary experiments indicate that the nitrogen fraction of the simulated acid rain solution is the predominant causative agent for the deleterious effect on both incidence and vigor of these short roots.

4. Nitrogen, deposited in excess, prolongs the growing season, delaying the cold hardening process, reducing the frost hardiness of forest trees. (Kramer and Kozlowski 1979, EPA 1985a.) Damage symptoms of this type have been observed in high elevation spruce forests in Vermont and North Carolina.

Turning to effects, Peterson and Sueker (1987) show there is considerable uncertainty across forest scientists concerning the characterization of ozone forest exposure-response functions and the effective dose. The EPA (1986b), using an analysis based on 18 tree species, listed the following concentrations and exposure times that are likely to cause foliar injury:

- a. 0.20 ppm to 0.51 ppm for 1 hour
- b. 0.10 ppm to **0.25 ppm** for 2 hours
- c. 0.01 ppm to 0.17 ppin for 4 hours.

Fewer studies have attempted to link ozone with physiological or growth responses. In an examination of eastern white pine from two ozone sensitivity classes growing in Virginia, the most sensitive classes exhibited **20-50%** less growth than trees from the least sensitive class (Benoit et al. 1983). A concomitant decrease in height growth and foliar biomass has been reported for saplings grown in unfiltered air compared to charcoal-filtered air (Skelly et al. 1983).

More recently, Wang et al. (1986) measured increased visible foliar injury, acceleration of leaf senescence, and reduction in growth of trembling aspen exposed to ambient air. For two of the three years of the study saplings exposed to ambient air had much less shoot growth (**12-14%**) than those grown in filtered air. Reich and Amundson (1985) reported that net photosynthesis decreased linearly with increasing ozone concentrations in sugar maple, easten

white pine, hybrid poplar, and northern red oak. No visible symptoms were apparent .

The most complete study of ozone effects at the forest ecosystem level was done in the San Bernardino National Forest, California, east of Los Angeles, an area typified by elevated ozone (3-4 pphm to **10-12** pphm) (Miller et al. 1977). This area served as the site of an intensive field and controlled environment research program from 1973 to 1978 (Miller et al. 1977). The study concluded that ozone induced foliar injury and premature leaf fall, leading to decreased photosynthetic capacity, decreased radial growth and reduced-nutrient retention in foliage and produced an 84 percent reduction in commercial yield of Jeffrey and ponderosa pine.

Limited summary estimates of forest yield decline have been produced (Wilhour 1986, Peterson and Sueker 1987, Miller et al. **1977**), but these may be useful in estimating marketable yield declines. **Economic studies** are even more limited. **Callaway** et al. (1986) examined the value of changes in forest productivity in the northeastern and southeastern U.S based upon the Timber Assessment Market Models (TAMM) and Timber Resources Assessment System (TRAS) developed by the U.S. Forest Service. The model examines losses for growth changes in softwoods and hardwoods for the two separate regions. The total present value of losses for a 10 percent change in yield for both softwoods and hardwoods for the entire region is estimated at \$3.4 billion. The corresponding annualized value is approximately \$42 million. Using the Wilhour estimates of the percent change in yield loss per 1 pphm ozone results in annualized best estimate of \$63.5 million/pphm ozone. Of interest is that the stream of damages per year decreases as offsetting production and harvesting measures are undertaken in the affected region and other regions of the country. Damages are roughly split **30%/70%** to the northeast and southeast.

Botkin and Devine (1986) have also developed a model for economic losses for changes in eastern (northeastern, southeastern and **midwest**) hardwoods and softwoods and applied it to potential yield losses from ozone. The model calculates county by county yield losses related to ozone changes using lowest, best and upper bound estimates of the percent yield change per pphm change in ozone of **.4**, 1.8, and 2.9 for softwoods and **.1**, 1.9 and 2.9 for hardwoods and

calculates present value of **yield changes** in the year trees **are** harvested using a constant increase of 2.5 percent per year in real prices. Pulpwood values are omitted and **may** represent an omission of as much as 25 percent of the estimated results. The final numbers for this model have not yet been received.

To estimate values associated with yield losses for ozone, the **Callaway** et al. and Botkin and Devine analyses provide potential benefit estimates for the Northeast and Southeast, but require further evaluation. For the west, the Miller et al. (1977) yield losses could be applied for all western forests experiencing ozone **above** selected thresholds. An inventory of ozone concentrations in commercial timberlands is found in Pinkerton and Lefohn (1986).

Visual aesthetic forest injury may also cause reduced welfare. Only one economic study has been completed to date. **Crocker** (1985) considered aesthetic impacts in the San Bernardino National Forests with a small contingent valuation study. Work on a related project is also underway at the University of Colorado, but will not be available till later in 1987. The Colorado work suggests potential substantive concerns with the **Crocker** and Vaux analysis, but the overall direction of bias, if any, is not clear. Further, the Colorado work suggests that aesthetic impacts are often transitory unless recurrent high peak ozone levels occur. In part, this may **be because** the most sensitive individuals are replaced in high ozone areas, or because individuals recover when not continually exposed **to** high peak levels. At best, the **Crocker** estimates could be applied to forests with ambient ozone levels similar to the San Bernardino NF, of which there are few. A procedure for applying these numbers can be found in Rowe et al. (1986).

Forest yield and **some** aesthetic injury can be quantified and valued with appropriate assumptions. Further review is required to determine whether the quantification is best listed as accepted or speculative and to determine appropriate lower, best and upper bound estimates. Other impacts to the forest ecosystem, including impacts on other vegetation, **animals**, water retention, fire susceptibility, etc. all may occur, but without further physical and economic evidence they must remain unquantified at this time.

5.0 INDIRECT EFFECTS THROUGH ACID DEPOSITION

Very few acid deposition physical effects and economic valuations results are available, resulting in **most** potential benefits remaining unquantified or listed as speculative.

5.1 ACID DEPOSITION AND AGRICULTURAL EFFECTS

Initial analyses by **Crocker** and Regens (1985) had suggested up to \$1 billion of U.S. agricultural **damages** due to acid deposition. However, recent NAPAP chamber study results incorporated into a mathematical programming model of U.S. agriculture suggest significantly different results (Adams et al. 1986). Adams et al. examine crop loss, reduced fertilizer and increased liming costs due to change in acid deposition. They conclude with a best estimate of crop damages due to a 50 percent increase in deposition equal to \$142 million (**\$1980**), with an upper bound of \$316 million under the assumption that all **soybeans** react equally to the most sensitive variety identified in NAPAP testing. However, when also considering increased fertilization (a benefit) and additional liming requirements (a cost), a 50% increase in acid deposition results in a point estimate of positive benefits of \$48 million, largely due to fertilization. Adams et al. indicate that the benefits of fertilization, which are for all crops, are likely to **be** overstated as the deposition is not uniform or only in the growing season when needed. Yield losses **may** also be understated due to the limited number of crops for which acceptable dose response information exists. These biases are likely to cancel **much** or all of the \$50 million in benefits in **the** best estimate, but substantial uncertainty exists. As a result, net agricultural damages due to a 50 percent increase in acid deposition are recommended to be categorized as speculative with a lower bound of \$50 million in benefits (**+\$1** million per 1% change), a best estimate of zero damages and an upper bound of \$200 million in damages (-\$4 million per 1% change).

One other recent study by Forster (1984) examined economic losses due to acid deposition in eastern Canada of \$100 million using an early corn damage function and considering fertilization and liming costs. This could be significant for U.S. control of **NO_x**. However, subsequent replications of the study generating the crop loss function and NAPAP experiments both show little corn yield effect

due to acid deposition. Therefore, the results of this study are best treated as being incorrect and not used.

5.2 ACID DEPOSITION AND MATERIAL DAMAGE

To impute values for the NO_x contribution to acid deposition impacts upon materials requires knowledge or assumptions concerning the share of local and non-local deposition due to NO_x emissions, the relative importance of sulfate versus nitrates in the material damage process, the physical-damage incurred and economic measures of damage incurred. We only address the physical and economic damage estimates presuming the previous information can be provided. Moreover, the estimates provided must be categorized as "speculative" based upon controversy surrounding the preliminary research upon which they are based.

Acid deposition is believed to be responsible for damage to paints, galvanized steel, mortar and stonework, outdoor artwork and other materials. An analysis of the potential physical and economic impact of these damages in major regions of the east was conducted by Horst et al. (1986). However, critical reviews of this work have found the underlying damage functions utilized for paints and mortar, which were the two largest damage categories, were probably unreliable. For example, the paint function was based upon erosion rather than what is perceived, but not known in fact, as the more operational measure of peeling and cracking. If acid deposition results in increased peeling and cracking at the same or faster rate than erosion, and peeling and cracking are the variables of interest to individuals, then the Horst et. al. paint damages could be understated, although the converse may also be true. The galvanized steel estimates were found to be acceptable, but of a relatively small magnitude.

The Horst et al. analysis was also criticized because values were calculated based upon optimal maintenance practice and costs, rather than perceived physical damages, actual behavior and actual costs or values, and because they omitted aesthetic damage. Overall, the direction and size of biases due to these considerations is not clear.

Additional work ongoing or **planned** at **the California Air Resources Board** and within EPA will shed new light on the limitations in the Horst et al. study and input assumptions, but not for several more years.

Physical and economic damages from acid deposition appear to occur. Selection of physical or economic damage estimates for materials other than zinc **must be** recognized as speculative based upon preliminary research. A conservative approach would **be** to use the galvanized steel estimates in Horst et al. for the quantified lower and **best estimates**. This amounts to about \$1.6 million per 1% reduction in acid deposition. While we are uncomfortable with and do not mean to attribute much accuracy to the Horst et al. estimates for other materials, and feel the results may be upwardly biased, use of the Horst et al. total lower estimate as a speculative upper bound may represent an improvement in information over using only galvanized steel economic damage estimates. This results in approximately \$7.2 million per one percent reduction in acid deposition.

5.3 OTHER ACID DEPOSITIONS EFFECTS

No evidence was identified on acid deposition impacts on ornamental vegetation. However, the Adams et al. (1986) analysis for acid deposition to agricultural crops suggests the net economic value of impacts on vegetation is near zero.

Acid deposition may also affect forest and human health but reliable quantitative studies for use in benefits analyses were not identified. A recent review of health effects and acid aerosols by CASAC may provide information for future analyses. Similarly, acid deposition **may** offset aquatic ecosystems, but evidence has yet to be obtained to combine the biologic **impacts**, fertilization and liming aspects into one economic measure for a balanced perspective in this report. As a result, the other effects are **best** left unquantified.

6.0 SUMMARY AND INPUT NEEDS

6.1 SUMMARY OF PHYSICAL AND ECONOMIC STUDY SELECTION

Table 6-1 summarizes the studies recommended for use in a quantitative physical effects and economic benefit assessment of NO_x control based upon currently available literature. The needed inputs to the assessment are discussed in Section 6.2. The suggested application of the studies for calculation of best, upper, and lower bounds are discussed in Chapters 3-5. Available research in some effects categories requires further evaluation prior to implementation due to the limited time to obtain documents and conduct this review. Note also, that the speculative quantified effects categories are generally based upon limited research often of uncertain validity. The available results may underestimate or overestimate these speculative benefits, but due to the speculative nature of the benefit category estimates, we recommend that they be included only as upper bound estimates.

6.2 INPUT NEEDS

The implementation of the NO_x benefits analysis will require two types of inputs: sociodemographic and meteorological. The first includes population characteristics such as baseline MRADS, population by region, prices, etc. These data can be readily obtained by the economic analyst and are not further discussed (see Krupnick et al. 1986a for much of these data).

Emissions and meteorological variables are needed to evaluate physical and economic functions. Optimally, separate estimates of these assumptions, variables or relationships would be made available by regional and urban/rural locations. Below, the input needs are identified.

Ideally a benefits analysis would be based upon estimates of changes in the exact variables in the damage functions. However, this may not be possible in the time and resources allocated to this analysis. Some needs may be reduced by using approximations based upon other physical and economic studies. For example, the studies by Chestnut et al. (1987) and Krupnick et al. (1986)

Table 6-1
Summary Of Study Selection'

Effect Category	Physical Effects Study Recommended	Economic Valuation Studies Recommended
I. <u>NO₂ Effects</u>		
Human Health		
- Eye Irritation	Schwartz et al. (1987) adults only	Krupnick et al. (1986a)
- Others	Possible further evaluation of Mullahy (1987) for MRADs and Lebowitz (1985) for asthmatics	
Visibility		
- Haze Use Values (values per household)	Tri jonis (1987)	Chestnut et al. (1986)
- Plume Use Values in West (per household)	?	Chestnut and Rowe (1983)
Materials	---	Barrett and Waddell (1973)
Agriculture, Vegetation and Forests	No significant effect	
II. <u>Nitrate Effects</u> (using nitrate or TSP or PM studies)		
Human Health		
- Mor tality	Evans et al. (1984) Schwartz et al. (1986)	Violette et al. (1986)
- Emerg. Room Visits	Samet et al. (1981)	Chestnut et al. (1987)
- Rest. Act. Days	Ostro (1987)	See Text
Visibility		
- Haze Use Values (Values per household)	Trijonis (1987)	Chestnut et al. (1986)
Materials (per household)	---	Manual et al. (1982)
Agriculture, Vegetation and Forests	No significant effect	

Table 6-1 (Continued)
Summary Of Study Selection'

Effect Category	Physical Effects Study Recommended	Economic Valuation Studies Recommended
III. <u>Ozone Effects</u>		
Health		
- Resp. Rest. Act. Days (for adults only in excess of asthma attacks for asthmatics)	Portney & Mullahy (1986)	Krupnick et al. (1986), Dickie et al. (1986)
- Eye irritation and cough symptom days in excess of RRAD (for adults only)	Schwartz et al. (1987)	Loehman et al. (1979), Tolley et al. (1986), Krupnick et al. (1986a)
- Asthma attacks (for asthmatics)	Whittemore and Korn (1980), Holguin (1985)	Krupnick et al. (1986a) Rowe & Chestnut (1985)
- Symptoms Aggregate	Krupnick et al. (1986b)	
Materials		
		McCarthy (1983) Barrett and Waddell (1973)
Agriculture		
		Kopp et al. (1984, 1986), Adams et al. (1985), Howitt et al. (1984), Rowe & Chestnut (1985)
Ornaments		
		Heintz (1976)
Forests ²		
- Yield Effects	Wilhour (1986), Miller (1977)	Callaway et al. (1986) Botkin & Devine (1986)
- Aesthetics	Miller (1977), Crocker (1985)	
IV. <u>Acid Deposition Effects</u>		
Materials		
-Galvanized Steel		Horst et al. (1986)

Table 6-1 (Concluded)
Summary Of Study Selection'

Ef fect Category	Physical Effects Study Recommended	Economic Valuation Studies Recommended
V. <u>Speculative Quantified Effects</u> ³		
NO₂ and nitrate non- use visibility values at national parks		
- Haze	Trijonis (1987)	Chestnut and Rowe (1983)
- Plumes	?	Chestnut and Rowe (1983)
Ozone chronic illness (for adults only)	Higgins et al. (1982) Mullahy (1987)	See Text
Acid deposition materials effects other than galvanized steel		Horst et al. (1986)
Acid depositions agriculture effects		Adams et al. (1986)

Notes:

1. See text for application discussion, especially with regard to best, lower and upper bound determinations and potentially overlapping categories.
2. Insufficient time has prevented thorough **review and** selection of all studies. Studies identified are studies of probable usefulness.
3. Speculative quantified effects are generally only suggested as upper bounds, or as otherwise indicated in the text.

provide results that **might be used to provide part or all of the needs** in 5, 10, and 11 below, although the results would be aggregate, or potentially biased toward conditions in specific regions. Other approximations may also be available **and** should **be** examined in the next phase of the effort. Finally, the ozone health effects could **be** estimated with modifications to OZ-ONE model developed by EPA as part of the ozone RIA process. While the model differs from some of the recommendations made here, most differences are **not** so significant as to radically alter the results.

Input needs are identified below in terms of the input, possible alternatives and the analysis component using the input. Suggestions for assumptions or relationships are not made at this time.

1. Current baseline tons of **NO_x** from emission inventories. (All analyses)
2. A relationship between percent change in **NO_x** emissions and a 1% and 10% change in ambient **NO₂** and nitrates. (**NO₂** and nitrate visibility haze analysis and **NO₂** materials effects)
3. Plume days at southwest national parks and integral vistas, and average plume **NO_x** tons. (**NO_x** visibility plume analysis)
4. Relationship between **NO_x** tons and TSP or FP or PM10 annual average and baseline **TSP/FP/PM10**. Alternatively, the functions could be converted to elasticities at alternative values requiring the percent change in **NO_x** for a percent change in the annual average for TSP or FP or **PM10**. One would still require baseline **TSP/FP/PM10** by region. (Nitrate health effects)
5. Change in **NO_x** to second high 24 hour TSP in $\mu\text{g}/\text{m}^3$. Alternatively, convert functions to elasticities at alternative baseline values requiring percent change in **NO_x** to percent change in second high hour (or a relationship for percent change in annual average to percent change in second high hour and use #4 above). (Nitrate materials effects)

6. Percent of total acid deposition associated with **NO_x** emissions, particularly in the **midwest** and east. (Acid deposition material effects)
7. Percent change in **NO_x** emissions that would relate to a 50 percent increase in acid deposition east of the Mississippi. (Acid deposition agricultural effects)
8. Percent change in **NO_x** resulting in a 10 percent change in ozone hourly average for 7 hour period during growing season. (Ozone agricultural)
9. Change in **NO_x** leading to **.01** ppm change in annual average ozone. Alternatively, convert current estimates to elasticities requiring percent change in **NO_x** to 1 percent change in annual average ozone. (Ozone material effects)
10. Change (or percent change) in **NO_x** leading to change (or percent change) in 2-week average daily high hour ozone in ppm. (Ozone **health-RRADs**)
11. Change (or percent change) in **NO_x** leading to change (or percent change) in daily 24-hour maximum ozone in ppm, and daily 12-hour maximum ozone reading. (Ozone health - symptoms and asthma)
12. Change in ozone leading to a 1 ppm change in annual average of 12 hour daytime ozone. (Ozone forest effects)