ESTIMATES OF THE PUBLIC HEALTH BENEFITS AND RISKS ATTRIBUTABLE TO EQUIPPING LIGHT DUTY MOTOR VEHICLES WITH OXIDATION CATALYSTS

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February 1, 1975

Introduction

National standards limiting emissions of carbon monoxide, hydrocarbons, and oxides of nitrogen from light duty motor vehicles were established primarily to protect public health. Since implementation of the first standards in the late 60's, the Federal Government has required further reductions in permissible emission levels with final statutory standards being established in the 1970 Clean Air Act Amendments and targeted for achievement in the mid-70's While the amendments, in effect, established the final emission levels of the pollutants, they did not permit EPA to establish the techniques by which such levels would be achieved. In meeting the standards for carbon monoxide and hydrocarbons, domestic automobile manufacturers chose to employ the oxidation catalyst for most engine families. While there is current debate over the statutory oxides of nitrogen standard, it is likely that reduction catalysts will be utilized to achieve the required emissions reduction of that pollutant should the statutory standard remain at the planned level.

The required reductions in the emissions of carbon monoxide, hydrocarbons, and oxides of nitrogen were based upon the need to protect public health from adverse effects attributable to carbon monoxide, nitrogen dioxide, and to oxidants, for which the key precursors are the hydrocarbons. The application of oxidation catalysts will make it possible to reduce substantially emission levels of carbon monoxide and hydrocarbons. EPA has long been aware that the three regulated pollutants

do not solely comprise the products emitted from light duty motor vehicles. Non-regulated emission products of both past and current concern include total particulates, particulate lead, polynuclear aromatic hydrocarbons, phenols, sulfur compounds, particulate metals, aldehydes, nitrogen compounds, and oxygenates. Emission control approaches employed to achieve the various emissions standards through the 1974 model year generally only altered the relative concentrations of species already present in the exhaust. The use of oxidation catalysts, on the other hand, alters emission products far more dramatically. Certain non-regulated emission products of public health concern are dramatically decreased, while others are created or dramatically increased.

Elucidating the health consequences of changes in environmental quality is one of the most challenging scientific tasks facing mankind today. Four major types of difficulties are customarily encountered when one attempts to develop the dose-response relationships linking environmental agents to adverse effects on human health. First, there is usually insufficient information regarding the magnitude and frequency of exposure to environmental agents because health-related environmental monitoring has been an underdeveloped activity and because the wide variations observed in human preferences and activity patterns make the translation of environmental monitoring into human exposure models a complex endeavor. Second, the links between exposure and disease are complex. For example, the effects of infrequent short-term peak exposures may well differ markedly from the effects of long-term exposures or frequent short-term exposures repeated over an extended time frame. The relationship between exposure and disease

may be obscured because the latency period between exposure and effects may be quite long. Furthermore, a single environmental agent may contribute to a number of different disorders and a single disorder may result from a combination of circumstances and not result from one or more environmental agents acting alone. Third, health effects studies are limited by the shortcomings of vital records and the imperfections in morbidity assessment. Fourth, one usually lacks a biologically coherent research data base with clearly interlocking and mutually supporting clinical, occupational, epidemiologic, and toxicologic studies. Progress in each of these areas has been made during recent months and years. However, the residual scientific uncertainties clearly demonstrate that our technical information base must be rapidly augmented if we are to assure a reasonable foundation for sound policy decisions affecting economic growth, transportation, power generation and other problems involving energy and our environment. Under optimum circumstances assembling the needed scientific information will require several years. In the meantime scientists must provide at least rough assessments for decision makers who face tight, legally required action schedules and deal with shifting political and social realities.

This paper attempts to put into perspective the benefit/risk aspect of equipping light duty motor vehicles with oxidation catalysts by examining the public health impact of changing emissions and changing air quality on the urban population of our nation. Major benefits should follow the expected reduction in exposures to carbon monoxide and photochemical oxidants. However, major risks are likely to accompany the increases in exposures

to acid-sulfate-aerosols which can be attributed to the use of oxidation catalysts. It is also well to recall that changes in exposures to a number of other pollutants whose effects are not yet well quantified could well have measurable public health impacts and that the temporal and geographic patterns of change can become an important consideration for decision makers.

METHODS AND ASSUMPTIONS

Comparison of public health benefits and risks requires a complex assessment with five major steps: First, emissions factors for a changing vehicle population are calculated. Second, the impact of emissions changes on ambient air quality are projected. Third, probable changes in human exposures to the pollutants of major interest are estimated. Fourth, dose-response functions for the adverse effects of greatest interest are constructed using the best-judgment assessments of available health intelligence. Fifth, public health benefits and risks are then estimated.

Calculation of Changes in Emissions: Projected changes in carbon monoxide and hydrocarbon emissions were made assuming that 1975 interim Federal Standards for the forty-nine states will be followed by statutory standards for 1976 and subsequent model years. These calculations were based on the 1975 Federal Test Procedures and they assume an age distribution for the automobile population like that seen in 1973, a miles-driven distribution like that seen in 1973, and emissions controls that function in individual vehicles at the standard specified for the model year.[1] Appropriate calculations were likewise made for a number of unregulated pollutants emitted by vehicles not equipped with

oxidation catalysts. Such pollutants include polynuclear aromatic hydrocarbons, phenols, aldehydes, particulate nitrogen, and lead. When considering lead, it was assumed that the 1975 models and subsequent model years would require lead-free fuel and that the EPA promulgated lead phase-down regulations would apply. When considering unregulated pollutants emitted from catalyst-equipped vehicles, the authors applied the previously-mentioned assumptions regarding the age and miles-driven distributions for future vehicle populations. Emissions factors for particulate sulfates and sulfuric acid, aluminum and its compounds, platinum and its compounds, and particulate nitrogen were based upon measurements made with prototype 1975 model year vehicles equipped with various types of oxidation catalysts.[2,3,4]

Impact of Emissions Changes on Ambient Air Quality: Since past air monitoring data for automotive emissions and for photochemical oxidants are limited, it was necessary to choose air quality distributions for carbon monoxide and for photochemical oxidants which provide an upper boundary for calculation of benefits associated with the use of catalytic converters. For carbon monoxide (Table 1) the average carboxyhemoglobin level in non-smokers was chosen as the most appropriate indicator of recent average exposures to carbon monoxide emitted primarily from mobile sources. When meteorological conditions are adverse, such exposures are likely to increase by a factor of about four and carboxyhemoglobin levels will increase by a factor of about three. It is thought that the carboxyhemoglobin distribution utilized reflects an estimate of baseline exposures for calculation of benefits attributable to carbon monoxide control. In the case of photochemical oxidants, the South Coast Air Basin of California was considered one part of the problem and other United States cities with populations over 100,000 were considered separately.

The maximum hourly oxidant concentrations for each day during a pre-catalyst year are assumed to average approximately 140 $\mu g/m^3$ (.07 ppm) in Los Angeles and 60 $\mu g/m^3$ (.03 ppm) elsewhere (see Table 1). In fact, oxidant estimates may somewhat overstate the severity of the oxidant problem in our major cities during 1973-1974. Unfortunately, air monitoring stations for oxidants outside California were few in number until state implementation plans recently required more extensive monitoring. Thus, it will be a year or more before the more adequate oxidant data are available to establish lower boundary and best judgment estimates for public health benefits associated with the use of oxidation catalysts.

For unregulated emissions, two different types of air quality assumptions were applied. In the case of lead, phenols and polynuclear aromatics, one assigned air quality levels to homes, arterial throughways, the workplace and commercial establishments and time-weighted averages based on activity levels were applied.[2,5,6] In the case of particulate sulfates and sulfuric acid, urban sulfate distributions for each region were estimated using water soluble sulfate measurements from National Air Sampling Network Stations.[7] Baseline air quality levels for aluminum and its compounds, particulate nitrogen and noble metals were also obtained from National Air Sampling Network reports.

Exposure Assessments Were Made: For carbon monoxide it was assumed that equipping the entire vehicle population with oxidation catalysts would eliminate exogenous carbon monoxide exposure for non-smokers. Their carboxyhemoglobin values were so adjusted. This, of course, overstates the exposure reduction

that is, in fact, achievable and it may seem to provide an upper boundary for calculating benefits. However, this is not necessarily the case as the dose-response function employed for carbon monoxide may actually underestimate the number of premature cardiac deaths resulting by several fold. For photochemical oxidants the report also assumed that catalytic converters would reduce urban levels so that only natural background levels, which are not now relatable to adverse health effects, would be encountered. For both photochemical oxidants and carbon monoxide, a simple proportionate rollback model was applied when assessing the benefits of interim years before the entire vehicle population is equipped with oxidation catalysts, thus, benefits are somewhat underestimated for the first few years and overestimated in the later years.

For particulate sulfate and sulfuric acid, incremental increases in exposures associated with the use of oxidation catalysts were estimated using the carboxyhemoglobin surrogate detailed in reference 2. The low COHb surrogate estimate was employed, which is likely to provide a low particulate sulfate and sulfuric acid exposure estimate for major urban centers and Southern California (see Table 2). The COHb surrogate provides a means of estimating urban population exposures to catalyst incremental particulate sulfate and sulfuric acid which is independent of any assumed human activity pattern and suggests incremental exposure estimates which are lower than those projected using the physical exposure models also discussed in reference 2.

An approximation of how regulated and unregulated mobile source emissions are likely to change is shown in Table 2. It is clear that substantial reductions in exposures to carbon monoxide, photochemical oxidants, phenols, polynuclear aromatic hydrocarbons, and lead particulate will occur. Exposures to oxides of nitrogen will not be measurably altered by oxidation catalysts. On the other hand, increased exposures to particulate sulfate and sulfuric acid, and possibly aluminum and its compounds, and platinum and its compounds will follow the introduction of vehicles equipped with oxidation catalysts.

Construction of Dose-Response Functions for Adverse Effects of Greatest Interest: For carbon monoxide, a dose-response function linking elevated levels of carboxyhemoglobin to excess death following mycardial infarction was established utilizing data from laboratory animals and from human volunteer studies of less severe cardiac effects.[9,10] While adverse effects might occur with very low carboxyhemoglobin levels, it was assumed for this report that no adverse effect could be demonstrated below a carboxyhemoglobin level of two percent. A linear increase in adverse effe

was assumed up to a carboxyhemoglobin level of 10 percent (See Table 3). Unfortunately, it is not now possible to assess some of the most important potential adverse effects of oxidant exposures like co-carcinogenesis, acceleration of aging, and mutagenesis. However, one can estimate benefits associated with the likely improvement of health in certain groups exposed to oxidants: less aggravation of asthma and chronic heart and lung disorders, and a reduced frequency of irritation symptoms in healthy persons.[8] It was assumed that oxidant levels which produced respiratory tract irritation in healthy young adults would likewise aggravate asthma or heart and lung disorders in susceptible population segments (See Table 3). For particulate sulfate and sulfuric acid, dose-response functions were constructed for increased daily mortality, aggravation of heart and lung disorders in the elderly, aggravation of asthma, increased frequency of acute lower respiratory illness, and increased frequency of chronic respiratory disease symptoms in adults (See Table 3).[7]

Estimating Public Health Benefits and Risks: Three steps are required to estimate how changes in air quality will influence public health. First, the population at risk must be specified. Second, air quality distributions must be linked with dose-response functions to calculate adverse health effects attributable to any specific pollutant exposure. Third, adverse health effects attributable to baseline pollutant exposures must be compared with effects from future projected air quality distributions. The choice of populations at risk and the assignment of baseline risks are summarized in Table 4. Procedures are detailed elsewhere for defining baseline risks and populations at risk including asthmatics, elderly persons with cardio-respiratory disorders, healthy adults, children, and those susceptible to a premature cardiac death.[7,10,11]

For each population segment, it was assumed that both the benefits and risks associated with the use of catalytic converters would be limited to urban regions of 100,000 or larger.[7]

RESULTS

General: Our best efforts allow only a rough approximation of the benefits and public health risks attributable to equipping light duty motor vehicles with oxidation catalysts. Two major problems are persistently encountered: First, best judgment estimates are framed in considerable uncertainty, and, second, we simply lack the ability to quantify a number of probably significant benefits. However, it is unlikely that major national decisions affecting public health, energy and transportation can wait until our ability to make benefit-risk analyses of motor vehicle emissions is significantly improved.

It is very important to remember that it is much easier to calculate the economic costs of emissions controls than to develop the health damage functions and calculate health costs. With our present limited health intelligence base and with the present methodological difficulties in assigning health costs, there would be a tendency to underestimate the true health costs. A cost-benefit approach will require rather precise dose response functions for each adverse effect related to the primary ambient air quality pollutants taken individually or in combination. Generating these functions would be a major scientific endeavor requiring substantial increments in public

investments for five to ten years. In our opinion, precipitous movement to a cost-benefit philosophy in the absence of greatly improved health damage functions would tend to slow drastically the air pollution control effort and leave a rather large but poorly defined residual of continuing ill health.

Mortality: One expects that reducing carbon monoxide exposures will reduce premature deaths from myocardial infarctions and that increasing particulate sulfate-sulfuric acid will increase the risk of premature death in elderly persons already afflicted with chronic heart and lung disorders. A benefit-risk comparison of the projected effects on mortality shows that oxidation catalysts should after ten model years prevent modest numbers of premature deaths (Table 5 and Appendix A). It is difficult to generalize, but one might hypothesize that deaths attributable to carbon monoxide exposure are more likely to occur in somewhat younger persons.

There are a number of other important aspects of the comparison: First, even our high estimates of benefits may prove too low by a factor of three or more if carbon monoxide exposures are higher than now estimated or if such exposures play a significant role in producing or accelerating atherosclerosis. Our estimates of carbon monoxide exposure are only approximate because human activity patterns have not been well described in the areas where large amounts of carbon monoxide are emitted at or near ground level. The time spent in a shopping center, in an urban street canyon, on a busy arterial throughway or adjacent to these sources is likely to be a more important determinant of exposure than general ambient levels away from such sources. Second, not all

regions equally share risks and benefits. Initially, benefits can be expected in large cities of both the eastern and western United States. Later, one can maintain net benefits in large western cities but a net public health risk for excess mortality will be created in the east. Third, most of the benefits attributable to catalyst use will occur in the first four to six model years when vehicles with more stringent controls are replacing uncontrolled or poorly controlled vehicles. The risk attributable to catalyst-equipped vehicles will rise almost linearly over time. There is little doubt that most of the mortality reduction attributable to use of oxidation catalysts during the first four model years would have likewise resulted over a somewhat longer period from the continued sales of new cars meeting the 1973-1974 national emissions standards for light duty motor vehicles. Unlike catalyst-equipped vehicles, 1973-1974 model year cars did not increase mortality risk because they did not emit appreciable amounts of particulate sulfates and sulfuric acid.

Other Effects of Carbon Monoxide: There are a number of other as yet poorly quantified benefits of carbon monoxide control including possible improvements in mental performance among drivers and in the workplace. These benefits would, of course, also be achieved if carbon monoxide exposures were reduced by means other than the use of oxidation catalysts. In summary it can be concluded that effective control of carbon monoxide emissions is necessary to protect public health even though all public health benefits cannot now be precisely quantified.

Aggravation of Asthma: Elevated ambient levels of photochemical oxidants and particulate sulfate-sulfuric acid can aggravate asthma. In other words, known asthmatics are more likely to experience one or more asthma attacks on more polluted days. However, it has proved very difficult to relate pollutant levels to the severity of any single asthmatic attack or to the number of attacks experienced by any individual patient on any given day. The present benefit-risk analysis (Table 6 and Appendix B) indicates that any reductions in asthma attributed to reductions in photochemical oxidants will be overwhelmed in all geographic areas by an increased risk attributable to emissions of particulate sulfate-sulfuric acid. Statistically, one expects increased risks for asthma to begin rather promptly with measurable adverse effects seen in most cities around the fourth model year. The dose-response function utilized for asthma probably overestimates the public health risk because one has assumed warmer temperatures than exist during much of the winter. In summary there is every indication that the use of oxidation catalysts will be accompanied by a net aggravation of asthma throughout the nation.

Aggravation of Heart and Lung Disease: Elevated ambient levels of photochemical oxidants and particulate sulfate-sulfuric acid are thought to aggravate symptoms experienced by elderly persons with chronic heart and lung disorders. In other words, elderly persons are more likely to report that they feel their own symptoms have worsened on more polluted days. At the present time we cannot reliably quantify what such aggravation means in terms of medical care.

We do, however, believe that these persons not only report a worsening of symptoms but that also more of them prematurely die on such days. Our present benefit-risk approximation (Table 7 and Appendix C) shows that a net increase in aggravation of heart and lung disorders can be expected to occur when the entire vehicle population is equipped with oxidation catalysts. Other observations may be equally important. First, geographically, one can expect most of the benefits in the far west and most of the increased risks in the eastern United States. Second, net benefits in Southern California can be expected for four to six model years whereas net benefits are not projected elsewhere at any time. Third, additional dose-response data might well substantially increase the benefits allocated to Southern California. One can, of course, retain the benefits in Southern California and not experienc the risks by desulfurizing gasoline.

Acute Lower Respiratory Disease in Children: Laboratory studies in animals indicate that exposure to elevated levels of photochemical oxidants are likely to increase the risk for excess acute respiratory disease in man. Existing epidemiologic studies have not yet been able to disentangle oxidant effects from the other major determinants of such illnesses. Nevertheless, it is the best judgment of the authors that measurable health benefits in the form of reduced acute respiratory morbidity would follow the reduction of peak oxidant exposures now experienced in Southern California. Against this benefit one must weigh the risks that are projected to occur nationally as vehicles are equipped with oxidation catalysts (Table 8 and Appendix D). Interestingly enough, the present analysis projects little or no catalyst associated risk among children living in Southern California but substantial

risks for children in the eastern United States. In summary, the use of catalysts in Southern California might well lead to a reduction in acute respiratory morbidity and thus represent a substantial net benefit not quantified by the present analysis.

Chronic Respiratory Disease Symptoms: Chronic respiratory disease symptoms of interest include persistent cough, production of sputum, shortness of breath and at times more severe symptoms accompanied by marked functional impairment. A variety of underlying pathologic conditions affecting the lower airways and alveoli are associated with these symptoms. In any case, an increased frequency of persistant cough accompanied by sputum among general population is considered to represent an adverse health effect. At present there is not a substantial body of laboratory or epidemiologic evidence indicating that either photochemical oxidants or carbon monoxide constitutes a risk factor for chronic respiratory disease. However, responsible scientists will not be surprised if future studies reveal a contributing role for photochemical oxidants in these disease processes.

The present benefit-risk analysis (Table 9 and Appendix E) shows that the use of oxidation catalysts will result in a substantial increased risk for chronic respiratory disease. Again, one is struck by geographical and temporal factors. Almost all of the projected risks occur in the eastern United States while most of the hypothesized benefits are likely to occur in the most impacted areas of the west. Substantial risks will begin occurring only after the expected exposures projected for two to four model years are maintained for several additional years. In other words, public

health risks are likely to increase rapidly after the fourth to sixth model year. If one were to equip Southern California vehicles with oxidation catalysts and desulfurize fuel supplies in that area, one could avoid any public health risk and maximize any reductions in chronic respiratory illness that might be achievable through the reduction of exposures to photochemical oxidants.

Prevalence of Irritation Symptoms Among Otherwise Healthy Adults: Physicians but not the general public, may at times be inclined to overlook the importance of rather subtle increases in eye irritation, transient cough, ill defined chest discomfort and the common headache. Increases in the frequency of such symptoms after laboratory or ambient exposures to elevated levels of photochemical oxidants, especially peroxyacylnitrate (PAN), are well documented. Increases in irritation symptoms, especially transient cough, can also be hypothesized to follow exposures to elevated levels of particulate sulfate-sulfuric acid but no dose-response function is available. Consequently, the present benefit-risk analysis projects that irritation symptoms will be clearly reduced especially in Southern California by the use of oxidation catalyst (Table 10 and Appendix F). Somewhat unexpectedly, the greatest projected lessening of "excess symptom days" occurs for the common headache. This projection is the least defensible of the group as causative mechanisms for aggravation of the complex symptom are not yet defined.

Other Significant Public Health Problems Where Benefits are Poorly Defined: Photochemical oxidants represent a complex group of pollutants indexed by ozone a including a number of the most biologically worrisome gaseous air pollutants currently encountered. There is reason to believe that photochemical oxidants

cause not only respiratory effects but a variety of systemic changes that constitute adverse health effects. Scientists are legitimately concerned that photochemical oxidants may act as carcinogens or co-carcinogens, that they can accelerate aging and that they may contribute to mutations. It is also known that catalysts will further reduce general population exposures to polynuclear aromatic and phenolic compounds and that this grouping contains a number of suspected carcinogens and co-carcinogens.[1,5] There is also reason to believe that photochemical oxidants and carbon monoxide may aggravate other clinical conditions that are less common than those previously discussed. For example, either of these pollutants might exacerbate the symptoms of certain hemolytic anemias or decrease the functional reserves of other presumably susceptible population segments, like newborn infants, children with congenital heart disease, cystic fibrosis patients, and apparently well but elderly persons. In any case, there is every reason to believe that the present analysis does not consider substantial benefits associated with the control of carbon monoxide and hydrocarbon emissions. Likewise, it is probably that increased exposures to particulate sulfate and sulfuric acid will also result in other health risks which are not well-defined in this analysis. In balance though, it is the opinion of the authors that the net effect of these poorly-defined risks would be in the direction of favoring the stringent control of automotive pollutants.

Health Problems Associated with Lead, Aluminum Compounds and Noble Metals: It is not within the scope of the present report to discuss in detail the health effects of airborne lead. Suffice it to say that the use of leaded gas is not considered compatible with oxidation catalysts and that the use of such devices will preclude the use of lead as a fuel additive in catalyst-

equipped vehicles, except under emergency conditions. If the lead content of fuel is not reduced for public health reasons, there still is every reason to believe that public health benefits relatable to decreased lead exposures can occur as catalyst-equipped vehicles come to dominate the vehicle population and thus lead to a reduction in the emissions of lead. In urban areas it is likely that blood lead levels and lead burdens would be significantly reduced among those not overly exposed to other sources of environmental lead. There is a growing suspicion that one should consider not only classical lead poisoning but also other chronic effects of lead exposure. For example, there is legitimate concern about the behavioral implications of elevated lead levels and a conjectured increase in the risk for respiratory cancer associated with simultaneous exposure to lead and polynuclear aromatic compounds. Health scientists in the Environmental Protection Agency strongly advocate reducing lead emissions from motor vehicles.

In the case of aluminum, aluminum compounds and noble metals, it is now thought unlikely that equipping light duty motor vehicles with oxidation catalysts will result in any direct inhalation hazard for the next few years. However, our information is not complete enough to assure that these substances will pose no hazard to public health or to the environment. More information on fabrication, use, disposal, exposures and effects will be required. It is known that both aluminum and noble metal compounds can, under some circumstances, be rather potent pulmonary irritants. Very little is known about the effects of chronic exposure to noble metals and their compounds. Hopefully, oxidation

catalysts will prove durable enough in the hands of the consumer so that exposures to these substances will continue to be minimal.

Discussion

When considering the net effect of all of the benefit-risk assessments individually discussed (Table 11), it does not appear that the use of oxidation catalysts for ten or more model years represents a consistent major net public health benefit. However, closer examination of geographic difference and probable temporal trends is necessary before reaching any final conclusions. Clearly, a large portion of the quantified benefits will accrue from the use of oxidation catalysts in Southern California while most health risks will be in the eastern United States. Likewise, the use of oxidation catalysts can be continued somewhat longer without the full range of major adverse effects in California than in other parts of the nation. Even so, somewhere between the fourth and tenth model year the use of oxidation catalysts appears to become a public health liability in every locale unless steps are taken to reduce emissions of particulate sulfates and sulfuric acid. One assumption of the present analysis is that only cities 100,000 or larger will be affected. This is probably not the case. An alternate analysis for all urban regions is included among the appendices but the conclusions reached by either route are the same. A persistent vexing aspect of the analysis is the realization that a number of probable health benefits are not presently quantified. Nevertheless, it is quite apparent that the continued use of oxidation catalysts to control carbon monoxide and hydrocarbon emissions from light-duty motor vehicles is at best an uncertain, mixed blessing.

The reader should recall the major directions of bias in the assumptions for this report and the reasons for these assumptions. When considering carbon monoxide, study assumptions undoubtedly overestimated the degree of control that is possible. In fact, all carbon monoxide exposure to persons who do not smoke tobacco will not be eliminated by the use of catalyst converters. This bias would tend to set an upper bound on benefits. It is thought that the assumed ambient exposures for carbon monoxide and human activity patterns are more or less reasonable approximations for the exposures and activity patterns of populations living in larger urban areas. In the case of photochemical oxidants, study assumptions are thought to overestimate existing exposures outside of California and to bound exposures within the Southern California. Furthermore, the analysis assumes control of mobile sources of hydrocarbons can, in fact, reduce oxidant exposures to natural background levels which are well below the known thresholds for adverse health effects. The net result is to assign an upper boundary for the quantifiable benefits of catalyst use. The analysis did not specifically consider the benefits of reducing photochemical oxidant exposures for less than or more than the peak hour experienced in a given day. When considering particulate sulfate and sulfuric acid, best judgment exposure estimates for each city size in each region were applied instead of applying upper boundary estimates of exposure. Furthermore, adverse effects attributable to short-term exposures lasting up to two or three hours could easily aggravate respiratory disorders and these are not considered. Thus, the net direction of bias is probably to underestimate the risks attributable to the use of oxidation catalysts. On the other hand, one does not have to consider as large a set of unquantifiable

major adverse health effects when considering exposures to particulate sulfates and sulfuric acid as was the case with photochemical oxidants.

The present analysis assumes that oxidation catalysts will be required on all new vehicles after the 1975 model year to achieve statutory emissions standards for hydrocarbons and carbon monoxide. A wide number of alternate emissions scenarios can be similarly evaluated. However, it should be pointed out that the present analysis is probably not sufficiently precise to quantify the public health impact of minor changes in emissions factors. It should be recalled that for the next five or six model years one anticipates replacing older vehicles having little or no emissions control with vehicles that have much more stringent controls. The real impact of modest changes in the relative effectiveness of alternate emissions standards will be seen in how well the control devices utilized function in the hands of the consumer and in other characteristics of the vehicle population such as number of cold starts. vehicle miles driven, growth in vehicle population, transportation controls, and impact of urban mass transit systems.

Characteristics of the vehicle population will also significantly affect several dimensions of the particulate sulfate-sulfuric acid exposure problem. The present analysis assumes no further growth in the vehicle population. Projections are that the present static vehicle population will continue to grow in the next few years, but the rate of future growth and the place of growth is not now clear. Failure to include some growth factor probably leads

also assumed fuel economy, vehicle model weight distributions, vehicle age distribution, and constant miles-driven distribution based on the 1973 vehicle populations. The authors expect that lighter vehicles with better fuel economy will be introduced during the period covered by this analysis. There is, however, as yet no reason to assume that the miles-driven or vehicle age distributions will significantly change. The net effect of such changes might lessen somewhat the public health risks that are projected to occur as vehicles equipped with catalysts comprise an even greater portion of the total population, although the use of the low carboxyhemoglobin exposure estimator in this analysis may tend to mitigate such inaccuracies, particularly in major urban centers. Another relevant consideration is that of catalyst durability in the hands of the consumer. A realistic concern is that the consumer may use leaded fuel should gasoline shortages recur or operating problems be encountered with low-octane unleaded fuel. In this case, the benefits and risks associated with oxidation catalysts will both be reduced as the catalyst loses effectiveness.

Other important sets of considerations not addressed in the present analysis are effects of controls on other air pollutants, welfare effects, and regional differences in sulfur content of gasoline, vehicle density, and vehicle model weight distributions. For example, reductions in hydrocarbons might alter the rate for atmospheric transformation of sulfur dioxide into sulfuric acid, control of oxidants should reduce plant damage, and increased urban levels of sulfuric acid could be expected to damage materials. It is not clear whether visibility would change. Because of high fuel sulfur, one would expect higher emissions of particulate sulfate and sulfuric acid in Southern California than was projected in this analysis.

How might mobile source emissions of particulate sulfate and sulfuric acid interact with changes in emissions of sulfur dioxide from stationary sources? The present analysis assumes that 1970 levels of particulate sulfate would remain unchanged as vehicles equipped with oxidation catalysts became predominant in the vehicle population. A comparison of catalyst associated exposures with 1970 baseline exposures shows that exposures attributable to oxidation catalysts become a significant part of the total exposure problem in all larger urban areas after two to four model years (Table 2). Actually, the lower exposure estimator mentioned in Table 2 was used in the present analysis. The sulfate problem would loom even larger if the higher exposure estimator thought more appropriate for the largest cities, and certainly Southern California, had been utilized for these cities. If one assumes that state implementation plans were able to reduce sulfur dioxide emissions and urban particulate sulfate levels by 50 to 60%, then the use of catalytic converters could completely negate any benefits from such control. This would also mean that the public health risks projected in this report would be greatly reduced if not entirely eliminated. More worrisome is the possibility that planned sulfur dioxide emissions limitations for stationary sources will be dramatically relaxed and unrestrained energy demands may accelerate growth in emissions of sulfur oxides. In that case, it is likely that the public health risks associated with catalytic converters would be substantially higher than these projected by the present analysis. Of course, if ambient sulfate levels are not greatly changed during the next few years, the estimates of risk projected in this analysis are less likely to be in error.

There are a number of regulatory options which can maximize the public health benefits attributable to emissions controls and minimize any health risks. One option is the desulfurization of gasoline, an option which might prove quite practical for an area like Southern California where the benefits of stringent emission controls are quite large. Another option is the application of a particulate emission standard which could effectively regulate mobile source emissions of lead, other metals, sulfates, and sulfuric acid. Such a standard, applied to new vehicles, has many desirable health aspects.

Major Caveats

Throughout this report, major caveats affecting benefit-risk assessments have been clearly stated but it is well to repeat the more important ones:

First, it is difficult to define precisely how the characteristics of vehicle population, and consequent emission factors and emissions will change. However, this factor does not seem as important as other major uncertainties.

Second, better estimates of the links between emissions, air quality, and human exposure are needed before benefit-risk models can precisely evaluate different emissions control scenarios.

Third, major uncertainties exist in existing dose-response functions for adverse health effects. Indeed, dose-response relationships simply are not well documented for a number of effects of greatest concern.

Fourth, existing air monitoring data do not allow more than boundary estimates of benefits associated with the use of oxidation catalysts.

Fifth, the impact of national decisions on the control of sulfur oxide emissions from stationary sources can drastically alter the projections of this report.

Conclusions

The introduction and continued sales of light-duty motor vehicles equipped with oxidation catalysts will probably result in a net public health risk unless the sulfur content of gasoline is reduced or particulate emissions are controlled by other means. Geographically, a large portion of the projected health benefits can be expected in Southern California while the health risks will be concentrated but not limited to the eastern United States. Temporally, benefits attributable to the first few model years might be greater than benefits occurring as later models are introduced because catalyst-equipped cars initially replace largely uncontrolled vehicles. In any case, it is likely that after four model years, public health risks will exceed benefits in all areas of the country unless other regulatory steps are taken. The use of oxidation catalysts clearly represents a complex issue involving public health, established and proposed regulatory mandates, energy considerations, and the national economy.

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Table 1

BASELINE AIR QUALITY DISTRIBUTIONS
ASSUMED FOR BENEFIT-RISK CALCULATION

Pollutant	Geographic Area	Data Reference	Geometric Mean	Standard Deviation of Geometric Mean
Carbon Monoxide	Major U.S. Cities	Los Angeles Survey by Stewart	1.8 percent* Carboxyhemo-globin	1.50
Photochemical Oxidant	Major Urban Centers	CAMP (Colorimetric)	60 $\mu g/m^3$ **	2.22
	S. California (Best judgment)	CHESS (Chemiluminescence)	140 $\mu g/m^3$ **	2.22
	S. California (Worst Case)	CARB (Colorimetric)	240 $\mu g/m^3$ **	2.22

*Equivalent to an average 8-hour carbon monoxide exposure of 13 to 16 mgm/m^3, depending on activity level. Peak 8-hour exposures during the year associated with this distribution range from 48 to 62 mgm/m^3 depending on the activity level.

**Maximum Hourly Values

TABLE 2

PREDICTED CHANGES IN 24-HOUR EXPOSURES TO POLLUTANTS
EMITTED BY LIGHT DUTY MOTOR VEHICLES

(For Persons Living Near and Traversing Major Arterial Throughways and Working in Urban Centers)

Pollutant	Direction of Predicted Change	Predicted Changes from Existing Urban Levels		
		After Two Model Years Are Catalyst Equipped	After Four Model Years Are Catalyst Equipped	After Ten Model Years Are Catalyst Equipped
Carbon Monoxide*	Decreased	Moderate (20%) Decrease	Significant (40%) Decrease	Significant (84%) Decrease
Oxidants**	Decreased	Moderate (30%) Decrease in Hydrocarbon Emissions Leads to Modest (14%) Decrease in Oxidants***	Significant (48%) Decrease in Hydrocarbon Emissions Leads to Moderate (22%) Decrease in Oxidants	Significant (86%) Decrease in Hydrocarbon Emissions Leads to Further (39%) Decrease in Oxidants
Phenols	Decreased	Significant (27%) Decrease	Significant (49%) Decrease	Significant (89%) Decrease
Polynuclear Aromatic* Hydrocarbons	Decreased	Moderate (23%) Decrease	Significant (45%) Decrease	Significant (82%) Decrease
Lead Particulate	Decreased	Significant (25%) Decrease	Significant (70%) Decrease	Significant (up to 95%) Decrease
Particulate Nitrogen**	Little or No Change	Less Than One Per Cent of Present Urban Nitrate Levels		
Particulate Sulfates** and Sulfuric Acid	Increased	Moderate (10 to 25%) Increase	Significant (25 to 60%) Increase	Significant (75 to 200%) Increase
Aluminum and Its Compounds	Increased	Small (2 to 6%) Increase	Modest (4 to 12%) Increase	Modest (10 to 30%) Increase
Platinum and Its Compounds	New Pollutant	Minute (up to .05 nanograms/M^3) Levels Not Measureable	Minute (up to .10 nanograms/M^3) Levels Not Measurable	Exposures First Become Measurable
Oxides of Nitrogen**	No Change	Oxidation Catalyst Should Have Little or No Effect on Oxides of Nitrogen		

*Applies to persons who do not smoke tobacco and are not occupationally exposed to these pollutants.
**In these cases one is dealing with changes in exposures involving large areas.
***This refers to mobile sources only. In 1970 mobile sources contributed about 45% of anthropogenic hydrocarbon emissions, but the proportion varied greatly from city to city.

Table 3
DOSE RESPONSE FUNCTIONS LINKING POLLUTANT EXPOSURES TO SELECTED ADVERSE HEALTH EFFECTS
(Best Judgment)

Pollutant	Adverse Health Effect*	Threshold Concentration of Pollutant and Exposure Duration (X_o)	Characteristic of Dose Response Function		
			Slope	Intercept (Y_o)	Acceptable Upper Limit of Prediction**
Carbon Monoxide	Excess Cardiac Deaths	Two percent carboxyhemoglobin	0.05	-0.1	Ten percent carboxyhemoglobin
Photochemical Oxidants	Aggravation of Heart and Lung Disease in Elderly Patients	540 $\mu g/m^3$ for one hour or longer	0.00017	0.2	$\sim$1400 $\mu g/m^3$
	Agravation of Asthma	540 $\mu g/m^3$ for one hour or longer	0.00017	0.02	$\sim$1400 $\mu g/m^3$
	Eye Discomfort	300 $\mu g/m3$ for one hour or more	0.00032	0.09	
	Cough	540 $\mu g/m^3$ for one hour or more	0.00017	0.125	$\sim$1400 $\mu g/m^3$
	Chest Discomfort	600 $\mu g/m^3$ for one hour or more	0.00010	0.03	$\sim$1400 $\mu g/m^3$
	Headache	100 $\mu g/m^3$ for one hour or more	0.00003	0.105	$\sim$1400 $\mu g/m^3$

TABLE 3 (continued)

Pollutant	Adverse Health Effect*	Threshold Concentration of Pollutant and Exposure Duration	Characteristic of Dose Response Function		
			Slope	Intercept	Acceptable Upper Limit of Prediction**
Particulate Sulfate-Sulfuric Acid	Increase Daily Mortality	25 $\mu g/m^3$ for 24 hours or longer	0.00252	-0.0631	$\sim$60 $\mu g/m^3$
	Aggravation of Heart and Lung Disease in Elderly Patients	9 $\mu g/m^3$ for 24 hours or longer	0.0141	-0.127	$\sim$60 $\mu g/m^3$
	Agravation of Asthma	6-10 $\mu g/m^3$ for 24 hours or longer	0.0335	-0.201	$\sim$35 $\mu g/m^3$
	Excess Acute Lower Respiratory Disease in Children	13 $\mu g/m^3$ for several years	0.0769	-1.000	$\sim$25 $\mu g/m^3$
	Excess Risk for Chronic Bronchitis				
	Non-smokers	10 $\mu g/m^3$ for up to 10 years	0.1340	-1.42	$\sim$30 $\mu g/m^3$
	Cigarette Smokers	15 $\mu g/m^3$ for up to 10 years	0.0738	-1.14	$\sim$30 $\mu g/m^3$

*Plotted as percent excess over base rate for each study in every effects category for CO and sulfates. For oxidants plotted as a "hockey stick" function of the general form

$$Y = Y_o \text{ for } XLX_o.\ Y = Y_o + b\ (X-X_o) \text{ for } X > X_o$$

where X_o, b, and Y_o are the threshold concentration, slope, and intercept respectively as shown above.

**Extrapolations above these limits are less reliable.

Table 4

BRIEF SUMMARY OF POPULATION-AT-RISK AND BASELINE ESTIMATES FOR ADVERSE HEALTH EFFECTS CONSIDERED IN THE BENEFIT-RISK ANALYSIS OF CATALYTIC CONVERTERS

Adverse Health Effect	Pollutant	Assumed Baseline Frequency of the Disorder	Definition of Population-at-Risk	Estimated Number at Risk	Reference
Increased Mortality	Particulate Sulfate-Sulfuric Acid	2137 Deaths per Day	Total 1970 Census population residing in urban areas of 100,000 or larger	83.4×10^6	7
	Carbon Monoxide	Prevalence of One out of 200 Persons per Year	One-sixth of persons suffering myocardial infarctions or sudden coronory death	7×10^4	10
Aggravation of Heart and Lung Disease in Elderly Patients	Photochemical Oxidants	One out of Five Persons with the Disorder Complains of Symptom Aggravation on Any Given Day	The prevalence of chronic heart and lung disease (.27) among those age 65 or older living in urban areas of 100,000 or larger	8.3×10^6	7
	Particulate Sulfate-Sulfuric Acid	Same	Same	Same	7
Aggravation of Asthma	Photochemical Oxidants	One out of 50 Persons Experiences an Attack Each Day or Seven Attacks Each Year for Each Person	The prevalence of asthma in the general population (.03) living in urban areas of 100,000 or larger	2.5×10^6	7
	Particulate Sulfate-Sulfuric Acid	Same	Same	Same	7
Excess Acute Lower Respiratory Disease in Children	Particulate Sulfate-sulfuric Acid	Six out of 100 Children Experience This Illness Each Year	Children from birth through age 13 living in urban areas of 100,000 or larger	22.0×10^6	7
Chronic Bronchitis Symptoms	Particulate Sulfate-Sulfuric Acid	Prevalence of Two Persons per 100 for Non-smokers	Sixty-two percent of the population age 21 or older living in urban areas of 100,000 or larger	31.2×10^6	7
		Prevalence of Ten Persons per 100 for Cigarette Smokers	Thirty-eight percent of the population age 21 or older living in urban areas of 100,000 or larger	19.2×10^6	7
Eye Discomfort	Photochemical Oxidants	Prevalence of Five Persons per 100 per Day	Otherwise healthy population living in urban areas of 100,000 or larger. This excludes persons with asthma or heart and lung disease	72.6×10^6	8,11
Cough	Photochemical Oxidants	Prevalence of Ten Persons per 100 per Day	Same	Same	8,11
Chest Discomfort	Photochemical Oxidants	Prevalence of Two Persons per 100 per Day	Same	Same	8,11
Headache	Photochemical Oxidants	Prevalence of Ten Persons per 100 per Day	Same	Same	8,11

*For the benefit-risk analysis these populations were separately identified for each electric reliability region (7) before national estimates were made.

Table 5

BENEFIT-RISK COMPARISON OF OXIDATION CATALYSTS:
MORTALITY ESTIMATES*

Number of Model Years Equipped**	Geographic Area	Mortality Estimates***					
		Reduction (Benefit) Associated with Decreased Carbon Monoxide Exposures		Increment (Risk) Associated with Sulfate-Sulfuric Acid Exposures Attributable to Use of Oxidation Catalysts		Net Benefit or (Risk) Attributable to Use of Oxidation Catalysts	
		Low	High	Low	High	Low	High
2	Total	~ 462	~1386	197	684	265	702
4	Total	~1047	~3141	435	1540	1004	1601
10	East	960	2880	1095	3641	(135)	(761)
	West****	400	1200	5	170	395	1030
	Total	1360	4080	1100	3811	260	269

*Assumes 1970 particulate sulfate-sulfuric acid exposures not altered by air pollution controls or growth.

**Vehicular assumptions detailed in reference 2.

***Best judgment estimates

****Roughly two thirds of the expected "west" benefit would occur in California

Table 6

BENEFIT-RISK COMPARISON OF OXIDATION CATALYSTS:
AGGRAVATION OF ASTHMA*

Number of Model Years Equipped**	Geographic Area	Estimates of Changes in Frequency in Asthma Attacks*** (thousands)					
		Reduction (Benefit) Associated with Decreased Photochemical Oxidants		Increment (Risk) Associated with Sulfate-Sulfuric Acid Exposures Attributable to Use of Oxidation Catalysts		Net Benefit or (Risk) Attributable to Use of Oxidation Catalysts	
		Low	High	Low	High*****	Low	High
2	Total	less than 24	less than 41	904	904	more than (880)	more than (863)
4	Total	less than 24	less than 41	1875	1875	more than (1851)	more than (1834)
10	East	1	5	2944	2944	(2943)	(2939)
	West	5	12	977	977	(972)	(965)
	Total	6	17	3921	3921	(3915)	(3904)
	West**** (worst case oxidants)	23	36	977	977	(954)	(941)

*Assumes 1970 particulate sulfate-sulfuric acid exposures not altered by air pollution controls or growth.

**Vehicular assumptions detailed in reference (2)
***Best judgment estimates
****Assumes worst case oxidant exposures in Southern California
*****Because of the shape of the dose-response function, it does not matter whether incremental sulfate-sulfuric acid exposures are in phase or reversed

Table 7

BENEFIT-RISK COMPARISON OF OXIDATION CATALYSTS:
AGGRAVATION OF HEART AND LUNG DISORDERS*

Number of Model Years Equipped**	Geographic Area	Days of Aggravation of Heart and Lung Disorders (thousands)***					
		Reduction (Benefit) Associated with Decreased Photochemical Oxidants		Increment (Risk) Associated with Sulfate-Sulfuric Acid Exposures Attributable to Use of Oxidation Catalysts		Net Benefit or (Risk) Attributable to Use of Oxidation Catalysts	
		Low	High	Low	High	Low	High
2	Total	less than 672	less than 1211	2322	2916	more than (1650)	more than (1705)
4	Total	less than 672	less than 1211	5146	6112	more than (4474)	more than (4901)
10	East	70	212	10,617	10,749	(10,547)	(10,537)
	West	187	392	1,892	2,408	(1,705)	(2,016)
	Total	257	604	12,509	13,157	(12,252)	(12,553)
	West**** (Worst Case)	602	999	1,892	2,408	(1,290)	(1,409)

*Assumes 1970 particulate sulfate-surfuric acid exposures not altered by air pollution controls or growth

**Vehicular assumptions detailed in reference (2)
***Best judgment estimates
****Assumes worst case oxidant exposures in Southern California

Table 8

BENEFIT-RISK COMPARISON OF OXIDATION CATALYSTS
EXCESS ACUTE LOWER RESPIRATORY DISEASE IN CHILDREN*

Number of Model Years Equipped**	Geographic Area	Estimates of Change in Frequency of Acute Lower Respiratory Disease (thousands)***					
		Reduction (Benefit) Associated with Decreased Photochemical Oxidants		Increment (Risk) Associated with Sulfate-Sulfuric Acid Exposures Attributable to Use of Oxidation Catalysts		Net Benefit or (Risk) Attributable to Use of Oxidation Catalysts	
		Low	High	Low	High****	Low	High****
2	Total	Benefits not quantified	Benefits not quantified	88	88	(88)	(88)
4	Total			184	184	(184)	(184)
10	East			407	407	(407)	(407)
	West			7	7	(7)	(7)
	Total			414	414	(414)	(414)

*Assumes 1970 particulate sulfate-sulfuric acid exposures not altered by air pollution controls or growth

**Vehicular assumptions detailed in reference (2)
***Best judgment estimates
****Becasue of the dose-response function shape, it does not matter whether incremental sulfate exposures are in phase or reversed.

Table 9

Benefit-Risk Comparison of Oxidation Catalysts:

Chronic Respiratory Disease Symptoms*

Number of Model Years Equipped	Geographic Area	Change in Prevalence of Chronic Respiratory Disease Symptoms (in thousands)***					
		Reduction (Benefit) Associated with Decreased Photochemical Oxidants		Increment (Risk) Associated with Sulfate-Sulfuric Acid Exposures Attributable to Use of Oxidation Catalysts		Net Benefit or (Risk) Attributable to Use of Oxidation Catalysts	
		Low	High	Low	High****	Low	High****
2	Total			205	205	(205)	(205)
4	Total	Benefit Not Quantified	Benefit Not Quantified	424	424	(424)	(424)
10	East			883	883	(883)	(883)
	West			51	51	(51)	(51)
	Total			934	934	(934)	(934)

* Assumes 1970 particulate sulfate-sulfuric acid exposures not altered by air pollution controls or growth.

** Vehicular assumptions detailed in reference (2).

*** Best judgment estimates.

**** Because of dose-response function shape, it does not matter whether incremental sulfate exposures are in-phase or reversed.

Table 10

BENEFIT-RISK COMPARISON OF OXIDATION CATALYSTS:
IRRITATION SYMPTOMS

Irritation Symptom	Number of Model Years Equipped**	Geographic Area	Estimates of change in Irritation Symptom Frequency (thousands)***					
			Reduction (Benefit) Associated with Decreased Photochemical Oxidants		Increment (Risk) Associated with Sulfate-Sulfuric Acid Exposures Attributable to Use of Oxidation Catalysts		Net Benefit or (Risk) Attributable to Use of Oxidation Catalysts	
			Low	High	Low	High	Low	High
Eye Discomfort	10	East	53	79	THIS ANALYSIS ASSUMES NO IRRITATION SYMPTOM INCREASE	THIS ANALYSIS ASSUMES NO IRRITATION SYMPTOM INCREASE	53	79
		West	83	107			83	107
		Total	136	186			136	186
		West (worst case)****	216	471			216	471
Transient Cough	10	East	21	58			21	58
		West	52	107			52	107
		Total	73	165			73	165
		West (worst case)****	182	304			182	304
Chest Discomfort	10	East	0	4			0	4
		West	2	5			2	5
		Total	2	9			2	9
		West (worst case)****	10	18			10	18
Headache	10	East	No Estimate	1520			No Estimate	1520
		West		889				889
		Total		2409				2409
		West (worst case)****		1066				1066

*Assumes 1970 particulate sulfate-sulfuric acid exposures not altered by air pollution controls or growth
**Vehicular assumptions detailed in reference (2)
***Best judgment estimates
****Assumes worst case oxidant exposures in Southern California

Table 11

SUMMARY OF NET BENEFITS OR (RISKS)
ATTRIBUTABLE TO USE OF OXIDATION CATALYSTS

Adverse Health Effect	Unit	Geographic Area	Net Benefit or (Risk Attributable to Equipping All Light Duty Motor Vehicles with Oxidation Catalysts	
			Low	High
Excess Premature Deaths	Thousands of Deaths	East West Total	(0.135) 0.395 0.260	(0.761) 1.030 0.269
Aggravation of Asthma	Thousands of Days with an Attack	East West Total	(2943) (972) (3915)	(2939) (965) (3904)
Aggravation of Heart and Lung Disorders	Thousands of Days Symptoms Worsened	East West Total	(10,547) (1705) (12,252)	(10,537) (2016) (12,553)
Excess Acute Lower Respiratory Disease in Children	Thousands of Illnesses	East West Total	(407) (7) (414)	(407) (7) (414)
Chronic Respiratory Disease Symptoms	Thousands of Individuals Affected	East West Total	(883) (51) (934)	(883) (51) (934)
Irritation Symptoms in Healthy Individuals Other than Headache	Thousands of Excess Symptom Days	East West Total	74 137 211	141 219 360
Headache	Thousands of Excess Headaches	East West Total	Not Estimated	1520 889 2409
Increased Risk of Cancer, Mutations, Accelerated Aging and Other Adverse Effects	Beneficial But Benefits Cannot Yet Be Quantified			

TABLE 12

SUSPENDED WATER SOLUBLE URBAN SULFATE LEVELS
BY ENERGY REGION AND POPULATION CLASS:
IMPACT OF CATALYST SULFATE EMISSIONS
ON 24-HOUR MEDIAN CONCENTRATIONS*,**

Urban Population Class: >2 Million[1]

Electric Power Region[2]	Median Sulfate Levels ($\mu gm/M^3$)(Base)	Incremental Percentage Increase in Sulfate Levels Attributable to Use of Oxidation Catalysts After: 2 years	4 years	10 years
Northeast and Mid Atlantic (NPCC - MAAC)	17.2	15	30	75
East Central (ECAR)	14.1	18	36	92
Mid-America (MAIN)	17.0	15	31	76
Southeastern (SERC)	14.0	19	37	93
Mid-Continental (MARCA)	-	-	-	-
Southwest Including Texas (SWPP - ERCOT)	4.8	54	108	270
Western (WSCC)	4.9	53	106	265

Urban Population Class: 100,000 to 2 Million[3]

Northeast and Mid Atlantic (NPCC - MAAC)	13.3	12	24	60
East Central (ECAR)	10.4	15	31	77
Mid-America (MAIN)	7.3	22	44	110
Southeastern (SERC)	8.2	19	39	97
Mid-Continental (MARCA)	5.8	28	55	138
Southwest Including Texas (SWPP - ERCOT)	5.2	31	61	154
Western (WSCC)	4.3	37	74	186

* Assumes no coversion of power plants to coal.

** Assumes median urban sulfate concentrations (24-hour) will not change from 1970 base levels.

1 Assumes high estimate catalyst sulfate exposure model (carboxyhemoglobin surrogate) for urban centers.

2 See figure A.1 for geographic boundaries of these regions

3 Assumes low estimate catalyst sulfate exposure model (carboxyhemoglobin surrogate) for suburban areas.

Appendix A

Mortality Estimates

FIGURE A.1. REGIONAL ELECTRIC RELIABILITY COUNCILS

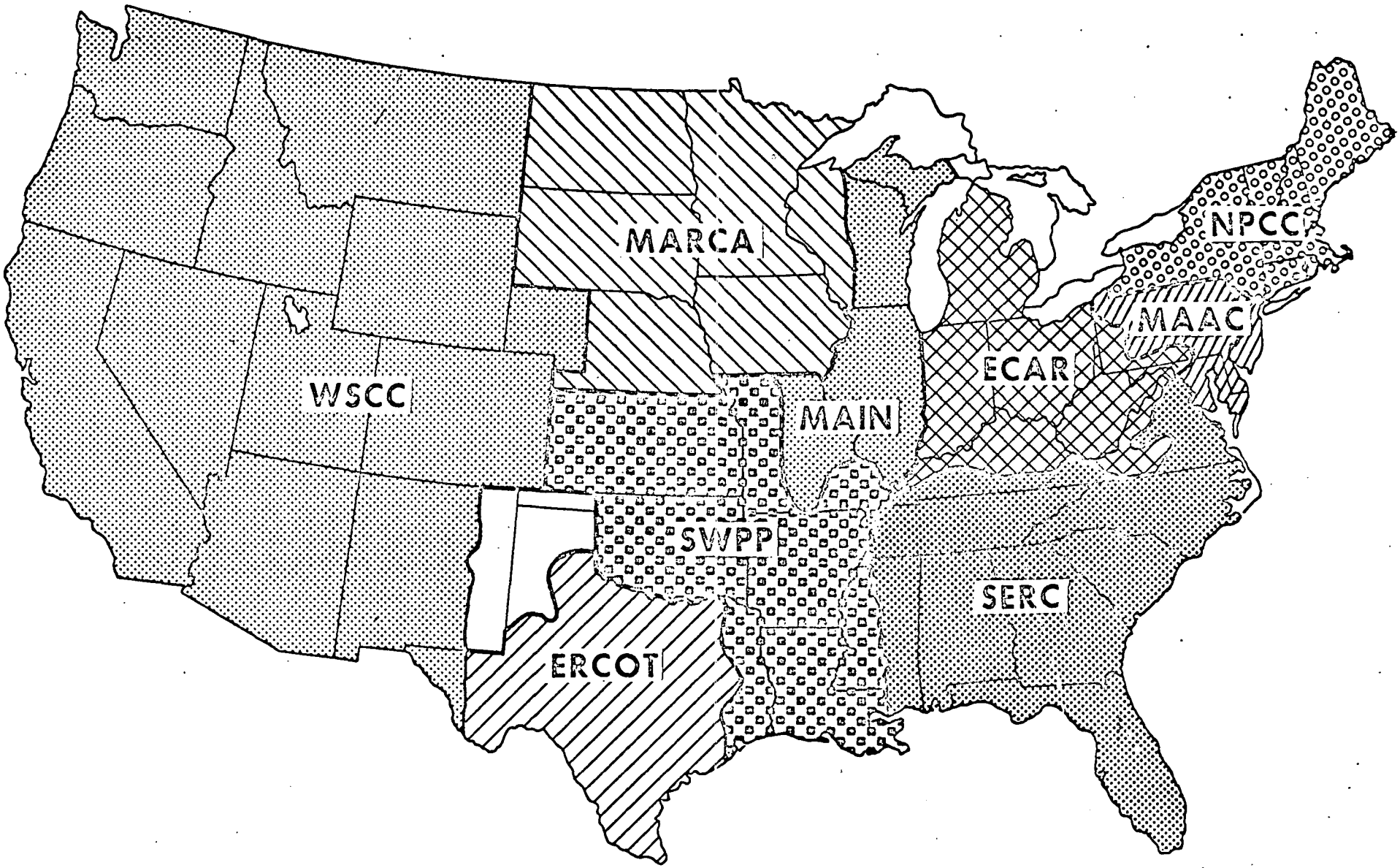

Table A.1. Baseline Estimates of Premature Mortality Attributable to Particulate Sulfate-Sulfuric Acid Exposures Distributed by Electric Power Reliability Regions

Electric Power Reliability Region	Baseline Mortality Attributed to Particulate Sulfate-Sulfuric Acid in 1970*	
	Under Best Judgment Estimate*	Under Least Square Estimates
Northeast Power Coordinating Council (NPCC) and Mid Atlantic Area Coordinating Group (MAAC)	1195	7540
East Central Area Reliability Coordination Agreement (ECAR)	217	1801
Mid America Interpool Network (MAIN)	434	2245
Southeastern Electric Reliability Council (SERC)	134	1527
Mid Continent Area Reliability Coordination Agreement (MARCA)	23	174
Southwest Power Pool (SWPP) and Electric Reliability Council of Texas (ERCOT)	17	341
Western Systems Coordinating Council (WSCC)	2	76
East**	1980	13,113
West**	42	591
Total	2022	13,704

*Best judgment functions are used in the formal benefit risk analysis

**East equals NPCC + MAAC + ECAR + MAIN + SERC
West equals SWPP + ERCOT + MARCA + WSCC

Table A.2. Estimated Mortality Increase Attributable to Carbon Monoxide Emitted from Motor Vehicles by Electric Power Region and Urban Population Exposure Category.

Electric Power Region	Premature Deaths Following Myocardial Infarction (MI)*			
	Immediately After MI		During Recuperative Period	
	Urban > 100,000	All Urban	Urban > 100,000	All Urban
NPCC-MAAC	150	246	250	410
ECAR	69	120	115	200
SERC	69	138	115	230
SWPP-ERCOT	39	90	65	150
MAIN	72	102	120	170
MARCA	9	30	15	50
WSCC	102	180	170	300
East	360	606	600	1010
West	150	300	250	500
Total	510	906	850	1510

*These are lower best judgment estimates. Upper best judgment estimates are at least threefold higher and the upper boundary estimates might be an order of magnitude higher.

Table A.3. Projected Mortality Increase from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	117	258	663	147	323	820
ECAR	19	42	104	26	57	141
SERC	14	31	79	17	38	96
SWPP-ERCOT	0	0	1	2	4	12
MAIN	46	102	249	47	104	253
MARCA	0	1	2	1	4	9
WSCC	1	1	2	1	1	2
East	196	433	1095	237	522	1310
West	1	2	5	4	9	23
Total	197	435	1100	241	531	1333

* Assumes incremental and base exposure distributions are reversed and best judgment dose-response function.

Table A.4. Projected Mortality Increase from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	376	831	1963	513	1144	2754
ECAR	81	189	493	126	298	797
SERC	67	159	431	97	239	695
SWPP-ERCOT	3	10	50	22	61	218
MAIN	147	321	754	155	343	833
MARCA	4	10	30	15	37	108
WSCC	6	20	90	6	22	114
East	671	1500	3641	891	2024	5079
West	13	40	170	43	120	440
Total	684	1540	3811	934	2144	5519

*Assumes incremental and base exposure distributions are in phase and best judgment dose-response function.

Table A.5. Projected Mortality Increase from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	952	2,200	4,920	1,270	2,963	7,294
ECAR	224	546	1,599	343	834	2,707
SERC	194	475	1,451	298	733	2,681
SWPP-ERCOT	10	24	160	76	188	942
MAIN	324	762	1,859	354	836	2,241
MARCA	8	18	79	25	58	226
WSCC	14	32	189	15	36	278
East	1,694	3,983	9,829	2,265	5,366	14,923
West	32	74	428	116	282	1,446
Total	1,726	4,057	10,257	2,381	5,648	16,369

* Assumes incremental and base exposure distributions are reversed and least squares dose-response function.

Table A.6 Projected Mortality Increase from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	1,202	2,450	5,039	1,783	3,675	7,695
ECAR	383	816	1,793	652	1,403	3,129
SERC	354	766	1,728	643	1,422	3,308
SWPP-ERCOT	60	167	523	244	584	1,527
MAIN	458	947	1,998	548	1,156	2,514
MARCA	28	65	167	92	213	545
WSCC	96	261	856	123	361	1,328
East	2,397	4,979	10,558	3,626	7,656	16,646
West	184	493	1,546	459	1,158	3,400
Total	2,581	5,472	12,104	4,085	8,814	20,046

*Assumes incremental and base exposure distributions are in phase and least squares dose-response function.

Appendix B

Excess Asthma Attack Estimates

Table B.1. Baseline Estimates of Excess Asthmatic Attributable to Particulate Sulfate-Sulfuric Acid Exposures Distributed by Electric Power Reliability Region

Electric Power Reliability Region	Baseline Excess Asthmatic Attacks (thousands) Attributed to Particulate Sulfate-Sulfuric Acid in 1970	
	Under Best Judgment Estimate	Under Least Squares Estimate
Northeast Power Coordinating Council (NPCC) and Mid Atlantic Area Coordinating Group (MAAC)	3421	3073
East Central Area Reliability Coordination Agreement (ECAR)	1118	1233
Mid America Interpool Network (MAIN)	1073	1028
Southeastern Electric Reliability Council (SERC)	1209	1469
Mid Continent Area Reliability Coordination Agreement (MARCA	125	188
Southwest Power Pool (SWPP) and Electric Reliability Council of Texas (ERCOT)	377	587
Western Systems Coordinating	136	432
East**	6821	6803
West**	638	1207
Total	7459	8010

*Best judgment estimates used in formal risk-benefit analysis
**East equals NPCC + MAAC + ECAR + MAIN + SERC
West equals SWPP + ERCOT + MARCA + WSCC

Table B.2. Estimated Excess Asthmatic Attacks Attributable to Oxidants Emitted from Motor Vehicles by Electric Power Region and Urban Population Exposure Category.

Electric Power Region	Excess Asthma Attacks (thousands)			
	.27 Threshold*		.20 Threshold*	
	Urban > 100,000	All Urban	Urban > 100,000	All Urban
NPCC-MAAC	1	1	2	3
ECAR	0	0	1	2
SERC	0	1	1	2
SWPP-ERCOT	0	0	1	1
MAIN	0	0	1	1
MARCA	0	0	0	0
WSCC-SC	0	0	1	2
East	1	2	5	8
West-SC	0	0	2	3
Total-SC	1	2	7	11
SC (.07)**	5	6	10	12
SC (.12)**	23	26	36	41

*.27 is low benefit estimate and .20 is high benefit estimates
** Best judgment - Southern California
*** Worst case - Southern California

Table B.3. Projected Asthma Increase in Thousands from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	311	624	1,249	499	1,003	2,015
ECAR	136	276	557	245	498	1,009
SERC	137	280	573	278	568	1,164
SWPP-ERCOT	62	138	313	156	331	710
MAIN	139	280	565	187	378	767
MARCA	16	34	74	51	110	243
WSCC	103	243	590	165	406	1,023
East	723	1,460	2,944	1,209	2,447	4,955
West	181	415	977	372	847	1,976
Total	904	1,875	3,921	1,581	3,294	6,931

*Assumes incremental and base exposure distributions are in phase and best judgment dose-response function. Due to the shape of the dose-response function, there is no change in estimates when incremental exposures are distributed in a reverse fashion, that is opposite to the baseline sulfate distribution.

Table B.4. Projected Asthma Increase in Thousands from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	192	383	766	311	621	1,243
ECAR	87	174	348	158	317	634
SERC	91	182	364	185	371	741
SWPP-ERCOT	58	115	230	121	242	483
MAIN	88	176	350	121	241	480
MARCA	13	26	52	44	88	176
WSCC	122	244	489	218	436	874
East	458	915	1,828	775	1,550	3,098
West	193	385	771	383	766	1,533
Total	651	1,300	2,599	1,158	2,316	4,631

*Assumes incremental and base exposure distributions are in phase and least squares dose-response function. Due to the shape of the dose-response function, there is no change in estimates when incremental exposures are distributed in a reverse fashion, that is opposite to the baseline sulfate distribution.

Appendix C

Estimates of Heart and Lung Disease Symptom Aggravation

Table C.1. Baseline Estimates of Aggravation of Heart and Lung Disorders Attributable to Particulate Sulfate - Sulfuric Acid Exposures bistributed by Electric Power Reliability Region (in thousands)

Electric Power Reliability Region	Baseline Symptom Aggravation Attributed to Particulate Sulfate-sulfuric Acid in 1970	
	Under Best Judgment Estimate	Under Least Squares Estimate
Northeast Power Coordinating Council (NPCC) and Mid Atlantic Area Coordinating Group (MAAC)	10,121	16,064
East Central Area Reliability Coordination Agreement (ECAR)	2,553	5,259
Mid America Interpool Network (MAIN)	3,118	5,187
Southeastern Electric Reliability Council (SERC)	2,509	6,263
Mid Continent Area Reliability Coordination Agreement (MARCA)	321	848
Southwest Power Pool (SWPP) and Electric Reliability Council of Texas (ERCOT)	695	2,290
Western Systems Coordinating Council (WSCC	179	1,107
East**	18,301	32,773
West**	1,195	4,245
Total	19,496	37,018

*Best judgment estimates used in formal risk-benefit analysis
**East equals NPCC + MAAC + ECAR + MAIN + SERC
West equals SWPP + ERCOT + MARCA + WSCC

Table C.3. Projected Increase for Aggravation of Heart and Lung Disease in Thousands from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	1,215	2,446	4,930	1,884	3,822	7,787
ECAR	420	870	1,820	739	1,540	3,249
SERC	414	869	1,857	806	1,710	3,702
SWPP-ERCOT	124	308	815	392	883	2,076
MAIN	512	1,042	2,142	653	1,348	2,823
MARCA	48	107	253	154	345	823
WSCC	183	470	1,340	260	716	2,213
East	2,561	5,227	10,749	4,082	8,420	17,561
West	355	885	2,408	806	1,944	5,109
Total	2,916	6,112	13,157	4,888	10,364	22,670

*Assumes incremental and base exposure distributions are in phase and best judgment dose-response function.

Table C.4. Projected Increase in Aggravation of Heart and Lung Disease in Thousands from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	1,159	2,411	4,917	1,666	3,653	7,720
ECAR	326	780	1,786	534	1,338	3,171
SERC	299	723	1,800	526	1,383	3,577
SWPP-ERCOT	34	101	691	189	553	1,905
MAIN	444	972	2,114	520	1,198	2,765
MARCA	19	51	224	59	151	702
WSCC	41	108	977	50	136	1,555
East	2,228	4,886	10,617	3,246	7,572	17,233
West	94	260	1,892	298	840	4,162
Total	2,322	5,146	12,509	3,544	8,412	21,395

* Assumes incremental and base exposure distributions are reversed and best judgment dose-response function.

Table C.5. Projected Increase for Aggravation of Heart and Lung Disease in Thousands from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	1,164	2,329	4,657	1,888	3,777	7,555
ECAR	467	934	1,868	849	1,699	3,401
SERC	497	996	1,996	1,013	2,029	4,066
SWPP-ERCOT	300	616	1,259	652	1,322	2,674
MAIN	529	1,060	2,121	725	1,452	2,907
MARCA	82	166	337	270	553	1,133
WSCC	530	1,127	2,382	933	2,000	4,245
East	2,657	5,319	10,642	4,475	8,957	17,929
West	912	1,909	3,978	1,855	3,875	8,052
Total	3,569	7,228	14,620	6,330	12,832	25,981

*Assumes incremental and base exposure distributions are in phase and Least squares dose-response function.

Table C.6. Projected Increase in Aggravation of Heart and Lung Disease in Thousands from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	1,164	2,328	4,657	1,887	3,776	7,555
ECAR	466	933	1,868	847	1,698	3,401
SERC	496	995	1,996	1,010	2,028	4,066
SWPP-ERCOT	288	611	1,259	640	1,317	2,674
MAIN	329	1,060	2,121	724	1,452	2,907
MARCA	79	165	337	255	547	1,132
WSCC	473	1,109	2,382	835	1,971	4,244
East	2,655	5,316	10,642	4,468	8,954	17,929
West	840	1,885	3,978	1,730	3,835	8,050
Total	3,495	7,201	14,620	6,198	12,789	25,979

*Assumes incremental and base exposure distributions are reversed and least squares dose-response function.

Appendix D

Estimates of Excess Acute Lower Respiratory Disease

Table D.1

Baseline Estimates of Acute Lower Respiratory Disease in Children Attributable to Particulate Sulfate-Sulfuric Acid Exposures Distributed by Electric Power Reliability Region

Electric Power Reliability Region	Baseline Cases of Acute Lower Respiratory Disease Attributed to Particulate Sulfate-Sulfuric Acid in 1970 (in thousands)	
	Under Best Judgment Estimate*	Under Least Squares Estimate
Northeast Power Coordinating Council (NPCC) and Mid-Atlantic Area Coordinating Group (MAAC)	187	214
East Central Area Reliability Coordination Agreement (ECAR)	25	58
Mid-America Interpool Network (MAIN)	69	68
Southeastern Electric Reliability Council (SERC)	23	36
Mid-Continent Area Reliability Coordination Agreement (MARCA)	0	0
Southwest Power Pool (SWPP) and Electric Reliability Council of Texas (ERCOT)	0	3
Western Systems Coordinating Council (WSCC)	0	0
East**	303	376
West**	0	3
Total	303	379

* Best Judgment Estimates used in formal risk-benefit analysis

** East equals NPCC+MAAC+ECAR+MAIN+SERC

West equals SWPP+ERCOT+MARCA+WSCC

Table D.2

Projected Acute Lower Respiratory Disease Increase in Thousands from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	46	92	184	75	149	299
ECAR	14	36	82	21	61	144
SERC	10	19	62	10	26	117
SWPP-ERCOT	0	0	0	0	0	33
MAIN	18	37	79	18	37	95
MARCA	0	0	3	0	0	7
WSCC	0	0	4	0	0	4
East	88	184	407	124	273	655
West	0	0	7	0	0	44
Total	88	184	414	124	273	699

*Assumes incremental and base exposure distributions are in phase and best judgment dose-response function.

Table D.3

Projected Acute Lower Respiratory Disease Increase In Thousands from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	28	54	109	45	88	177
ECAR	14	27	54	25	49	98
SERC	14	28	55	28	56	111
SWPP-ERCOT	0	0	18	10	19	57
MAIN	13	26	52	18	36	72
MARCA	0	2	6	0	6	20
WSCC	0	6	37	0	6	47
East	69	135	270	116	229	458
West	0	8	61	10	31	124
Total	69	143	331	126	260	582

*Assumes incremental and base exposure distributions are in phase and least squares dose-response function.

Appendix E

Estimates of Excess Chronic Respiratory Disease

Table E.1

Baseline Estimates of Chronic Respiratory Disease (CRD) Symptoms Attributable to Particulate Sulfate-Sulfuric Acid Exposures Distributed by Electric Power Reliability Region

Electric Power Reliability Region	Baseline Estimates of CRD Attributed to Particulate Sulfate-Sulfuric Acid in 1970			
	Under Best Judgment Estimate*		Under Least Squares Estimate	
	Non-Smokers	Cigarette Smokers	Non-Smokers	Cigarette Smokers
Northeast Power Coordinating Council (NPCC) and Mid-Atlantic Area Coordinating Group (MAAC)	265	149	339	150
East Central Area Reliability Coordination Agreement (ECAR)	51	7	79	5
Mid-America Interpool Network (MAIN)	79	56	98	57
Southeastern Electric Reliability Council (SERC)	31	11	49	10
Mid-Continent Area Reliability Coordination Agreement (MARCA)	0	0	0	0
Southwest Power Pool (SWPP) and Electric Reliability Council of Texas (ERCOT)	0	0	2	0
Western Systems Coordinating Council (WSCC)	0	0	0	0
East**	426	223	565	222
West**	0	0	2	0
Total	426	223	567	222

* Best Judgment Estimates used in formal risk-benefit analysis

** East equals NPCC+MAAC+ECAR+MAIN+SERC

West equals SWPP+ERCOT+MARCA+WSCC

Table E.2

Projected Chronic Respiratory Disease Increase in Thousands for Nonsmokers from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	41	81	163	66	132	264
ECAR	17	35	70	31	64	128
SERC	11	29	65	27	64	139
SWPP-ERCOT	0	0	14	6	19	59
MAIN	15	33	69	18	43	93
MARCA	0	1	6	0	1	19
WSCC	0	0	31	0	0	31
East	84	178	367	142	303	624
West	0	1	51	6	20	109
Total	84	179	418	148	323	733

*Assumes incremental and base exposure distributions are in phase and best judgment dose-response function.

Table E.3

Projected Chronic Respiratory Disease Increase in Thousands for Nonsmokers from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	45	90	180	73	146	292
ECAR	19	39	78	35	71	142
SERC	19	40	81	40	83	166
SWPP-ERCOT	0	0	24	14	28	80
MAIN	19	38	78	26	53	108
MARCA	0	3	9	0	8	28
WSCC	0	6	52	0	6	63
East	102	207	417	174	353	708
West	0	9	85	14	42	171
Total	102	216	502	188	395	879

*Assumes incremental and base exposure distributions are in phase and least squares dose-response function.

Table E.4

Projected Chronic Respiratory Disease Increase In Thousands for Smokers from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	66	134	272	73	184	407
ECAR	17	33	87	17	33	132
SERC	13	27	57	13	27	83
SWPP-ERCOT	0	0	0	0	0	10
MAIN	25	51	102	25	51	107
MARCA	0	0	0	0	0	0
WSCC	0	0	0	0	0	0
East	121	245	516	128	295	729
West	0	0	0	0	0	10
Total	121	245	516	128	295	739

*Assumes incremental and base exposure distributions are in phase and best judgment dose-response function.

Table E.5

Projected Chronic Respiratory Disease Increase In Thousands for Smokers from Sulfate Aerosols Emitted from Motor Vehicles by Electric Power Region, Motor Vehicle Model Years, and Urban Population Exposure Category.*

Electric Power Region	Urban > 100,000			All Urban (towns > 2,500)		
	Model Years			Model Years		
	2	4	10	2	4	10
NPCC-MAAC	69	142	288	70	189	426
ECAR	18	35	91	18	35	136
SERC	14	28	58	14	28	81
SWPP-ERCOT	0	0	0	0	0	8
MAIN	27	54	108	27	54	112
MARCA	0	0	0	0	0	0
WSCC	0	0	0	0	0	0
East	128	259	545	129	306	755
West	0	0	0	0	0	8
Total	128	259	545	129	306	763

*Assumes incremental and base exposure distributions are in phase and least squares dose-response function.

Appendix F

Estimates of Irritation Symptom Frequency

Table F.1

Estimated Adverse Health Effects (000) Attributable to Oxidants Emitted from Motor Vehicles by Electric Power Region and Urban Population Exposure Category.

Electric Power Region	Eye Discomfort				Cough			
	.15 Threshold*		.13 Threshold**		.27 Threshold*		.20 Threshold	
	Urban > 100,000	All Urban	Urban > 100,000	All Urban	Urban > 100,000	All Urban	Urban > 100,000	All Urba
NPCC-MAAC	23	37	34	55	8	13	25	40
ECAR	10	18	15	27	4	7	11	20
SERC	10	21	15	31	4	8	11	22
SWPP-ERCOT	6	13	9	19	2	4	7	14
MAIN	10	14	15	20	5	6	11	15
MARCA	2	6	2	7	1	2	2	6
WSCC-SC	6	15	9	22	2	5	6	16
East	53	90	79	133	21	34	58	97
West-SC	14	34	20	48	5	11	15	36
Total-SC	67	124	99	181	26	45	73	133
SC (.07)***	69	79	87	100	47	54	92	106
SC (.12)****	208	231	235	269	177	203	289	331

* low benefit estimate

** high benefit estimate

*** best judgment - Southern California

**** worst use - Southern California