FINAL
APPENDIX I

HEALTH EFFECTS

NOVEMBER 1996

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Table of Contents

APPENDIX I

HEALTH EFFECTS

Health Effects of Air Pollution

Ozone

Particulate Matter (PM_{10})

Carbon Monoxide (CO)

Nitrogen Dioxide (NO_2)

Sulfur Dioxide (SO_2)

Sulfates

HEALTH EFFECTS OF AIR POLLUTION

Ambient air pollution is a major public health concern. Excess deaths associated with air pollution episodes have been documented as early as 1930 in Meuse Valley, Belgium; 1948 in Donora, Pennsylvania; and 1952 in London. Although acute air pollution episodes associated with such readily-evident excess deaths are now unlikely in the United States, air pollution continues to be linked as a cause of respiratory illness (morbidity) and slight increase in death rates (mortality).

The adverse health effects associated with air pollution are diverse and include:

a) Increased health care utilization (hospitalization, physician and emergency room visits)

b) Increased respiratory illness (symptoms, infections, and asthma exacerbation)

c) Reduction in life-span

d) Potential increased risk of developing cancer

e) Decreased breathing capacity

f) Lung inflammation

g) Potential immunological changes

h) Increased airway reactivity to a known chemical exposure - a method used in laboratories to evaluate the tendency of airways to have an increased possibility of developing an asthmatic response

i) A decreased tolerance for exercise.

The evidence linking these general effects to air pollution, and specific effects to a specific pollutant(s) is derived from population-based observational and field studies (epidemiological) as well as controlled laboratory studies involving human subjects and animals. There have been an increasing number of studies focusing on the mechanisms and specific pollutant(s) responsible for individual effects, yet these effects are not always clearly understood. Long-term effects of exposure, being more difficult to identify and measure, require further research and evaluation.

Although individuals inhale pollutants as a mixture under ambient conditions, a focus on specific pollutants occurs because the regulatory framework and the control measures developed therefrom are mostly pollutant-specific. Results from recent studies have raised doubts about the adequacy of the existing National Ambient Air Quality Standards (NAAQS) for ozone and PM_{10} in protecting public health. Hence, U.S. EPA recently

reviewed these NAAQS and recommended revised NAAQS for ozone and PM_{10}. A brief overview of the effects observed and attributed to various air pollutants is presented in this document.

The summary presented is drawn from: (1) a two-part article: "State of the Art: Health Effects of Outdoor Air Pollution" prepared by a committee of the Environmental and Occupational Health Assembly of the American Thoracic Society (presented as an appendix to this document); (2) draft versions of U.S. EPA's Staff Paper on the review of NAAQS for ozone(1995) and PM_{10} (1996); and (3) a report to the Health Effects Institute on Phase 1B: Particle Epidemiology Evaluation Project (1995).[1]

OZONE

The major subgroups of population considered to be at increased risk from ozone exposure are outdoor exercising individuals including children and people with preexisting respiratory disease(s) such as asthma. The data base identifying the former group as being at increased risk to ozone exposure is much stronger and more quantitative than that for the latter group, probably because of a larger number of studies conducted with healthy individuals. The adverse effects reported with short-term ozone exposure are greater with increased activity because activity increases the volume of air reaching the lungs, resulting in an increased amount of ozone reaching the lungs.

The number of hospital admissions for all respiratory causes (infections, respiratory failure, chronic bronchitis, etc.) including asthma show a consistent increase as ambient ozone, sulfate or sulfur dioxide levels increase in a community. These excess hospital admissions and emergency room visits are observed when hourly ozone concentrations are as low as 0.08 to 0.10 ppm. However, limitations in the study designs preclude attributing the effects solely to ambient ozone concentrations.

Several population-based studies suggest that asthmatics are more adversely affected by ambient ozone levels, as evidenced by increased hospitalizations and emergency room visits. Laboratory studies have attempted to compare the degree of lung function change seen in age and gender matched healthy individuals versus asthmatics and those with chronic obstructive pulmonary disease. While the degree of change evidenced did not differ significantly, that finding may not accurately reflect the true impact of exposure on these respiration-compromised individuals. Since the respiration-compromised group may have lower lung function to begin with, the same degree of change may represent a substantially greater adverse effect overall.

In addition, human and animal studies involving both short-term (few hours) and long-term (months to years) exposures indicate a wide range of effects induced or associated

[1] All of the studies referred to in this Appendix are cited in the above noted sources. Specific references to individual studies will not be made in this summary.

with ambient ozone exposure (Table 1). The effects reported include decreased breathing capacity and exercise performance; increased airway resistance and responsiveness (indicative of involvement of both smaller distal and larger proximal airways); impaired host defenses; excess hospital admissions and emergency room visits; acute inflammation of the lung tissue; and respiratory cell damage.

Some lung function responses (volume and airway resistance changes), observed after a single exposure to ozone, exhibit attenuation or a reduction in magnitude with repeated exposures. Although it has been argued that the observed shift in response is evidence of a probable adaptation phenomena, it appears that while functional changes may exhibit adaptation, biochemical and cellular changes which may be associated with episodic and chronic exposure effects may not exhibit similar adaptation.

In a laboratory, exposure of human subjects to low levels of ozone causes reversible decrease in lung function as assessed by various measures such as respiratory volumes, airway resistance and reactivity, irritative cough and chest discomfort. Lung function changes have been observed with ozone exposure as low as 0.08 to 0.12 ppm for 6-8 hours under moderate exercising conditions. Similar lung volume changes have also been observed in adults and children under ambient exposure conditions (0.10 - 0.15 ppm)[2]. The responses reported are indicative of decreased breathing capacity and are reversible.

In laboratory studies, cellular and biochemical changes associated with respiratory tract inflammation have also been consistently reported in the airway lining fluid after low level exposure to ozone . These changes include an increase in specific cell types and in the concentration of biochemical mediators of inflammation and injury such as cytokines and fibronectin. These inflammatory changes can be observed in healthy adults exposed to ozone in the range of 0.08 to 0.10 ppm.

Finally, the lack of consistency between population and laboratory studies also suggests that the susceptibility observed under ambient conditions could be due to the combination of pollutants (not ozone alone) which co-exist in the atmosphere, or that ozone may actually sensitize these subgroups to the effect(s) of other pollutants.

Some animal studies show results which indicate possible chronic effects including functional and structural changes of the lung. These changes indicate that repeated inflammation associated with ozone exposure over a lifetime may result in sufficient damage to respiratory tissue such that individuals later in life may experience a reduced quality of life in terms of respiratory function and activity level achievable. An autopsy study involving Los Angeles County residents provided supportive evidence of lung tissue damage (structural changes) attributable to air pollution.

TABLE 1

[2] Federal Standard .12 ppm, 1-hour average. State Standard .09 ppm, 1-hour average.

Adverse Health Effects of Ozone (O_3)
(Summary of Recent Key Studies)

0_3 Concentration and Exposure Hr, ppm	Health Effect
Ambient air containing 0.10 - 0.15 daily 1-h max over days to weeks	Decreased breathing capacity), in children, adolescents, and adults exposed to 0_3 outdoors Exacerbation of respiratory symptoms (e.g., cough, chest pain) in individuals with preexisting disease (e.g., asthma) with low ambient exposure, decreased temperature, and other environmental factors resulting in increased summertime hospital admissions and emergency department visits for respiratory causes
≥0.12 (1-3h) ≥0.08 (6.6h) (chamber exposures)	Decrements in lung function (reduced ability to take a deep breath), increased respiratory symptoms (cough, shortness of breath, pain upon deep inspiration), increased airway responsiveness and increased airway inflammation in exercising adults
≥0.12 (1-3 h) ≥0.08 (6.6h) (chamber exposures)	Effects are similar in individuals with preexisting disease except for a greater increase in airway responsiveness for asthmatic and allergic subjects
≥0.12 (1-3h) ≥0.08 (6.6h) (chamber exposures)	Older subjects (>50 yr old) have smaller and less reproducible changes in lung function Attenuation of response with repeated exposure
≥0.12 with prolonged, repeated exposure (chamber exposures)	Changes in lung structure, function, elasticity, and biochemistry in laboratory animals that are indicative of airway irritation and inflammation with possible development of chronic lung disease Increased susceptibility to bacterial respiratory infections in laboratory animals

A few studies have suggested that population exposures to community air pollution (ozone and particulate matter), are related to genetic toxicity and increased incidence of cancer. In recent years, an increase in daily mortality has also been reported from studies involving Southern California's population. Because of limitations in study design in a number of studies, as well as assumptions involved and coexisting pollutants, the severity of chronic effects associated with ozone exposure alone cannot be ascertained from these studies.

In summary, acute adverse effects associated with ozone exposures have been well documented, although the specific causal mechanism is still somewhat unclear. Additional research efforts are required to evaluate the long-term effects of air pollution and to determine the role of ozone in influencing chronic effects.

PARTICULATE MATTER (PM_{10})

Inhalability and deposition characteristics of particles less than 10 micrometers in size, which could potentially damage the lower respiratory tract and the gas-exchange region of the lung were the driving force to set the NAAQS for PM_{10}.[3] U.S. EPA in its recent PM_{10} NAAQS review has concluded that the difference in exposure relationships, and the strong likelihood of the fine mode fraction of PM_{10} being significant contributors to PM-related health effects in sensitive populations, are sufficient to justify the consideration of fine and coarse mode particles in PM_{10} as separate classes of pollutants. The major categories of adverse health effects associated with PM_{10} include:

a) Increased mortality (acute and chronic)

b) Exacerbation of preexisting respiratory and cardiovascular diseases leading to an increase in hospital admissions, emergency room visits, school absences, lost work days and restricted activity days

c) Changes in lung function and structure

d) Altered defense mechanisms

e) Increased risk of developing cancer

Short-Term Exposure Effects

Epidemiological studies have provided continued and consistent evidence for most of the effects listed above. An association between increased daily or several-day-average concentrations of PM_{10} (measured or estimated) and excess mortality as well as morbidity is consistently reported from studies involving communities across the U.S. as well as in Europe and South America. A review and analysis of recent epidemiological literature for acute adverse effects was undertaken by Dockery and Pope to estimate these effects as percent increase in mortality associated with each incremental increase of PM_{10} by 10 $\mu g/m^3$. The estimates are presented in Table 2. It appears that individuals who are elderly or have preexistent lung or heart disease are more susceptible than others to the adverse effects of PM_{10}.

[3] Federal Standard 50 $\mu g/m^3$, annual arithmetic mean; 150 $\mu g/m^3$, 24-hour average. State Standard 30 $\mu g/m^3$, annual geometric mean; 50 $\mu g/m^3$, 24-hour average.

TABLE 2

Combined Effect Estimates of Daily Mean
Particulate Pollution

	% Change in Health Indicator per each 10 μg/m^3 Increase in PM_{10}
Increase in daily mortality	
Total deaths	1.0
Respiratory deaths	3.4
Cardiovascular deaths	1.4
Increase in hospital usage (all respiratory diagnoses)	
Admissions	1.4
Emergency department visits	0.9
Exacerbation of asthma	
Asthmatic attacks	3.0
Bronchodilator use	12.2
Emergency department visits*	3.4
Hospital admissions	1.9
Increase in respiratory symptom reports	
Lower respiratory	3.0
Upper respiratory	0.7
Cough	2.5
Decrease in lung function	
Forced expiratory volume	0.15
Peak expiratory flow	0.08

* One study only

(Source: American Journal of Respiratory and Critical Care Medicine, Vol 153, 113-50, 1996)

A positive association between short-term exposures to PM and hospital admissions for respiratory or cardiac diseases is reported in several studies. When results from many epidemiological studies are viewed together, the observed greater effect on admissions for chronic obstructive respiratory disease is consistent with the observation from mortality studies -- suggesting stronger associations between respiratory related mortality and PM exposure.

Similarly, school absences, lost work days and restricted activity days have also been used in some studies as indirect indicators of acute respiratory conditions. The results are suggestive of both immediate and delayed impact on these parameters following elevated PM exposures. These observations are consistent with the hypothesis that increased susceptibility to infection follows PM exposures.

Some studies have reported that short-term PM exposure is associated with changes in lung function (lung capacity and breathing volume); upper respiratory symptoms (hoarseness and sore throat); and lower respiratory symptoms (increased sputum, chest

pain and wheeze). The severity of these effects is widely varied and is dependent on the population studied, such as adults or children with and without asthma.

In contrast to the availability of data from population based studies, data from laboratory studies (human and animal) is insufficient to fully explain the potential causal mechanism associated with the severity of effects related to an incremental increase of 10 $\mu g/m^3$ of PM_{10}. In addition, most of the population-based studies suggest a linear relationship between morbidity and mortality versus ambient PM_{10} levels. However, the expected severity of effects is not always observable in areas with high ambient PM_{10} levels. This may be due to the fact that pollutant sources, as well as the concentration and combination of pollutants, vary significantly among different geographical areas.

Long-Term Exposure Effects

While many studies have evaluated the acute effects, two studies specifically focused on evaluating the effects of chronic exposure to PM_{10} and $PM_{2.5}$. These studies have analyzed the survival data of adults living in different U.S. cities. After adjusting for important risk factors, these studies found a consistent positive association of deaths and exposure to fine particles. A similar association was observable in both total number of deaths and deaths due to cardiorespiratory causes. A shortening of lifespan was also reported in these studies. The estimated range of relative risk of dying from long-term exposure to PM_{10} in U.S. cities is 17 to 37 percent. This estimate is substantially higher than the 3 to 20 percent estimated from acute effects studies.

In addition, long-term effects studies have reported an increased risk of mortality from lung cancer associated with PM exposures. A study involving Seventh Day Adventists has reported an association of cancer in females with increased particulate matter (total suspended particles) exposure. It is not clear from these studies whether the association relates to causation of disease, or whether individuals with cancer are more susceptible to other effects of particles leading to the observed mortality association.

Evaluating mortality effects in a public health context is a complex task and requires information regarding the extent of life shortening or prematurity of death associated with PM exposures. The relative risk estimates vary considerably depending on the type of study (acute vs. chronic) and also among studies within each group. Some attempts have been made in a broad sense to calculate the number of deaths associated with PM_{10} exposure by applying different statistical methods using the relative risk estimates of specific studies. However, U.S. EPA has concluded that although a substantial portion of deaths associated with long-term PM_{10} exposure may be independent of the daily deaths, quantification of the extent of life- shortening or the number of deaths directly associated with PM_{10} exposure from either types of studies is difficult.

Despite data gaps, the extensive body of epidemiological studies have both qualitative and quantitative consistency suggestive of causality. A considerable body of evidence

from these studies suggests that ambient particulate matter, in concentrations below the current NAAQS alone or in combination with other coexisting pollutants is associated with significant increases in mortality and morbidity in a community.

Effects Associated with Fine and Coarse Fraction

The data to compare the adverse impact of fine versus coarse particles is quite limited in comparison to that of PM_{10} (which contains both coarse and fine mode fractions). However, multiple indicators of fine fraction ($PM_{2.5}$, sulfate, coefficient of haze, black smoke, carbonaceous material and hydrogen ion levels) are associated with short-term effects in over 15 cities located in different parts of the world. The health outcomes studied include mortality in sensitive population subgroups, increased hospitalization, respiratory symptoms and decreased lung function. In a long-term effects study involving over 500,000 people living in 151 U.S. cities, the mortality association was statistically more significant with fine particles and sulfates than with coarse particles.

In addition, controlled laboratory studies involving exposure of humans and animals to high levels of pollutants suggest: a) a higher percentage of fine fraction particle deposition in the lungs than of coarse fractions; b) a relative increase in cellular and immunological toxicity with fine fraction particle exposure (ammonium sulfate and nitrate) than with the coarse fraction (road dust); c) a wide range of short and long-term toxic effects with exposures to acid aerosols which are present in the fine fraction; and d) possible additive or synergistic effects when exposures include fine particles and other pollutants such as ozone, nitrogen dioxide or acids either in aerosol or vapor phase. Further research is required to evaluate whether the reported asthmatic effects are more closely associated with the coarse fraction of particles. Evidence is accumulating to indicate that fine particles are probably more toxic than coarse particles and that the fine fraction is responsible for most of the reported PM effects. In view of these observations, U.S. EPA has opted to consider the two fractions in PM_{10} as separate classes of pollutants and has recommended the development of a $PM_{2.5}$ NAAQS which will assist in developing future controls to regulate this class of pollutants.

In summary, the scientific community concurs that: a) an increased risk of mortality and morbidity is associated with PM_{10} at levels below the current NAAQS; b) the evidence for PM effects is mostly derived from population studies; c) there are data gaps of supportive evidence from clinical and animal studies; d) although most of the effects are attributable to $PM_{2.5}$, co-pollutant effects cannot be ruled out on the basis of existing studies; and e) the difficulty of separating the effects may be due to the fact that $PM_{2.5}$ levels may co-vary with other combustion source pollutants, and hence, may serve as an index of air pollution from these sources instead of a separate pollutant by itself.

CARBON MONOXIDE (CO)

The high affinity of CO to bond with oxygen-carrying proteins (hemoglobin and myoglobin) results in reduced oxygen supply in the bloodstream of exposed individuals. It is that reduced oxygen supply which appears to be responsible for the toxic effects of CO which are typically manifested in the oxygen-sensitive organ systems. The effects have been studied in controlled laboratory environments involving exposure of humans and animals to CO, as well as in population-based studies of ambient CO exposure effects. People with deficient blood supply to the heart (ischemic heart disease) are known to be susceptible to the effects of CO, and protection of this group is the basis of the existing NAAQS for CO.[4]

Inhaled CO has no direct toxic effect on lungs but rather appears to exert its effects by interfering with oxygen transport through the formation of carboxyhemoglobin (COHb, chemical complex of CO and hemoglobin). Exposure to CO is often evaluated in terms of COHb levels in blood measured as percentage of total Hb bound to CO. COHb levels in non-smokers range between 0.3 and 0.7% and 5 to 10% in smokers. COHb levels in excess of 1.5% in a significant proportion of urban nonsmoking populations can be considered as evidence of widespread exposure to environmental CO.

Under controlled laboratory conditions, healthy subjects exposed to CO sufficient to result in 5% COHb levels, exhibited reduced duration of exercise performance and consumption of oxygen. Studies involving subjects with ischemic heart disease who engaged in exercise during CO exposures have shown that COHb levels as low as 2.2% can lead to: a) earlier onset of electrocardiograph changes indicative of increased deficiency of oxygen supply to the heart; b) earlier onset of chest pain; c) increase in the duration of chest pain; and d) decrease in oxygen consumption.

Animal studies associated with long-term exposure to CO resulting in COHb levels that are equivalent to those observed in smokers have shown indication of reduction in birth weight and impaired neurobehavior in the offspring of exposed animals. These changes, however, are unlikely at observed ambient CO levels.

NITROGEN DIOXIDE (NO_2)

Evidence for low-level NO_2 exposure effects is derived from laboratory studies of asthmatics and indoor population-based studies of homes with gas stoves as the exposure source. Additional supportive evidence is derived from animal studies.

Epidemiological studies using the presence of an unvented gas stove as a surrogate for NO_2 exposures suggest an increased incidence of respiratory infections or symptoms in children. The same results have not been seen in similarly exposed adults. These studies have provided the basis for the consideration of the margin of safety provided by California's short-term standard (0.25 ppm with a one hour averaging period).

[4] Federal Standard 9 ppm, 8-hour average; 35 ppm, 1-hour average. State Standard 9 ppm, 8-hour average; 20 ppm, 1-hour average.

Collectively, results from controlled exposure studies of asthmatics demonstrate an increase in the tendency of airways to contract in response to a chemical stimulus (bronchial reactivity) on exposure to <0.3 ppm NO_2 for a period ranging from 30 minutes to 3 hours. A similar response is reported in some studies with healthy subjects at higher levels of exposure (1.5 - 2.0 ppm). Mixed results have been reported when people with chronic obstructive lung disease are exposed to low levels of NO_2.

Short-term controlled studies of animals exposed to <0.3 ppm of NO_2 over a period of three hours indicate cellular changes associated with allergic and inflammatory response and interference with detoxification processes in the liver. In some animal studies the severity of the lung structural damage observed after relatively high levels of short-term ozone exposure is observed to increase when animals are exposed to a combination of ozone and NO_2.

In animals, longer-term (3-6 months) repeated exposures at 0.25 ppm appear to decrease one of the essential cell-types (T-cells) of the immune system. Non-specific changes in cells involved in maintaining immune functions (cytotoxic T cells and natural killer cells) have been observed in humans after repeated exposure (4-6 days) to >0.6 ppm of NO_2 (20 min. - 2 hours). All these changes collectively support the observation reported both in population and animal studies of increased susceptibility to infections, as a result of NO_2 exposure.

SULFUR DIOXIDE (SO_2)

Controlled laboratory studies involving human volunteers have clearly identified asthmatics as the most sensitive group to the effects of ambient SO_2 exposures. Healthy subjects have failed to demonstrate any short-term respiratory functional changes at exposure levels up to 1.0 ppm over 1-3 hours.

In asthmatics, brief (10 minutes) exposure to SO_2 at levels as low as 0.25[5] ppm can result in significant increases in airway resistance as well as decreases in breathing capacity. In some, the exposure can result in severe symptoms necessitating the use of medication for relief. The response to SO_2 inhalation is observable within 2 minutes of exposure, increases further with continuing exposure up to 5 minutes then remains relatively steady as exposure continues. SO_2 exposure is generally not associated with any delayed reactions or repetitive asthmatic attacks.

No significant changes have been reported from studies which have evaluated the effects of exposure to co-pollutants (ozone or nitrogen dioxide) prior to or in conjunction with SO_2 exposure.

Animal studies have shown that despite SO_2 being a respiratory irritant, it does not cause substantial acute or chronic toxicity in animals exposed at ambient concentrations.

[5] Federal Standard .03 ppm, annual average; .14 ppm, 24-hour average. State Standard .05 ppm, 24-hour average; .25 ppm, 1-hour average.

However, relatively high exposures (10 ppm of SO_2 for 72 hours) in mice can lead to tissue damage, fluid accumulation and sloughing of respiratory lining. Sensitization to allergies is observable in guinea pigs repeatedly exposed to high levels (72 ppm) of SO_2. This effect needs further evaluation in clinical and population studies to identity the chronic exposure impact on both asthmatic incidence and attacks in a population.

Some epidemiological studies indicate that the mortality and morbidity effects associated with the fine fraction of particles show a similar association with ambient SO_2 levels. In these studies, efforts to separate the effects of SO_2 from fine particles have not been successful. Thus, it is not clear whether the two pollutants act synergistically, or whether being generated from similar combustion sources they represent the same pollution index for the observed effects.

SULFATES

Based on a level determined necessary to protect the most sensitive individuals, the California Air Resources Board in 1976 adopted a standard of 25 $\mu g/m^3$ (24-hour average) for sulfates. In addition, U.S. EPA has concluded that 24-hour average sulfate concentrations of 8-10 $\mu g/m^3$ can aggravate symptoms in people with respiratory disease(s) and can affect lung function in children.

In recent years, a vast majority of effects (mortality and morbidity) associated with fine particles ($PM_{2.5}$) and sulfur dioxide have shown a similar association with ambient sulfate levels in some population studies. The efforts to fully separate the effects of sulfates from other coexisting pollutants have not been successful. This may be due to the fact that these pollutants co-vary under ambient conditions having been emitted from common sources and the effects observed may be due to the combination of pollutants, rather than a single pollutant.

Clinical studies involving exposure of human subjects to sulfuric acid aerosol (<100 $\mu g/m^3$) have indicated that adolescent asthmatics may be a susceptible population subgroup. Lung function changes (volume and resistance to air flow) are observable in this subgroup as well as in people with chronic obstructive lung disease such as chronic bronchitis and emphysema.

Results from animal studies involving exposures to sulfuric acid aerosol, ammonium bisulfate and ammonium sulfate indicate that acidic particles (former two) are more toxic than non-acidic particles (latter). In addition, the severity or magnitude of both mortality and morbidity effects is relatively higher in population studies of the eastern United States and Canada where sulfate concentrations are higher than for those observed in the western United States. Mixed results have been reported from studies which attempted to ascertain the role of acidity in determining the observed toxicity.

CONCLUSION

The vast body of scientific evidence shows that the adverse impacts of air pollution in human and animal health are clear. A considerable number of population-based and laboratory studies have established a link between increased morbidity and in some instances, earlier mortality and air pollution.

ATTACHMENT 1

STATE OF THE ART - HEALTH EFFECTS OF OUTDOOR AIR POLLUTION

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