

PAPER 9<sup>1</sup>**OZONE HEALTH EFFECTS****1. INTRODUCTION**

Ozone is a principal component of photochemical air pollution endogenous to numerous metropolitan areas. It is primarily formed by the oxidation of  $\text{NO}_x$  in the presence of sunlight and reactive organic compounds. Ozone is a highly active oxidizing agent capable of causing injury to the lung (Mustafa and Tierney 1978). Lung injury may take the form of irritant effects on the respiratory tract that impair pulmonary function and result in subjective symptoms of respiratory discomfort. These symptoms include, but are not limited to, cough and shortness of breath, and they can limit exercise performance. Some of the toxicological properties of ozone were first investigated over 100 years ago by Schonbein (1851), who found it to be a lethal gas when inhaled by mice in sufficient quantities. When Schonbein accidentally inhaled ozone gas, it produced cough and pain in the chest; this is still a common finding. The effects of ozone observed in humans have been primarily limited to alterations in respiratory function, and a range of respiratory physiological parameters have been measured as a function of ozone exposure in adults and children. These effects have been observed under widely varying (clinical experimental and environmental settings) conditions (Adams 1987).

The vast data base on the effects of ozone on humans and animals provides abundant evidence of the adverse acute effects of ozone. Laboratory-based human and animal studies have suggested effects on pulmonary host defenses and the immune system, for example, decreased particle clearance rate in rats. Pinkerton et al. (1989) and Amoruso et al. (1989) observed a modification of alveolar macrophages with respect to a decreased production of superoxide radicals in mice and rats. Orlando et al. (1988) observed possible adverse effects on the immune system with respect to a decrease in the T-cell mitogen-induced blastogenesis of circulating T-cells in humans. Burleson (1988) found a decrease in pulmonary natural killer cell activity in rats. Short-term effects on lung structure have also been observed in animal models such as shortened or less dense cilia in the trachea and bronchi of monkeys (Castleman et al. 1977); damaged cilia in rats (Schwartz et al.

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<sup>1</sup>Based largely on a working paper by C. Easterly.

1976), and elevated collagen synthesis rates and histologically discernible fibrosis in rat lung tissue (Last et al. 1979).

In addition to acute effects, a wide range of subchronic and chronic effects have been identified in laboratory-based animal studies. Because chronic exposures are some cumulative function of a series of acute exposures, a linkage exists between acute and chronic exposures, but the mechanisms, at present, are not fully defined. Some of the effects seen in animals subjected to subchronic and chronic exposures to ozone are: an increase in respiratory bronchioles in monkeys and a thickening of their walls (Tyler et al. 1988; Moffatt et al. 1987); a thickening of the alveolar septa (Moffatt et al. 1987); increased numbers of inflammatory cells and fibroblasts in the interstitia of monkeys' (Tyler et al. 1988, Moffatt et al. 1987) and rats' (Pickrell et al. 1987) lungs; increases in the number of epithelial cells (Boorman et al. 1980); altered morphology and cell type shifts in rats (Huang et al. 1988; Grose et al. 1989); and an increase in amorphous extracellular matrix in rats (Huang et al. 1988; Grose et al. 1989). Finally, Zelikoff et al. (1991) have demonstrated that rabbits exposed to ozone exhibit compromised macrophage (MO) responses in lung tissue. Because the MO provides important functions in tumor surveillance, susceptibility to pulmonary cancer may be effected by ozone exposure.

The results of studies in animals and the range of chronic effects observed suggests that there is significant potential for chronic effects in humans. In addition, the types of morphological changes caused by ozone in animals are also observed in the lungs of cigarette smokers. These changes are generally interpreted as representing early stages of chronic lung disease in smokers. Several epidemiological studies tend to support a concern about the potential for chronic effects in humans (Detels et al. 1987; Knudson et al. 1983; and Kilburn et al. 1985). While there are acknowledged imperfections in these studies, they suggest an increased rate of lung function decline with ozone exposure that has also been observed in animal studies. Notwithstanding, there is no definitive evidence from epidemiological studies that ambient ozone exposures cause chronic effects in humans. Consequently, there will be considerable uncertainty in any estimates of externalities associated with the chronic effects of ozone on health.

NAPAP Report 22 (EPA 1990) identified a set of observations that are considered to provide clear and consistent evidence from human clinical, epidemiological and field studies regarding acute effects of ozone on human pulmonary function:

1. Inhalation of ozone causes concentration-dependent mean decrements in lung volumes and flow rates during forced expiratory maneuvers.

2. The mean decrements increase with increases in minute ventilation as a function of increasing exercise.
3. A wide range of reproducible individual responsiveness exists among healthy young adults, with the upper end of the distribution constituting the most responsive group of individuals.
4. There are small differences in average ozone responsiveness (based on lung function test changes) among different population groups. Healthy young adults and children appear to be slightly more responsive than older adults. Asthmatics have greater increases in airway resistance and smokers have smaller changes in spirometry after ozone exposure than healthy subjects.
5. Repetitive, daily exposures at levels initially producing a functional response may lead to an enhanced response on the second day, but responsiveness diminishes on subsequent consecutive days of exposure. This "attenuation" phenomenon is transient, disappearing about 1 week after termination of continued daily exposure. Responsiveness may have a seasonal pattern of variation; that is, there may be less responsiveness in the fall after repeated summertime high-ozone exposures. This seasonal variation may complicate or compromise the ability of epidemiological studies to detect effects.
6. Multihour or multiday exposures more likely lead to pulmonary response than 1-hour peak exposures. A pattern of simultaneous or sequential exposure to other pollutants such as acidic aerosols is, likewise, more likely to lead to pulmonary response.
7. In adults, ozone exposure causes respiratory symptoms such as cough and chest discomfort.

## 2. CHOICE OF MODEL

Risk estimates for a number of urban areas have been performed using existing or projected levels of ozone (e.g., Hayes et al. 1987; Whitfield 1988; Krupnick and Kopp 1988; Hayes et al. 1989; and Hayes et al. 1990). These estimates were developed for both pulmonary function and lower respiratory tract symptoms. In the willingness-to-pay paradigm that is used in our study, the most fully investigated health endpoint, pulmonary function, cannot be used because pulmonary decrements themselves have not been evaluated. Pulmonary

decrements have not been linked to specific symptoms of ill health by the medical community and without a symptom, it is difficult to obtain a measure of the willingness to pay to avoid the pulmonary decrement. Because the focus of the present study is on willingness to pay, and because Krupnick and Kopp (1988) have evaluated eight symptomatic endpoints for use in that particular economic evaluation, we adopt their study for our use. Krupnick and Kopp (1988) draw on some of the same health studies as Hayes et al. (1987, 1989, 1990).

Krupnick and Kopp (1988) identified eight symptoms from five studies and reduced the quantitative information to mathematical models, usually probit functions (Schwartz, Hasselblad, and Pitcher 1989; McDonnell et al. 1983; Krupnick et al. 1987; Portney and Mullahy 1986; Holguin et al. 1985). One study (McDonnell et al. 1983), was reanalyzed by Krupnick and Kopp (1988) from the raw data in order to make the results more useful for an economic analysis. Krupnick and Kopp (1988) made several assumptions in order to fit the epidemiologic or clinical studies to the needs of an economic analysis. For the symptom endpoints, it was necessary to convert symptom incidence to symptom-day. Because the symptoms are associated with elevated breathing rates, it was necessary to identify the percentage of the time an average person spends engaged in heavy exercise, adjusted for indoor and outdoor activities. Baselines were also identified for several symptoms. Because Krupnick and Kopp (1988) investigated issues related to regulatory statutes, their assessment was designed around two key types of ozone measurements. These key measures are the maximum 1-hour ozone concentrations, and the average for a 2-week period of daily 1-hour maximum ozone concentrations. These two key measurements fit well into the present study because measurements can be readily identified for the baseline (present) conditions at the two sites. Further, atmospheric transport models can be used to estimate the increased concentrations of ozone due to emissions from coal-fired power plants.

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The particular symptoms chosen for our analysis, based on the earlier development of Krupnick and Kopp (1988), are:

*Epidemiologically-Based Endpoints*

1. Total Respiratory Restricted Activity Days (TRRAD), used by Portney and Mullahy (1986). This measure is based on symptoms identified by adults over a 2-week recall period. Their health effects model was based on an average for a 2-week period of daily 1-hour maximum concentrations of ozone, as recorded within a 20-mile radius.
2. Any-Symptom Day (Krupnick, Harrington, and Ostro 1987). This study resulted in a variety of response functions for a variable that took the value of one if any of 19 symptoms or conditions were present on a given day and zero otherwise. Except for eye irritation and headache, these symptoms and conditions were all respiratory related. The response function is based on adults and daily 1-hour maximum ozone concentrations. In the accounting framework, the total number of Any-Symptom Days is reduced to the extent that there is no double counting of separately calculated symptoms.
3. Asthma-Attack Day (Holguin et al. 1985). Based on a 12-hour period of observations on identified asthmatics, and related to total oxidants, this study was modeled by Krupnick and Kopp (1988). They used two time periods in order to do statistical fits with daily 1-hour maximum ozone concentrations.
4. Eye-Irritation Day (Schwartz, Hasselblad, and Pitcher 1989).
5. Days of Coughing (Schwartz, Hasselblad and Pitcher 1989). This study investigated the relationship between total oxidants, coughing, eye irritation and chest tightness. Only the first two symptoms were found to be significantly associated with oxidant exposure to members of the total population.

*Clinical Study-Based*

6. Cough Incidence (McDonnell et al. 1983).
7. Shortness of Breath (McDonnell et al. 1983).
8. Pain upon Deep Inspiration (McDonnell et al. 1983). This study found the difference in symptom scores taken before and after 2-hour

ozone exposures in a clinical setting. Krupnick and Kopp (1988) obtained the raw data from this study and performed a re-analysis, and then developed a procedure for adapting results from 2-hour incidences to a symptom-day measure.

Several steps are required to apply the Krupnick and Kopp (1988) results to estimate the effects of ozone on health at a reference site:

1. The concentration-response functions from Krupnick and Kopp (1988) were coded into a simple Fortran program using the middle value coefficients plus the upper and lower 75% confidence limits.
2. For the months of May, June, July, August, and September, during which ozone production is significant at the Southeast site, daily 1-hour maxima were transcribed from the EPA's Aerometric Information Retrieval System (AIRS) data base, modified by a factor of 0.773 as described in Paper 3 of this document, and used as data input to which incremental ozone values were added. These increases in ozone concentrations were obtained from the modeling described in Paper 3 of this document using an estimate of median ozone conditions. The baseline and its increment were used as input to the health effects algorithms.
3. In the execution of the computer code, the AIRS data (the baseline) when combined with the additional amount attributed to the reference plant were checked for values below 0.08 ppm. This level was considered to be a practical threshold for the present study.
4. The populations used for this evaluation were divided into local (within 50 mile radius of the power plant) and regional (beyond 50 miles) populations.

Results of the computations for the 5-month ozone season were summed over the appropriate populations. This calculation provided an estimate of the numbers of cases.

The following dose-response functions provide relevant calculational information:

Dose Response Functions:  
OZONE

Incidences of pain upon deep inspiration (PDI): Based on McDonnell et al. (1989)

$$\Delta C = \{ [1/(1+\exp(-\gamma-\beta\omega X_1))] - [1/(1+\exp(-\gamma-\beta\omega X_0))] \} f\theta(\text{mpop})$$

where

- $\Delta C$  = change in number of PDI incidences for 2-hour period t
- $X_0$  = daily maximum hourly ozone concentration, baseline in reference environment
- $X_1$  = daily maximum hourly ozone concentration including reference plant
- $\gamma$  = -0.799
- $\beta$  = 3.456, 6.946, 10.436
- mpop = entire population
- $\theta$  = percent of a 2-hour period the population is exercising
- f = the incidence-day factor
- $\omega$  = the scaling factor for 2-hour period t

Dose Response Functions (continued)  
OZONE

Any symptom or condition (ARD): Based on Krupnick, Harrington and Ostro (1987)

$$\Delta\text{ARD} = \beta^* (X_1 - X_0) (\text{apop})$$

where

$\Delta\text{ARD}$	=	change in the number of days of "any" symptoms/conditions
$\beta^*$	=	marginal change in the stationary probability of experiencing any symptom/condition
	=	$p_0(1-p_1)\beta[p_1+(1-p_0)]/(1-p_1+p_0)^2$ , where $p_0$ is the conditional probability of illness on day $t$ given wellness on day $t-1$ , $p_1$ is the conditional probability of illness on day $t$ given illness on day $t-1$ , and $\beta$ is the ozone coefficient from the logit model regression.
	=	0.13, 0.20, 0.27
$X_0$	=	daily maximum ozone concentration, baseline in reference environment
$X_1$	=	daily maximum ozone concentration including reference plant
apop	=	adult population

Dose Response Functions (continued)  
OZONE

Total respiratory-related restricted activity days (TRRADs): Based on Portney and Mullahy (1986),

$$\Delta\text{TRRAD} = \text{TRRAD} [\exp [\beta(X_1 - X_0)] - 1] (\text{apop})$$

where

$\Delta\text{TRRAD}$	=	change in number of respiratory-related restricted activity days for the 2-week period
$\text{TRRAD}$	=	baseline per capita TRRADs for a 2-week period
$X_0$	=	average daily 1-hour maximums of ozone concentrations for each 2-week period, baseline in reference environment
$X_1$	=	average daily 1-hour maximums of ozone concentrations for each 2-week period including reference plant
$\text{apop}$	=	adult population
$\beta$	=	2.63, 7.99, 13.34

Dose Response Functions (continued)  
OZONE

Asthma attacks: Based on Holguin et al. (1985)

$$\Delta a = [m/(1+m) - p] (\text{apop})$$

where

$$m = [p/(1-p)] \exp (\beta\omega X_1 - \beta\omega X_0)$$

and

$\Delta a$  = change in number of asthma attacks for the 7AM-7PM or 7PM-7AM period

$p$  = baseline number of attacks per asthmatic for the day

$X_0$  = maximum 1-hour ozone concentration for 7AM-7PM, baseline in reference environment

$X_1$  = maximum 1-hour ozone concentration for 7AM-7PM including reference plant

$\text{apop}$  = asthmatic population

$\omega$  = scaling factors for half-day periods

$\beta$  = 3.58, 6.20, 8.82

Dose Response Functions (continued)  
OZONE

Incidences of coughing: Based on McDonnell et al. (1983),

$$\Delta C = \{[1/(1+\exp(-\gamma-\beta\omega X_1))] - [1/(1+\exp(-\gamma-\beta\omega X_0))]\} f\theta(\text{mpop})$$

where

- $\Delta C$  = change in number of coughing incidences in two-hour period
- $X_0$  = daily maximum hourly ozone concentration, baseline in reference environment
- $X_1$  = daily maximum hourly ozone concentration including reference plant
- $\gamma$  = -1.742
- $\beta$  = 10.961, 14.1, 17.239
- mpop = entire population
- $\theta$  = percent of a two-hour period the population is exercising
- f = the incidence-day factor
- $\omega$  = the scaling factor for two-hour period t

Dose Response Functions (continued)  
OZONE

Incidences of shortness of breath: Based on McDonnell et al. (1989)

$$\Delta C = \{ [1/(1+\exp(-\gamma-\beta\omega X_1))] - [1/(1+\exp(-\gamma-\beta\omega X_0))] \} f\theta(\text{mpop})$$

where

$\Delta C$  = change in number of shortness of breath incidences for two-hour period

$X_0$  = daily maximum hourly ozone concentration, baseline in reference environment

$X_1$  = daily maximum hourly ozone concentration including reference plant

$\gamma$  = -0.076

$\beta$  = 4.938, 7.265, 9.562

mpop = entire population

$\theta$  = percent of a two-hour period the population is exercising

f = the incidence-day factor

$\omega$  = the scaling factor for two-hour period t

**Table 1. Health effects estimated to occur from ozone exposure (in thousands) for the maximum extent of the ozone plume (about 215 km)**

<b>Southeast Reference site</b>	<b>Lower</b>	<b>Mid</b>	<b>Upper</b>
1. Total restricted activity days	4.2	13	24
2. Any-symptom day	11	27	43
3. Asthma-attack day	0.85	1.5	2.2
4. Eye-irritation day	30	36	43
5. Cough day	9.5	15	20
6. Cough	60	84	109
7. Shortness of breath	34	49	62
8. Pain upon deep inspiration	22	47	73

**Table 2. Health effects estimated to occur from ozone exposure within 50 miles (80 km) of the plant (in thousands)**

<b>Southeast Reference site</b>	<b>Lower</b>	<b>Mid</b>	<b>Upper</b>
1. Total restricted activity days	3.6	11	20
2. Any-symptom day	9.1	23	37
3. Asthma-attack day	0.73	1.3	1.9
4. Eye-irritation day	26	31	36
5. Cough day	8.1	12	17
6. Cough	51	72	93
7. Shortness of breath	29	42	53
8. Pain upon deep inspiration	19	40	62

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**NUSAP: OZONE HEALTH EFFECTS<sup>2</sup>**


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**Numerical Information:** Symptoms (Any-symptom day, Asthma-attack day, Cough-day, Cough incidence, Shortness of breath, Respiratory restricted activity days, Eye irritation-day, and Pain upon deep inspiration)

**Units**

*Measurement Units:* symptom days/incidences

*Statistical Unit:* Lower Bound, Mean, Upper Bound

**Spread**

*Confidence Level:* 75%

*Upper Bound:* x 2 to 4

*Lower Bound:* ÷ 2 to 4

Eight endpoints are examined in this work; typically the lower to the high estimates are within a factor of 2 to 4. A formal sensitivity analysis has not been done; however, a 1% increment in effect is affected only slightly by ambient concentration.

**Assessment**

*Informative Value Based on Spread:* LOW

*Informative Value Based on Application:* LOW

Damages vary one to one with impacts.

*Generalizability to Other Applications:* MEDIUM

*Robustness of Value over Time:* MEDIUM

Studies useful for this assessment are expensive and will be relatively rare, thus present findings will not be challenged often. Assumptions within the secondary analyses necessary to utilize data in the economic analysis may be the subject of second guessing; however, most changes should only marginally affect overall results.

**Pedigree** (credibility of the entry's origin)

*Theoretical Basis:* FAIR

No theory needed. Data tell the story. However, good laboratory data support the findings of adverse effects.

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<sup>2</sup>Refer to Part VI for a description of NUSAP.

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*Data Inputs:*                      GOOD

Some data come directly from environmental epidemiology, other come from clinical work. In both cases, adaption was necessary to fit analysis needs. These adaptations probably represent the weakest link in that any assumption which moves outside the direct realm of the original data is an extrapolation.

*Estimation Methods:*        GOOD

Estimation methods are basically fitting data to a curve and then applying conditions to meet analysis needs.

*Estimation Metric:*            EXCELLENT

Symptoms are what is valued and this is what is measured.

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**Note: Health effects analyses are dependent on atmospheric modeling of ozone. Significant uncertainty exists in this source of information.**

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## **PART IV**

### **ECONOMIC VALUATION**

- PAPER 10 BENEFITS FROM REDUCING RISK OF DEATH**
- PAPER 11 THE BENEFITS OF REDUCED MORBIDITY**
- PAPER 12 THE BENEFITS OF VISIBILITY IMPROVEMENTS**
- PAPER 13 THE BENEFITS OF IMPROVING RECREATION QUALITY AND QUANTITY**
- PAPER 14 CALCULATING EXTERNALITIES FROM DAMAGES IN OCCUPATIONAL HEALTH AND SAFETY**
- PAPER 15 COAL TRANSPORTATION ROAD DAMAGE**

