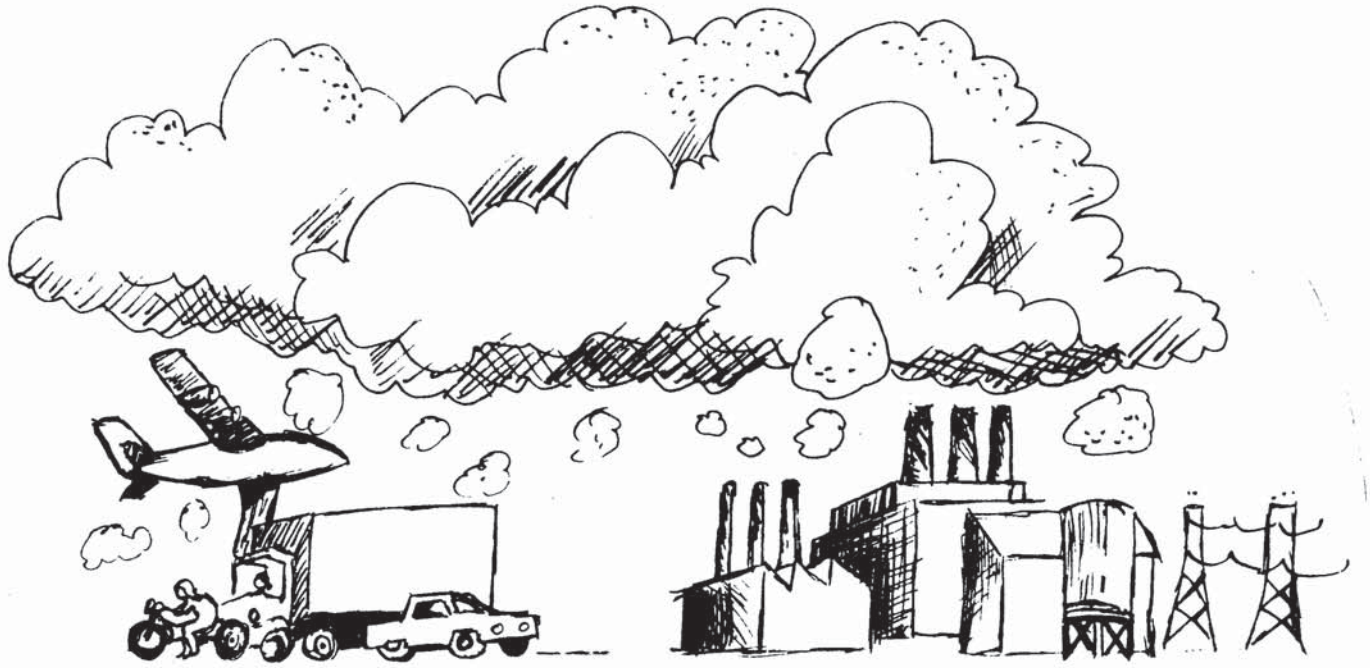
THE MEASUREMENT OF AIR QUALITY IN THE COMMONWEALTH



Research Report No. 179

LEGISLATIVE RESEARCH COMMISSION
Frankfort, Kentucky

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THE MEASUREMENT OF AIR QUALITY IN THE COMMONWEALTH

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Research Report No. 179

Legislative Research Commission
Frankfort, Kentucky
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FOREWORD

Concern has been voiced in several areas regarding the methods and procedures used to designate "non-attainment" areas in Kentucky for meeting national ambient air quality standards. This concern has been most pronounced for the air pollutant ozone. The 1980 House of Representatives, by passage of House Resolution 154, and the 1980 Senate, by passage of Senate Resolution 83, requested the Legislative Research Commission to conduct a study by which air quality in metropolitan areas is measured. The present report is in response to that request.

We thank the staff of the Division of Air Pollution Control in the Department for Natural Resources and Environmental Protection for their assistance in the course of this study. We especially want to thank Mr. Greg H. Lazarus of the National Conference of State Legislatures for his invaluable assistance and cooperation in conducting the study. We also appreciate the many other suggestions and ideas provided the staff by local officials concerned about air quality.

This report was prepared by Charles Hardin with the assistance of Peggy Hyland and was edited by Dr. Charles Bush.

It should be noted that Section 323 of the 1977 federal Clean Air Act directed the establishment of the National Commission on Air Quality to evaluate the Act and examine alternative methods of achieving its public health and welfare goals. This Commission has completed its evaluation and submitted a report with many recommendations which may have significant impact on the Act. However, this report was not available at the time of this writing.

VIC HELLARD, JR.
Director

The Capitol
Frankfort, Kentucky
April, 1981

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SUMMARY

In 1977 Congress made significant amendments to the Clean Air Act. These amendments required states to revise their State Implementation Plans and explain how they would meet the requirements of the federal Act. The federal Act specified dates by which programs established to meet the provisions of the Act must be in place. The Act required that areas which will not meet the ambient air standards for photochemical oxidants or carbon monoxide by December 31, 1982, must implement inspection and maintenance programs to control automobile emissions. The Act also provided for certain sanctions which could be brought against a state for not having an approved plan by the specific dates.

Kentucky's Air Pollution Program

The Commonwealth of Kentucky addressed the subject of air pollution as early as 1952, by the passage of legislation by the General Assembly to allow for the establishment of local air pollution control districts. Several other pieces of legislation relating to air quality were passed by later sessions of the General Assembly.

The Department for Natural Resources and Environmental Protection (DNREP) has implemented a very comprehensive air pollution program to control the quality of air in Kentucky. Additionally, under approval of DNREP, Jefferson County/Louisville also maintains a comprehensive local air pollution program. Both programs are periodically reviewed for compliance by the U.S. EPA with federal standards and guidelines.

For air quality control, the Commonwealth has been divided into nine Air Quality Control Regions, some of which include counties from other states. Although many regions have similar air pollution problems, some problems are common to only a few counties or areas within a region. As required by the federal Clean Air Act, areas which do not meet ambient air quality standards have been identified as "non-attainment" areas. Such areas require specific plans and strategies to reach the goal of attainment.

Monitoring and Reporting

The surveillance and monitoring of the atmosphere for air pollutants is difficult and complex. The monitoring program established by DNREP is a comprehensive one, which has in the past received approval from the U.S. EPA in meeting their criteria for sampling, measurement, analysis, and reporting.

Ozone Formation and Dispersion

Although many "unknowns" about the formation and dispersion of ozone in the atmosphere remain, much has been learned over the last ten years. The formation of ozone involves complex chemical reactions, which in nature follow a cyclical pattern. Human-made precursors, such as hydrocarbons, produce an imbalance in the cycle, which leads to an increase in the formation of ozone.

The dispersion and transport of ozone is dependent upon many meteorological factors, such as air stability, wind speed and wind direction. Ozone, or the precursors to ozone formation, can

travel many miles from the source of origin. Although much has been learned over the last ten years about the dispersion of ozone, more research is needed to fully understand the transport of this air pollutant.

The major human-made sources of ozone precursors are fuel-related stationary sources and transportion-related sources. Because these types of sources are concentrated in metropolitan areas, the level of ozone is usually higher in these metropolitan areas. There is but limited information available regarding unusual sources of ozone, such as ozone production from street lights. Some studies have observed ozone formation from the coronal discharges around high voltage transmission lines, but most investigations have indicated this source contributes little, if any, ozone to the atmosphere.

Motor Vehicle Inspection and Maintenance

The issue of mandatory automobile inspection and maintenance (I/M) has been the subject of much debate. Two metropolitan areas in Kentucky which have been designated "non-attainment" for ozone and/or carbon monoxide, are required by the Clean Air Act to implement some type of mandatory I/M program. These areas are Jefferson County/Louisville and the three counties of Boone, Campbell, and Kenton in northern Kentucky.

Unless state or local authorization is provided for the establishment of these I/M programs, these areas of Kentucky would be placed under sanction by EPA, and thus would stand to lose significant funds and/or potential for industrial growth. The Kentucky General Assembly considered legislation for state I/M programs in 1978 and 1980 but passed none. In the absence of state action in this area, the counties of Jefferson and Boone have adopted local ordinances providing legal authorization for the establishment of I/M programs in their counties. No such ordinances have been adopted by the other two Kentucky counties involved—Kenton and Campbell—and as a result, the U.S. EPA on December 12, 1981, announced the institution of sanctions on these counties. The three northern counties have petitioned the federal court to review this federal sanction.

One of the reasons the northern Kentucky counties have been reluctant to adopt local ordinances for I/M is that a technical advisor hired by the counties has raised questions regarding the data which led to "non-attainment." These questions were addressed by the U.S. EPA and by an independent source upon request by LRC staff. Both of these sources concluded that the data by which the area was designated non-attainment was accurate.

Other State I/M Programs

Of the twenty-eight states and Washington D. C. which are required to comply with I/M provisions of the Clean Air Act, all but California and the northern Kentucky counties of Kenton and Campbell have authorized such programs. Various state or local governments which have implemented I/M programs have generally shown that I/M programs have been effective in reducing carbon monoxide and hydrocarbon emissions at the tailpipes of automobiles. The correlation of this tailpipe pollutant reduction to ozone reduction in the atmosphere has been difficult to show. This lack of correlation is due to complex factors of ozone formation and transport.

Capital and operating costs for I/M programs vary from program to program. A key to the questions of cost-effectiveness is the "cost" in terms of human health and property if an I/M program is not implemented.

CHAPTER I

INTRODUCTION

The 1980 House of Representatives passed House Resolution 154, and the Senate passed an identical resolution, Senate Resolution 83, which requested the Legislative Research Commission to conduct a study of the systems by which air quality in metropolitan areas is measured. This report has been prepared in response to the request made by those resolutions (Appendices A and B).

Before discussing the details of methods of measurements, and other specific items identified by HR 154 and SR 83, we should consider the background of air pollution problems and the laws which have been enacted to address these problems. Congress has enacted several laws which have addressed the air pollution problems in the United States. These include the "Clean Air Act of 1963" (PL 88-206), amendments made by the "Motor Vehicle Air Pollution Control Act" (PL 89-272), the "Clean Air Act Amendments of 1966" (PL 89-675), the "Air Quality Act of 1967" (PL 90-148), the "Clean Air Amendments of 1970" (PL 91-604), the "Comprehensive Health Manpower Training Act of 1971" (PL 92-157), the "Energy Supply and Environmental Coordination Act of 1974" (PL 93-319), the "Clean Air Act Amendments of 1977" (PL 95-95), the "Safe Drinking Water Act of 1977" (PL 95-190), and the "Health Research, Health Statistics, and Health Care Technology Act of 1978" (PL 95-623).

The present report is concerned primarily with the "Clean Air Act Amendments of 1977" (PL 95-95). The 1970 Act required states to develop State Implementation Plans (SIP's) to implement, maintain, and enforce National Ambient Air Quality Standards (NAAQS) within each air quality control region (AQCR) within the state. Such state SIP's were to be approved by the EPA Administrator by May 31, 1975 (or as late as May 31, 1977, in cases where certain conditions for an extension had been met).

The development of the SIP's was a staggering job imposed on state agencies. Moreover, time deadlines were extremely tight. The many problems of meeting these stringent requirements, despite significant improvements in air quality, meant that much of the country failed to meet the May 13, 1975 deadline.

In August of 1977, Congress made several changes to the Act. These 1977 Amendments set a deadline of December 13, 1982, for meeting all primary national ambient air quality standards. The deadline may be extended until December 31, 1987, under certain conditions, for achievements of NAAQS for two pollutants linked closely to automobile emissions—carbon monoxide and ozone. The 1977 Amendments directed the states to submit to EPA by January 1, 1979, revised SIP's for achieving the requirements of the Act for each area within a state designated as not meeting a NAAQS. Such areas are designated as "non-attainment areas."

The State Implementation Plan consists of a description of air quality in each region (air quality control region, as identified by Section 107 of the Act), an inventory of emission sources, emission limitations and compliance schedules for each source, a permit program for review of new source construction, transportation control strategies, monitoring, reporting and enforcement procedures, and other measures which, when approved by EPA, constitute a binding agreement for federal—state air pollution control efforts.

States which fail to attain approval of their SIP's by the submission deadlines are subject to the imposition of severe sanctions. Specifically, Section 176(a) of the Act provides for EPA to withhold funds for any projects authorized by the "Clean Air Act" or any highway projects authorized by Title 23, U.S. Code; Section 316(b) of the Act provides for EPA to withhold. impose conditions upon, or restrict the making of any grant for the construction of sewage treatment works; and Section 110(a)(2)(1) of the Act provides for EPA to withhold permits for the construction or modification of major stationary sources proposed in any non-attainment area.

The minimal goals to be attained by the states SIP's are meeting the NAAQS. There are two categories of NAAQS, primary and secondary standards. National *primary* ambient air quality standards define levels of air quality which the Administrator of EPA judges to be necessary, with adequate margin of safety, to protect the public health. National *secondary* ambient air quality standards define levels of air quality which the Administrator judges necessary to protect the public from any known or anticipated adverse effects of a pollutant. Table 1 gives a summary of the NAAQS.

These national standards were adopted to provide protection of the public health and the environment from the identified air pollutants. The development of the standards was based on specific health and other considerations identified in criteria documents issued by the National Air Pollution Control Administration. A summary of the effects of air pollution on human health and the environment is presented in Appendices C through I.

EPA is required under the 1977 Clean Air Act to review all existing air quality standards and criteria by the end of 1980, and within 5-year intervals thereafter, and revise the standards as appropriate. EPA has revised the ozone standard and has issued draft criteria documents for other criteria pollutants (e.g., sulfur oxides, particulates, nitrogen oxides and carbon monoxide). It should be noted that a Clean Air Scientific Advisory Board, an independent body of scientists and other experts from outside EPA, was formed as a result of the 1977 Act.. This group reviews all draft and final EPA criteria documents for scientific accuracy and consistency.

Table 1

Summary of National Ambient Air Quality Standards

Air Pollutant and Averaging Time	Primary Standard ug/m^3	Primary Standard ppm	Secondary Standard ug/m^3	Secondary Standard ppm
Sulfur Oxides (measured as sulfur dioxide)				
Annual arithmetic mean	80	0.03		
maximum 24-hour Concentration (a)	365	0.14		
maximum 3-hour Concentration (a)	--	--	1,300	0.05
Particulate Matter				
Annual arithmetic mean	75	--	60(b)	--
Maximum 24-hour Concentration (a)	260	--	150	--
Carbon Monoxide				
Maximum 8-hour Concentration (a)	10,000	9.0	Same as primary	
Maximum 1-hour Concentration (a)	40,000	35.0	Same as primary	
Ozone (measured as photochemical oxidants)				
1-hour Concentration (c)	235	0.12	Same as primary	
Hydrocarbons (corrected for methane) (d)				
Maximum 3-hour Concentration (6 to 9 a.m.) (a)	160	0.24	Same as primary	
Nitrogen Dioxide				
Annual arithmetic mean	100	0.05	Same as primary	
Lead & Compounds (measured as elemental lead)				
Arithmetic mean average over a calendar quarter	1.5	--	Same as primary	

(a) Not to be exceeded more than once per year.

(b) As a guide to be used in assessing implementation plans to achieve the 24-hour standard.

(c) The standard is attained when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 part per million (235 ug/m^3) is equal to or less than 1.

(d) The hydrocarbons standard is for use as a guide is devising implementation plans to achieve oxidant standards.

Source: Code of Federal Regulations, Title 40, Part 50.

CHAPTER II

KENTUCKY'S AIR POLLUTION PROGRAM

The first significant action taken by the Commonwealth of Kentucky to address air pollution was the enactment of Senate Bill 147 (KRS Chapter 77) by the 1952 General Assembly. This Act authorized the establishment of air pollution control districts to be activated by any county or groups of counties.

The 1966 General Assembly enacted very comprehensive legislation relating to air pollution, by the passage of House Bill 259 (KRS Chapter 224), creating a state air pollution control commission with broad authority to develop and carry out a comprehensive program to abate, prevent, and control air pollution. The federal Clean Air Act (as amended) imposed certain legal requirements which rendered the 1966 Air Pollution Control Law deficient. These deficiencies were remedied by amendments by the General Assembly in 1972, 1974, and 1978.

In addition to the legislative authority granted by KRS Chapter 224 to the Kentucky Department for Natural Resources and Environmental Protection (DNREP) to control air pollution, KRS 224.450 provides authority to DNREP to grant concurrent jursidiction to a local air pollution control district, provided such local districts have adopted standards and procedures and have the necessary staff to implement an air pollution program consistent with the objectives of KRS Chapter 224.

Chapter 77 of the Kentucky Revised Statutes provides authority for counties, and cities of the first and second class, by proper ordinances, to implement and control air pollution programs in their jurisdiction. The statute provides for the establishment of an air pollution control board as the governing body of an air pollution control district (KRS 77.115).

In July 1952, Jefferson County/Louisville established the Jefferson County/Louisville Air Pollution Control District, with a governing air pollution board. The Jefferson County/Louisville Air Pollution Control District has been granted authority by DNREP to implement certain portions of the state Plan (See Appendix J).

The federal "Air Quality Act" of 1967 required the establishment of air quality control regions. Kentucky's first air quality control region in the Cincinnati area was designated in May of 1969. In 1971, the federal EPA designated nine air quality control regions (AQCR) in Kentucky. These nine regions covered all 120 Kentucky counties. Four of the regions are completely within Kentucky: South Central, Appalachian, Bluegrass, and North Central.

The other five Regions are *interstate*, each including a portion of other states. The counties for these interstate regions, including other state counties are shown below:

1. Evansville—Owensboro—Henderson Interstate AQCR
   (Ky.—7 counties, Ind.—8 counties)
   Kentucky: Daviess, Hancock, Henderson, McLean, Ohio, Union, and Webster
   Indiana: Dubois, Gibson, Perry, Pike, Posey, Spencer, Vanderburg, and Warrick

2. Huntington—Ashland—Portsmouth—Ironton Interstate AQCR
(Ky.—15 counties, Ohio—5 counties, West Virginia—3 counties)
Kentucky: Bath, Boyd, Bracken, Carter, Elliott, Fleming, Greenup, Lawrence, Lewis, Mason, Menifee, Montgomery, Morgan, Robertson, and Rowan
Ohio: Adams, Brown, Gallia, Lawrence, and Scioto
West Virginia: Cabell, Mason, and Wayne
3. Louisville Interstate AQCR
(Ky.—1 county, Ind.—2 counties)
Kentucky: Jefferson
Indiana: Clark and Floyd
4. Metropolitan Cincinnati Interstate AQCR
(Ky.—8 counties, Indiana—2 counties, Ohio—4 counties)
Kentucky: Boone, Campbell, Carroll, Gallatin, Grant, Kenton, Owen, Pendleton
Indiana: Dearborn and Ohio
Ohio: Butler, Clermont, Hamilton, and Warren
5. Paducah—Cairo Interstate AQCR
(Ky.—17 counties, Ill.—6 counties)
Kentucky: Ballard, Caldwell, Calloway, Carlisle, Christian, Crittenden, Fulton, Graves, Hickman, Hopkins, Livingston, Lyon, Marshall, McCracken, Muhlenberg, Todd, and Trigg
Illinois: Alexander, Johnson, Massac, Pope, Pulaski, and Union.

Figure 1 is a map showing each of the nine Kentucky Air Quality Control Regions.

The 1970 Act required states to develop SIP's to include provisions for the following:

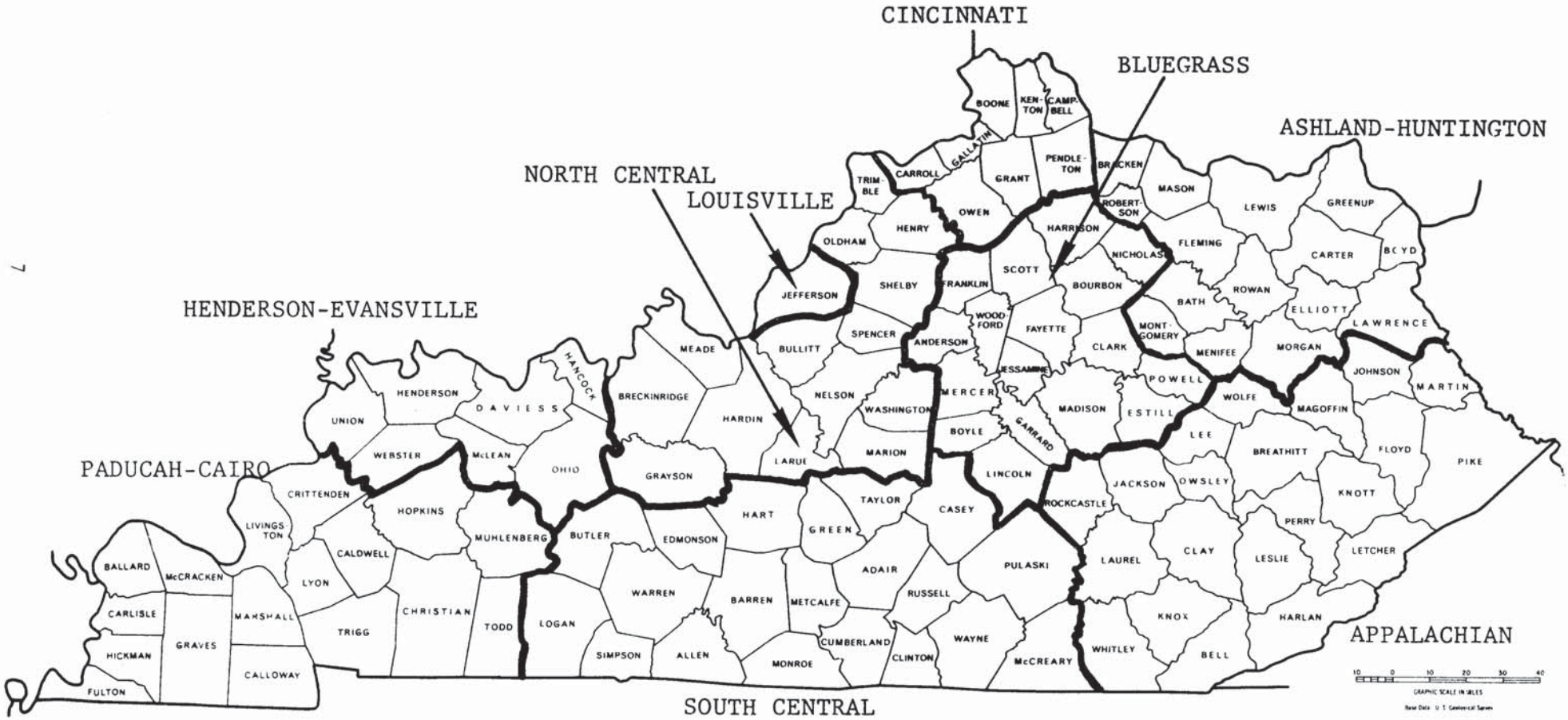
1. A *control strategy*, setting forth a combination of pollution reduction measures, such as stack emission limitations, restrictions on sulfur content in fuels, and transportation control plans;
2. *Legal authority* to adopt and enforce all applicable requirements;
3. *Compliance schedules*, establishing timetables for installation of control measures by individual sources;
4. *Preconstruction review of new sources*, to prevent the construction or modification of stationary sources that would interfere with attainment or maintenance of NAAQS;
5. *Emergency episode procedures*;
6. *Surveillance system*;
7. A description of the needed personnel and financial resources; and
8. A plan for *interstate cooperation*.

The Commonwealth adopted and submitted its first SIP to the federal EPA in February of 1972, which was approved in May 1972. Further revision was made to the 1972 plan, which was approved in 1974, with the exception of a Kentucky regulation relating to Alternate Control Strategy provisions. The SIP was again revised in 1976.

As previously indicated, the 1977 amendments required each state to revise its SIP and to specifically identify *non-attainment* areas in the revised plan. On December 1, 1977, the DNREP submitted to the federal EPA a list of non-attainment, attainment and unclassifiable areas in the Commonwealth. The determination of the designations by DNREP was based on air quality data obtained by DNREP for the four quarters covering the period October 1, 1976, through September 30, 1977, and the four quarters covering the period October 1, 1975, through September 30, 1976.

FIGURE 1

MAP OF KENTUCKY
Air Quality Control Regions



SOURCE: State Implementation Plan - 1979, KY Department for Natural Resources & Environmental Protection

The criteria used in identifying the attainment status were:

1. Areas where the SIP revision was needed.
2. Any area with measured violations.
3. Areas in which technical models showed violation due to inadequate emission standards in SIP.
4. Areas where a major source was not presently in compliance with an emission standard.
5. Areas where violation was due exclusively to rural fugitive dust were identified as "attainment."
6. Areas where OAQPS Guidelines No. 1.2—015 applied. That is, each monitoring site within the designated area had to meet the standards or the area was considered non-attainment for the specific pollutant.[1]

Procedures that were followed for each pollutant were as follows:

Total Suspended Solids and Sulfur Dioxide

Measured data, as well as modeling data, wherever available, were examined. If the measured data showed attainment but modeling data, using 1976 SIP emission standards, showed non-attainment, that area was identified as non-attainment. If a major source was not in compliance with 1976 emission standards (even though it may have been on a compliance schedule) the area in which it was located was identified as non-attainment. Areas with no monitoring data but expected to have no violations were classified attainment.

If violation of the standards was due to rural fugitive dust and there were no major sources impacting on the monitor, the area was classified as attaining the standard.

Photochemical Oxidants

The Department stated that it fully understood the argument that the oxidant problem is a transport problem and that large urban areas are repsonsible. The issue of considering the entire State as non-attainment area was examined. Several factors were analyzed: (1) the type and number of sources in the rural counties, (2) the motor vehicle emission concentration in the State, (3) the resulting return of applying a full control strategy to rural counties, (4) wind flow patterns and effects of some areas on others due to transport, and (5) resource allocation vs. net return.

The Department came to the conclusion that only those counties which had shown violation of the ambient standard should be designated non-attainment areas. These include nine counties, four of which were identified for SIP revision in 1976 by the U.S. EPA.

Carbon Monoxide

Whenever a violation was measured in a downtown area of an urban county the entire county was identified as "non-attainment." Those counties with no measured violation of air quality were identified as "attainment." Such

determination was based on recent data rather than using two-year old data. The rationale is that the percentage of controlled auto emission was higher in 1977 than in 1975, so it was assumed that the improvement in air quality is genuine rather than due to favorable meteorological conditions.

Nitrogen Dioxide

No area within the State where measured data was available was violating the ambient standard; therefore, these areas were identified as "unknown/attainment," as they were not expected to be in violation.[2]

Non-attainment areas in the Commonwealth were identified by the procedures above.[3] A summary of the non-attainment areas, as shown in the 1979 SIP, is presented in Table 2. Figures 2, 3, and 4 show non-attainment areas of Kentucky, by pollutants.

It should be noted that since the completion of the 1979 plan, Kentucky has requested EPA to make changes in the non-attainment designations as follows:

Daviess, McCracken: ozone to attainment (approved by EPA)
Greenup: SO_2 to attainment (approved by EPA)
City of Henderson: SO_2 to attainment (proposed for approval)
City of Owensboro: SO_2 to attainment (approved by EPA)
Boyd County (upper half): SO_2 to attainment (approved by EPA)
Henderson County: ozone to attainment (proposed for approval)

DNREP is authorized, under KRS 224.033, to adopt regulations to protect the air quality in Kentucky. Under the authority of this statute, the Department has adopted comprehensive regulations to address air quality in the Commonwealth. These regulations, as published in the 1980 *Kentucky Administrative Regulations Service*, include Title 401, Chapters 50, 51, 53, 55, 57, 59, 61, 63, and 65. The Ambient Air Quality Standards adopted by DNREP are shown in Table 3. They parallel the national standards for sulfur oxides, particulate matter, carbon monoxide, ozone, hydrocarbons, nitrogen dioxide and lead. However, the Kentucky standards also include hydrogen sulfide, gaseous fluorides, total fluorides, and certain odors, which are not in the national standard.

The Department's basic approach to assuring air quality in Kentucky is contained in the concept of air resource management. Air resource management is a systems approach to maximum utilization of the atmosphere without causing adverse effects on potential receptors (people, plants, other animals and property). It begins with a determination of existing conditions on a regional basis—air quality, meteorological patterns, and an inventory of emissions. Next, goals or objectives for an air resource management program are selected. These constitute the air quality standards. Finally, a control strategy is designed which will lead from existing conditions to the desired goal. Where the existing conditions need to be improved to meet the goal, appropriate measures must be provided. These measures may include the establishment of emission standards which limit the poundage production of air pollutants from such sources as steel mills, automobiles, or burning trash. Such emission standards are found in various Chapters of Title 401 of the Kentucky Administrative Regulations.

To obtain NAAQS by the specified dates, the DNREP has developed various control strategies. An emission control strategy consists of a set of emissions standards for each pollutant for specific types of operations, e.g., fuel combustion, industrial process, and solid waste disposal; the

combination of the several standards included within each strategy defines the allowable emission levels from each source in an air quality control region or a county within the state.

Each designated non-attainment area was examined to determine if the emission limitations were sufficient to attain the primary NAAQS by the date specified in the 1977 Amendments. Whenever those emission limitations were not satisfactory or when anticipated growth would prevent attainment by the end of 1982, the DNREP determined those source categories for which additional control would be necessary and the extent of such control. In order to make this determination, comprehensive point and area source emission inventories were developed for the base year and the projection years of 1982 and 1987. These emissions data were input to an air quality dispersion model or used in linear rollback calculations to provide a basis for determining the appropriate control strategy for each area.

The strategies to be used to meet the standard are based on a *design value*. The design value is determined from the ambient air concentrations which, according to EPA guidelines, would result in the most stringent control requirements.

TABLE 2
SUMMARY OF NON-ATTAINMENT AREAS
IN THE COMMONWEALTH OF KENTUCKY

AREA	POLLUTANT
Bell County	TSP
Boone County	O_x
Boyd County	TSP, SO_2, O_x
That portion of Bullitt County in Shepherdsville	TSP
Campbell County	O_x
That portion of Capmbell County in Newport	TSP
Daviess County	O_x
That portion of Daviess County in Owensboro	TSP, SO_2
Fayette County	O_x
Greenup County	SO_2
Henderson County	O_x
That portion of Henderson County in Henderson	TSP, SO_2
Jefferson County	TSP, SO_2, O_x, CO
Kenton County	O_x
That portion of Lawrence County in Louisa	TSP
McCracken County	TSP, SO_2, O_x
That portion of Madison County in Richmond	TSP
Marshall County	TSP
Muhlenberg County	TSP, SO_2
That portion of Perry County in Hazard	TSP
That portion of Pike County in Pikeville	TSP
Webster County	SO_2
That portion of Whitley County in Corbin	TSP

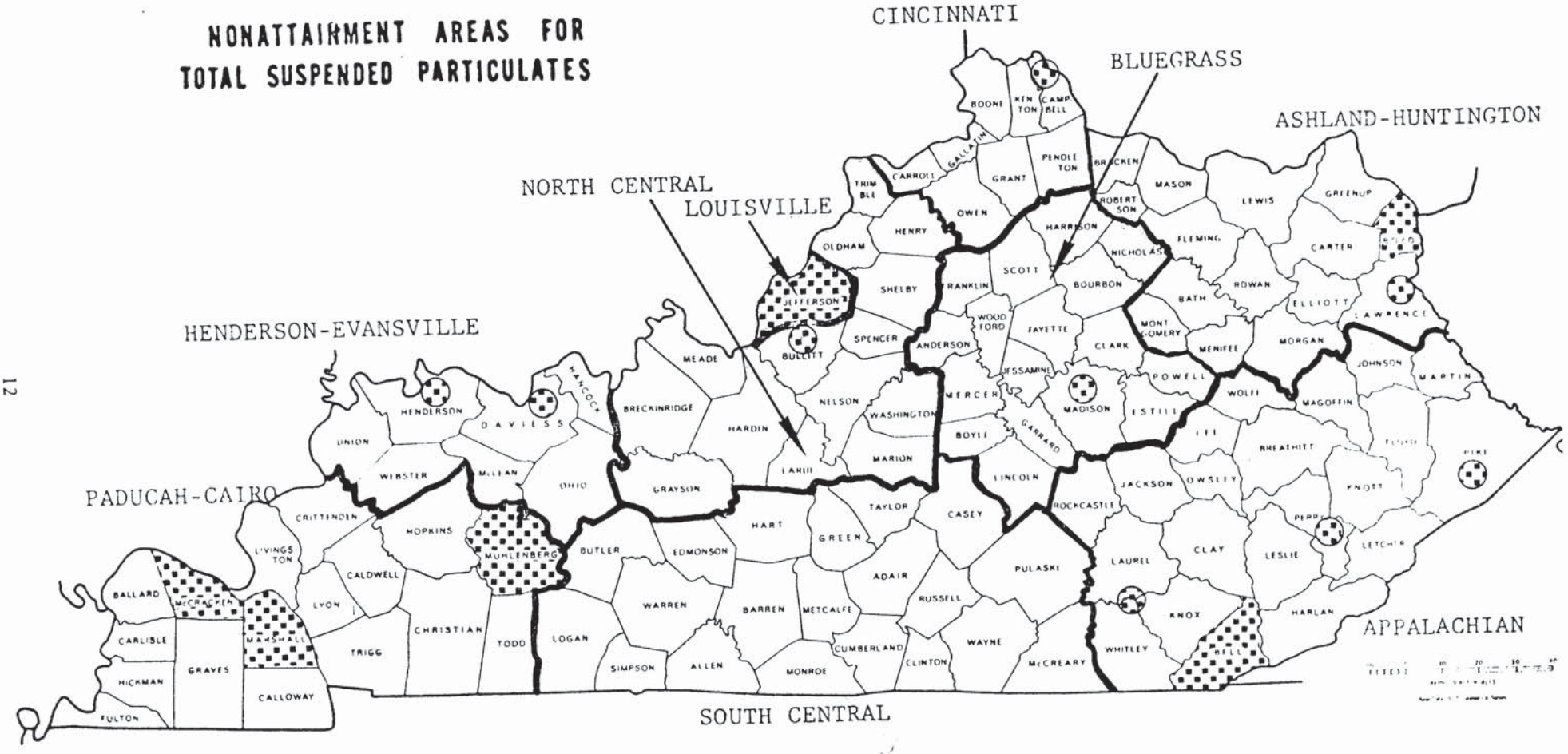
TSP = Total Suspended Particulates
SO_2 = Sulfur Dioxide
O_x = Photochemical Oxidants
CO = Carbon Monoxide

SOURCE: KY Department for Natural Resources and Environmental Protection, State Implementation Plan - 1979, page 2-19.

FIGURE 2

MAP OF KENTUCKY
Air Quality Control Regions

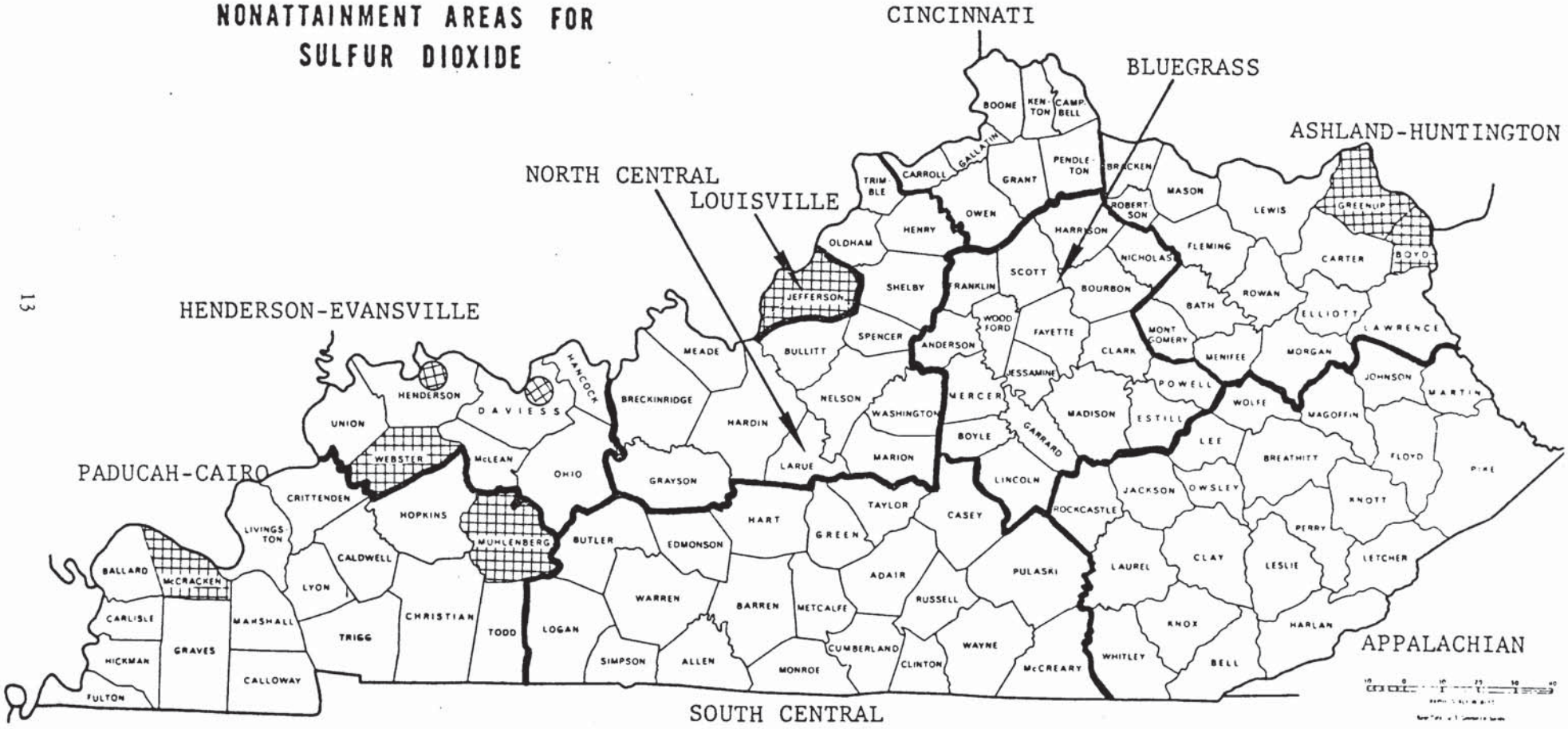
NONATTAINMENT AREAS FOR TOTAL SUSPENDED PARTICULATES



SOURCE: State Implementation Plan-1979, Kentucky Department for Natural Resources & Environmental Protection, page 2-21.

MAP OF
KENTUCKY
Air Quality Control Regions

FIGURE 3

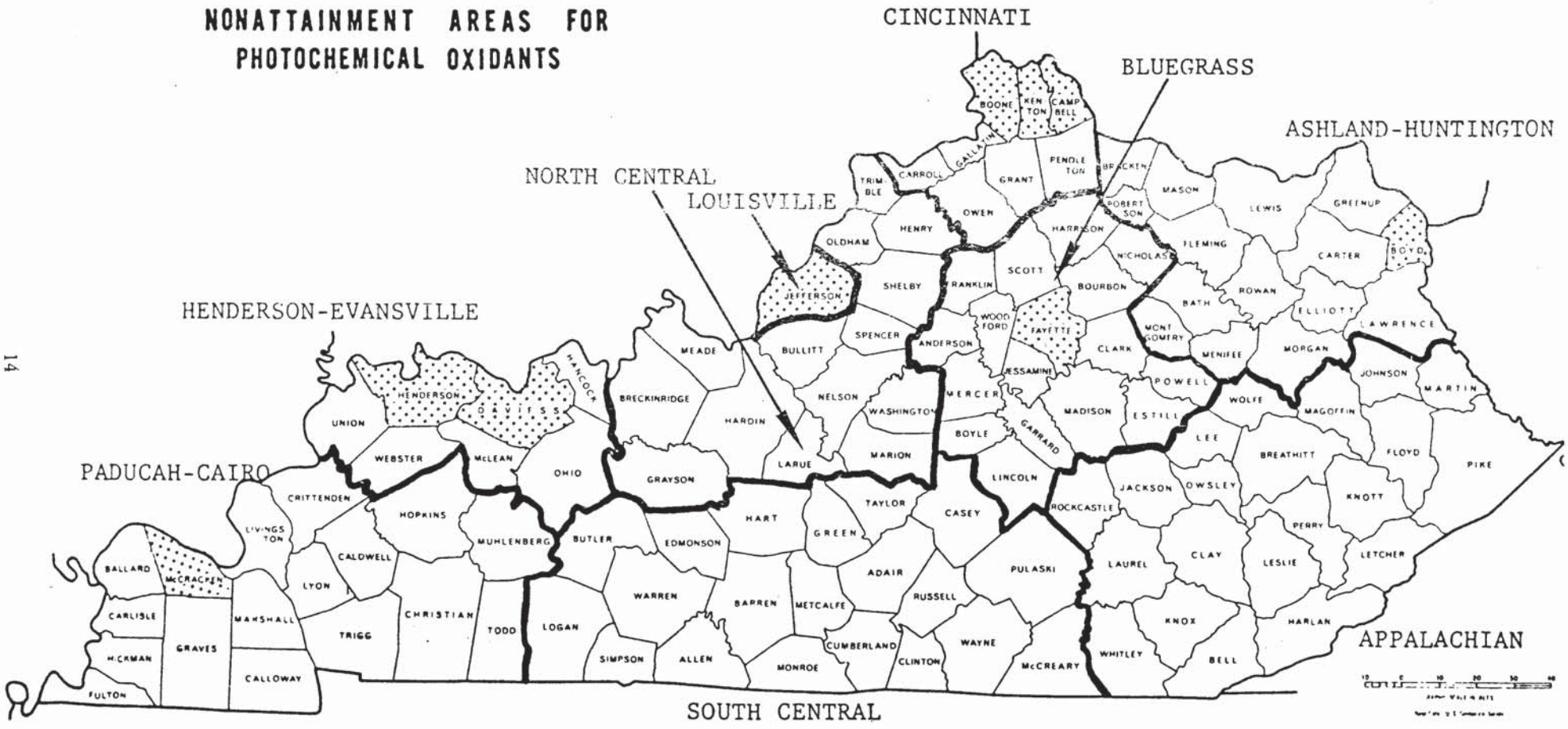


SOURCE: State Implementation Plan-1979, Department for Natural Resources & Environmental Protection, pg. 2-22

FIGURE 4

MAP OF KENTUCKY
Air Quality Control Regions

NONATTAINMENT AREAS FOR PHOTOCHEMICAL OXIDANTS



SOURCE: State Implementation Plan-1979, Department for Natural Resources & Environmental Protection, pg. 2-23

TABLE 3

Kentucky
Ambient Air Quality Standards[4]

The following air contaminant concentrations shall apply at any single point location:

CONTAMINANT	PRIMARY STANDARD	SECONDARY STANDARD
Sulfur Oxides (Sulfur Dioxide) - $\mu g/m^3$		
Annual Arithmetic Mean, not to exceed	80 (0.03 ppm)	---
Maximum Twenty-Four-Hour Average	365 (0.14 ppm)†	---
Maximum Three-Hour Average	---	1300 (0.50 ppm)†
Particulate Matter - $\mu g/m^3$		
Annual Geometric Mean, not to exceed	75	60‡
Maximum Twenty-Four-Hour Average	260†	150†
Carbon Monoxide - mg/m^3		
Maximum Eight-Hour Average	10 (9 ppm)†	Same as primary
Maximum One-Hour Average	40 (35 ppm)†	Same as primary
Ozone - $\mu g/m^3$		
Maximum Average	235 (0.12 ppm)Φ	Same as primary
Hydrocarbons - $\mu g/m^3$◇		
(measured as CH_4 and corrected for Methane)		
Maximum Three-Hour Morning Average (6-9 A.M.)	160 (0.24 ppm)†	Same as primary
Nitrogen Dioxide - $\mu g/m^3$		
Annual Arithmetic Mean, not to exceed	100 (0.05 ppm)	Same as primary
Lead - $\mu g/m^3$		
Maximum Arithmetic Mean averaged over a calendar quarter	1.5	Same as primary
Hydrogen Sulfide - $\mu g/m^3$		
Maximum One-Hour Average	---	14 (0.01 ppm)†
Gaseous Fluorides - (expressed as HF) - $\mu g/m^3$		
Annual Arithmetic Mean, not to exceed	400 (0.5 ppm)	0.50 (0.60 ppb)†
Maximum One-Month Average	---	0.80 (0.97 ppb)†
Maximum One-Week Average	---	2.86 (3.50 ppb)†
Maximum Twenty-Four-Hour Average	800 (1.0 ppm)†	2.86 (3.50 ppb)†
Maximum Twelve-Hour Average	---	3.68 (4.50 ppb)†
Total Fluorides - ppm		
Dry weight basis (as fluoride ion) in and on forage for consumption by grazing ruminants		
The following concentrations are not to be exceeded:		
Average concentration of monthly samples over growing season (not to exceed 6 consecutive months)	---	40 ppm (w/w)
Two-Month Average	---	60 ppm (w/w)
One-Month Average	---	80 ppm (w/w)
Odors		At any time when 1 volume unit of ambient air is mixed with 7 volume units of odorless air, the mixture must have no detectable odor.

Footnotes:

† This average is not to be exceeded more than once per year.

‡ This secondary annual standard is to be used as a guide in assessing implementation plans to achieve the twenty-four (24) hour standard.

Φ The standard is attained when the expected number of days per calendar year with maximum hourly average concentrations above 0.12 ppm (235 $\mu g/m^3$) is equal to or less than 1, as determined by Appendix H of 40 CFR 50.

◇ This standard is to be used as a guide in devising implementation plans to achieve photochemical oxidant standards.

CHAPTER III

MONITORING AND REPORTING

The proper surveillance and monitoring of the atmosphere for pollutants represents one of the most difficult challenges to the scientific community. This difficulty is that so many variables affect the concentration and movement of pollutants in the atmosphere, and, that some of the reactions in the atmosphere are not fully understood. The concentration of pollutants is affected by rate of release to the atmosphere, concentration at time of release, height of release, stability of the air, wind direction and velocity, and many other variables. Once the pollutant has been released into the atmosphere, it is subject to various physical or chemical changes.

As a result of these many complex factors, the sampling, analyzing, interpretation, and reporting must likewise be complex. The proper interpretation and application of the data obtained from monitoring stations usually requires the application of computerized models.

The Clean Air Act provides for a state to establish an air surveillance program, which is subject to approval of the Administrator of EPA. Prior to the regulation amendments of 1979, the federal requirements for an adequate air quality surveillance program were found in Title 40, Part 51, of the Code of Federal Regulations (since those amendments, they are now found in Title 40, Part 58). These regulations require a state, as a minimum, to include a description of the air quality monitoring system, the basis for the design and selection of samplers and sampling stations, the locations of samplers by grid coordinates, sampling schedules, methods of sampling and analysis, methods of data handling and analysis procedures. They also require that most methods used for measurement be performed in accordance with specific established reference methods or equivalent methods. Additionally, guidelines were provided by EPA in the performance of measurement, especially in the type and use of models in data interpretation and predictive analysis.

The Kentucky Department for Natural Resources and Environmental Protection has established an air quality surveillance program which has been approved by the EPA Administrator. This program includes sampling and analytical procedures which conform to federal reference or equivalent methods.

In 1977 the Kentucky surveillance program included over 150 monitoring stations at various sites strategically located throughout the state. These site locations included the areas of Lexington, Newport, Boone County, Owensboro, Henderson, Ashland, Paducah, McCracken County, Frankfort, Madisonville, Middlesboro, Elizabethtown, London, Corbin, Ballard County, Louisville, Jefferson County, Pikeville, Calvert City, Beuchel, Alexander, Ft. Thomas, Erlanger, Hopkinsville, Prestonsburg, Carrollton, Maysville, Carroll County, Gallatin County, Greenup County, Catlettsburg, Hancock County, Richmond, Winchester, Danville, Hazard, Berea, Bowling Green, Cynthiana, Lawrence County, Carter County, Shelbyville, Leitchfield, Oldham County, Central City, Princeton, Russellville, Glasgow, Somerset, Franklin County, Trimble County, Hardin County, Florence, Webster County, Beaver Dam, Ohio County, Harlan County, Mayfield, Smithland, Cleaton, Flat Lick, Shepherdsville, and Walton.

These various monitoring stations are operated in accordance with the criteria established by the federal regulations (see documents from U.S. EPA in Appendix K).

The criteria for selecting these sites are identified in the Code of Federal Regulations (CFR), Title 40, Part 58, Appendices D and E. Among other requirements, these sites were chosen to meet the following objectives:

(1) To determine the highest concentrations expected to occur in the area covered;

(2) To determine representative concentrations in areas of high population density;

(3) To determine the impact on ambient pollution;

(4) To determine general background concentration levels.

The methods used to measure the pollutants from the air samples from the established monitoring stations are referenced in 401 KAR 50:015 of the Kentucky Administrative Regulations Service. These referenced methods are those contained in 40 CFR 50, and are listed below. They describe in detail the principle and applicability, range and sensitivity, interferences, precision, accuracy, and stability of the method, and the type of apparatus and procedure to follow in performing the analysis. The methods also explain how to obtain the sample and prepare such for analysis. The referenced methods adopted by DNREP are:

40 CFR 50

1 Appendix A: Reference Method for the Determination of Surfur Dioxide in the Atmosphere (Pararosaniline Method).

2. Appendix B: Reference Method for the Determination of Suspended Particulates in the Atmosphere (High Volume Method).

3. Appendix C: Measurement Principle and Calibration Procedure for the Continuous Measurement of Carbon Monoxide in the Atmosphere (Non-Dispersive Infrared Spectrometry).

4. Appendix D: Measurement Principle and Calibration Procedure for the Measurement of Photochemical Oxidants Corrected for Interferences due to Nitrogen Oxides and Sulfur Dioxide.

5. Appendix E: Reference Method for the Determination of Hydrocarbons Corrected for Methane.

6. Appendix F: Measurement Principle and Calibration Procedure for the Measurement of Nitrogen Dioxide in the Atmosphere (Gas Phase Chemiluminescence).

7. Appendix G: Reference Method for the Determination of Lead in Suspended Particulate Matter Collected from Ambient Air.

8. Appendix H: Interpretation of the National Ambient Air Quality Standards for Ozone.[5]

In addition to these specifically referenced federal methods, the department has also adopted by regulation, under 401 KAR 50:015, specific methods of the Association of Official Analytical Chemists, the American Society of Testing Materials, the Technical Association of the Pulp and Paper Industry, the American Association of State Highway and Transportation Officials, and other federal test methods. Ultraviolet photometry, based on the absorption by ozone (O_3) in air of ultraviolet radiation at a wavelength of 254 nanometers (NM), will now be used by EPA as a calibration procedure for O_3 (reference method). This technique is important to note, as it will be more ac-

curate than the buffered or unbuffered potassium iodide (KI) procedures used in the past. Using UV photometry should probably lead to slightly lower O_3 readings, as several past experiments have shown the 1 percent and 2 percent neutral buffered potassium iodide (NBKI) procedures to give higher O_3 values (15-25 percent higher) when compared with UV photometry. The Kentucky Division of Air Pollution also has several specific Kentucky generated methods of reference.

Under most circumstances, the proper application and interpretation of data obtained from the state surveillance system involves the use of air quality models. The department has adopted, through regulation (401 KAR 50:040), the air quality models recommended in the U.S. EPA publication "Guidelines on Air Quality Models," OAQPS 1.2-080. This document identifies specific recommended models, data requirements for input into the models, and methods of model validation and calibration.

The recommended models are of four general classifications: (1) Gaussian, (2) Numerical, (3) Statistical or Empirical, and (4) Physical. The Gaussian models are generally considered to be state-of-the-art techniques for estimating the impact of nonreactive pollutants. Numerical models are more appropriate for multi-source applications which involve reactive pollutants. Statistical or empirical models are frequently employed in situations where incomplete scientific understanding of the physical or chemical processes makes the Gaussian or numerical model impractical. Physical modeling may be useful in evaluating the air quality impact of a source or group of sources in a geographic area limited to a few square miles.

Finally, in discussing any monitoring program, the method of quality assurance must be addressed. Appendix A of 40 CFR 58 contains quality assurance criteria to be followed by states in the operation of their air quality monitoring systems. This Appendix requires, as a minimum, that each quality control program have operational procedures for each of the following activities:

(1) Selection of methods, analyzers, or samplers.

(2) Installation of equipment.

(3) Calibration.

(4) Zero/span checks and adjustments of automated analyzers.

(5) Control checks and their frequency.

(6) Control limits for zero/span and other controls checks, and respective corrective actions when such limits are surpassed.

(7) Calibration and zero/span checks for multiple range analyzers.

(8) Preventive and remedial maintenance.

(9) Quality control procedures for air pollution episode monitoring.

(10) Recording and validating data.

(11) Documentation of quality control information.

For recording and reporting requirements, the department adheres to the EPA requirements of Appendix F of 40 CFR 58, which include an annual report, identifying for each pollutant the sampling station location, the number of observations, and specific statistics of the analysis results. As an example, for ozone, the annual summary statistics must identify the four highest daily maximum hour values (ppm) and their dates and time of occurrence, the number of times of exceeding the daily maximum 1-hour primary NAAQS, and the number of daily maximum hour concentrations in ranges:

Range	Number of Values
.0 to .04 (ppm)	
.05 to .08	
.09 to .12	
.13 to .16	
.17 to .20	
.21 to .24	
.25 to .28	
Greater than .28	

In compliance with this requirement, the department issues an annual report which identifies the monitoring methods, monitoring sites, and specific statistical data for each pollutant measured at each site. An example of the statistical data reported by DNREP is shown in Table 4.

Table 4

KENTUCKY DIVISION OF AIR POLLUTION CONTROL

OZONE STATISTICS
UNITS = PARTS PER MILLION
1 HOUR AVERAGES

Site No./Location (Saroad ID)	Period From - Thru	Parameter	No. Obs.	Max	Second Max	Times > 0.12	No. Days in Violation Actual	Expected
AQCR – 102 BLUEGRASS								
0002-003 Lexington (18 2300-002 F01)	79/01-79/05	Ozone	2844	.087	.074	0	0	0
0002-227 Fayette County (18 1160-001 F01)	79/01-79/12	Ozone	8509	.124	.111	0	0	0
0002-254 Harrodsburg (18 1680-001 F05)	79/08-79/09	Ozone	1125	.107	.102	0	0	0
0002-256 Danville (18 0900-004 F05)	79/09-79/09	Ozone	329	.039	.034	0	0	0
0002-263 Lexington (18 2300-012 F01)	79/12-79/12	Ozone	622	.046	.035	0	0	0
AQCR – 079 CINCINNATI METRO. INTERSTATE								
0003-189 Boone County (18 0280-003 F03)	79/01-79/08	Ozone	5388	.130	.120	2	1	2
0003-198 Florence (18 1220-002 F05)	79/01-79/08	Ozone	5599	.120	.116	0	0	0
0003-257 Dayton (18 0960-001 F01)	79/09-79/12	Ozone	2351	.105	.080	0	0	0
AQCR – 077 EVANSVILLE-HENDERSON INTERSTATE								
0004-058 Owensboro (18 3140-005 F01)	79/01-79/12	Ozone	8399	.106	.106	0	0	0
0004-155 Henderson (18 1740-008 F01)	79/01-79/12	Ozone	8306	.107	.107	0	0	0
AQCR – 103 HUNTINGTON-ASHLAND INTERSTATE								
0005-246 Ashland (18 0080-010 F01)	79/02-79/12	Ozone	5484	.170	.163	5	2	3
AQCR – 078 LOUISVILLE INTERSTATE								
0006-054 Jefferson Co. (18 1920-027 G03)	79/01-79/12	Ozone	8011	.126	.120	1	1	1
0006-055 Louisville (18 2380-021 G01)	79/01-79/10	Ozone	7034	.158	.129	6	3	4
0006-079 Jefferson (18 1920-034 G03)	79/07-79/12	Ozone	3976	.140	.140	15	8	18
0006-080 Jefferson (18 1920-033 G03)	79/01-79/12	Ozone	8665	.154	.138	14	5	5
006-084 Jefferson Co. (18 1920-041 G03)	79/07-79/12	Ozone	3254	.185	.160	37	13	38
AQCR – 072 PADUCAH-CAIRO INTERSTATE								
0008-120 Paducah (18 3180-020 F01)	79/01-79/12	Ozone	6483	.047	.038	0	0	0

CHAPTER IV

OZONE FORMATION & DISPERSION

House Resolution 154 and Senate Resolution 83 specifically requested that this study include the state of scientific knowledge regarding the formation and dispersion of ozone pollutants. A discussion of the health and environmental effects of photochemical oxidants, including ozone, is given in Appendix F.

Photochemical oxidants are products of atmospheric reactions between sunlight and organic pollutants, nitrogen oxides (NOx), and oxygen. They consist mostly of ozone (O_3), nitrogen dioxide (NO_2) and peroxyacetylnitrate (PAN). Photochemical oxidants originate mainly from volatile organic and NOx emissions produced by human activities. Photochemical oxidant formation is a complex function of emissions and meteorological patterns.

Peak concentrations of oxidants, expressed as ozone, are generally higher in urban and suburban areas than in rural areas, reaching levels in excess of 590 ug/m^3 (0.3 ppm). In rural areas, peak concentrations are lower but often exceed the 1-hour National Ambient Air Quality Standard. However, dosages or average concentrations in rural areas are comparable to or even higher than those in urban areas. Because of pollutant transport, oxidant pollution is a regional rather than a local problem.[7]

As identified previously, there are several photochemical oxidants which may pollute the air. However, the remainder of this chapter will concentrate only on ozone, which is the major photochemical oxidant.

Ozone Formation

In February of 1979, the U.S. EPA designated ozone (O_3) as the air pollutant of interest for control purposes. The reason for this was that O_3 is the major oxidant in the category of air pollutants known as photochemical oxidants. This chapter will therefore only discuss the formation of ozone, although it should be noted that the oxidant air pollution problem is significantly more complex.

To understand the formation of ozone in the atmosphere, it is necessary to discuss the processes which cause the formation and buildup of ozone. For simplicity, and because the process is not completely understood, the following description omits many of the atmospheric chemical reactions. However, much progress has been made in the last ten years in understanding the sources and roles of chemical "radicals" in promoting ozone formation.

The process begins when the nitrogen dioxide (NO_2) molecule absorbs sunlight and breaks apart (undergoes photolysis), as shown by the following reaction:

(1) NO_2 + light $\longrightarrow$ O + NO

From the above equation, it can be seen that nitrogen dioxide has been broken into an atom of oxygen (O) and nitric oxide (NO).

The oxygen atom then reacts with molecular oxygen (O_2) to form ozone, as follows:

(2) O + O_2 $\longrightarrow$ O_3

To complete the atmospheric ozone formation mechanism, ozone combines with nitric oxide to form nitrogen dioxide and molecular oxygen as follows:

(3) O_3 + NO $\longrightarrow$ NO_2 + O_2

Nitrogen dioxide then undergoes the reaction shown in equation (1), thus creating a cycle process. Since the above process shows a "build-up" and "break-down" cycle of ozone, this process is not sufficient to cause ozone formation of the magnitude that is currently observed in metropolitan air pollution. To understand why ozone builds to levels which can violate the national ambient air quality standard, other air pollutants must be considered.

In the last ten years, scientific research has led to the conclusion that various oxidation cycles involving reactive hydrocarbons, aldehydes, and carbon monoxide serve to cause nitric oxide to be "pumped" to nitrogen dioxide, thus leading to a build-up of ozone.

In the presence of photochemically reactive organic compounds, such as hydrocarbons, free radicals are formed. These free radicals react with oxygen and nitric oxide to produce nitrogen dioxide, which in turn serves to increase ozone production above its equilibrium cyclic state as described above. Rather than consuming ozone and balancing out the reaction, the nitric oxide is occupied by reactions involving the chemical radicals, and therefore is unable to react with the ozone. The ozone formed by this process is a *secondary* pollutant, as it is formed in the atmosphere from such primary pollutants as hydrocarbons, aldehydes, and nitrogen oxides. Again it should be noted that ozone is not the only pollutant formed in the complex chemical reactions in the atmosphere, although ozone is the only one for which a national air quality standard has been set.

In addition to contributing to ozone formation, nitrogen dioxide, a reddish-brown gas, absorbs light, reducing visibility and discoloring the atmosphere.

The preceding is a very brief description of the formation of ozone. A great deal of work in understanding the process remains. Chemical models have been useful, but these models are mainly derived from laboratory situations, which may not fully represent complex atmospheric processes.

Since ozone is a secondary pollutant, it is necessary to identify where the primary pollutants or precursors originate. These precursors are both natural and human-made in origin, although human-made ones are much more significant, especially in urbanized areas. The precursors of ozone are nitrogen oxides (collectively referred to as NOx) and hydrocarbons, referred to as volatile organic compounds (VOC).

The two major emission sources for NOx are transportation-related sources and stationary fuel combustion. Data from EPA statistics in 1977 estimated that nationwide NOx emissions from transportation-related sources (highway and non-highway) were 9.2 million metric tons. Emissions of NOx from stationary fuel combustion sources (electric utilities, industries, residences, commercial enterprises, and institutions) were estimated at 13 million metric tons.

Emissions of hydrocarbons are simply referred to as volatile organic compounds (VOC), since available data does not usually distinguish between the different types of hydrocarbons and other reactive organic compounds. The 1977 EPA data estimated that 11.5 million metric tons of VOC were released to the atmosphere from transportation-related sources. Industrial processes were estimated to contribute 10.1 million metric tons of VOC. According to EPA, transportation-related emissions of VOC nationwide have decreased from 1970 to 1978 by 1 million tons per year. However, stationary sources VOC emissions increased by 800,000 tons per year, which virtually offset the decline in mobile source emissions.

Pollutant Transport

A report to the National Commission on Air Quality[8] indicates that the local or regional

transport of ozone and its precursors (hydrocarbons, nitrogen oxides) is accepted by scientific investigators as common. However, the report also states that:

> . . . the principal problems connected with transport are a lack of analytical methods and meteorological and pollutant data for use in accounting quantitatively for the role of transport in the ozone problem of any given geographical area.

There are three broad categories of pollutant transport:

(1) *Urban scale transport*—Ozone and primary emissions originate in the urban area, but *peak ozone* concentrations occur downwind of the city-core in surburban areas.

(2) *Mesoscale transport*—Mesoscale is a meteorological term which indicates weather conditions occurring over a 10-100 mile range in a period of hours to days. In terms of ozone transport, the term indicates the formation of ozone plumes which can affect downwind ozone levels up to 100 miles away. Mesoscale conditions involve general weather and wind patterns from water bodies and land surfaces.

(3) *Synoptic Scale Transport*—Synoptic phenomena are generally what are seen on television weather reports and involve weather conditions from 100-1000 miles over a period of days to weeks. High and low pressure systems and cold and warm air fronts are plotted by taking simultaneous observations at different points. This scale of transport usually involves ozone carried by high pressure systems traveling from 100-1000 miles downwind of the source(s).

Each of these types of ozone transport has been observed and reported on in many research studies. For purposes of a brief review of the general *process* of ozone transport, one page from the recent report of the National Commission on Air Quality has been reproduced in Appendix L.[9]

As stated earlier, the process of ozone transport has come to be widely recognized, although the impact of transport on local ozone levels cannot be accurately quantified. Although this phenomenon is important and possibly crucial to local success in achieving ozone reductions to meet air quality standards, there are a large number of complicating factors in quantifying transported ozone levels.

For example, it is necessary to know the following types of information in order to begin estimating the amount of ozone for ozone precursor transport:

Chemical—Sources of nitrogen oxides and hydrocarbon emissions and their emission strengths and rates; chemical species (and reaction rates, molecular weights); the ratio of nitrogen oxides to hydrocarbons, the presence of other organic compounds in the atmosphere:

Meteorological—Prevailing winds (direction, speed); humidity; temperature; sunlight intensity and variation; frequency and duration of inversions; air turbulence and mixing capabilities;

Geographic/Topographic—Terrain, latitude and longitude; altitude; ventilation characteristics.

Natural levels of ozone appear to come from two sources: (1) natural organic emissions (e.g., terpenes) from vegetation or other sources which may form some ozone in rural areas, and (2) intrusion of ozone from the stratosphere. According to EPA research, natural hydrocarbon emissions in rural areas are able to form only negligible amounts of ozone, and are probably not capable of surviving long enough to be a significant influence on urban ozone levels. EPA estimates that the contribution of natural organic emissions to ambient air ozone levels is less than 0.01 part per million (ppm).

For purposes of air quality planning, the intrusion of stratospheric ozone into the troposphere is also of no significance, although it appears to contribute more than natural precursors (discussed above) to background ozone levels. Stratospheric ozone source mechanisms are not well understood but seem to impact the lower atmosphere either through slow diffusion and turbulent atmospheric mixing, or sporadic "intrusions" caused by very turbulent weather phenomena, such as jet streams, thunderstorms, or major weather front activity. In total, EPA uses a value of 0.04 parts per million (ppm) to represent the contribution of ozone from natural organic emissions and stratospheric sources. This estimated background ozone level in rural areas has been calculated to generally add about 0.02 ppm to urban ozone levels because of transport conditions. EPA also feels that modeling results suggest that rural precursors do not contribute significantly to urban ozone formation.

In a background document for EPA's Empirical Kinetic Modeling Approach (EKMA), used to calculate VOC (hydrocarbons) reductions necessary to meet the ozone air standards, EPA used the model to estimate the impact of ozone transport on peak levels in urban areas.

Transported ozone itself, rather than its chemical precursors, should be accurately monitored to estimate local air quality impacts. However, as stated previously, the impact of ozone transport varies with dilution rates, relative emission rates, the ratio of hydrocarbons to nitrogen oxides, light intensity and other factors, and *transported ozone levels cannot simply be added to local emission levels for regulatory purposes.* The reason for this is that the factors affecting ozone are interactive. Thus, ozone transported into an urban area may be reduced by nitrogen oxides emissions or other reactions, or may stimulate a higher NO_2/NO ratio, thus forming more ozone. Rather than attempting to quantify the variable effects of transported ozone, EPA has relied on control programs at the local level to reduce the amount of ozone formed and, subsequently, transported downwind.

The recent ozone report[10] from the National Commission on Air Quality examines EPA's present policies and the state-of-the-art regarding the examination of ozone transport. The Commission's report discusses the limitations of current chemical kinetic modeling and smog chamber research and recommends that the Clean Air Act "recognize [pollutant] transport and provide a regulatory framework for dealing with it." However, the report does not recommend any particular regulatory strategies for accomplishing this. In general, the report recommends further research in modeling ozone transport processes, and the gathering of more complete regional data on emissions and meteorology, in order to better understand and quantify transported ozone and its chemical precursors.

Other Sources of Ozone

The resolutions passed by the 1980 House and Senate also requested the study to include the effect of new systems on ozone formation.

There appears to be limited information on the formation of ozone from other human-made sources besides those discussed above. The resolution specifically mentioned the possibility of such new systems as street and highway lighting. These systems are usually incandescent lamps, mercury vapor lamps, or high-pressure sodium vapor lamps. There appears to be no data in the literature which would indicate that these lighting sources contribute ozone to the atmosphere. Some studies suggest that coronal discharges around high voltage transmission lines may be a possible source of ozone formation, although several research investigations have indicated that this is not the case, at least that the amount of such ozone is not of any significance.

The resolution also addressed the effect systems for stack gas cleanups might have on ozone formation. Cleanup systems to improve emissions from stationary sources may be used to meet emission standards. These systems, or other methods of NOx reductions, must be applied if a source does not meet the emission standards, and therefore they will affect ozone formation. However, the strategies identified by DNREP to meet the emission standards would include stack gas cleanup, industrial process changes, or other methods which would result in the reduction of pollutant emissions.

CHAPTER V

MOTOR VEHICLE EMISSION INSPECTION AND MAINTENANCE

The issue of vehicle emissions inspection/maintenance (I/M) has been the subject of much debate since Congress passed the Clean Air Act Amendments of 1977.

According to the Act, air quality regions which did not foresee attainment of national primary ambient air quality standards for photochemical oxidants (now measured as ozone-O_3) or carbon monoxide, by December 31, 1982, were required to submit in their State Implementation Plan (SIP) revisions of January 1, 1979, the following items in addition to other SIP requirements:

1. Establishment of a program requiring an environmental and social review prior to the issuance of a permit for construction or modification of a major emitting facility;
2. Establishment of "a specific schedule for implementation of a vehicle emission control inspection and maintenance program"; and
3. Identification of other measures necessary to provide for attainment of the applicable national ambient air quality standard not later than December 31, 1987.

EPA policy specified that extensions might be granted to July 1, 1980, if one or both of the following considerations were demonstrated:

1. There has been insufficient opportunity to conduct necessary technical analysis.
2. The legislature has had no opportunity to consider any necessary enabling legislation.

According to EPA policy, the following are minimum legal requirements for I/M enabling legislation:

1. Authorization of periodic inspection of all vehicles for which emission reduction credits are claimed. Regulations must be sufficient to produce a 25 percent reduction in hydrocarbons (HC) and a 25 percent reduction in CO by December 13, 1987.
2. Authorization of the imposition of penalties on vehicle owners not complying with the regulations.
3. Provisions for the staff, funding and facilities necessary to implement regulations.

A bill (HB 388) was introduced in the 1978 General Assembly which would have granted authority to DNREP to implement a state I/M program, but it did not pass. On August 18, 1978, in response to a request for an opinion from the Jefferson County Air Pollution Control District, the Attorney General, under OAG 78-550, expressed the opinion that the fiscal court of Jefferson County had authority to enact an ordinance establishing a mandatory motor vehicle emission inspection/maintenance program. Also, on August 25, 1978, in response to a request for an opinion from the *Kentucky Post Bureau*, the Attorney General expressed the opinion (OAG 78-582) that the Department for Natural Resources and Environmental Protection had the power to require mandatory automobile emissions tests. However, the opinion went on " . . . it is reasonable to think that the Department might not wish to do so [institute auto emissions testing] in light of the action by the General Assembly earlier this year." The 1980 General Assembly considered a state auto emission inspection program under Senate Bill 315. However, the bill did not pass.

Since Jefferson County/Louisville faced possible federal funding sanctions from EPA if authority for an I/M program were not established, the fiscal court of Jefferson County adopted Or-

dinance No. 21, Series 1980 (July 8, 1980), authorizing the Air Pollution Control Board to administer an auto emission inspection program.

Also, faced with similar loss of federal funds, the County of Boone on July 11, 1980, adopted Ordinance No. 520.2, delegating authority to the County Judge/Executive to administer a Boone County auto emission inspection program.

Since the other areas of northern Kentucky not meeting ozone standards—specifically the counties of Kenton and Campbell—had no local ordinance authorizing an I/M program, the U.S. EPA, in the Federal Register notice of September 19, 1980, announced that "the Agency was initiating the process to cut off certain Federal funds to the counties of Boone, Kenton, and Campbell. . . ." (Note: In a later Federal Register of September 22, 1980, EPA indicated it proposed to approve the Boone County portion of the state SIP). Further the EPA announced in the same issue of the Federal Register that they were invoking the ban on construction of major new or modified stationary sources of hydrocarbons in the Kentucky part of the Metropolitan Cincinnati Interstate AQCR.

On November 12, the Fiscal Courts of Boone, Campbell, and Kenton Counties, through the U. S. Court of Appeals, Sixth Circuit, petitioned the Court to review the Final Rule Making Action of the U.S. EPA, as was published in the September 22, 1980 Federal Register. No action had been taken on this petition at the time of this writing.

On December 12, the EPA officially announced "that federal highway and sewage treatment funds were being cut off in Kenton and Campbell Counties."[11]

One of the major reasons for some of the northern Kentucky counties being hesitant about adopting ordinances authorizing I/M programs is their questioning of the monitoring data and other assumptions which caused the northern Kentucky area to be designated "non-attainment" for ozone. A special technical advisor was employed by the three northern counties to review that data. In his review the advisor was very critical of several areas of the data. He raised questions on the validity of data from the Atkinson Square monitoring station, in Cincinnati, Ohio, the sensitivity of the statistical application of the data, and the model which was applied in determining a "non-attainment" status for northern Kentucky.

These questions on the monitoring data were made available as a report to EPA. After review of the report made by the special technical advisor, the EPA wrote the Governor: "We have reviewed the report carefully and have determined that it does not support the contention that attainment of the ozone standard can be reached by 1982." The letter further stated, "It also throws no doubt on the validity of the Kentucky State Implementation Plan (SIP)."[12]

For an independent review of the questions raised by the special technical advisor to the northern counties, the Legislative Research Commission staff requested assistance from an individual trained in air pollution who is with the National Conference of State Legislatures. His main conclusion, after review of the data, and the questions raised about the data was "that the use of the Atkinson Square monitoring data was correct, and that the 40% hydrocarbon reduction requirement for the Cincinnati air quality region is accurate."[13] Documentation for this opinion was provided in his letter and report to LRC dated November 5, 1980.

I/M Rationale

The basic reasoning behind inspection/maintenance programs has been explained by the

Kentucky Department for Natural Resources and Environmental Protection, Congress, EPA, the GAO and others: Vehicles in use are not meeting federal emission standards for all of their useful life.

The federal Clean Air Act authorizes the administrator of EPA to set standards applicable to the emission of air pollution by any class or classes of new motor vehicles or new motor vehicle engines. The Federal Motor Vehicle Emission Control Program (FMVCP) consists of a certification program for new vehicle designs, attempting to assure that they will meet federal emission standards. Assembly line testing is used to confirm that the vehicles do meet emission standards when new. Also if in-use surveillance shows up design flaws, resulting in failure to meet emissions standards, manufacturers may be required to recall those vehicle types. EPA estimates that about 80 percent of the 100 million vehicles presently in use do not meet applicable federal standards. Sixty percent of one-year-old cars exceed federal new car standards, and 75 percent exceed those standards after four years.

There are several likely explanations for vehicles not meeting federal new car standards: the federal EPA program is not an adequate indication that production-line vehicles are capable of meeting emission standards during all of their useful life; manufacturers' recommended maintenance intervals are not being observed by vehicle owners; or vehicles are being improperly adjusted or tampered with. EPA believes that proper maintenance must be performed in order that vehicles continue to meet emission standards.

The GAO Report issued in January, while recommending revisions to federal certification testing procedures, also concluded that inadequate maintenance and improper adjustment were responsible for in-use vehicles failing to meet federal standards.[14]

A May 1979 report of the National Conference of State Legislatures contained an EPA policy paper dated July 1978, in which EPA recommended that I/M programs should focus on metropolitan areas and should include the entire urbanized area and adjacent fringe areas of development: "For urbanized areas of 200,000 population or greater which need I/M to obtain an extension of the 1982 attainment date, full mandatory I/M must be implemented by the deadlines. . . ."[15]

Nine counties in the Commonwealth were declared non-attainment for ozone by the DNREP (later reduced to seven). However, since only areas of 200,000 population or greater must be addressed at this time, only four counties (Jefferson, Boone, Kenton, and Campbell) are required to implement an I/M program. Jefferson County is the only area in Kentucky shown to be non-attainment for carbon monoxide.

Louisville Interstate Air Quality Control Region

Although the Kentucky portion of this region contains only one county, it is important to note that the Indiana counties of Floyd and Clark are included in both the non-attainment designation and in the regional definition. On February 6, 1978, the Governor of Kentucky designated the Kentuckiana Regional Planning and Development Agency (KIPDA) as the agency responsible for planning and evaluating transportation control strategies for the Kentucky portion of the Louisville Interstate Air Quality Control Region.

Table 5 presents ozone monitoring data for Jefferson County for January 1, 1975 through November 1978. The number of ozone ambient air quality standard violations that occurred in the metropolitan areas has varied, depending on the meteorological conditions during the year, as well as the actual number of air monitoring instruments. The only ozone monitor located in Clark County or Floyd County is located at Green Valley Elementary School in New Albany.

TABLE 5

SUMMARY OF OZONE AIR QUALITY DATA
FOR JEFFERSON COUNTY

Site Location	Reporting Period	Maximum (PPM)	Second Maximum (PPM)	No. of Violations*
Reynolds Building (18 2380 011)	75/01-75/12	0.185	0.155	5
	76/01-76/12	0.120	0.110	0
	77/01-77/01	0.015	0.015	0
Floyd & Jefferson (18 2380 020)	75/01-75/12	0.200	0.200	5
	76/01-76/12	0.090	0.075	0
	77/01-77/06	0.135	0.130	4
Mellwood Avenue (18 2380 021)	75/06-75/12	0.275	0.185	11
	76/01-76/12	0.165	0.150	4
	77/01-77/12	0.195	0.160	9
	78/01-78/11	0.200	0.198	21
Bates School (18 1920 027)	76/04-76/12	0.180	0.160	6
	77/01-77/12	0.140	0.140	4
	78/01-78/11	0.182	0.162	7
1509 S. 6th (18 2380 034)	77/04-77/06	0.065	0.055	0
Valley High School (18 1920 034)	78/02-78/08	0.143	0.139	3
Freys Hill (18 1920 033)	77/08-77/12	0.125	0.115	1
	78/01-78/11	0.171	0.165	13

* Based on 0.12 ppm standard, number of days experiencing violations.

SOURCE: Kentucky Department for Natural Resources and Environmental Protection, State Implementation Plan—1979, p. 5-2.

The criterion for selection of the design air quality value—that is, the value to be used in control strategy work—is based upon the Table Look-Up Method in EPA's "Guideline for Interpretation of Ozone Air Quality Standard," published in January 1979. The Ozone Air Quality value corresponding to the appropriate amount of available data (2-3 years, 730-1094 daily observations) was used. Thus, for the purpose of control strategy development, the design value was determined to be 0.198 ppm, which was the second highest ozone reading during the time period studied.

Listed in Table 6 are the DNREP's 1976 and projected volatile organic compounds (VOC) or hydrocarbons emissions for the Jefferson County area. The totals include growth but do not project reductions from any new strategies for stationary sources, Vehicle Inspection and Maintenance, or transportation elements. Reductions due to the Federal Motor Vehicle Emission Control Program (FMVCP) are accounted for. Although Table 6 indicates reductions in total VOC emissions, these reductions are not adequate to meet national ambient standards.

TABLE 6

TOTAL EXISTING & PROJECTED
VOC EMISSIONS INVENTORY
TONS PER YEAR FOR
LOUISVILLE INTERSTATE AIR QUALITY CONTROL REGION

	1976	1979	1982	1987
Mobile Source				
Jefferson	32300	25700	18000	11700
Floyd	2040	1890	1350	880
Clark	5200	4510	3130	1900
Area				
Jefferson	6930	7850	9770	9730
Floyd	650	800	840	900
Clark	1130	1190	1250	1380
Stationary				
Jefferson	33500	34590	31450	31930
Floyd	500	500	600	600
Clark	4160	4160	4050	4050
Subtotal				
Mobile Sources	39540	32100	22480	14490
Area	8710	9840	11860	12010
Stationary	38160	39250	36100	36580
Total	86410	81190	70440	63080

SOURCE: Kentucky Department for Natural Resources and Environmental Protection, State Implementation Plan—1979, p. 5-4.

The mobile emissions were based on estimates of vehicle miles of travel (VMT) in the area, as supplied by the Kentuckiana Regional Planning and Development Agency.

The modified linear rollback (MLR) model, discussed in Appendix J of the 1979 State Implementation Plan, was the method chosen for determining emission reductions needed to achieve the ozone standard. By applying this model, DNREP calculated that a 39% reduction in VOC emissions would be required to attain the standard. To obtain this 39% reduction, control strategies would be applied to stationary and mobile sources.

The strategies and anticipated reductions of hydrocarbons for mobile sources are shown in Table 7. It should be noted that the most effective strategy for the hydrocarbon reduction is inspection and maintenance.

TABLE 7

MOBILE
EMISSION REDUCTIONS FROM PROPOSED STRATEGIES BY COUNTY
TONS PER YEAR REDUCTION FOR LOUISVILLE INTERSTATE
AIR QUALITY CONTROL REGION

		Jefferson	Floyd	Clark
	Year	HC	HC	HC
Metro Carpool Program	1982	77	4	15
	1987	48	3	14
Improvement of Public Mass Transit	1982	168	—	—
	1987	110	—	—
Highway Improvement Projects	1982	717	52	128
	1987	590	45	105
Five Year TARC Improvement Program	1982	194	—	—
	1987	127	—	—
Center City Transportation Study	1982	305	—	—
	1987	203	—	—
Long Range Bicycle Plan Jefferson County	1982	4	—	—
	1987	3	—	—
Implementation of All Programs	1982	1,465	56	143
	1987	1,081	48	119
Inspection Maintenance	1982	2,210	70	150
	1987	3,726	230	450
Grand Total with I/M	1982	3,675	126	293
	1987	4,807	278	569

SOURCE: Kentucky Department for Natural Resources and Environmental Protection, State Implementation Plan—1979, p. 5-12.

Northern Kentucky
(Metropolitan Cincinnati Interstate Air Quality Control Region)

Although the Kentucky portion of this region contains eight counties, only three (Boone, Kenton and Campbell) are included in the non-attainment designation for ozone. This is because these counties are part of a major metropolitan area.

According to DNREP, as stated in the 1979 State Implementation Plan, "Because these counties are part of an interstate region, the design value from which reduction estimates must be calculated is the highest value measured in the entire region, not just those counties comprising the Kentucky portion of the region."[16] The design value used in Northern Kentucky occurred in the City of Cincinnati, Ohio, which had a measurement of 0.233 ppm at the Cincinnati-Atkinson Square monitoring site in August of 1976.

Table 8 presents ozone monitoring data for Northern Kentucky (metropolitan Cincinnati Interstate Air Quality Control Region) for January 1, 1975 through December 1978. Table 9 is the

DNREP's 1976 and projected VOC emissions for the Cincinnati and Northern Kentucky area. The totals include growth, where appropriate, but do not include reductions from any new strategies. Reductions due to the Federal Motor Vehicle Emission Control Program are accounted for. Although Table 9 indicates reduction in total VOC emissions, these reductions are not sufficient to meet national ambient standards.

TABLE 8

OZONE AIR QUALITY DATA FOR
METROPOLITAN CINCINNATI INTERSTATE
AIR QUALITY CONTROL REGION

Location	Year	Maximum (PPM)	Second Maximum (PPM)	No. of Violations*
Cincinnati-Grooms Rd. (36 2720 006)	1975	.21	.21	—
Cincinnati-Atkinson Sq. (36 6140 002)	1976	.223	.213	—
Cincinnati-Gest St. (36 1220 019)	1977	.210	.208	—
Newport-Newport Mall (18 3020 001)	1/75-12/75	.164	.150	10
	1/76-1/76	.145	.138	5
	1/77-6/77	.123	.115	1
Florence-7209 U.S. 42 (18 1220 002)	7/75-12/76	.138	.135	4
	1/76-1/76	.025	.025	0
	6/77-12/77	.150	.135	6
	1/78-12/78	.155	.120	1
Covington-4101 Dixie Hwy. (18 0800 007)	8/75-12/75	.128	.118	1
	1/76-1/76	.025	.025	0
Boone Co.-Ky 338-Ky 536 (18 0280 003)	7/77-12/77	.145	.140	8
	1/78-12/78	.175	.125	2

* Based on 0.12 ppm standard, number of days experiencing violations.

SOURCE: Kentucky Department for Natural Resources and Environmental Protection, State Implementation Plan—1979, p. 5-24.

TABLE 9

TOTAL EXISTING & PROJECTED VOC FOR METROPOLITAN CINCINNATI INTERSTATE AIR QUALITY CONTROL REGION

	EMISSIONS INVENTORY (Tons per Year)		
	1976	1982	1987
Mobile Sources			
Boone	2,947	2,123	1,417
Campbell	2,765	1,585	939
Kenton	5,025	2,926	1,720
Butler	9,895	5,972	3,716
Clermont	4,890	2,935	1,785
Hamilton	37,657	19,072	11,441
Warren	5,205	3,218	1,977
Area			
Boone	1,158	1,566	1,906
Campbell	1,004	1,028	1,038
Kenton	1,489	1,594	1,683
Butler	1,616	1,746	1,854
Clermont	1,158	1,292	1,362
Hamilton	5,626	5,952	6,123
Warren	615	690	757
Stationary			
Boone	1,116	992	992
Campbell	245	245	245
Kenton	1,178	1,186	1,186
Butler	6,935	11,682	11,746
Clermont	641	633	567
Hamilton	24,142	26,058	27,854
Warren	1,744	2,407	2,254
Subtotals			
Mobile Sources	68,384	37,831	22,995
Area	12,666	13,868	14,723
Stationary	36,001	43,203	44,844
Totals	117,051	94,902	82,562

SOURCE: Kentucky Department for Natural Resources and Environmental Protection, State Implementation Plan—1979, p. 5-25.

Inspection and Maintenance: Other States

Since the I/M issue has been so controversial, a review of other states' experiences with I/M would be helpful in assessing the issue.

Twenty-eight states and Washington, D.C. are required to comply with the I/M provisions of the federal Clean Air Act. Table 10 lists the affected states. In only two cases have acceptable I/M programs failed to be authorized—in California and Kentucky's Kenton and Campbell counties. California has I/M programs for vehicles for which the ownership and registration is being transferred. However, the inspections required by these programs are considered too infrequent to produce sufficient emission reductions. The state legislature failed to enact statewide legislation authorizing the operation and enforcement of I/M programs.

TABLE 10

AREAS OR STATES CONTAINING AREAS
REQUIRED TO IMPLEMENT
I/M PROGRAMS

Arizona	New Jersey
California	New Mexico
Colorado	New York
Connecticut	North Carolina
Delaware	Ohio
Georgia	Oregon
Illinois	Pennsylvania
Indiana	Rhode Island
Kentucky	Tennessee
Maryland	Texas
Massachusetts	Utah
Michigan	Virginia
Missouri	Wisconsin
Nevada	Washington

Washington, D.C.

Because the federal Clean Air Act gives no specific direction as to how I/M is to be implemented, the details of the various proposed and operating programs vary from state to state. Table 11 shows some of the more important features of some programs already in operation.

The core of the I/M program is the requirement that certain classes of motor vehicles be tested periodically to determine their exhaust emission levels (inspection). When emission levels exceed the standard, repairs or adjustments must be made (maintenance) to bring the emissions levels back within the standards.

As can be seen from Table 11, programs differ in terms of the type of vehicles required to undergo testing, administering agencies (local or state), frequency of inspection, enforcement mechanisms, cost of inspections and repair limits.

For example, some states are phasing in mandatory I/M programs by requiring mandatory inspections with *voluntary* maintenance for a period of time before mandatory inspections and *mandatory* maintenance will be required. By December 31, 1982, however, all programs will have to have mandatory inspection and mandatory maintenance.

TABLE 11

DESCRIPTION OF OPERATING I/M PROGRAMS

ITEM	NEW JERSEY	CINCINNATI	OREGON	ARIZONA	NEVADA	RHODE ISLAND	CALIFORNIA
Geographic Location	Entire State	Cincinnati & Norwood, Ohio	Portland	Pima and Miracopa Cty.	Clark & Washoe Cty.	Entire State	South Coast Air Basin (LA Area)
Date of Inspection Mandatory Voluntary	Feb. 1, 1974 July 5, 1972	Jan. 1, 1975	July 1, 1975	Jan. 1, 1977 Mandatory Repairs. Jan. 1, 1976 Mandatory Inspection/Voluntary Repairs	Change of Owner 7-1-74 + New Regis. Owner 7-1-77. Annual (Clark Only) Jan. 1, 1980	Jan. 1, 1979 Nov. 1, 1977	March 19, 1979
Coverage (LDV-Light Duty Vehicle)	All LDV's less than 6000 lbs. GVW (Gross Vehicle Weight)	All LDV's less than 6000 lbs. 160,000 vehicles	All vehicles 500,000 vehicles	All vehicles 1,200,000 vehicles	All LDV's less than 6000 lbs. GVW. 330,000 vehicles	All LDV's less than 8000 lbs. GVW. 500,000 vehicles	All LDV's less than 8500 lbs. GVW. 1,200,000 vehicles
Exemptions	Diesels, vehicles less than 50 cu. in. pre 68 2 stroke Saabs, new cars for first 2 yrs.	Diesels (emissions only). Motorcycles, historical vehicles (over 25 yrs., collectors items)	HDV diesels over 8500 lbs GVW, motorcycles, farm plated vehicles, fixed & restricted load vehicles. Interstate vhls.	Vehicles over 13 yrs. old. Prorated vehicles (Interstate vhls)	65 and over 13 yrs. old Prorated vehicles (Interstate vehicles)	Diesels, new vehicles for 12 months or 12,000 miles. Farm vehicles over 25 yrs. old, motorcycles.	Diesels, motorcycles dual fuel or complete fuel conversions.

TABLE 11 (con't.)

DESCRIPTION OF OPERATING I/M PROGRAMS

ITEM	NEW JERSEY	CINCINNATI	OREGON	ARIZONA	NEVADA	RHODE ISLAND	CALIFORNIA
Type of Program	Centralized - State Oper.	Centralized - City Oper.	Centralized - State Oper.	Centralized - Contractor Oper.	Decentralized-Private Garage	Decentralized-Private Garage	Centralized-Contractor
Administering Agency	Dept. Motor Vehicles/ Dept. Env. Prot.	Cinn. Dept. of Sewers	Ore. Dept. of Environmental Quality	Bur. of Veh. Emission Insp. Div. of Environ. Health Services	Dept. of Motor Vehicles	Rhode Island Dept. of Transportation	Bur. Auto Repair/ Air Res. Board
Inspection Frequency	Annual	Annual	Biennial-LDV's Annual-HDV's	Annual	Change of Owner/Annual (Clark Cty Only)	Annual	Change of Owner/ New Registered Owner
Enforcement/Fines	Sticker & Registration $100 max.	Sticker-Cinc. $11-35 Norwood $15	Registration $100 max.	Registration $8 late reg.	Registration up to 6 mo. and $500	Sticker-Road Checks $15	Registration Variable fines
Inspection Cost	$2.50 (incl. safety) $1.00 for reinspection at private garages	$3.75 (incl. safety) free retests	$5.00 free retests	$5.00 incl. one free retest	$12.50 - $17.00	$4.00 incl. safety	$11.00 $7.00 reinspection

SOURCE: Greg H. Lazarus. Survey of Operating and Proposed State and Local Vehicle Emissions Inspection/Maintenance (I/M) Programs. Denver: National Conference of State Legislatures. August 20, 1980.

States also differ regarding which vehicles come under the I/M program. Some programs include all motor vehicles; others include only light duty vehicles (less than 6,000-10,000 lbs.). Exemptions may exist for vehicles which are over a certain number of years old. Arizona and Nevada exempt vehicles over thirteen years old, as well as all interstate vehicles. New Jersey, Rhode Island, and California all exempt diesels.

Programs may apply statewide or may apply only to specific counties. The inspections may be conducted by government established inspection stations (centralized) or by government contract (centralized) or by private garages (decentralized). EPA distinguishes between centralized and decentralized programs, requiring that decentralized programs be in operation by December 31, 1981 and centralized by December 31, 1982. (An extension of one year for decentralized programs may be obtained if computerized instruments for analyzing emissions are to be used.) In most cases inspections are conducted annually. They may involve only an analysis of the exhaust pipe emissions or they may include visual inspections for tampering. The test may be conducted while the vehicle is idling in place or it may be done by equipment designed to test emissions at a variety of simulated speeds.

In most states, if repairs for a vehicle which fails the emission test cost above a given amount, a waiver may be granted. Some states also offer training programs to familiarize mechanics with the major problems associated with emission control system failures and the ways to correct them.

Of the programs currently operating, inspection fees range from $2.50 in New Jersey to $12.50-$17.00 in Nevada.

One of the most important features of any I/M program is the manner in which it is enforced. There are two methods of enforcement. One is the sticker approach; the other is the registration approach. Under the sticker approach a vehicle owner is given a sticker to place in a visible spot on the car (windshield or bumper) to indicate that the vehicle has successfully passed the emission test. A vehicle granted a waiver receives a different sticker. Any vehicle required to be inspected which does not display a valid sticker is subject to a fine. Under a registration program, a person cannot renew the annual car registration without verification that the vehicle has passed inspection or received a legal waiver. Since the registration approach checks every vehicle annually it is considered by far a more effective enforcement approach.

The actual standards which exhaust emissions are required to meet also vary from program to program. EPA has set the goal of a 25 percent reduction by 1987 in levels of hydrocarbon (HC) and carbon monoxide (CO) emissions from automobiles compared to the levels projected for 1987 without I/M. Consequently, the level set by each program is designed to meet this goal. Thus "cut points" (the level of emissions distinguishing those who pass from those who fail) are established to achieve a desired emission reduction and at the same time "fail" a number that will generally be in an acceptable range to the public and the repair industry.

Consequently, there is a range of options open to a locality charged with implementing I/M. These options are limited by U.S. EPA reguirements. The Tenth Annual Report of the Council on Environmental Quality suggests the need to analyze the source of the problem and to design a site-specific program.

> Inspection and maintenance programs should be tailored to the cause of the problem. For example, if an unacceptable percentage of car owners tamper with their control systems, a roadside spot-check program may be the most appropriate strategy. However, if most owners are simply failing to maintain the

devices, an annual inspection and maintenance program for all may be a better choice.[17]

The Impact of I/M on Air Quality

States with I/M programs that have been in operation a reasonable length of time show the following impacts on air quality improvement, according to a 1980 report by the National Conference of State Legislatures. Note the distinction between reductions in CO and HC emissions of the tailpipe and changes in the overall quality of the ambient air for CO, HC and ozone.

New Jersey

The New Jersey Department of Environmental Protection (DEP) estimates that exhaust hydrocarbon (HC) emissions in 1979 were reduced by 15 percent, and carbon monoxide (CO) emissions by 26 percent. The DEP reports an 8 percent per year monitored improvement in ambient CO levels with I/M compared to an estimated average 5 percent improvement since 1974 in areas without the programs. Although ozone (O_3) reductions are difficult to quantify, the DEP reports fewer violations of the ozone air quality standard since the beginning of the I/M program in 1974.

The difficulty of extrapolating the reduction in auto exhaust hydrocarbon emissions to changes in ozone levels in the air is summarized as follows by Mr. John Elston, Chief of the New Jersey Bureau of Air Quality, Management and Surveillance:

> The ozone problem in New Jersey is difficult to quantify for several reasons. For one, the New York-New Jersey-Pennsylvania area contains substantial amounts of stationary hydrocarbon (HC) sources (petroleum refineries, chemical plants, etc.). For another, the northeast is generally downwind of HC sources during much of the ozone season. When these non-vehicular and transport effects are added to other variabilities, such as seasonal meteorology and the fact that hydrocarbons are precursors and not the primary pollutant, drawing conclusions about I/M and its impact on ozone is difficult indeed.[18]

Oregon

The Department of Environmental Quality (DEQ) estimates tailpipe emission of CO reduced 25%, and HC emissions reduced 8% (since 1976). The DEQ estimates that ambient CO levels have been reduced by 20% since 1974, and HC levels by 15%. Studies of tailpipe emission reductions in Portland show that I/M may be more effective than predicted in reducing auto emissions. The impact on ozone levels in the ambient air is undergoing further study.

Arizona

Test data indicates that exhaust CO levels have been reduced about 36% from the 1976 levels. The reduction for exhaust HC levels are estimated to be 56.3% (at idle), 51.2% (low speed cruise), and 47.5% (high speed cruise). When corrections are made for the increase in vehicle miles traveled, a 25% improvement in CO ambient levels has been reported. However, no improvement in ambient ozone levels has been quantified.

In Arizona, I/M was implemented primarily for control of CO rather than ozone.[19] The lack of change in ozone concentrations in the atmosphere has been attributed to the numerous other

sources of hydrocarbons besides auto emissions, including gasoline service stations and tank farms, manufacturing operations, painting, printing and dry cleaning.[20]

Data on the programs in California and Nevada are limited.[21]

California

Exhaust reductions from the change of ownership program are: 11% for HC, 15% for CO, and 2% for nitrogen oxides (NOx). The program has not been in operation long enough to quantify any ambient air quality improvement.

Nevada

Exhaust emissions of CO are estimated to be 39% lower than before the I/M program began. HC exhaust reductions are estimated at 33%. Data on improvements in ambient air quality is not yet available.

It appears generally that I/M has proven to be effective in reducing CO and HC emissions at the tailpipe. In addition, in the case of CO, this reduction has corresponded with decreases in the concentration of CO in the ambient air. In the case of ozone, the tailpipe HC reductions have not been specifically related to changes in ozone levels in the ambient air. This lack of specific correlation may be attributed to the complex factors involved in ozone formation and transport and the variety of sources emitting hydrocarbons. Additional studies on the relationship between ozone levels in the air and HC emissions from automobile exhausts are now in progress.

This correlation between tailpipe emission reductions and improvements in overall ambient air quality for CO and ozone is a key point in the controversy over the federal requirement for implementation of I/M programs.

Opponents of I/M consider it to stand alone as a control strategy and question its necessity in controlling ozone levels. This group predicts that the ozone standard for ambient air quality will be reached in most communities without I/M as new cars meeting more strict emission standards replace older cars.

Proponents of I/M consider it not as an alternative to the federal motor vehicle emission control program (FMVCP) but as complementary to it. They hold that emissions from new cars will only continue to stay within the standards if the cars are properly tuned and maintained. I/M is a check to assure that proper car maintenance and repairs are done and that emission control equipment remains in working order.

An example of the interrelatedness of I/M and new car emission standards can be seen from an analysis of the New Jersey Program. New Jersey was successful in reducing CO levels 8% per year, whereas an improvement of 5% per year was calculated for areas without I/M. One could assume that this 3% difference is the result of the I/M program. However, an analysis done by the University of Wisconsin concludes the following:

1) Statistically significant improvement was monitored from all the carbon monoxide stations examined;

2) the improvement in carbon monoxide levels could not be attributed to meteorological conditions;

3) the improvement in carbon monoxide occurred at a time when traffic volume was increasing (apart from a temporary and short-lived reduction in traffic due to the oil embargo of 1973-74); and

4) the improvement *can* be attributed to the progressively more stringent federal motor vehicle program *and* (emphasis added) the New Jersey I/M program.[22]

In other words, contributions from I/M and the FMVCP in CO reductions are "best interpreted jointly."[23]

The Council on Environmental Quality, in its Tenth Annual Report based on a study by Kay H. Jones, points toward a similar conclusion concerning automobile-related pollutants:

> CEO has calculated that if all counties were to achieve a 40-percent reduction through any combination of controls during the next decade, then the number of counties (excluding those in the Los Angeles Basin) in non-attainment for ozone would decline from 184 in 1979 to 85 in 1982. This projection is based on the assumption that most counties will be able to achieve a 40-percent reduction in ozone levels simply through the gradual introduction of autos and trucks equipped with emission controls. By 1988, 16 counties outside of Los Angeles would still require more stringent and complex control strategies.[24]

However, CEO points out that automobile emission controls will have the greatest impact on attaining CO and ozone standards and that "vehicle emission control systems must not only be in place; they must be performing at an acceptable level."[25] This is where the significance of an I/M program emerges. Consequently, I/M programs are required by Congress in areas where the ozone standard will not be attained by 1982, to assure proper maintenance on vehicles to control their emissions.

On the other hand, some independent studies reported by Jones (1980) conclude that although data in New Jersey shows improvement in air quality at a rate greater than the national trend for CO, the same is not true for Portland or Phoenix, both of which have I/M programs. When CO data in Portland was compared with data for Seattle—in the same geographic area but without I/M—no significant improvement could be shown for Portland.[26]

Jones also challenges the argument that I/M is needed to achieve ambient CO and ozone standards by 1987 in many areas or that I/M is justified from a public health perspective.

A key factor in all of this is how fast deterioration occurs; that is, at what rate vehicle emission levels get worse as new cars age.

Using data for the Philadelphia Air Quality Control Region for 1976-1978, and a different deterioration factor from that of EPA, Jones found that the region would attain the ozone standard by 1987 without I/M.[27]

In order to determine deterioration rates the U.S. EPA is conducting a study of automobiles in Portland, Oregon, which has an I/M program, and Eugene, Oregon, which does not. The findings regarding deterioration, as reported in June, 1980, are as follows:

> Results from this part of the study, as reported last year, show that emission reductions immediately following maintenance are substantial, and that benefits accrued over a year's time are also large, even after deterioration. The 1975-77 failed vehicles experienced a 47% reduction in CO emissions following maintenance, and a corresponding 42% reduction in HC emissions. Over the course of a year, HC emissions deteriorated almost back to original levels, but about 40% of the initial reduction in CO emissions was still present at the end of the year. Thus, benefits were derived from I/M over a full year. Total CO emissions over the year were 36% lower for 1975-77 cars than they were in Eugene (the non-I/M case). HC emissions were 20% lower.[28]

For 1972-74 model year cars the final benefit over the year was calculated to be 16% for HC and 35% for CO. The study showed that HC levels return to premaintenance levels by the end of the third quarter. The CO benefit, however, remains significant throughout the entire year.[29]

Preliminary data from the Portland study also indicates that the tailpipe emission reductions from I/M are greater than those originally predicted by EPA.

Today there are more questions than answers regarding the impact of I/M on overall air quality and subsequently on human health. HC and CO emissions from auto exhaust have been reduced by I/M. How these reductions correlate with improvements in overall air quality is still under study, particularly where ozone is concerned. Will changes in auto exhaust emissions from new car standards be sufficient to attain ozone standards by 1987 even without I/M? What are the health implications of *not* implementing I/M programs to hasten attainment of ozone standards? Do EPA modeling programs actually reflect and accurately predict what will happen in the field? There appears to be no general agreement in these areas; the dispute is likely to continue as field studies progress and data analysis continues.

I/M: Cost and Cost-Effectiveness

Tables 12 and 13 show some basic capital and operating costs for I/M programs compiled by the National Conference of State Legislatures. This analysis shows that capital costs run from $.08 per car in Cincinnati to $1.67 per car in Rhode Island. Operating costs run from $.33 per car in Cincinnati to $4.00 per car in Oregon. Table 11 sets out the specifics of these operating programs which will have a bearing on cost per car.

Some data from the Portland, Oregon, program relate total emission reductions to cost. These figures are based on the total tons of HC and CO calculated to have been eliminated and the total cost for inspection and repair. Data for 1975-77 model cars put cost per ton of pollutant eliminated for one year at $1,400 for HC and $60 for CO.[30] For 1972-74 model year cars the cost per ton of pollutant eliminated was about $1,200 for HC and $40 for CO.[31]

Based on its own models, EPA estimates the range of I/M cost-effectiveness for the first year to run from $600-$1,500 per ton of HC eliminated and $30-$50 per ton of CO. No health and welfare benefits and no fuel economy benefits are included in these calculations.

> EPA goes on to clarify, however, that the cost-effectiveness of a program can be affected by such factors as test mode, stringency of program, cost or age waivers, emission deterioration with and without I/M, mechanic training programs and type of program (centralized or decentralized). Although the effects of some of these factors on costs and effectiveness have been investigated, no comprehensive quantitative summary of the sensitivity of cost-effectiveness to these various factors is available now.[32]

A study on the cost of the I/M program in New Jersey was conducted by Palmini and Rossi in 1980.[33] Their analysis attempts to assess total costs to the state government and to the motoring public. Costs to the private service station industry are not included, since these costs are recouped in the charges to the vehicle owner.

TABLE 12

CAPITAL COSTS

	NEW JERSEY	CINCINNATI	OREGON	ARIZONA	CALIFORNIA	NEVADA	RHODE ISLAND
Land				$ 99,000			
Test Facilities			$ 80,000				$750,000 bldg. improvements for safety test of public vehicles.
Analyzers & Calib. Materials	$250,000 (1972) 582,000 (1980)	$12,600	$217,500				
Office Space/ Supplies				$270,000			
Laboratory/Challenge Station						$42,000	0 - Use safety facilities
Laboratory Equipment	$100,000			$150,000	$ 10,000	$20,000	$ 10,000
Calibration Equipment	$ 10,000						
Data Processing Equipment (Software development, etc.)	$ 20,000				$260,000		$ 54,000
Vehicles	$287,000				$ 30,000	$ 6,000	
Enforcement Equipment	$ 40,000						
Public Information Material							$20,000 (EPA funded)
TOTAL	$707,000 (1972) $1.039.000 (1980)	$12,600	$297,500	$519,000	$300,000	$68,000	$834,000
(Per Car)	(.19)	(.08)	(.60)	(.43)	(.25)	(.20)	(1.67)

SOURCE: Greg H. Lazarus. Survey of Operating and Proposed State And Local Vehicle Emissions Inspection/Maintenance (I/M) Programs. NCSL. August, 1980.

TABLE 13

OPERATING COSTS

	NEW JERSEY (Incremental for I/M)	CINCINNATI (Incremental for I/M)	OREGON* (Incremental for I/M)	ARIZONA (Incremental for I/M)	CALIFORNIA (Incremental for I/M)	NEVADA (Incremental for I/M)	RHODE ISLAND Total for I/M and Safety
Vehicle Inspectors Salaries	$600,000 (re-classification to include I/M)	$50,000	$950,000	0			$ 200,000
Other Salaries (Supervisor, Q.A., Trainers, etc.)	$1,400,000		$330,000	$389,000	$1,500,000	$201,000	$ 760,000
Maintenance & Office Supplies	$ 75,000		10,000	38,000	Incl. w/ misc.	$ 35,000 ($15,000 for printing)	0
Travel	120,000		20,000	24,000	75,000	7,500	52,000
Data Processing	5,000			35,000	65,000		0
Leases			300,000	25,000	Incl. w/ misc.		
Miscellaneous			400,000 (payroll acct., word process., and other gen. DEQ charges)	6,000	760,000	$5,000 prep. of documented vehicles for enforcement	
TOTAL	$2,200,000	$50,000	$2,000,000	$517,000	$2,400,000	$248,500	$1,012,000
(Per Car)	(.58)	(.33)	(4.00)	(.41)	(2.00)	(.73)	(2.02)

*Biennial

SOURCE: Greg H. Lazarus. Survey of Operating and Proposed State and Local Vehicle Emissions Inspection/Maintenance (I/M) Programs. NCSL. August, 1980.

Table 14 shows the annual I/M program costs calculated for New Jersey in 1977. It should be noted that in New Jersey the I/M program was coupled with the safety inspection program; this practice would keep overall costs somewhat lower than those of an independent program. Also the figures in Table 14 differ from those in Tables 12 and 13 for the New Jersey program, because different costs were included in each analysis. The Net Annual Cost to the Motoring Public includes values for the time involved in the initial emission inspection, repair, and reinspection; cost of traveling for repairs and reinspection; cost of fines for driving without a current inspection sticker; as well as fuel economy gains and engine maintenance benefits from tuneups. Total government costs include general planning, equipment purchase and installation, initial training programs and cost associated with private reinspection systems. The values for annual operating and maintenance costs include administration and cost associated with private reinspection systems, labor and equipment maintenance and repair, and training and public education programs. Using EPA models, the estimated motor vehicle emissions for 1977 were calculated under conditions without an I/M program and then with an I/M program. The difference in tons of pollutant emitted resulted in a value for emissions reduction due to I/M. This amounted to 30,900 tons of HC and 603,000 tons of CO. The cost of the program per ton of pollutant reduction was then determined, as shown in Table 15.

TABLE 14

ANNUAL EMISSIONS INSPECTION/MAINTENANCE PROGRAM COSTS, 1977

	Aggregate Value	Cost/Vehicle
Cost to Motoring Public		
Total Cost	$16,799,200	$4.57
Less: Value of fuel economy gains	- 7,319,700	- 1.99
Net annual cost to motoring public	$ 9,479,500	$2.58
Cost to state government		
Annualized capital costs	216,600	0.06
Annual operating and maintenance	840,800	0.23
Total	1,057,400	0.29
Net total program cost	$10,536,900	$2.86

SOURCE: Dennis Palmini and Daniel Rossi, "What Price Air Quality? The Cost of New Jersey's Inspection/Maintenance Program," *Journal of the Air Pollution Control Association*, Volume 30, No. 10 (October, 1980), p. 1084.

TABLE 15

COST OF INSPECTION/MAINTENANCE PER UNIT REDUCTION IN EMISSIONS

Cost item	Cost per ton reduction		
	Hydrocarbons	Carbon monoxide	Total pollutants
Cost to motoring public			
Total Cost	$543.66	$27.86	$26.50
Net Cost	306.77	15.72	14.96
Cost to state government	34.22	1.75	1.67
Total program cost	577.88	29.61	28.17
Net program cost	$341.00	$17.47	$16.62

SOURCE: Dennis Palmini and Daniel Rossi, "What Price Air Quality? The Cost of New Jersey's Inspection/Maintenance Program," *Journal of the Air Pollution Control Association*, Volume 30, No. 10 (October, 1980) p. 1085.

Consequently the cost of I/M programs have been analyzed in a variety of different ways. As in many environmental areas, the cost of a program is much easier to determine than the monetary value of the benefits derived. As we have seen, the case of I/M is especially complicated because the reduction of emissions from automobile exhaust is difficult to translate into improved air quality. However, a general picture of benefits from improved air quality can be drawn.

A study done for the Council on Environmental Quality attempted to review and standardize the major analyses which have been done over the years to calculate pollution control benefits. The study presents a range of estimates for benefits from pollution control and indicates a value that from all available data appears to be the most reasonable within that range. Table 16 shows the ranges calculated in that study for benefits from air pollution control since 1970. The "benefits" used in calculating these values include:

> improvements to human health, reduced soiling and cleaning costs for households, reduced damages to vegetation and crops, and reduced damages to materials. It also reviewed estimates of air pollution control benefits derived from studies of property value differences. Property value measures will tend to capture benefits in some or all of the above four categories. Thus to add them to the other measures would likely involve double counting. However, the property value measures may also capture aesthetic values, which are not included in any of the above categories. On the basis of a survey of households in Los Angeles conducted by David Brookshire and others at the University of Wyoming, it was estimated that 30 percent of property value benefits were the result of improvements in aesthetic values and should be added to the benefit estimates in the other categories.[34]

The author of the study cautions that these benefit figures do not include values for prevention of further degradation of the air due to environmental controls; nor do they include benefits to Canada and Mexico from a decrease in transport of pollutants across the border; nor do they contain values for improved visibility. In other words, they could be considered conservative figures.

As shown in Table 16, the total benefits for health, vegetation, materials and property values attributed to control of mobile sources ranged from $.5 to $5.1 billion dollars, the most reasonable figure being around $1.5 billion (in 1978 dollars).

TABLE 16
AIR POLLUTION CONTROL BENEFITS SINCE 1970 BEING ENJOYED IN 1978
(IN BILLIONS OF 1978 DOLLARS)

CATEGORY	REALIZED BENEFITS RANGE	REALIZED BENEFITS MOST REASONABLE POINT ESTIMATE
1. Health		
A. Stationary Source	$ 2.8 - 27.8	$13.9
Mortality		
Morbidity	.29 - 11.5	2.9
TOTAL	$ 3.1 - 39.3	$16.8
B. Mobile Source	0 - .4	.2
TOTAL HEALTH	$ 3.1 - 39.7	$17.0
2. Soiling and Cleaning	$ .5 - 5.0	$ 2.0
3. Vegetation		
A. Stationary Source	0	0
B. Mobile Source	$ .2 - 2.4	$ .7
TOTAL VEGETATION	$ .2 - 2.4	$ .7
4. Materials		
A. Stationary Source	$ .4 - 1.1	$ .7
B. Mobile Source	.1 - .3	.2
TOTAL	$ .5 - 1.4	$ .9
5. Property Values		
A. Stationary Source	$ .9 - 6.9	$ 2.3
B. Mobile	.2 - 2.0	.4
TOTAL	$ 1.1 - 8.9	$ 2.7
GRAND TOTAL*	$ 4.6 - 51.2	$21.4

* Because of overlap, only 30 percent of property value benefits are added to other categories (See text).

SOURCE: A. Myrick Freeman III. "The Benefits of Air and Water Pollution Control: A Review and Synthesis of Recent Estimates". A report prepared for the Council on Environmental Quality, December, 1979.

How these figures might apply to an individual I/M program would depend on a site-specific analysis. They do indicate, however, that there are health and property benefits from improved air quality from mobile source controls. The cost of I/M and its benefits are two sides of the same coin; one must not only consider the cost of an I/M program but also the "cost" to the community of not having one. A variety of complex factors is involved and the outcome depends on the specific analysis which is made and the weight given to the various factors.

One might also want to consider the cost of alternatives to I/M for pollution control. The primary alternative to controlling HC emissions is the control of industry. In many cases industry has already been required to control its HC emissions. The cost of additional control on industry will depend on, among other things, the degree to which HC emissions are already controlled. Each increment closer to 100% control becomes more and more costly from a monetary perspective. In fact as control systems attempt to remove 90% plus of the pollutants involved, the costs rise almost exponentially, thus making the effort less and less cost-effective. Cost per ton of removal would increase dramatically.

Carbon monoxide (CO) is produced almost solely from auto exhaust and I/M is currently considered the most effective means for controlling CO levels.

Table 17 summarizes other alternatives for control of auto-related pollutants. Their costs, their effectiveness in reducing emissions, their applicability on a large scale, and their current technological feasibility all vary. Their current usefulness in addressing the problems associated with auto emissions control appear minimal.

TABLE 17

AUTO EMISSIONS: ALTERNATIVE CONTROLS

TYPE ALTERNATIVE ENGINES	ADVANTAGES	DISADVANTAGES
Diesel Engine	Can meet HC and CO standards. Better fuel mileage. Low evaporation losses of fuel.	Health related - Emits Carcinogenic carbon compounds. May not meet NO_x standards.
*Stirling Engine (External Combustion Engine)	Low pollution emissions. Good fuel economy. Low noise and vibration. Uses a wide range of fuels.	Technology still undergoing research; not expected to reach market before 1988.
*Gas Turbines (External Combustion Engine)	Low pollution emission.	Technical problems with fuel economy requiring high operation temperatures and special ceramic parts. May not meet NO_x standards. Practicality probably not known until 1984
Electric Vehicle	Emits no pollutants. Saves petroleum based fuels.	Small size. High Cost. Power and range between charges.
ALTERNATIVE FUELS		
Synthetic Petroleum Fuels (from oil shale and coal)	Saves petroleum based fuels.	Pollution control problems for HC. CO, and NO_x same as with petro-based fuels Possible emissions of carcinogens.
Gasohol	Saves petroleum based fuels. Can be used with conventional engines.	No pollution control advantages because the alcohol content is small (Around 10% usually).
Pure Alcohol Fuels	Lower emissions of CO, HC, and NO_2 (compared to gasoline)	Require major modifications in existing engines. Emissions of aldehydes; technical problems with starting and corrosion.
Hydrogen	No HC or CO emissions except from burning of lubrication oil. Can be used in conventional engines.	Still at research level. Problems with combustion and high NO_2 emissions.

*In external combustion engines fuel is burned at a steady pressure and temperature, as contrasted to internal combustion which have cyclic explosive combustion.

Source: Council on Environmental Quality, Tenth Annual Report, 1979.

CONCLUSIONS

In developing this report, many documents relating to air quality were reviewed, and many personal contacts were made by LRC staff with local, state, and federal officials knowledgeable or concerned about the quality of air in Kentucky. Based on this literature review and personal contacts, the following conclusions have been made:

(1) The understanding and control of human-made pollutants in the atmosphere is very complex and difficult. In an attempt to understand air pollutants, substantial scientific research has been conducted, and in an attempt to control these pollutants very comprehensive laws and regulations have been developed.

(2) Many pollutants in our atmosphere originate from both natural and human-made sources. However, due to the rapid industrial and technical expansion of society, the ratio of human-made to naturally-generated pollutants has increased substantially, especially in metropolitan and highly industrial areas.

(3) For some air pollutants, the biological and enivonmental effects seem well established, whereas such effects for other pollutants appear less certain, and require additional research.

(4) The federal government, under authority and requirement of the Clean Air Act, as amended, has established stringent requirements and timetables for states to meet primary national ambient air quality standards.

(5) The federal requirements for the proper surveillance and monitoring of pollutants in the atmosphere are comprehensive and detailed. The methods of site selection, sample collection, sample analysis and interpretation, quality assurance, type of instrumentation and procedures of reporting are detailed in various federal guidelines, regulations, and standards. The Kentucky Department for Natural Resources and Environmental Protection (DNREP) has adopted and now uses appropriate methods and procedures to obtain reliable monitoring data, and meets federal criteria for acceptable monitoring procedures. Although DNREP applies the best available models in predicting long-range pollution impact and required reduction to meet standards, it would appear that there is a need to improve the accuracy of some of these models, especially as related to ozone.

(6) The formation and dispersion of ozone in the atmosphere is very complex, both physically and chemically. Although much progess has been made in the last ten years in understanding the formation and dispersion of ozone and other photochemical oxidants, there are still a great many "unknowns." Continued research is required to more precisely understand and predict the formation and movement of ozone in the atmosphere.

(7) It appears there is limited information available on the formation of ozone from new or unusual human-made sources. Present research shows the two major human-made emission source categories for oxides of nitrogen are transportation-related sources and stationary fuel combustion. Emissions of volatile organic compounds (VOC), or hydrocarbons, are primarily from transportation-related sources and such industrial processes as chemical manufacturing, petroleum refining, oil and gas production and marketing and industrial organic solvent use.

There appears to be no data to indicate significant generations of ozone from street and highway lighting systems. Some researchers have indicated the formation of ozone from coronal discharges around high voltage transmission lines, although most researchers believe the amount of such ozone formation, if any, is insignificant.

(8) The reduction of ozone formation, or more specifically, the reduction of ozone precursors, by the installation of gas clean-up systems, is a method which an operator of generating sources can apply to meet the emission standards. Such systems may be necessary to meet emissions standards for VOC and oxides of nitrogen, which lead to ozone formation.

(9) Facing potential financial loss of federal funds and other sanctions, due to being designated "non-attainment" for ozone, Jefferson County/Louisville and Boone County fiscal courts have adopted ordinances providing authority for the implementation of a mandatory automobile inspection and maintenance program. The two counties of Campbell and Kenton are also facing federal sanctions, since these counties have not adopted such ordinances. In light of these federal sanctions, the three northern counties (Boone, Campbell, and Kenton) have filed a petition in 6th District, U.S. Court of Appeals, requesting the Court to review the EPA's decision on levying sanctions. (At the time of this writing, no action had been taken on the petition.)

(10) A special technical consultant to the three northern counties has raised several questions on the validity of the data used to determine "non-attainment" status for ozone in the metropolitan Cincinnati Interstate Air Quality Control Region. However, the U.S. EPA, after review of the issues raised by the consultant, concluded the decision for designating the area non-attainment was correct, and that the conclusion of the consultant "throws no doubt on the validity of the Kentucky State Implementation Plan." Additionally, a requested independent review of the question raised by the consultant by an individual trained in air pollution indicated that the identified specific reduction of hydrocarbons in the Cincinnati Area was accurate.

(11) Twenty-eight states and Washington, D.C. are required to comply with the I/M provisions of the Clean Air Act. All but California and the counties of Boone and Kenton in Kentucky have authorized the necessary programs. Because the federal Clean Air Act gives no specific direction as to how I/M is to be implemented, the details of various operating and proposed programs vary from state to state, including the type of vehicles required to undergo testing, the frequency and cost of inspections, the emission levels which must be met, and the enforcement mechanism. Requiring evidence that testing has been done for renewal of licenses is the most effective enforcement option.

(12) The impact of I/M on air quality has been examined by using data from programs already in operation. It appears that I/M has generally proven to be effective in reducing CO and HC emissions at the tailpipe. The impact on ambient air quality is not so clear cut. In the case of CO there is some correlation between reduction of tailpipe emissions and reduction of CO in the ambient air. Since 90% of CO comes from vehicles, this is to be expected. In the case of ozone, the tailpipe HC reductions have not been specifically translated into changes in ozone levels in the ambient air. This lack of correlation is due to the complex factors involved in ozone formation and transport and the variety of sources emitting hydrocarbons. Further studies are being conducted to establish the impact of I/M programs on ambient ozone levels.

(13) The correlation between tailpipe emission reductions and improvements in the ambient air quality is a key point in the controversy over the requirement for implementation of I/M programs. Opponents of I/M claim that the ozone standards for ambient air quality will be reached in most communities without I/M as new cars meeting more strict emissions standards replace older cars. Proponents of I/M point out that the new cars will continue to stay within the standards only if they are properly tuned and maintained, a purpose that I/M programs serve.

(14) Capital and operating costs for I/M programs vary from program to program. A variety of analyses have been done to arrive at program costs. A key to the question of cost-effectiveness is

how much it will "cost" in terms of human health and property if an I/M program is not implemented. I/M is the most effective method of controlling CO emissions, since vehicles are the major source of CO in the air. The alternative to controlling HC emissions from automobiles is to control emissions from industry. The cost of additional control of HC emissions on industry depends on the degree to which HC emissions are already controlled; as control systems approach the removing of 90% plus of the pollutants involved, costs rise almost exponentially. Alternatives to I/M for control of auto-related pollutants, such as new engine types or new fuel types, are generally so far from being ready for mass marketing that they would not have any significant impact in the immediate future on air quality.

FOOTNOTES

1. *Revisions to the Implementation Plan—For the Attainment and Maintenance of the National and State Ambient Air Quality Standards*, Kentucky Department for Natural Resources and Environmental Protection (June, 1979), p. 2-20.

2. *State Implementation Plan*—1979, p. 2-20 and p. 2-24.

3. *Identification of Non-Attainment Areas in the Commonwealth of Kentucky*, A report submitted to the Administrator, U.S. EPA, by Kentucky Department for Natural Resources and Environmental Protection (December 5, 1977), p. 3.

4. *Kentucky Administrative Regulations Service*, 401 KAR 53:010, Appendix A (1980).

5. *Kentucky Administrative Regulations Service*, 401 KAR 50:015 (1980).

6. *Kentucky Ambient Air Quality 1979 Annual Report*, Kentucky Department for Natural Resources and Environmental Protection (1979), p. 28.

7. *Air Quality Criteria for Ozone and Other Photochemical Oxidants*, U.S. EPA publication EPA-600/8-78-004 (April 1978), p. 1.

8. Report of National Commission on Air Quality, as quoted from *Ozone: Formation, Transport, and Control*, Greg H. Lazarus, National Conference of State Legislatures (August 1980), p. 6.

9. Ibid.

10. *Ozone: Formation, Transport, and Control*, Lazarus, p. 8.

11. *Federal Register*, Volume 45, No. 241 (December 12, 1980), p. 81752.

12. Letter to Governor John Y. Brown from David G. Hawkins, Assistant Administrator for Air, Noise and Radiation, dated August 8, 1980.

13. Letter to Charles Hardin, LRC Staff, from Greg H. Lazarus, Staff Associate, Office of Science and Natural Resources, National Conference of State Legislatures, dated November 5, 1980.

14. *Better Enforcement of Car Emission Standards—A Way to Improve Air Quality*, a report of the U.S. General Accounting Office (January 23, 1979).

15. Policy for the Development and Implementation of Inspection/Maintenance Programs, included in *Motor Vehicle Emissions Inspection/Maintenance*, Greg H. Lazarus, National Conference of State Legislatures (May, 1979).

16. State Implementation Plan—1979, p. 5-22.

17. Council on Environmental Quality, *Environmental Quality—1979*, The Tenth Annual Report (Washington, D.C.: U.S. Government Printing Office, December 1979), p. 63.

18. Letter from Mr. John Elston, Chief of the New Jersey Bureau of Air Quality, Management and Surveillance, to Mr. Charles Hardin, LRC Staff, dated November 10, 1980.

19. Letter from Mr. R. Fred Iacobelli, Chief, Bureau of Vehicular Emissions, Arizona Department of Health Services, to Mr. Charles Hardin, LRC Staff, dated October 21, 1980.

20. R. Fred Iacobelli. *Effectiveness of the Arizona Emissions Inspection/Maintenance Program*. SAE Technical Paper Series: Warrendale, PA: Society of Automotive Engineers, August, 1980.

21. Greg H. Lazarus. *Survey of Operating and Proposed State and Local Vehicle Emissions Inspection/Maintenance (I/M) Programs*. Denver: National Conference of State Legislatures, August, 1980.

22. John Elston. "The Calm After the Storm," *EPA Journal*, Washington, D.C.: Office of Public Awareness, October, 1978, p. 20.

23. Ibid.

24. Council on Environmental Quality, *Environmental Quality—1979*, p. 62.

25. Ibid., p. 63.

26. Kay H. Jones. "Are I/M Programs Necessary to Achieve the Nation's Air Quality and Public Health Goals?" Presentation at the Eighth Annual North American Motor Vehicle Emissions Control Conference, Atlantic City, October 1, 1980.

27. Ibid.

28. James A. Rutherford and Rebecca L. Waring. "Update on EPA's Study of the Oregon Inspection/Maintenance Program." Presentation at the 73rd Annual Meeting of the Air Pollution Control Association, June 24, 1980, p. 4.

29. Ibid., p. 5.

30. Ibid., p. 4.

31. Ibid., p. 5.

32. U.S. EPA. "Questions and Answers Concerning the Technical Details of Inspection and Maintenance." Emission Control Technology Staff, April, 1979, No. IMS-002/QA-1, p. 21.

33. Dennis Palmini and Daniel Rossi. "What Price Air Quality? The Cost of New Jersey's Inspection/Maintenance Program," *Journal of the Air Pollution Control Association*, October, 1980. Volume 30, No. 10, pp. 1081-1088.

34. A. Myrick Freeman III. "The Benefits of Air and Water Pollution Control: A Review and Synthesis of Recent Estimates," A report prepared for the Council on Environmental Quality, December, 1979.

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36. Ibid., Part 31:2101 to 31:2106 (1970).

37. Ibid., Part 31:1951 to 31:1955 (1970).

38. Ibid., Part 31:2151 to 31:2162 (1978).

39. Ibid., Part 31:1981 to 31:1983 (1970).

40. Ibid., Part 31:1985 to 31:1996 (1971).

41. Ibid., Part 31:2301 to 31:2312 (1978).

42. *State Implementation Plan*—1979, p. 12-12.

43. *Ozone: Formation, Transport and Control*, Lazarus, Appendix I.

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U.S. Government. *Federal Register*. Volume 45, No. 241, pp. 81752-81757. December 12, 1980.

APPENDICES

APPENDIX A

IN HOUSE

REGULAR SESSION 1980

House Resolution No. 154

April 15, 1980

Representative Carl Nett introduced the following resolution which was ordered to be printed.

A RESOLUTION requesting the Legislative Research Commission to study the systems by which air quality in metropolitan areas are measured.

WHEREAS, Kentucky's major metropolitan areas are threatened with a cut-off of federal permits and funds in connection with their air quality programs; and

WHEREAS, at the base of this problem are systems for measuring and monitoring the quality of the air in the regions, information from which is reported to federal agencies; and

WHEREAS, there is evidence that these systems may not be providing a reliable picture of the air quality in the metropolitan areas; and

WHEREAS, there is also doubt about the scientific bases for determining the source and information of some of the air pollutants; and

WHEREAS, there have been recent changes in some of the factors that contribute to the problem of maintaining air quality; and

WHEREAS, unless these changes and uncertainties are properly taken into consideration, the net results of current and future air quality programs could be negative;

NOW, THEREFORE,

Be it resolved by the House of Representatives of the

General Assembly of the Commonwealth of Kentucky:

Section 1. That the Legislative Research Commission is requested to conduct a study of the systems by which the air in the metropolitan areas are sampled, analyzed and reported, for the general purpose of assuring that measures that are taken to improve the quality of the air are effective and commensurate with overall community interests.

Section 2. In conducting the study, the Legislative Research Commission should consider, but not be limited to, the state of scientific knowledge regarding the formation and dispersion of ozone pollutants; and of the effect on ozone formation of new systems such as those for street and highway lighting, for stack gas cleanups and for altering motor vehicle emissions, with the objectives of obtaining a more accurate understanding of the air pollution problems in the metropolitan areas and of the causes of these problems.

Section 3. Staff services to be utilized in completing this study are estimated to cost $20,000. These staff services shall be provided from the regular Commission budget and are subject to the limitations and other research responsibilities of the Commission.

APPENDIX B

IN SENATE

REGULAR SESSION 1980

Senate Resolution No. 83

April 15, 1980

Senators Daniel Meyer and W. L. Quinlan introduced the following resolution which was ordered to be printed.

A RESOLUTION requesting the Legislative Research Commission to study the systems by which air quality in metropolitan areas are measured.

WHEREAS, Kentucky's major metropolitan areas are threatened with a cut-off of federal permits and funds in connection with their air quality programs; and

WHEREAS, at the base of this problem are systems for measuring and monitoring the quality of the air in the regions, information from which is reported to federal agencies; and

WHEREAS, there is evidence that these systems may not be providing a reliable picture of the air quality in the metropolitan areas; and

WHEREAS, there is also doubt about the scientific bases for determining the source and information of some of the air pollutants; and

WHEREAS, there have been recent changes in some of the factors that contribute to the problem of maintaining air quality; and

WHEREAS, unless these changes and uncertainties are properly taken into consideration, the net results of current and future air quality programs could be negative;

NOW, THEREFORE,

Be it resolved by the Senate of the General Assembly of

the Commonwealth of Kentucky:

Section 1. That the Legislative Research Commission is requested to conduct a study of the systems by which the air in the metropolitan areas are sampled, analyzed and reported, for the general purpose of assuring that measures that are taken to improve the quality of the air are effective and commensurate with overall community interests.

Section 2. In conducting the study, the Legislative Research Commission should consider, but not be limited to, the state of scientific knowledge regarding the formation and dispersion of ozone pollutants; and of the effect on ozone formation of new systems such as those for street and highway lighting, for stack gas cleanups and for altering motor vehicle emissions, with the objectives of obtaining a more accurate understanding of the air pollution problems in the metropolitan areas and of the causes of these problems.

Section 3. Staff services to be utilized in completing this study are estimated to cost $20,000. These staff services shall be provided from the regular Commission budget and are subject to the limitations and other research responsibilities of the Commission.

APPENDICES C—I

APPENDIX C[35]

Sulfur Oxides—Health & Other Effects

The following adverse effects of sulfur oxides represent the EPA's best judgment of the effects that may occur when various levels of pollution are reached in the atmosphere.

Effects on Health

Analyses of numerous epidemiological studies clearly indicate an association between air pollution, as measured by sulfur dioxide, accompanied by particulate matter, and health effects of varying severity. This association is most firm for the short-term air pollution episodes.

There are probably no communities which do not contain individuals with impaired health who are particularly susceptible to the adverse effects of elevated levels of sulfur oxides and particulate matter. However, to show small changes in deaths associated with coincident higher levels of air pollutants requires extremely large populations. In small cities, these changes are difficult to detect statistically.

The epidemiologic studies concerned with increased mortality also show increased morbidity. Again, increases in morbidity as measured, for example, by increases in hospital admissions or emergency clinic visits, are most easily detected in major urban areas.

It is believed that, for the large urban communities which are routinely exposed to relatively higher levels of pollution, sound statistical analysis can detect with confidence the small changes in daily mortality which are associated with fluctuation in pollution concentrations.

The association between long-term community exposures to air pollution and respiratory disease incidence and prevalence rates is conservatively believed to be intermediate in its reliability. Because of the reenforcing nature of the studies conducted to date, the conclusions to be drawn from this type of study can be characterized as probable.

The association between long-term residence in a polluted area and chronic disease morbidity and mortality is somewhat more conjectural. However, in the absence of other explanations, the findings of increased morbidity and of increased death rates for selected causes, independent of economic status must still be considered consequential.

Based on the above guidelines the following conclusions are listed in order of reliability, with the more reliable conclusions first.

1. AT CONCENTRATIONS OF ABOUT 1500 ug/m³ (0.52 ppm) of sulfur dioxide (24-hour average), and suspended particulate matter measured as a soiling index of 6 cohs or greater *increased mortality* may occur.

2. AT CONCENTRATIONS OF ABOUT 715 ug/m³ (0.25 ppm) of sulfur dioxide and higher (24-hour mean), accompanied by smoke at a concentration of 750 ug/m³ *increased daily death rate* may occur.

3. AT CONCENTRATIONS OF ABOUT 500 ug/m³ (0.19 ppm) of sulfur dioxide (24-hour mean), with low particulate levels *increased mortality rates* may occur.

4. AT CONCENTRATIONS RANGING FROM 300 ug/m³ (0.11 ppm to 0.19 ppm) of sulfur dioxide (24-hour mean), with low particulate levels, *increased hospital admissions* of older persons for respiratory disease may occur, *absenteeism* from work, particularly with older persons, may also occur.

5. AT CONCENTRATION OF ABOUT 715 ug/m³ (0.25 ppm) of sulfur dioxide (24-hour mean), accompanied by particulate matter, *a sharp rise in illness rates* for patients over age 54 with severe bronchitis may occur.

6. AT CONCENTRATIONS OF ABOUT 600 ug/m³ (about 0.21 ppm) of sulfur dioxide (24-hour mean), with smoke concentrations of about 300 ug/m³, patients with chronic lung disease may experience *accentuation of symptoms*.

7. AT CONCENTRATIONS RANGING FROM 105 ug/m³ to 265 ug/m³ (0.037 ppm to 0.092 ppm) of sulfur dioxide (annual mean), accompanied by smoke concentration of about 185 ug/m³, *increased frequency of respiratory symptoms and lung disease* may occur.

8. AT CONCENTRATIONS OF ABOUT 120 ug/m³ (0.046 ppm) of sulfur dioxide (annual mean), accompanied by smoke concentrations of about 100 ug/m³, *increased frequency and severity of respiratory diseases* in school children may occur.

9. AT CONCENTRATIONS OF ABOUT 115 ug/m³ (0.04 ppm) of sulfur dioxide (annual mean),accompanied by smoke concentrations of about 160 ug/m³ *increase in mortality* from bronchitis and from lung cancer may occur.

Effects on Visibility

AT A CONCENTRATION OF 285 ug/m³ (0.10 ppm) of sulfur dioxide, with comparable concentration of particulate matter and relative humidity of 50 percent *visibility may be reduced* to about five miles.

Effects on Materials

AT A MEAN SULFUR DIOXIDE LEVEL OF 345 ug/m³ (0.12 ppm) accompanied by high particulate levels, the *corrosion rate* for steel panels may be increased by 50 percent.

Effects on Vegetation

For plants such as maple trees, spinach, and sweet potatoes that are sensitive to sulfur dioxide, damage or reduction in growth or yield may result from short-term exposures as low as 131 to 1316 ug/m³ (0.05 to 0.5 ppm) over periods of 8 hours or 2620 to 10,480 ug/m³ (1.0 to 4.0 ppm) over periods of ½ hour. More resistant plants such as oak trees and corn may require exposures of over 5240 ug/m³ (2 ppm) for the 8-hour period or over 26,200 ug/m³ (10 ppm) for the ½-hour period.

Growing season average concentrations as low as 26 to 66 ug/m³ (0.010 to 0.025 ppm) have been reported to affect a large number of agronomic species. These averages were associated with maximum 30-minute values of 2096 to 4978 ug/m³ (0.8 to 1.9 ppm).

Foliar and growth effects of mixtures of SO2 with other pollutants may be greater than the effects of SO2 alone. Mixtures of both SO2 and ozone and SO2 and nitrogen dioxide have been found to produce greater effects than either pollutant alone.

RESUME

In addition to health considerations, the economic and aesthetic benefits to be obtained from low ambient concentrations of sulfur oxides as related to visibility, soiling, corrosion, and other effects should be considered by organizations responsible for promulgating ambient air quality standards. Under the conditions prevailing in areas where the studies were conducted, adverse health effects were noted when 24-hour average levels of sulfur dioxide exceeded 300 ug/m³ (0.11 ppm) for 3 to 4 days. Adverse health effects were also noted when the annual mean level of sulfur dioxide exceeded 115 ug/m³ (0.04 ppm). Visibility reduction to about 5 miles was observed at 285 ug/m³ (0.10 ppm); adverse effects on materials were observed at an annual mean of 345 ug/m³ (0.12 ppm); and adverse effects on vegetation were observed at an annual mean of 85 ug/m³ *and below* (0.03 ppm). It is reasonable and prudent to conclude that, when promulgating ambient air quality standards, consideration should be given to requirements for margins of safety which take into account long-term effects on health, vegetation, and materials occurring below the above levels.

APPENDIX D[36]

Particulate Matter—Health & Other Effects

The following adverse effects of particulates represent the EPA's best judgment of the effects that may occur when various levels of pollution are reached in the atmosphere.

Effects on Health

Analyses of numerous epidemiological studies clearly indicate an association between air pollution, as measured by particulate matter accompanied by sulfur dioxide, and health effects of varying severity. This association is most firm for the short-term air pollution episodes.

There are probably no communities which do not contain individuals with impaired health who are particularly susceptible to the adverse effects of elevated levels of particulate matter and sulfur oxides. However, to show small changes in deaths associated with coincident higher levels of air pollutants requires extremely large populations. In small cities, these changes are difficult to detect statistically.

The epidemiologic studies concerned with increased mortality also show increased morbidity. Again, increases in morbidity as measured, for exampie, by increases in hospital admissions or emergency clinic visits, are most easily demonstrated in major urban areas.

For the large urban communities which are routinely exposed to relatively high levels of pollution, sound statistical analysis can show with confidence the small changes in daily mortality which are associated with fluctuation in pollution concentrations.

The association between longer-term community exposures to particulate matter and respiratory disease incidence and prevalence rates is conservatively believed to be intermediate in its reliability. Because of the re-enforcing nature of the studies conducted to date, the conclusions to be drawn from this type of study can be characterized as probable.

The association between long-term residence in a polluted area and chronic disease morbidity and mortality is somewhat more conjectural. However, in the absence of other explanations, the findings of increased morbidity and of increased death rates for selected causes, independent of economic status must still be considered consequential.

Based on the above guidelines the following conclusions are listed in order or reliability, with the more reliable conclusions first. . . .

1. AT CONCENTRATIONS OF 750 $\mu g/m^3$ and higher for particulates on a 24-hour average, accompanied by sulfur dioxide concentrations of 715 $\mu g/m^3$ and higher, *excess deaths* and a considerable *increase in illness* may occur. . .

2. A DECREASE FROM 140 $\mu g/m^3$ *to* 60 $\mu g/m^3$ (annual mean) in particulate concentrations may be accompanied by *a decrease in mean sputum volume* in industrial workers. . . .

3. IF CONCENTRATIONS ABOVE 300 $\mu g/m^3$ for particulates persist on a 24-hour average and are accompanied by sulfur dioxide concentrations exceeding 630 $\mu g/m^3$ over the same average period, *chronic bronchitis* patients will likely suffer *acute worsening of symptoms*. . . .

4. AT CONCENTRATIONS OVER 200 $\mu g/m^3$ for particulates on a 24-hour average, accompanied by concentrations of sulfur dioxide exceeding 250 $\mu g/m^3$ over the same average period, *increased absence of industrial workers due to illness* may occur. . . .

5. WHERE CONCENTRATIONS RANGE FROM 100 $\mu g/m^3$ to 130 $\mu g/m^3$ and above for particulates (annual mean) with sulfur dioxide concentrations (annual mean) greater than 120 $\mu g/m^3$, children residing in such areas are likely to experience *increased incidence* of certain *respiratory diseases.*

6. AT CONCENTRATIONS ABOVE 100 $\mu g/m^3$ for particulates (annual geometric mean) with sulfation levels above 30 mg/cm^2-mo., *increased death rates* for persons over 50 years of age are likely. . . .

7. WHERE CONCENTRATIONS RANGE FROM 80 $\mu g/m^3$ *to* 100 $\mu g/m^3$ for particulates (annual geometric mean) with sulfation levels of about 30 mg/cm^2-mo., *increased death rates* for persons over 50 years of age may occur. . . .

Effects on Direct Sunlight

AT CONCENTRATIONS RANGING FROM 100 $\mu g/m^3$ to 150 $\mu g/m^3$ for particulates, where large smoke turbidity factors persist, in middle and high latitudes direct sunlight is reduced up to one-third in summer and two-thirds in winter. . . .

Effects on Visibility

AT CONCENTRATIONS OF ABOUT 150 $\mu g/m^3$ for particulates, where the predominant particle size ranges from 0.2μ to 1.0μ and relative humidity is less than 70 percent, *visibility is reduced* to as low as 5 miles. . . .

Effects on Materials

AT CONCENTRATIONS RANGING FROM 60 $\mu g/m^3$ (annual geometric mean), to 180 $\mu g/m^3$ for particulates (annual geometric mean), in the presence of sulfur dioxide and moisture, *corrosion of steel and zinc* panels occurs at an accelerated rate. . .

Effects on Public Concern

AT CONCENTRATIONS OF APPROXIMATELY 70 $\mu g/m^3$ for particulates (annual geometric mean), in the

presence of other pollutants, public awareness and/or *concern for air pollution* may become evidence and increase proportionately up to and above concentrations of 200 $\mu g/m^3$ for particulates. . . .

The economic and aesthetic benefits to be obtained from low ambient concentrations of particulates as related to visibility, soiling, corrosion, and other effects should be considered by organizations responsible for promulgating ambient air quality standards. On the basis of the foregoing information and data, it is reasonable and prudent to conclude that ambient concentrations of particulates of 80 $\mu g/m^3$ (annual average) or more in the atmosphere may produce adverse health effects in particular segments of the population. In the promulgation of ambient air quality standards, it should be recognized that circumstances existing within given region (as well as requirements for margins of safety) may warrant more stringent standards than those indicated by these criteria.

APPENDIX E[37]

Carbon Monoxide—Health & Other Effects

The following adverse effects of carbon monoxide represent the EPA's best judgment of the effects that may occur when various levels of pollution are reached in the ambient air.

Effects on Health

1. Experimental exposure of nonsmokers to a concentration of 35 mg/m^3 (30 ppm) for 8 to 12 hours has shown that an equilibrium value of 5 percent COHb is approached in this time; about 80 percent of this equilibrium value, i.e., 4 percent COHb, is present after only 4 hours of exposure. These experimental data verify formulas used for estimating the equilibrium values of COHb after exposure to low concentrations of CO. These formulas indicate that continuous exposure of nonsmoking sedentary individuals to 23 mg/m^3 (20 ppm) will result in a blood COHb level of about 3.7 percent, and an exposure to 12 mg/m^3 (10 ppm) will result in a blood level of about 2 percent.
2. Experimental exposure of nonsmoker to 58 mg/m^3 (50 ppm) for 90 minutes has been associated with impairment in time-interval discrimination. This exposure will produce an increase of about 2 percent COHb in the blood. This same increase in blood COHb will occur with continuous exposure to 12 to 17 mg/m^3 (10 to 15 ppm) for 8 or more hours.
3. Experimental exposure to CO concentrations sufficient to produce COHb levels of about 5 percent (a level producible by exposure to about 35 mg/m^3 for 8 or more hours) has provided in some instances evidence of impaired performance on certain other psychomotor tests, and an impairment in visual discrimination.
4. Experimental exposure to CO concentrations sufficient to produce blood COHb levels above 5 percent (a level producible by exposure to 35 mg/m^3 or more for 8 or more hours) has provided evidence of physiologic stress in patients with heart disease.

The following table presents the above conclusions in tabular form.

EFFECTS OF CARBON MONOXIDE

Environmental conditions		Effect	Comment
35 mg/m^3 (30 ppm)	for up to 12 hours	Equilibrium value of 5 percent blood COHb is reached in 8 to 12 hours; 80 percent of this equilibrium value, (4 percent COHb) is reached within 4 hours.	Experimental exposure of nonsmokers. Theoretical calculations suggest exposure to 23 (20 ppm) and 12 mg/m^3 (10 ppm) would result in COHb levels of about 3.7 and 2 percent, respectively, if exposure was continuous for 8 or more hours.
58 mg/m^3 (50 ppm)	for 90 minutes	Impairment of time-interval discrimination in nonsmokers.	Blood COHb levels not available, but anticipated to be about 2.5 percent. Similar blood COHb levels expected from exposure to 10 to 17 mg/m^3 (10 to 15 ppm) for 8 or more hours.
115 mg/m^3 (100 ppm) intermittently through a facial mask		Impairment in performance of some psychomotor tests at a COHb level of 5 percent.	Similar results may have been observed at lower COHb levels, but blood measurements were not accurate.
High concentrations of CO were administered for 30 to 120 seconds, and then 10 minutes was allowed for washout of alveolar CO before blood COHb was measured.		Exposure sufficient to produce blood COHb levels above 5 percent has been shown to place a physiologic stress on patients with heart disease.	Data rely on COHb levels produced rapidly after short exposure to high levels of CO; this is not necessarily comparable to exposure over a longer time period or under equilibrium conditions.

RESUME

An exposure of 8 or more hours to a carbon monoxide concentration of 12 to 17 mg/m^3 (10 to 15 ppm) will produce a blood carboxyhemoglobin level of 2.0 to 2.5 percent in nonsmokers. This level of blood carboxyhemoglobin has been associated with adverse health effects as manifested by impaired time interval discrimination. Evidence also indicates that an exposure of 8 or more hours to a CO concentration of 35 mg/m^3 (30 ppm) will produce blood carboxyhemoglobin levels of about 5 percent in nonsmokers. Adverse health effects as manifested by impaired performance on certain other psychomotor tests have been associated with this blood carboxyhemoglobin level, and above this level there is evidence of psysiologic stress in patients with heart disease.

There is some epidemiological evidence that suggests an association between increased fatality rates in hospitalized myocardial infarction patients and exposure to weekly average CO concentrations of the order of 9 to 16 mg/m^3 (8 to 14 ppm).

Evidence from other studies of the effects of CO does not currently demonstrate an association between existing ambient levels of CO and adverse effects on vegetation, materials, or other aspects of human welfare.

It is reasonable and prudent to conclude that, when promulgating air quality standards, consideration should be given to requirements for margins of safety that would take into account possible effects on health that might occur below the lowest of the above levels.

APPENDIX F[38]

Photochemical Oxidants—Health & Other Effects

In this section, the influence of exposure to ozone and other photochemical oxidants on physiology and health is assessed, beginning with a brief discussion of the concept of "threshold pollutant concentrations" and its application to the protection of public health. Then, the strength of association between exposure to ozone and other oxidants and changes in several types of biomedical indicators is evaluated.

For each type of indicator, the discussion addresses four main topics: (1) the degree to which changes in each type constitute impairments in public health; (2) the available scientific evidence relating ozone and other oxidant exposures to changes within each type; (3) the reliability of existing scientific evidence; and (4) where appropriate, the confidence with which findings may be attributed to ozone alone, as opposed to other substances or combinations of substances.

Whenever possible, clinical (human experimental) and epidemiologic studies will be discussed together. However, due to the great uncertainty inherent in the quantitative extrapolation of results of animal studies to humans, human and animal studies will be discussed separately.

1. Discussion of "Threshold Concentrations"

The Clean Air Act directs that national primary ambient air quality standards shall be standards ". . . the attainment and maintenance of which in the judgment of the Administrator, . . . allowing an adequate margin of safety, are requisite to protect the public health." The confidence with which a margin of safety can be defined depends upon the precision with which a "threshold pollutant concentration," above which exposure to ozone or other oxidants promotes impairment of health and below which it does not, can be determined.

In practice, no single overall "threshold concentration" for ozone or other pollutants exists. "Thresholds" have been shown to vary widely with the population segment studied and the biologic indicators measured. Also, since the great majority of known dose-response relationships do not show sharp discontinuities, it is most unlikely that a discrete "threshold pollutant concentration" can be established even for a single population segment and a single biomedical indicator.

Despite these limitations, the environmental decision maker may find it useful to incorporate the concept of "threshold concentrations" into the standard-setting process. For a given population segment, a "threshold concentration" may be operationally defined as occurring somewhere between a concentration at which no effect on health or function has been observed, and a concentration at which such an effect has been demonstrated. In protecting public health, the population segments of primary concern are the most susceptible groups, in whom exposure to ozone or other pollutants is most likely to promote impairment of health. Such groups may include those with underlying illness, the very old, the very young, and the pregnant. (It has not been determined whether such susceptible population segments differ from the most sensitive population segments, which comprise those individuals most likely to show measurable responses to very low pollution concentrations.)

Ideally, the environmental decision maker would know the pollutant concentrations with which no adverse effects are associated in susceptible groups, as well as the concentrations with which such adverse effects are unambiguously associated. Unfortunately, knowledge in both of these areas remains sparse. Recent experimental studies of healthy people and animals have greatly advanced our knowledge of the health effects of ozone. However, "threshold concentrations" deducible from such studies may not apply to the potentially susceptible groups described above. The opportunity to study such groups experimentally is severely limited by practical and ethical constraints. The relatively few epidemiologic studies of these groups that are extant have yielded inconclusive results.

2. Health Effects on Mechanical Function of Lung

There is considerable room for honest disagreement on whether pollution-induced alterations in mechanical lung function, in and of themselves, constitute *bona fide* impairments of health. In the great majority of experimental studies in which oxidant exposures have produced changes in lung function in healthy subjects, function has returned to normal within a few hours. Thus, there is no reason to suspect that, in healthy individuals, such changes promote any measurable increase in risk of future illness. Nor does any available evidence suggest that, in healthy individuals, a small change in ventilatory function, unaccompanied by symptoms or impairment of oxygen uptake or work capacity, would interfere with normal activity or task performance.

However, three considerations suggest that oxidant-associated changes in lung function may signal impairment of public health. First, in people with underlying respiratory illness such as asthma, chronic bronchitis, and emphysema, even small decrements in lung function often interfere with normal activity. Second, at experimental ozone concentrations as low as 0.30 ppm, decrements in lung function have usually been accompanied by physical discomfort, as manifested in symptoms such as sore throat, chest pain, cough, and headache. At times this discomfort has been great enough to prevent the completion of experimental protocols, particularly when subjects have been exercising vigorously. It appears quite likely that the pulmonary irritant properties of ozone (and perhaps other oxidants) underlie both the discomfort and the decrements in function. Thus, at least when asociated with ozone exposure, changes in lung function often represent a level of discomfort which, even among healthy people, may restrict normal activity or impair the performance of tasks.

Summary of data. Human experimental studies have demonstrated that the subject's level of exercise during ozone exposure is directly related to the magnitude of change in lung function and the severity of symptoms at any given ozone concentration. During exercise, subjects increase their

expiratory flow rates, and they tend to breathe through their mouths. These factors increase the total dose of ozone delivered to the lung and may increase the depth to which it is delivered in the respiratory tree.

After 2 hours of resting exposure to 1470 $\mu g/m^3$ (0.75 ppm), healthy young adults showed small changes in lung function in a study by Bates et al. Folinsbee et al. observed changes in respiratory pattern (increased respiratory frequency and decreased tidal volume) and reductions of vital capacity in healthy young adults exercising submaximally after a similar resting exposure. Immediately after 2 hours of exposure to 1470 $\mu g/m^3$ (0.75 ppm) ozone, during which they performed intermittent light exercise, subjects showed quite pronounced changes in lung function in studies by Folinsbee et al. and Hazucha et al. In subjects exposed under the same conditions, Folinsbee et al. observed changes in respiratory pattern, though not in minute volume or oxygen uptake.

In subjects exercising maximally after 2 hours of exposure to 1470 $\mu g/m^3$ (0.75 ppm) ozone and intermittent light exercise, Folinsbee et al. observed decrements in maximum work load, tidal volume, heart rate, and oxygen uptake.

Folinsbee et al. observed decreases in tidal volume and maximum expiratory flow rate at 50 percent of vital capacity (V_{50}) in subjects exercising submaximally after 2 hours of exposure to 980 $\mu g/m^3$ (0.5 ppm) ozone and intermittent light exercise. In subjects giving no history of cough, chest discomfort, or wheezing in response to allergy or air pollution exposure ("unreactive subjects"), few changes in lung function occurred after 4 hours of exposure to 980 $\mu g/m^3$ (0.50 ppm) and intermittent light exercise (Hackney et al.). However, in subjects giving such a history ("reactive subjects"), and receiving an identical exposure, the same investigators observed decrements in 8 of 14 measured parameters. Interestingly, in a second group of "unreactive subjects," the same investigators observed substantial decrements in lung function after only 2 hours of exposure to 980 $\mu g/m^3$ (0.5 ppm) ozone and intermittent light exercise.

Statistically significant changes in forced vital capacity, maximum mid-expiratory flow rate, and airway resistance in 22 young males after 2 hours of exposure to 0.4 ppm ozone and intermittent moderate exercise were observed by Knelson et al. After subjects had been exposed for 4 hours, changes in these parameters had increased, and several other flow parameters had also changed significantly in comparison with control values.

In studies by Hazucha and Bates, Hazucha et al., and Folinsbee et al., subjects showed changes in lung function after 2 hours of exposure to 730 $\mu g/m^3$ (0.37 ppm) ozone and intermittent light exercise. Hackney et al. observed such changes in "reactive subjects" but not in "unreactive subjects." In a separate study by Hackney and colleagues, four Canadians and four southern Californians were exposed in the Los Angeles area to 730 $\mu g/m^3$ (0.37 ppm) ozone for 2 hours. The Californians showed few changes in lung function, whereas the Canadians showed decrements in most parameters measured. (Due at least partly to small sample sizes, no observed changes were statistically significant.) In subjects exposed to 730 $\mu g/m^3$ (0.37 ppm) ozone together with 1000 $\mu g/m^3$ (0.37 ppm) SO_2, Hazucha and Bates observed an effect on lung function substantially larger than the sum of the separate effects of the individual pollutants.

DeLucia and Adams observed changes in lung function and respiratory pattern in healthy subjects exercising steadily and fairly heavily over a 1-hour exposure to 590 $\mu g/m$ (0.30 ppm) O_3. Two of six subjects experienced such discomfort as to prevent them from completing the experimental protocol.

Hazucha observed small changes in lung function in three non-smokers exposed for 2 hours to 490 $\mu g/m^3$ (0.25 ppm) ozone and intermittent light exercise. No lung function changes of note were seen by Hackney et al. even among "reactive subjects" who were similarly exposed.

After 1 hour of exposure to 290 $\mu g/m^3$ (0.15 ppm) ozone and steady, fairly heavy exercise, subjects observed by DeLucia and Adams showed changes in respiratory pattern. In two of six subjects, the same investigators noted inconsistent increases in residual volume.

Small but statistically significant increases in airway resistance, as measured by plethysmography, were observed in two of four healthy subjects immediately after a 1-hour exposure to 200 $\mu g/m^3$ (0.1 ppm) ozone. The investigators (Goldsmith and Nadel) did not state whether the subjects exercised during exposure.

Von Nieding and Wagner reported that subjects showed decrements in arterial O_2 pressure (Pa_{O_2}) and airway resistance after 2 hours of exposure to 200 $\mu g/m^3$ (0.10 ppm) ozone and intermittent light exercise.

Kagawa and Toyama reported the results of Japanese epidemiologic studies relating lung function in Japanese elementary schoolchildren to daily air pollution level. In approximately 25 percent of the children studied, lung function parameters were significantly correlated with the average ozone concentration in the 2 hours prior to testing. During these studies, the ozone concentration ranged between 20 and 590 $\mu g/m^3$ (0.01 and 0.30 ppm). Correlations of lung function with ozone exposure were generally greater than with total-oxidant exposure, and correlations of ozone exposure with parameters reflecting upper airway function were generally greater than with those reflecting lower airway function. The design of these studies precludes the inference of specific dose-response relationships.

Reliability of evidence. Available evidence showing an effect of 730 $\mu g/m^3$ (0.37 ppm) ozone on the lung function of lightly exercising subjects is convincing. Though it remains unreplicated, the study of DeLucia and Adams raises the distinct possibility that an ozone concentration of 590 $\mu g/m^3$ (0.30 ppm) exerts a temporary effect on the lung function of healthy subjects exercising fairly strenuously. The same investigators have raised the question whether ozone concentrations as low as 290 $\mu g/m^3$ (0.15 ppm) exert effects in a portion of healthy subjects exercising strenuously.

Nadel and Goldsmith's findings suggest that certain changes in lung function may be detectable in some healthy subjects who have been exposed to 200 $\mu g/m^3$ (0.1 ppm) of ozone. For three reasons, however, these findings do not suggest any endangerment of public health. First, the subjects experienced no physical discomfort while exposed to 200 $\mu g/m^3$ (0.1 ppm) ozone. Second, the observed changes in airway resistance were small. Third, the two subjects who showed significant increases in airway resistance after exposure to 200 $\mu g/m^3$ (0.1 ppm) ozone showed smaller, non-significant increases in airway resistance after exposure to 780 $\mu g/m^3$ (0.4 ppm) ozone.

Von Nieding and Wagner's studies, though interesting, are unconfirmed. These investigators did not use standard techniques to measure airway resistance. Nor did they draw arterial blood for PaO_2 measurements, but instead drew

"arterialized" blood from subjects' ear lobes. Thus, until confirmed with generally accepted methods, these studies must be interpreted with caution.

Kagawa and Toyama's studies, like those of DeLucia and Adams and of Hackney, suggest that even among healthy people there is a considerable range of sensitivity to ozone exposure. Whether the lung function of respiratory disease patients is more sensitive to such exposure than that of healthy people has not been determined. However, as mentioned above, the clinical significance of changes in lung function is likely to be greater in respiratory disease patients than in healthy people.

Hackney and colleagues' experimental study of Canadians and Californians is consistent with the hypothesis that repeated oxidant exposures promote adaptation toward the maintenance of full lung function. More study is required to confirm this hypothesis and to investigate whether adaptation in lung function is of any long-term consequence to health.

Attributability of effects to ozone. Experimental studies have shown that ozone at concentrations observed in the ambient air can produce changes in mechanical lung function. Kagawa and Toyama's epidemiologic studies, in which lung function was correlated more strongly with ozone exposure than with total oxidant exposure, are consistent with this finding. However, the degree to which epidemiologic observations may be attributed specifically to ozone remains in doubt, since Kagawa and Toyama were not able fully to separate the effects of ozone from the effects of other environmental factors.

Finally, the work of Hazucha and Bates suggests that the effect on lung function of ozone at 730 $\mu g/m^3$ (0.37 ppm) may be enhanced by an identical concentration of sulfur dioxide. Observation of this enhancement argues in favor of providing a margin of safety in a primary National Ambient Air Quality Standard for ozone. However, this observation does not support a quantitative recommendation for a safety margin, since it has not yet been determined whether such enhancement occurs at lower ozone concentrations or with substances other than sulfur dioxide.

3. Health Effects on Impairment of Physical Performance Decrements in physical performance are deterrents to personal satisfaction. In this respect, such decrements constitute impairment of public health. The degree to which oxidant exposures may promote decrements in physical performance has not been determined. However, Wayne et al. assessed the association between hourly oxidant concentrations and the proportion of a high-school cross-country team failing to improve running times between successive track meets in southern California. Over six cross-country seasons, the correlation of average oxidant concentration in the hour before the race with the proportion of runners failing to improve times was 0.88. The corresponding correlation for both the first and second three-season periods was 0.945. A correlation this high denotes a very close numerical relationship between two variables.

During the period studied, hourly oxidant concentrations ranged from approximately 60 to 590 $\mu g/m^3$ (0.03 to 0.30 ppm). Inspection of Wayne et al.'s data reveals no obvious relationship between unimproved running time and oxidant concentrations below 200 to 290 $\mu g/m^3$ (0.10 to 0.15 ppm), in spite of the high overall correlations mentioned above. Also, since the authors did not consider ozone separately from other oxidants, the specific contribution of ozone to the observed results cannot be determined from this study.

As far as can be ascertained, no replication of Wayne et al.'s study has appeared in the published literature. For three reasons, however, Wayne et al.'s results are more trustworthy than most results of a single epidemiologic study. First, the correlations between hourly oxidant concentration and unimproved running time were unusually high. Second, as mentioned in Chapter 10 of this document, Herman, at the University of North Carolina, has analyzed Wayne et al.'s data as well as data from two additional seasons, and has observed results similar to those of Wayne et al. Third, Wayne et al.'s results are qualitatively consistent with the results of the experimental lung function studies mentioned above, especially those of Folinsbee et al. In view of these experimental studies, it would also appear plausible that ozone contributed significantly to Wayne et al.'s results.

4. Health Effects on Asthmatics In the United States, there are an estimated 6,000,000 to 8,000,000 asthmatics, about 70 percent of whom are estimated to live in urban areas. Thus, the number of asthmatics who may be exposed to elevated oxidant concentrations is substantial.

Available epidemiologic evidence on the relationship between oxidant exposure and exacerbation of asthma is very limited. In 1961, Schoettlin and Landau reported that the proportion of selected asthmatics in the Pasadena area having attacks was significantly greater ($p < 0.05$) on days when the maximum hourly oxidant concentration exceeded 490 $\mu g/m^3$ (0.25 ppm) than on days when the corresponding concentration was below this level. However, the proportion of asthmatics having attacks on days of maximum hourly oxidant concentration above 250 $\mu g/m^3$ (0.13 ppm) was not significantly different from the corresponding proportion when the maximum hourly concentration was below this level. The authors did not state the actual percentage of asthmatics having attacks on days in any exposure category, though they did state that asthma attacks tended to coincide with elevated oxidant levels in 8 (6 percent) of 137 patients studied.

Because it does not present asthma attack rates, Schoettlin and Landau's report gives no indication whether increases in attack rates might have been expected at maximum hourly oxidant concentrations below 490 $\mu g/m^3$ (0.25 ppm). Nor does it allow any judgments as to the extent to which increased attack rates might be attributable specifically to ozone. Despite the considerable attention it has received, this report should be considered the preliminary investigation its authors intended it to be.

5. Health Effects on Respiratory Symptoms and Headache As mentioned above, increased rates of respiratory symptoms and headache constitute impairment of public health. Even when mild, such symptoms are annoying. Even when reversible, they may restrict normal activity or limit the performance of tasks.

Summary of data. In nearly all experimental studies in which ozone exposures have been sufficient to produce changes in lung function, most subjects have reported respiratory symptoms. The most common symptoms have been throat tickle, substernal tightness, pain on deep inspiration, and cough. Wheezing, dyspnea, and headache have occurred less commonly. Symptom severity has increased with ozone concentration and exercise. In studies of heavy exercise, symptoms have occasionally been severe enough to prevent subjects from completing experimental protocols.

In an epidemiologic study in southern California, Hammer et al. assessed the association between daily maximum hourly

oxidant concentration and rates of chest discomfort, cough, and headache among student nurses. Rates of each of these symptoms, whether unadjusted or adjusted for fever, began to increase in the following oxidant concentration ranges: chest discomfort, 490 to 570 $\mu g/m^3$ (0.25 to 0.29 ppm); cough, 590 to 760 $\mu g/m^3$ (0.30 to 0.39 ppm); and headache, 290 to 370 $\mu g/m^3$ (0.15 to 0.19 ppm). Adjusted and unadjusted rates of headache, however, were not unequivocally elevated below maximum hourly oxidant concentrations 590 to 760 $\mu g/m^3$ (0.30 to 0.39 ppm).

Several Japanese investigators have assessed the association between daily pollutant concentrations and symptom rates among students. In one Japanese study, rates of sore throat, dyspnea, and headache were somewhat higher during the summer months on days when the oxidant concentration exeeded 200 $\mu g/m^3$ (0.10 ppm) than on days when it did not. Over a 1-year period, rates of respiratory symptoms and headache were higher on days when the oxidant concentration exceeded 290 $\mu g/m^3$ (0.15 ppm) than on days when it was lower than 200 $\mu g/m^3$ (0.10 ppm). Though there is reason to believe that the stated oxidant concentrations were daily maximum hourly averages, averaging times were not clearly presented in the report of this study.

Reliability of evidence. Because of their close correlation with the results of experimental studies, it appears that Hammer et al.'s results appear quite reliable, even though unconfirmed in this country. Their reliability is enhanced by the large number of person-days of observation, about 53,000, encompassed by the study. Though ozone levels were not considered in Hammer et al.'s study, it is reasonable to hypothesize, in view of experimental studies, that ozone contributed substantially to observed increases in rates of cough, chest discomfort, and headache.

As nearly as can be determined from translations of original articles, the Japanese epidemiologic studies cited in this document were appropriately designed. However, it is very difficult to interpret their results. Also, at least at present, the applicability of these results to any oxidant pollution problem in the United States must be considered very limited.

In data analyses, Japanese investigators (like many U.S. investigators) have not been able fully to separate the effects of individual pollutants. It is conceivable that combinations of pollutants unique to Japan were necessary to promote the increased symptom rates observed there. Averaging times for pollutant measurements were not clearly stated in the Japanese studies. Therefore, it is often impossible to draw specific inferences as to dose and response. In addition, the degree to which differences in Japanese and U.S. cultural responses to air pollution may differentially affect symptom perception in the two countries has not been determined.

A problem more fundamental than any of these specific reservations, however, is that Japanese and U.S. investigators as yet have great difficulty in exchanging specific scientific concepts. Until the quality of scientific communication between these groups of investigators increases, the ability to interpret the Japanese studies and to apply them to situations in the United States will remain severely limited.

6. Health Effects on Eye Irritations — In that it is annoying and uncomfortable, the reversible eye irritation produced by exposure to ambient photochemical oxidants may legitimately be considered a marginal impairment of public health. However, whether such eye irritation is sufficient to impair performance or restrict normal activity has not been determined. Nor has any association between oxidant-mediated eye irritation and chronic eye disease been observed.

In epidemiologic studies, no symptom has been more consistently linked to oxidant exposure than eye irritation. In most studies reported before the publication in 1970 of *Air Quality Criteria for Photochemical Oxidants*, rates of eye irritation were observed to increase fairly steadily when oxidant concentrations ranged from 200 to 880 $\mu g/m^3$ (0.10 to 0.45 ppm). In Hammer et al.'s study mentioned above, rates of eye discomfort began to increase at oxidant concentrations of 290 to 370 $\mu g/m^3$ (0.15 to 0.19 ppm).

Evidence linking ambient photochemical oxidant exposures to eye irritation is convincing. However, the specific etiologic agent or agents remain unknown. Experimental studies have shown quite conclusively that ozone at ambient concentrations is not an eye irritant.

7. Oxidants and Mortality — Review of existing studies shows no consistent association between daily oxidant concentrations and daily mortality rates. As far as can be ascertained, no studies of oxidant exposures and mortality have been performed since the publication in 1970 of *Air Quality Criteria for Photochemical Oxidants*.

8. Other Effects of Short-Term Ozone & Oxidant Exposure

a. *Changes in erythrocytes.* In an experimental study, Buckley et al. observed increased rates of erythrocyte lysis in H_2O_2 after a 2¾-hour exposure of healthy subjects to 980 $\mu g/m^3$ (0.5 ppm) ozone and intermittent light exercise. These investigators also noted changes in the activity of several erythrocytic enzymes. Hackney et al. observed an increased *in vitro* lysis rate in Canadians' erythrocytes, but not in Californians' erythrocytes, following a 2-hour exposure of subjects to 730 $\mu g/m^3$ (0.37 ppm) ozone and intermittent light exercise.

b. *Chromosomal aberrations.* Merz et al. reported chromosomal abnormalities in the lymphocytes of six subjects after they were exposed to 980 $\mu g/m^3$ (0.5 ppm) ozone for 6 or 10 hours. However, a study by McKenzie et al. showed no increased rate of leukocyte chromosomal aberration in the lymphocytes of 30 subjects exposed for 4 hours to 780 $\mu g/m^3$ (0.4 ppm) ozone. In the few epidemiologic studies of oxidant exposure and chromosome morphology performed to date, factors other than differences in oxidant exposure have confounded observed results to the extent that no inferences can be drawn.

c. *Biochemical parameters.* Buckley et al. observed reduced glutathione reductase activity and increased vitamin E and lipid peroxidation in human serum following exposure of subjects to 980 $\mu g/m^3$ (0.5 ppm) ozone and intermittent light exercise for 2¾ hours. Following a 2-hour exposure to 730 $\mu g/m^3$ (0.37 ppm) ozone and intermittent light exercise, the Canadians and Californians studied by Hackney et al. showed increases in serum vitamin E levels; but only in the Canadians were these increases statistically significant.

After subjects exercised steadily and vigorously throughout a 1-hour exposure to 590 $\mu g/m^3$ (0.30 ppm) ozone, De Lucia and Adams observed no changes in the following biochemical blood parameters: hemoglobin level, non-protein sulfhydryl level, erythrocyte glucose-6-phosphate dehydrogenase activity, and glutathione reductase activity,

d. *Clinical significance.* The clinical significance of ozone-mediated changes observed in studies of blood is not yet

known. As yet, an epidemiologic study of oxidant effects on anemic individuals has not been done. Whether or not oxidant exposures promote changes in leukocyte chromosome morphology, the significance of the changes themselves is unknown. Finally, changes in serum parameters of the magnitude observed in experimental ozone studies have not yet been linked to any clinical diseases.

9. Long-Term Oxidant Exposures

With the exception of Hackney et al.'s study of Californians and Canadians, no experimental studies of humans have as yet assessed the effects of long-term oxidant exposures. The few available epidemiologic studies of such exposures have yielded inconclusive results. Mahoney has observed an association between broad patterns of oxidant distribution and annual respiratory disease mortality in the Los Angeles area. However, no convincing association between lung cancer mortality and oxidant exposure has been shown. In some studies, a limited association between the frequency of chronic obstructive lung disease and oxidant exposure has been observed. In other studies, however, no such association has been apparent. No relationship between long-term oxidant exposure and acute respiratory disease incidence or change in lung function has been observed, though neither of these areas has been extensively investigated.

Most available epidemiologic studies of long-term oxidant exposure are difficult to interpret. As usually acknowledged by the authors themselves, factors other than pollution exposure may often have influenced the results observed. Some studies have proved difficult to evaluate because areas in which health variables were compared did not show clear-cut differences in pollution exposure. Thus, human studies cannot yet provide the environmental decision maker with concrete information as to the effects of long-term oxidant exposures on public health.

10. Effects on Vegetation & Certain Microorganisms

Since injury to vegetation by oxidants was first identified in 1944 in the Los Angeles Basin, our understanding of oxidant effects and of the widespread nature of their occurrence has increased substantially. The major phytotoxic components of the photochemical oxidant complex are ozone and peroxyacetylnitrate (PAN), although some data suggest that other phytotoxicants are also present. The peroxyacylnitrates are the most phytotoxic of the known photochemical oxidants; however, because it is ubiquitous and is associated with widespread injury to vegetation, ozone is the most important phytotoxic component of the photochemical oxidant complex.

The effects of photochemical oxidants on vascular plants occur at several levels, ranging from the subcellular to the organismic, depending on the concentration and duration of exposure to the pollutant and the interval between cessation of exposure and examination of the plant.

The earliest effect is an increase in cell membrane permeability. Following that, cellular and biochemical changes take place that are ultimately expressed on the organismic level as visible foliar injury, increased leaf drop, reduced plant vigor, reduced plant growth, and death. Such biochemical modifications in an individual plant are manifested by changes in plant communities and, finally, in whole ecosystems.

Leaf stomata are the principal sites through which ozone and PAN enter plants. Oxidants affect photosynthesis, respiration, transpiration, stomatal opening, and metabolic pool development, as well as biochemical pathways and enzyme systems.

Visible injury is identifiable as pigmented, chlorotic, or necrotic foliar patterns. Metabolic cellular disturbances can occur without visible injury and may be reversible. However, most of the growth effects reported until recently were accompanied by visible injury.

Classic ozone injury is demonstrated by the upper-surface leaf fleck of tobacco and the leaf stipple of grape. Many plants show an upper-surface response with no associated injury to the lower surface of leaves. However, in monocotyledonous plants such as grasses or cereals and in some non-monocotyledonous plants, there is no division of mesophyll tissue and bifacial necrotic spotting (flecking) is a common symptom of ozone injury.

Coniferous trees exhibit different symptoms. Ozone is probably the cause of emergence tipburn in eastern white pine (white pine needle dieback) and of "chlorotic decline," a needle injury of Ponderosa pine.

Classic PAN injury appears as a glaze, followed by bronzing, of the lower leaf surface of many plants. Complete collapse of leaf tissue can occur if concentrations are sufficiently high. Early leaf senescence and abscission usually follow the chronic symptoms. Patterns of chronic injury are generally not characteristic and may be confused with symptoms caused by biotic diseases, insect infestation, nutritional disorders, or other environmental stresses.

A great deal of research has been done to define more accurately the effects of oxidants on plant growth and yield. Studies comparing the growth of plants in field chambers provided with carbon-filtered or nonfiltered ambient air containing oxidants have reported up to 50 percent decreases in yield of citrus (orange and lemon) exposed to oxidants; 10 to 15 percent suppression in grape yield in the first year and 50 to 60 percent reduction over the following 2 years; and a 5 to 29 percent decrease in yield of cotton lint and seed in California. Losses of 50 percent in some sensitive potato, tobacco, and soybean cultivars have been reported in the eastern United States. It is apparent that oxidants in the ambient air reduce the yields of many sensitive plant cultivars.

Experimental chambers with controlled environments have been used to study both short-term and long-term effects of exposure to ozone. Radishes given one, two, or three acute exposures (785 $\mu g/m^3$, 0.40 ppm) of 1.5 hours each at 7, 14, and/or 21 days of age exhibited reductions in root growth. The reductions in root growth from the multiple ozone exposures were equal to the additive effects of the single exposures. In other words, the temporal distribution was not a significant factor. When soybean plants were exposed to 1468 $\mu g/m^3$ (0.75 ppm) of ozone for 1 hour, root growth was consistently reduced more than top growth. There were also reductions in nodule weight and number. The greater reduction of root growth compared to top growth is related to the transport of photosynthate. Ozone also affects nitrogen fixation in clover, soybean, and pinto bean through reduction in nodule number, even though nodule size and efficiency of nitrogen fixation are not influenced. The effect of ozone on the number of nodules formed by legumes, if widespread, could have a major impact on plant communities and could affect fertilizer requirements. There are indications that the effect of ozone on nodulation may be related to the carbohydrate supply in the host plant.

Experimental long-term exposures to ozone of a variety of

crops, as well as of ornamental and native plants, have resulted in a reduction in growth and/or yield. Exposure of 14 species, representative of the aspen plant community, to ambient air containing 98 to 137 $\mu g/m^3$ (0.05 to 0.07 ppm) of ozone and to ozone at 290 and 588 $\mu g/m^3$ (0.15 and 0.30 ppm) for 3 hours a day, 5 days a week, and to charcoal-filtered air throughout the growing season, resulted in foliar injury to all species at the highest pollutant concentration. The growth of two soybean cultivars (Hood and Dare) was inhibited by intermittent exposure to ozone at 196 $\mu g/m^3$ (0.10 ppm) for 3 weeks. Both root and top growth was decreased. Similar results were noted with radish except that a lower concentration of ozone (98 $\mu g/m^3$, 0.05 ppm) inhibited growth. In these studies, the reduced growth occurred even though there were few visible symptoms of plant injury.

A 30 percent reduction in the yield of wheat occurred when wheat at anthesis was exposed to ozone at 392 $\mu g/m^3$ (0.2 ppm), 4 hours a day for 7 days. A significant reduction in the yield of tomato was noted when plants were experimentally treated with ozone at 686 $\mu g/m^3$ (0.35 ppm); fewer fruit set and thus fewer fruit were harvested. Chronic exposures to ozone at 98 to 290 $\mu g/m^3$ (0.05 to 0.15 ppm) for 4 to 6 hours a day reduced yields in soybean and corn grown under field conditions. The threshold for measurable effects for ozone appears to be between 98 to 196 $\mu g/m^3$ (0.05 to 0.10 ppm) for sensitive plant cultivars. This is well within the range of ozone levels monitored in the eastern United States. Growth or flowering effects were reported for carnation, geranium, radish, and pinto bean grown in greenhouse chambers and exposed to ozone at 98 to 294 $\mu g/m^3$ (0.05 to 0.15 ppm) for 2 to 24 hours per day.

The two most critical factors in determining plant response to air pollution are duration of exposure and concentration of pollutants. These two factors describe exposure dose. In determining the response of vegetation to oxidants, concentration is more important than time.

The concept of limiting values was used by Jacobson to define a boundary between doses of a pollutant that are likely to injure vegetation measurably and those that are not. Foliar injury was used as the index of plant response. The ranges for limiting values for effects of ozone are:

1. Trees and shrubs—
400 to 1000 $\mu g/m^3$ (0.2 to 0.51 ppm) for 1 hour
200 to 500 $\mu g/m^3$ (0.1 to 0.25 ppm) for 2 hours
120 to 340 $\mu g/m^3$ (0.06 to 0.17 ppm) for 4 hours
2. Agricultural crops—
400 to 800 $\mu g/m^3$ (0.2 to 0.41 ppm) for 0.5 hour
196 to 500 $\mu g/m^3$ (0.1 to 0.25 ppm) for 1 hour
75 to 180 $\mu g/m^3$ (0.04 to 0.09 ppm) for 4 hours

Limiting values for PAN are:
1000 $\mu g/m^3$ (0.2 ppm) for 0.5 hour
500 $\mu g/m^3$ (0.1 ppm) for 1 hour
175 $\mu g/m^3$ (0.035 ppm) for 4 hours

Doses of ozone or PAN greater than the upper limiting values are likely to cause foliar injury.

The data points used to determine the limiting values listed above are not necessarily "threshold values," but are based on available published research data. Any limitations which were present in the experimental techniques used in the studies are, therefore, expressed in the data points. The number of studies used to derive the data points for ozone exposure of trees and shrubs and for PAN is another limitation on the values given above.

For agricultural crops, the inaccuracies in measurements make the interpretation of results of repeated long-duration exposures difficult; therefore, limiting values for ozone concentrations below 100 $\mu g/m^3$ (0.05 ppm) are not useful.

An ozone concentration of 98 to 137 $\mu g/m^3$ (0.05 to 0.07 ppm) for 4 to 6 hours per day for 15 to 133 days can significantly inhibit plant growth and yield of certain species.

Plant sensitivity to ozone and PAN is conditioned by many factors. Genetic diversity in sensitivity to ozone between species and between cultivars within a species is well documented. Variations in sensitivity to ozone within a natural species are well known for several pine species, including white, loblolly, and Ponderosa. Plant sensitivity to oxidants can be changed by both climatic and edaphic factors. A change in environmental conditions can initiate a change in sensitivity at once, but it will be 3 to 5 days before the response of the plant is totally modified. Plants generally are more sensitive to ozone when grown under short photoperiods, medium light, medium temperature, high humidity, and high soil moisture. Injury from PAN may increase with an increase in light intensity. Conditions during exposure and growth affect the response of plants to oxidants in similar ways. In general, environmental conditions optimum for plant growth tend to increase the sensitivity to ozone. Factors that increase water stress at the time of exposure tend to make plants more tolerant to ozone. Soil moisture is probably the most important environmental factor that affects plant response to oxidants during the normal growing season. Physiologic age affects the response of the leaf to oxidants. Young leaf tissue is most sensitive to PAN, whereas newly expanding and maturing tissue is most sensitive to ozone. Light is required for plant tissue to respond to PAN; a similar light requirement is not needed for plants to respond to ozone.

The majority of effects observed, such as suppression of root growth, mineral uptake, and nitrogen fixation, apparently result from a suppression of photosynthesis and modifications in photosynthate distribution. This suppression of metabolic reserves ultimately slows plant growth and renders the plant more sensitive to other stresses. Physiological changes can provide a sensitive means of monitoring the health and vigor of the plant with or without visible injury. Ozone affects pollen germination in some species and may affect yield through incomplete pollination of flowers. Investigations with *Arabidopsis thaliana* showed no mutagenic effects from ozone over seven generations.

Mixtures of ozone and SO_2 can cause effects below the levels caused by either gas alone; however, there is some disagreement concerning the interactions of ozone with other gases. Ratios of gas mixtures, intermittent exposures, sequential exposures to pollutants, and predisposition by one pollutant to the effects of a second pollutant may be important factors in nature, but insufficient knowledge is available for elucidation of the effects.

The response of plants to oxidants may be conditioned by the presence or absence of biotic pathogens. Depending on the plant and the pathogen, oxidants may cause more or less injury to a given species. Oxidant injury to Ponderosa pine predisposes the trees to later invasion by bark beetles. Ozone and ozone-sulfur dioxide mixtures can decrease the population of some plant-parasitic nematodes. Variable plant responses have been noted when herbicides were used in the presence of high oxidant concentrations.

Little research on the effects of oxidants on ferns, non-

vascular green plants, and microorganisms has been reported. Lichens and mosses are responsive to acid gases, but there is no definite evidence that they respond to oxidants. Ferns may be especially sensitive, but their injury response is different from that of higher vascular plants. Growth and sporulation of fungi on surfaces are usually, but not always, affected. Ozone from 0.1 to several milligrams per liter of solution is required to kill many microorganisms in liquid media. Most work with microorganisms has been done to study the effectiveness of ozone as a biocide in the storage of vegetation or in the treatment of water or sewage.

11. Effects on Ecosystems

Plants, animals, and microorganisms usually do not live alone but exist as populations. Populations live together and interact as communities. Communities, because of the interactions of their populations and of the individuals that comprise them, respond to pollutant stress differently from individuals. Man is an integral part of these communities and, as such, is directly involved in the complex ecological interactions that occur within the communities and within the ecosystem of which the communities are a part.

The stresses placed on the ecosystems and their communities can be far-reaching, inasmuch as the changes that occur may be irreversible. For example, it has been suggested that the arid lands of India are the result of defoliation and elimination of vegetation, which in turn induced local climatic changes that were not conducive to the reestablishment of the original vegetation.

An ecosystem (e.g., the planet earth, a forest, a pond, or a fallen log) is a major ecological unit comprised of living (biotic) and physical (abiotic) components through which the cycling of energy and nutrients occurs. A structured relationship exists between the various components. The biotic units are linked together by functional interdependence, while the abiotic units comprise all of the physical factors and chemical substances that interact with the biotic units. The processes occurring within the biotic and abiotic units and the interactions between them can be influenced by the environment.

Ecosystems tend to change with time. Adaptation, adjustment, and evolution are constantly taking place as the biotic component, the populations, and the communities of living organisms interact with the abiotic component in the environment. Recognizable sequential changes occur. With time, populations and communities may replace one another. This sequential change, termed succession, may culminate in climax communities. Climax communities are structurally complex, are more or less stable, and are held in a steady state through the operation of a particular combination of biotic and abiotic factors. The disturbance or destruction of a climax community or ecosystem results in its being returned to a simpler stage. Existing studies indicate that changes occurring within ecosystems, in response to pollution or other disturbances, follow definite patterns that are similar even in different ecosystems. It is, therefore, possible to predict broadly the basic biotic responses to the disturbance of an ecosystem.

Diversity and structure are most changed by pollution as a result of the elimination of sensitive species of flora and fauna and of the selective removal of the larger overstory plants in favor of plants of small stature. The result is a shift from the complex forest community toward the less complex hardy shrub and herb communities. The opening of the forest canopy changes the environmental stresses on the forest floor, causing differential survival and, consequently, changed gene frequencies in subcanopy species.

Associated with the reduction in diversity and structure is a shortening of food chains, a reduction in the total nutrient inventory, and a return to a simpler successional stage.

It should be emphasized that ecosystems are usually being subjected to a number of stresses at the same time, not just a single perturbation such as oxidant pollution.

The effects of oxidants on the mixed-conifer forest of the San Bernardino Mountains graphically demonstrate the changes which occur in natural ecosystems as discussed above. Since the early 1940's, the San Bernardino Forest has been undergoing stress from oxidants transported long-range from Los Angeles, 140 miles away. Losses of Ponderosa and Jeffrey pines, the overstory vegetation, have increased dramatically as pollutant levels have risen. Black oak has also suffered oxidant injury. The composition of both plant and animal populations has been altered by the death of the Ponderosa and Jeffrey pines.

The interaction of pollutant and inversion layers at the heated mountain slope results in the vertical venting of oxidants over the mountain crest by up-slope flow, thus establishing an elevational gradient of oxidant concentrations. Oxidant concentrations ranging from 100 to 200 $\mu g/m^3$ (0.05 to 0.10 ppm) at altitudes as high as 2432 m, approximately 1033 m above the mountain crest, have been measured by aircraft.

Total oxidant concentrations in the San Bernardino Mountains have been measured continuously from May through September since 1968 at the Rim Forest-Sky Forest monitoring station. During each of the first 7 years of monitoring, between June and September, the total number of hours in which concentrations of ozone were 160 $\mu g/m^3$ (0.08 ppm) or more was never less than 1300. The number of hours in which the total oxidant concentration was 390 $\mu g/m^3$ (0.20 ppm) or higher increased from less than 100 in 1969 to nearly 400 in 1974. It was not uncommon to observe momentary oxidant peaks as high as 1180 $\mu g/m^3$ (0.60 ppm). The duration of oxidant concentrations exceeding 200 $\mu g/m^3$ (0.10 ppm) was 9, 13, 9, and 8 hours per day going from the lower- to the higher-altitude stations.

The most recent data firmly indicate that oxidant concentrations in the San Bernardino Forest will either increase annually or oscillate around the mean of present high concentrations in the foreseeable future.

The transport of the urban plume from the coast northeastward to the mountains can be readily demonstrated. Because of this transport, the permanent vegetation constituting natural ecosystems receives much greater chronic exposure, while the short-lived vegetation constituting the economically more valuable agroecosystem of the Los Angeles coastal plain can be subject to injurious doses, but in intermittent short-term fumigations. Each situation has measurable economic and aesthetic consequences, but on different time scales. The single-species agricultural ecologic system (the agroecosystem) has little resilience to pollutant stress; losses are sometimes immediate and occasionally catastrophic. The complex natural ecosystem is initially more resistant to pollutant stress, but the longer chronic exposures cause disruption of both structure and function in the system that may be irreversible.

The oxidant injury to the mixed conifer stands of the San

Bernardino Mountains that began in the early 1940's, as indicated above, is well advanced. A similar problem is developing in the forests of the southern Sierra Nevada Mountains. Both areas show direct as well as indirect effects on all subsystems of the forest ecosystem: producers, consumers, and decomposers.

12. Effects on Materials

Ozone is a major factor in the overall deterioration of several different types of organic materials. In fact, certain specific organic compounds are more sensitive to ozone attack than are humans or animals. The magnitude of damage is difficult to assess because ozone is one of many oxidizing chemicals in the atmosphere which contribute to the "weathering" of materials. Nevertheless, researchers have shown that ozone accelerates the deterioration of several classes of materials, including elastomers (rubber), textile dyes and fibers, and certain types of paints and coatings.

Although many organic materials have been shown to be susceptible to ozone attack, only certain paints, elastomers, and dyes sustain damage representing significant economic loss. Even the measures to prevent ozone damage to elastomers and dyes constitute a major cost.

APPENDIX G[39]

Hydrocarbons—Health & Other Effects

The conclusions that follow are derived from a careful evaluation of the studies cited in this document, representing the National Air Pollution Control Administration's best judgment of the effects that may occur when various levels of hydrocarbons are reached in the ambient air.

1. Our present state of knowledge does not demonstrate any direct health effects of the gaseous hydrocarbons in the ambient air on populations, although many of the effects attributed to photochemical smog are indirectly related to ambient levels of these hydrocarbons.

2. Injury to sensitive plants has been reported in association with ethylene concentrations of from 1.15 to 575 $\mu g/m^3$ (0.001 to 0.5 ppm) over a time period of 8 to 24 hours.

3. Examination of air quality data indicates than an early morning (6:00 to 9:00 a.m.) concentration of 200 $\mu g/m^3$ (0.3 ppm C) nonmethane hydrocarbon can be expected to produce a maximum hourly average oxidant concentration of up to 200 $\mu g/m^3$ (0.1 ppm).

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Studies conducted thus far of the effects of ambient air concentrations of gaseous hydrocarbons have not demonstrated direct adverse effects from this class of pollution on human health. However, it has been demonstrated that ambient levels of photochemical oxidant, which do have adverse effects on health, are a direct function of gaseous hydrocarbon concentrations; and when promulgating air quality standards for hydrocarbons, their contribution to the formation of oxidant should be taken into account.

An analysis of 3 years of data collected in three American cities shows that on those several days a year when meteorological conditions were most conducive to the formation of photochemical oxidant, nonmethane hydrocarbon concentrations of 200 $\mu g/m^3$ (0.3 ppm C) for the 3-hour period from 6:00 to 9:00 a.m. might produce an average 1-hour photochemical oxidant concentration of up to 200 $\mu g/m^3$ (0.10 ppm) 2 to 4 hours later. The hydrocarbon measurements were confined to 200 $\mu g/m^3$ (0.3 ppm C), or above, because of instrumentation limitations. However, if the functional relationship between the hydrocarbon and photochemical oxidant measurements were extended to include the lowest levels at which photochemical oxidant has been observed to adversely affect human health, the corresponding hydrocarbon concentration would be approximately 100 $\mu g/m^3$ (0.15 ppm).

APPENDIX H[40]

Nitrogen Oxides—Health & Other Effects

1. Introduction

Although the essential role of NO_x in the production of photochemical oxidants is treated from the physical-chemical standpoint in this document, little research has been done to demonstrate the significance of the indirect effects of NO_x on health, vegetation, and materials through the photochemical reaction mechanism; thus, only the direct effects of NO_x are treated here. A P C O publication AP-63, *Air Quality Criteria for Photochemical Oxidants,* provides a comprehensive review of photochemical oxidant effects.

Units of pollution concentration used in this document are expressed as both mass per unit volume (e.g., micrograms per cubic meter, $\mu g/m^3$) and as volume-ratios (e.g., parts per million, ppm). Conversion between these units requires a knowledge of the gas density, which varies with temperature and pressure measurement. In this document 25°C (77° F) has been taken as standard temperature, and 760 mm Hg (atmospheric pressure at sea level) as standard pressure. All references to NO_x are expressed in terms of NO_2 mass per unit volume on the basis of the conversion formula: ppm x 1880 = $\mu g/m^3$ at 25° C, 760 mm Hg, unless otherwise specified. Similarly, hydrocarbons and oxidant concentrations are expressed as mass of methane and ozone per unit volume, respectively.

2. Effects on Light Transmission

Visibility reduction is common in polluted atmospheres. Scattering and absorption of light rays by particles and gases reduce the brightness and contrast of distant objects. The degree of reduction depends on the concentration and properties of the pollutants. Nitrogen dioxide absorbs light energy over the entire visible spectrum, although primarily in the shorter, blue-wavelength regions; thus, NO_2 can by itself reduce visibility. At present, however, under most ambient conditions, aerosols make the major contributions to visibility reduction.

3. Effects on Materials

Significant effects of NO_x have been observed and studied on three classes of materials: textile dyes and additives, natural and synthetic textile fibers, and metals.

The most pronounced problem is associated with textile dyes and additives. Fading of sensitive disperse dyes used on cellulose acetate fibers has been attributed to NO_2 levels below 188 mg/m^3 (<100 ppm). Loss of color, particularly in blue- and green-dyed cotton and viscose rayon, has occurred in gas dryers where NO_x concentrations range from 1.1 to 3.7 mg/m^3 (0.6 to 2 ppm). Yellow discoloration in undyed white and pastel-colored fabrics has recently been attributed to NO_x by controlled laboratory experiments.

Laboratory and field observations have shown that cotton and Nylon textile fibers can be deteriorated by the presence of NO_x, but specific reactants and threshold levels are undetermined at this time.

Failure of nickel-brass wire springs on relays has been related to high particulate nitrate levels. This type of stress corrosion has been observed when surface concentrations of particulate nitrates have exceeded 2.4 $\mu g/cm^2$ and relative humidity was greater than 50 percent. Another type of this corrosion has been associated with annual average particulate nitrate concentrations of 3.0 and 3.4 $\mu g/m^3$ with corresponding NO_x levels of 124 and 158 $\mu g/m^3$ (0.066 and 0.084 ppm).

4. Health Effects

Both of the prominent oxides of nitrogen present in ambient air are potential health hazards. At ambient concentrations, NO presents no direct threat to general health; NO_2 does, however.

The toxicology of nitrous oxide (N_2O) and other oxides of nitrogen does not appear to be relevant to the problems of ambient air pollution at the present time.

1. Nitric Oxide

NO is not an irritant and is not considered to have adverse health effects at concentra-

tions found in the atmosphere. Its greatest toxic potential at ambient concentrations is related to its tendency to undergo oxidation to NO_2. A 12-minute exposure to 3,075 mg/m^3 (2,500 ppm) NO has proved lethal to mice. In addition, NO has been observed to inhibit bacterial hydrogenase activity at lower concentrations–24.6 mg/m^3 (20 ppm). This inhibition was reversible, however, until the exposure reached about 12,300 mg/m^3 (10,000 ppm).

2. Nitrogen Dioxide

NO_2 exerts its primary toxic effect on the lungs. High concentrations, greater than 188 mg/m^3 (100 ppm), are lethal to most animal species; 90 percent of the deaths are caused by pulmonary edema.

The concentration time product determines nonlethal morbidity effects of NO_2 exposures. At 940 $\mu g/m^3$ (0.5 ppm) for 4 hours or 1.9 mg/m^3 (1.0 ppm) for 1 hour, mast cells of rat lungs became degranulated, possibly signifying the onset of an acute inflammatory reaction. These cells returned to normal 24 hours after exposure was terminated. Lung proteins, collagen and elastin, were found to be altered structurally in rabbits exposed to 1.9 mg/m^3 (1 ppm) NO_2 for 1 to 4 hours. The condition was also reversible within 24 hours. Similar changes were observed in rabbits exposed to 470 $\mu g/m^3$ (0.25 ppm) NO_2, 4 hours a day for 6 days, except that recovery was delayed and some denaturation was still apparent 7 days after the final exposure. Denaturation of collagen and elastin associated with repeated exposure to NO_2 has been suggested as a possible factor in the pathogenesis of pulmonary emphysema.

Early pulmonary emphysema-type lesions have been observed in dogs exposed continuously to 47.0 mg/m^3 (25 ppm) for 6 months. In lung tissue from monkeys exposed to 18.8 to 94.0 mg/m^3 (10 to 50 ppm) NO_2 for 2 hours, alveoli were expanded and had thin septal walls. This response involved increasing numbers of alveoli as the NO_2 concentration was increased. Hyperplasia has been observed in respiratory bronchiolar epithelium of hamsters exposed to 94.0 mg/m^3 (50 ppm) for 10 weeks, and a similar response was noted in major bronchi and distal portions of the respiratory tract of hamsters exposed to 18.8 mg/m^3 (100 ppm) for 6 hours.

Long-term exposures to NO_2 concentrations that do not produce acute inflammatory responses have a cumulative, sustained effect, suggestive of a pre-emphysematous condition. Examination of lung tissue from rats exposed to 3.8 mg/m^3 (2 ppm) for their natural lifetimes showed loss of cilia; decreased bronchiolar blebbing; and intercellular, crystalloid, rod-shaped, inclusion bodies. Similar effects have been seen in lungs of rats continuously exposed to 1.5 mg/m^3 (0.8 ppm). Alveoli in lungs of mice exposed to 940 $\mu g/m^3$ (0.5 ppm) for 3 to 12 months on 6-, 18-, and 24-hour daily schedules have shown increase in size from distension rather than from septal breakage. The accompanying inflammation of the bronchiolar epithelium and reduction in distal airway size suggested the development of early focal emphysema.

Rats chronically exposed to 18.8 to 47.0 mg/m^3 (10 to 25 ppm) NO_2 developed compensatory changes, such as polycythemia and thoracic kyphosis, with lateral flaring of the ribs.

Since certain pathological changes seen in animals after experimental NO_2 exposure are similar to changes that occur in the pathogenesis of chronic obstructive pulmonary disease in man, it is suggested that long-term, low-level exposures to NO_2 may play a significant role in the development of chronic lung disease.

Exposure of mice, hamsters, and squirrel monkeys to NO_2 increased susceptibility to bacterial pneumonia and influenza infection. The susceptibility has been demonstrated by a significantly increased mortality, decreased survival time, and a reduction in ability to clear infectious agents from the lungs. In mice, threshold for increased susceptibility to *Klebsiella pneumoniae* occurred after exposure to 6.6 mg/m^3 (3.5 ppm) NO_2 for 2 hours, if the infectious challenge was given within 1 hour after the NO_2 exposure. Squirrel monkeys exposed to 18.8 mg/m^3 (10 ppm) NO_2 for 2 hours and then challenged with *K. pneumonia* aerosol retained the infectious agent in their lungs for extended periods of time.

In long-term studies of mice, significantly increased susceptibility to infection occurred after continuous daily exposure to 940 $\mu g/m^3$ (0.5 ppm) NO_2 for 3 months, and after 6- and

18-hour daily exposures for 6 months. A significant increase in susceptibility to influenza virus or *K. pneumoniae* was also seen in squirrel monkeys continuously exposed to 18.8 and 9.4 mg/m^3 (10 and 5 ppm) NO_2 for 1 and 2 months, respectively. In addition, interferon formation has been impaired and resistance to viral infection has decreased following exposure of rabbits to 47.0 mg/m^3 (25 ppm) NO_2 for 3 hours. Researchers conjecture that such increased susceptibility to infection may also be significant in the pathogenesis of human lung disease.

Inhalation of NO_2 can produce other systemic effects, although these are generally secondary to the effects on the lungs. In monkeys exposed to 28.2 to 94.0 mg/m^3 (15 to 50 ppm) NO_2 for 2 hours, cellular changes appeared in heart, liver, and kidney tissue. A circulating substance, possibly a lung antibody, has been detected in the blood of guinea pigs exposed to 9.4 mg/m^3 (5.0 ppm) for 4 hours daily, 5 days per week for 5.5 months. Rats and monkeys continuously exposed to 3.8 mg/m^3 (2.0 ppm) NO_2 for 3 weeks developed marked polycythemia. Methemoglobin has been detected in the blood of several species exposed to NO_2 concentrations greater than 122 mg/m^3 (70 ppm) for 1 hour.

The small amount of information available concerning the toxicological effects of the oxides of nitrogen in man pertains to levels higher than those found in ambient air. Experimental exposure of volunteer subjects to 9.4 mg/m^3 (5 ppm) NO_2 for 10 minutes has produced a substantial, but transient, increase in airway resistance. Other data, derived from occupational exposure to high-concentration mixtures of NO and NO_2, are complicated by the presence of other pollutants.

5. Effects on Vegetation

The degree of injury occurring with the lower concentrations of NO_2 present in the atmosphere remains to be determined. Exposure of many kinds of plants to concentrations of NO_2 above 47 mg/m^3 (25 ppm) for any extended period causes acute necrotic leaf injury. Such lesions are usually characteristic for each plant, but their nonspecific character in relation to other toxicants renders these symptoms of little value in diagnosing NO_2 damage in the field.

The 1-hour visible-injury-threshold value for NO_2 can be achieved by exposing plants to 18.8 to 28.2 mg/m^3 (10 to 15 ppm). Increasing the exposure time, however, obviates the threshold level; 4.3 to 6.6 mg/m^3 (2.3 to 3.5 ppm) NO_2 administered for 8 to 21 hours and 1.9 mg/m^3 (1 ppm) NO_2 for 48 hours cause equivalent leaf injury. Continuous fumigation with 940 μg/m^3 (0.5 ppm) NO_2 for 35 days resulted in leaf drop and chlorosis in citrus, but no actual necrotic lesions developed.

The effects of exposure to low levels of NO_2 for extended periods are less evident. Recently completed studies suggested that 470 μg/m^3 (0.25 ppm) or less of NO_2, supplied continuously for 8 months will cause increased leaf drop and reduced yield in navel oranges.

The mechanism(s) by which NO_x causes direct injury to plants can only be postulated at this time. Evidence of diurnal fluctuation in sensitivity to NO_2 has been presented, and could indicate that the pollutant is reacting with a particular plant metabolite, which only accumulates at certain periods during the day. The absence of a protective metabolite within the plant at certain periods would also cause a diurnal sensitivity.

Limited information regarding the effect of nitric oxide on photosynthesis indicates that NO would reduce the growth of plants if concentrations in the range of 3.8 to 7.5 mg/m^3 (2.0 to 4.0 ppm) persisted continuously.

APPENDIX I[41]

Lead—Health & Other Effects

1. Health Effects

There are various physiological levels and exposure ranges at which the effects of lead in man occur. Furthermore, lead affects man at the subcellular, cellular, and organ system levels.

Among subcellular components, both nuclei and mitochondria generally show the most pronounced responses to cellular invasion by lead. The mitochondria, however, are most vulnerable and sustain the greatest functional impairment. Mitochondrial injury, both in terms of cellular energetics and morphological aberration, has been shown in a number of experimental animals. In man, the evidence for mitochondrial impairment has been morphological rather than functional. Those subcellular changes observed are primarily the development of nuclear inclusion bodies in kidney cells as well as mitochondrial changes in renal tubular cells in people exposed occupationally.

Any discussion of the subcellular effects of lead must consider the question of chromosomal aberrations and carcinogenesis. At the present time, no conclusive statements can be made about the induction of chromosomal damage by lead. The literature on this issue either yields conflicting information or describes studies that are difficult to compare with each other. Some experimental animal studies relate the development of cancer to relatively high doses of lead, but as is true in the case of other suspected carcinogens, there are no data corroborating these findings in man.

Among the systemic and organic effects of lead, important areas are its hematologic, neurobehavioral, and renal effects. Attention must also be given, however, to the effects of lead on reproduction and development as well as its hepatic, endocrine, cardiovascular, immunologic, and gastrointestinal effects.

A number of significant effects of lead on the hematopoietic system in humans has been observed in lead poisoning. These effects are prominent in clinical lead poisoning but are still present to a lesser degree in persons with a lower level of lead exposure.

Anemia is a clinical feature of lead intoxication, resulting from both increased erythrocyte destruction and decreased hemoglobin synthesis. In children, a threshold blood-lead level for production of these symptoms of anemia is approximately 40 μg lead/dl, while the corresponding value for adults appears to be 50 μg lead/dl.

Hemoglobin synthesis is impaired by lead via inhibition of synthesis of the globin moiety and inhibition at several steps in the synthesis of heme. Also inhibited by lead is the incorporation of iron into protoporphyrin to form heme, the prosthetic group of hemoglobin. This results in the accumulation of coproporphyrin, which is excreted in the urine, and of protoporphyrin, which is retained in the erythrocytes. The overall effect of lead is a net decrease in heme synthesis.

An increase in free erythrocyte protoporphyrin (FEP) occurs at blood lead levels of 16 μg/dl in children. In adult females, this threshold is probably similar. In adult males, the value is 20 to 25 μg/dl. To the extent that the protoporphyrin elevation is a likely indicator of the impairment of mitochondrial function in erythroid tissue, it may be even more important. For these reasons, physicians who participated in the development of the 1975 statement by the Center for Disease Control and the American Academy of Pediatrics reached a consensus that elevated FEP should be used as an indicator of increased exposure to lead.

The effects of lead on the nervous system range from acute intoxication and fatal encephalopathy to subtle behavioral and electrophysiologic changes associated with lower level exposures. Changes throughout the range of effects are related to blood lead levels.

It would appear that surprisingly low levels of blood lead may sometimes be associated with the most extreme effects of lead poisoning — severe, irreversible brain damage as indexed by the occurrence of acute or chronic encephalopathy symptoms and/or death. While for most adults such damage does not occur until blood-lead levels substantially exceed 120 μg/dl, some evidence suggests that acute encephalopathy and death may occur in some adults at blood-lead levels slightly below 100 μg/dl. For children, the effective blood-levels for producing encephalopathy or death are lower than for adults, with such effects being seen somewhat more often, starting at approximately 100 μg/dl. Again, however, evidence exists for the occurrence of encephalopathy in a very few cases at lower levels, down to about 80 μg/dl.

It should be noted that once encephalopathy occurs, then death can be a frequent outcome, regardless of the level of medical intervention at the time of the acute crisis. It is also crucial to cite the rapidity with which acute encephalopathy or death can develop in apparently asymptomatic individuals or in those apparently only mildly affected by elevated body burdens of lead. It is not unusual for rapid deterioration to occur, with convulsions or coma suddenly appearing and progressing to death within 48 hours.

This suggests that at high blood-lead levels, even when individuals are asymptomatic, rather severe neural damage can likely exist without overt manifestations. Studies showing that apparently "asymptomatic" children having high blood lead levels of over 80 to 100 μg/dl are permanently impaired cognitively, as are individuals who survive acute episodes of lead encephalopathy, tend to support the hypothesis that significant if albeit subtle changes in neural function occur at what were once considered tolerable blood-lead levels.

Other evidence tends to confirm rather well that some types of neural damage does exist in "asymptomatic" children and not necessarily only at very high levels of blood lead. The body of studies on "low" or "moderate"

level lead effects on neurobehavioral functions present, overall, an impressive array of data pointing to that conclusion. Several well-controlled studies find effects that are clearly statistically significant, while many others report non-significant but "borderline" effects. Since some of the effects at low levels of lead exposure discussed in this document are of a subtle nature, the findings are not always striking in individual cases. Nevertheless, when the results of all of the studies on neurologic and behavioral effects at "subclinical" exposures are considered in an overall perspective, a rather consistent pattern of impaired neural and cognitive functions appears to be associated with blood levels below those producing the overt symptomatology of lead encephalopathy. The blood lead levels at which neurobehavioral deficits occur in otherwise "asymptomatic" children appear to start at a range of 50 to 60 μg/dl, although some evidence tentatively suggests that such effects may occur at slightly lower levels for some children.

Data obtained for the effects of lead on the nervous system in laboratory animals are also quite extensive. Encephalopathy is produced by high-level perinatal exposure to lead; in different species this occurs to varying degrees as characterized by the relative extent of neuronal degeneration and vasculopathy. It seems clear that the animal data support the contention that the developing organism represents the population at greatest risk for central nervous system toxicity.

There is also good evidence that perinatal exposure to lead in laboratory animals, at moderate exposure levels, will produce delays in both neurological and sexual development. Since these effects have been demonstrated to occur in the absence of either undernutrition or growth retardation, it has been suggested that they may represent more or less direct effects of lead in the respective systems.

In animal studies, locomotor activity has been the most commonly used behavioral index of lead toxicity. Data indicate that increased locomotor activity in young animals occurs only at moderately high exposure levels. It may be, in view of the levels, that the changes in activity currently reported in laboratory animals are more diagnostic of a post-encephalopathic hyperactivity than of subclinical effects. Interestingly, the reactivity changes seen in older animals are associated with much lower blood lead levels.

Finally, reports on the effects of lead exposures on the acquisition and/or performance of operant responses indicate that perinatal exposure to moderate or low levels of lead may disrupt this type of behavior. Thus, at blood lead levels in the range 30 to 80 μg/dl, cognitive function appears to be disrupted in animals.

Excessive lead exposure can result in acute as well as chronic renal injury in man. The acute renal effects of lead are seen in persons dying of acute lead poisoning where lead-induced anemia and/or encephalopathy may also be seen. These effects are manifested by nonspecific degenerative changes in renal tubular lining cells, cloudy swelling, and some degree of cellular necrosis. In addition, nuclear inclusion bodies form in tubule cells and there are functional and ultrastructural changes in tubular mitochondria. Aminoaciduria, glycosuria, and hyperphosphaturia are noted, with aminoaciduria being a rather consistent feature of tubular damage in children. These effects are usually reversible. It is not possible at the present time to state what level of lead in blood is associated with aminoaciduria or any of the other specific indices of acute renal injury.

Prolonged lead exposure in humans can result in chronic lead nephropathy. The pathology of these chronic changes is different than that seen in acute renal injury. It is characterized by the gradual onset of pronounced arteriosclerotic changes, fibrosis, glomerular atrophy, hyaline degeneration, and reduction in kidney size. This can be a progressive, irreversible condition resulting in death from renal failure. A threshold of lead exposure for those chronic changes, however, cannot yet be stated due to the typical inaccessability of data needed for the accurate assessment of the preceding long-term exposure history.

Considerable evidence for the adverse effects of lead on reproduction and development in man has been accumulating for many years. Many of the early data on the induction of abortions, stillbirths, and neonatal deaths were for occupationally exposed pregnant women, where such effects were demonstrated at high blood lead levels. Of more pressing present interest are certain recent studies in this area focusing on two aspects of the effects of "low"[f] to "moderate" lead exposure on reproduction: gametotoxicity and post-conception events.

In regard to potential lead effects on human ovarian function, one study has shown that short-term exposure at ambient air levels of less than 7 $\mu g/m^3$ may cause an increase in the anovular cycle and disturbances in the lutein phase. This study, however, requires confirmation before conclusive statements can be made. Another recent report involving occupational exposure similarly suggests that moderately increased lead absorption (blood lead mean = 52.8 μg/dl) may result in direct testicular impairment; however, the design of this study is such that this observation also requires verification.

Thus, it is clear that gametotoxic, embryotoxic, and teratogenic effects at a gross level can be induced in laboratory animals with lead, but it should be emphasized that the production of such effects probably requires acute, high exposures. Unfortunately, a paucity of information exists on the teratogenicity and developmental toxicity of chronic "low" or "moderate" lead exposures. Available data on the subject do suggest, however, that chronic low-level lead exposure may induce postnatal developmental delays in rats.

Our present knowledge about the effects of lead in man on the hepatic, cardiovascular, immunologic, and endocrine systems is fragmentary, rendering it difficult to make any conclusive statements about quantitative relationships. For example, effects of lead on the endocrine system are not well defined at present. Thyroid function in man, however, has been shown to be decreased in occupational plumbism. Also, effects of lead on pituitary and adrenal function in man have been observed, decreased secretion of pituitary gonadotrophic hormones being noted but adrenal function effects being a less consistent finding.

The response of the hepatic system to lead has not been well characterized in man; instead, much of the literature is on hepatic effects in experimental animals. Lead-

poisoned animals show significantly impaired drug-metabolizing activities, suggesting an effect on the hepatic mixed-function oxidase system. Since detoxification in animals depends on the microsomal heme protein, cytochrome P450, and since heme biosynthesis is impaired in lead exposure, such an effect is a logical consequence of lead poisoning.

Of more direct interest in terms of reproductive efficiency are the effects of lead exposure on pregnant women, not only fetal health and development but also maternal complications. Placental transfer of lead has been demonstrated both by fetal tissue analysis and comparison of newborn umbilical cord blood lead with maternal blood lead. One must not only consider the resulting absorption of lead by the fetus but also the specific points in embryonic development at which exposure occurs. Fetal tissue uptake of lead occurs by the end of the first trimester, which may be a sensitive period in embryonic development of the nervous system.

Studies comparing umbilical cord blood lead levels in newborns with simultaneously sampled maternal blood show that the newborn and maternal levels are closely correlated. The studies have also shown that the newborn of mothers in an urban setting are born with higher blood levels, in general, than corresponding newborns from rural areas.

That the prenatal exposure of the fetus to lead, even in the absence of teratogenic effects, is of consequence for adverse health effects is shown by studies relating fetal levels to changes in fetal heme synthesis and to the incidence of premature births. Some suggestions in the literature that heme biosynthesis in a newborn may be affected require confirmation.

In evaluating maternal complications related to lead exposure, one must consider that pregnancy is a physiological stress that may place the pregnant woman at higher risk to lead exposure effects. Both iron and calcium deficiency increase the susceptibility of an individual to lead toxicity. Women have an increased risk of both deficiencies during pregnancy and postpartum.

Some information is available, but requires confirmation, that the risk of premature rupture of the amniotic membrane may be higher in cases of elevated exposure than in age-matched controls not having such exposure.

The literature leaves little doubt about deleterious health effects of lead on reproduction, but most reports do not provide specific descriptions of exposure levels at which specific reproductive effects are noted. Maternal blood lead levels of approximately 30μg/dl may be associated with a higher incidence of premature delivery and premature membrane rupture, but these observations require confirmation. In adult males, levels of 50 to 80 μg/dl may be sufficient to induce significant spermatotoxic effects but this has not been conclusively demonstrated.

Lead has not been shown to be teratogenic in man, but animal experiments have demonstrated that high levels of lead which are still compatible with life in sexually mature animals interfere with normal reproduction; these studies include assessment of lead effects in both parents. Reduction in offspring number, weight, and survival and an increase in fetal resorption is a consistent finding in rats, mice, and other species over a range of high-level lead exposures. Effects on offspring have been shown to involve the gametotoxic effect of lead on males as well as females in a number of animal species.

At lead levels presently encountered in occupational exposure, no significant cardiovascular effects are discernible. Clinical data for children suffering from chronic lead poisoning resulting in death indicate that extensive myocardial damage occurs. It is not clear that the associated morphological changes are a specific response to lead intoxication. However, in many instances where encephalopathy is present, the electrocardiographic abnormalities disappear with chelation therapy.

There are insufficient data pertaining to the effects of elevated blood lead levels and the incidence of infectious diseases in man to allow the derivation of a dose-response relationship. Neither can a dose-response relationship be defined for the effects of elevated blood lead levels on the gastrointestinal tract, even though colic is usually a consistent early symptom of lead poisoning, in adults exposed occupationally and in infants and young children.

2. Effects on the Ecosystem

As a natural constituent, lead does not usually pose a threat to ecosystems. The redistribution of naturally occurring lead in the environment, however, has now caused some concern that lead may represent a potential threat to the ecosystem. For example, studies have shown a fivefold increase in lead in tree rings during the last 50 years: this accumulation may serve as a useful index of patterns of environmental lead accumulation.

There are also documented effects of lead on domestic animals, wildlife, and aquatic life. Lead poisoning in domestic animals produces varying degrees of derangement of the central nervous system, gastrointestinal tract, muscular system, and hematopoietic system. As is true in man, younger animals appear to be more sensitive than older ones.

Wildlife are exposed to a wide range of lead levels. Toxic effects from ingestion of lead shot have long been recognized as a major health problem in waterfowl. Several species of small mammals trapped along roadways were tested for lead concentrations. All but one of the species living in habitats adjacent to high-volume traffic showed higher concentrations of lead. This was especially true in urban areas.

Lead toxicity in aquatic organisms has been observed and studied experimentally. Symptoms of chronic lead poisoning in fish include anemia, possible damage to the respiratory system, growth inhibition, and retardation of sexual maturity.

There is evidence that lead has both harmful and beneficial effects on plants. Plants are exposed to lead through the leaves, stems, bark, or roots and the extent of the effects depends upon the form, amount, and availability of that lead. The morphology of the plant surface plays the major role in determining the type and quantity of material retained by plants. Meteorological factors are also important in determining the fate of lead that comes into contact with plants. Large deposits of inert insoluble metal compounds on the leaves are probably of little consequence to plants as the most important factor is the solubility of the metal. Thus, because inorganic lead compounds are generally of low solubility, there is little incorporation and accumulation within the leaves of plants.

The majority of studies reporting lead toxicity in plants have been conducted with plants grown in artificial nutrient culutre. These studies have promoted the concept that the effects of lead are dependent on a variety of environmental factors, including anions and cations within the plant and in the growth media, and the physical and chemical characteristics of the soil itself. As lead interacts with many environmental factors, specific correlations between lead effects and lead concentrations are extremely difficult to predict.

3. Effects on Populations

The frequency distribution of blood lead levels in homogeneous human populations has almost invariably been found to be lognormal. Most data sets of homogeneous populations display a geometric standard deviation (GSD) of 1.3 to 1.5. This would roughly correspond, for example, to an arithmetic standard deviation of 5.3 to 8.5 μg/dl at a mean blood lead of 20μg/dl.

From the lognormal distributiion, given a mean blood lead level and estimated GSD, it is possible to predict the percentage of a population whose blood lead levels exceed or fall below a specified value. It is also possible to estimate the probable increase in mean blood lead levels for a population exposed to specific increases in environmental lead. These two procedures, used together, provide a method by which air quality standards may be chosen to protect the health of the population.

Blood lead levels vary with geographic location. They are lowest in some remote populations, higher in most rural settings, higher still in suburban areas, and highest in inner-city areas. This gradient follows the presumed lead exposure gradient. Blood lead values also vary by age, sex, and race, although in a somewhat more complex fashion. Generally, young children have the highest levels, with little difference noted between sexes at this age. In older segments of the population, after elimination of occupational exposure in lead workers, males still have a higher blood lead than females. Only limited published data are available comparing the blood levels of the various racial and ethnic groups of the population. Those data suggest that urban Blacks have higher lead levels than Whites, with levels in Puerto Ricans frequently being intermediate.

Results of the numerous studies of environmental exposures of man have indicated strongly that man does indeed have cumulative uptake from each source to which he is exposed. Equally important, these studies have shown that the blood lead level represents a summation of the absorption from each of these sources.

Data for the two most widespread environmental sources of lead other than food permit summary statements concerning their quantitative relationship with blood lead levels: air and soil/dust. Blood lead levels were found to increase with rising air lead concentrations. The relationships were found to be either log-linear or log-log. Evaluation of the equations at various commonly observed air lead levels revealed that the ratio between changes in blood and changes in air lead varied generally between 1 and 2 and that it was not constant over the range of air exposures. This implies that an increase of 1 μg/m3 of air lead results in an average increase of 1 to 2 μg/dl in blood lead levels. Suggestive evidence indicates that children may have higher ratios than adults and that males may have higher ratios than females.

One of the most extensive data sets on blood lead in children comes from a study by the United States Department of Housing and Urban Development on the blood lead values of approximately 180,000 children in New York City. These data covered the period March 1970 through December 1976. A preliminary analysis of these important research findings was presented to the Subcommittee on Lead of EPA's Science Advisory Board in October 1977. The following patterns appear to be indicated by these data:

1. There is a definite difference in blood lead values for racial and ethnic groups, Blacks having higher mean lead levels and Hispanics and whites having lower levels.

2. Analysis shows that the mean blood lead level is related to race and ethnicity, age, and year of sampling. This age dependence is similar for all years; the 1- to 12-month-old group has the lowest levels and, generally, the maximum is found in 2- to 4-year-olds.

3. There was a consistent decrease of mean Pb-B levels over the course of the study. This decrease was coincident with a reduction in lead levels in gasoline in the New York City area.

4. There appears to be a likely corresponding decrease in the air lead. However, it should be pointed out that air lead data for New York City are sparse and that it would be unwise to assume that the air lead level as measured at a single location would be the same for all locations. Because of the height of the sampler, it is also questionable whether the air lead level would represent the level to which the population is exposed.

Consistent relationships between blood lead levels and exposure to lead-containing soil have been shown. Also, children exposed to higher concentrations of soil and house dust lead have been shown to have elevated concentrations of lead on their hands. The intermediate link, from elevated hand levels of lead to elevated blood levels, has not yet been established. Quantitatively, blood lead levels have been shown to increase 3 to 6 percent given a doubling of the soil or dust lead content.

Significant water lead exposures in this country have only occurred in places having a soft water supply and using leaded pipes. Such exposures have been shown to be associated with significant elevations of blood lead. They have also been linked to cases of mental retardation.

Exposure to leaded paint still comprises a very serious problem for American children in urban settings. Although new regulations of the lead content of paint should alleviate the problem in new housing, the poorly enforced regulation and lack of regulation of the past have left a heavy burden of lead exposures. Most of the studies on lead poisoning in children have assumed an association with leaded paint. It is very difficult in these studies to measure the actual amount of exposure. There is, nevertheless, incontrovertible evidence that the contribution from this source is very significant for certain segments of the population.

Food lead exposures are thought to be a source of a significant portion of blood lead. Precise quantitative es-

APPENDIX J[42]

State Plan Implementation by Jefferson County/Louisville Air Pollution Control District

(1) Conduct air contaminant source registration in a manner consistent with the provisions of 401 KAR 50:030. Maintain an updated file of registration. Submit to the Department on an annual basis a listing of the updated registration file and on a semi-annual basis a listing of the new or corrected registrations. Such reporting is to be made according to a format supplied by the Department.

(2) Process permit applications for installation or operation of air contaminant sources in a manner consistent with 401 KAR 50:035. Submit to the Department on a semi-annual basis a listing of the permits granted, renewed, denied, or revoked. Such reporting is to be made according to a format supplied by the Department.

(3) Require the owner or operator of any air contaminant source to install, use and maintain monitoring equipment in a manner consistent with 401 KAR 50:050. Submit to the Department on a semi-annual basis a listing of these sources that were required to install or have installed the monitoring devices. Maintain a file of the periodic emission reports submitted by the sources for inspection by the Department.

(4) Report to this Department on a semi-annual basis a listing of shutdown of air pollution control equipment as required by 401 KAR 50:055.

(5) Identify and require major air contaminant sources that emit 100 tons/year or more of any air pollutant to submit emission reduction standby plans for air pollution emergencies as required by 401 KAR 55:020. Review, approve, or disapprove such plans, in conjunction with the Department. Enforce the provisions of such plans during localized air pollution emergencies which do not extend beyond Jefferson County. Assist the Department in enforcing the provisions of such plans during air pollution emergencies that are interstate in scope. Maintain an emergency control center as described in Chapter VI of this Plan.

(6) Maintain an updated emission inventory of air pollutants for point sources and area sources. Submit to the Department on an annual basis an updated emission inventory outlining the assumptions made and method used in calculations, and according to a format supplied by the Department.

(7) Enforce the prohibiton of open burning according to the provisions of 401 KAR 63:005 as a minimum. Report to the Department on a semi-annual basis a listing of violations and enforcement actions taken according to a format supplied by the Department.

(8) Enforce as a minimum the provisions of 401 KAR Chapters 59 and 61. Report to the Department on a semi-annual basis all enforcement actions taken according to a format submitted by the Department.

(9) Conduct a program of continuous and intermittent air quality monitoring as outlined in Chapter VIII of this Plan as a minimum, using sampling and analysis methods approved by the Department. Maintain necessary per-

timates of the relationship between food and blood lead, however, are not available. Similarly, precise quantitative estimates are not available for the relative contributioins of different sources to the total amount of lead in the diet. It is clear, however, that probably the largest proportion of dietary lead is derived from food processing, e.g., from solder in the seams of cans, and some is also derived from lead in the air and the soil.

sonnel, equipment and supplies to a high degree of data integrity. Submit to the Department on a quarterly basis, air quality monitoring data and statistical summaries according to a format supplied by the Department.

(10) Conduct all field emission surveillance in a manner consistent with the work plan outlined in Chapter X of this Plan. Report to the Department on a semi-annual basis a summary of the inspection and enforcement actions according to a format furnished by the Department.

(11) Administer the District's program as required by KRS Chapter 77 for fiscal and administrative matters.

(12) Semi-annual periods shall be January 1 - June 30 and July 1 - December 31. Quarterly periods shall be January 1 - March 31, April 1 - June 30, July 1 - September 30, and October 1 - December 31.

(13) Implement all procedures and control measures associated with alert, warning, and emergency episodes as specified in Title 401, Chapter 55 of the regulations of this Department.

APPENDIX K

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Region IV, Surveillance and Analysis Division
College Station Road, Athens, GA 30601

SUBJECT: Evaluation of Kentucky Division of Air Pollution Laboratory and Monitoring Activities

DATE: FEB 4 1976

FROM: James H. Finger

TO: G. Tom Helms

SUMMARY

In accordance with 40 CFR 35.410(a) the laboratory and ambient air monitoring activities of the Kentucky Division of Air Pollution were evaluated to determine agency performance in meeting commitments for Air Objective IV-1-b in the FY-76 Program Plans. The laboratory was evaluated via a mailed questionnaire.

The subject agency's operations and maintenance schedules for the operation of laboratory facilities for sample analysis and data reduction meet the above commitments.

The subject agency's operations, calibration and maintenance schedules for the operation of monitoring facilities for ambient air monitoring and data reduction meet the above commitments.

The subject agency's laboratory has acceptably implemented a quality assurance program.

The subject agency's quality assurance program has been acceptably implemented in ambient air monitoring.

ACTION

We recommend that Federal funding for the subject agency be continued contingent upon continued progress in present programs.

BACKGROUND

- FY-76 Program Plans
- 40 CFR Part 35.527b(3) and 35.530(c)
- Guidelines for Development of a Quality Assurance Program.
  - Reference Method for the Measurement for:
    1) Carbon Monoxide EPA-R4-73-028a
    2) Suspended Particulates EPA-R4-73-208b

3) Photochemical Oxidants EPA-R4-73-028c

4) Sulfur Dioxide EPA-R4-73-028d

5) National Ambient Air Quality Standards Which Have Been Promulgated EPA-R4-73-028e.

- Quality Control Practices In Processing Air Pollution Samples APTD 1132.

- Guidelines For Technical Services Of A State Air Pollution Control Agency APTD 1347.

cc: Jim Wilburn
Tom Bennett
Doyle T. Brittain

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Region IV, Surveillance & Analysis Division, College Station Rd., Athens, GA 30605

DATE: OCT 13 1977

SUBJECT: Evaluation of the Air Laboratory and Monitoring Activities of the State of Kentucky's Department for Natural Resources and Environmental Protection

FROM: Billy H. Adams for
James H. Finger, Director
Surveillance and Analysis Division

TO: Asa Foster, Director
Air and Hazardous Material Division

SUMMARY

In accordance with 40 CFR 35.410(a), the laboratory and ambient air monitoring activities of the subject agency were evaluated to determine agency performance in meeting air objective eleven (11) in the FY-77 Program Plans. The evaluation was conducted by an EPA Surveillance and Analysis Division team, using the "Criteria and Procedures for the Evaluation of Ambient Air Monitoring Programs, " Draft III, November 3, 1976. The laboratory activities were evaluated by utilizing the survey questionnaire completed on May 17, 1977. The field activities were visited and evaluated on July 19, 1977. Specific details of the evaluation are contained in the attached questionnaires. In general, the findings of the evaluation were as follows:

Laboratory

- This agency's laboratory activities continue to serve as an excellent model of how a laboratory should function.

The subject agency's laboratory activities exceed the minimum criteria that an air pollution control agency laboratory must meet for Regional approval of the ambient air surveillance program.

Field

- The agency is generating data of high quality. However, in some cases the documentation is not available to support it.

- The agency is placing confidence limits on the continuous data and is preparing to generate confidence limits on the data from the manual instruments.

The subject agency's field-monitoring activities meet the minimal criteria that an air pollution control agency's field monitoring program must meet for Regional approval of the ambient air surveillance programs.

ACTION

One of the attached copies is for your information. We request that, in the very near future, the additional copy be forwarded to the director of the subject agency.

BACKGROUND

- Criteria and Procedures for the Evaluation of Air Monitoring Programs, Laboratory and Field, Draft III, November 3, 1976.
- FY-77 Program Plans

cc: Paul Traina
Bobby Carroll
Doyle Brittain

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Region IV, Surveillance and Analysis Division, College Station Road, Athens, GA 30605

DATE: OCT 17 1978

SUBJECT: Evaluation of the Air Laboratory and Monitoring Activities of the Department for Natural Resources and Environmental Protection, State of Kentucky

FROM: James H. Finger, Director
Surveillance and Analysis Division

TO: George L. Harlow, Acting Director
Air and Hazardous Materials Division

SUMMARY

In accordance with 40 CFR 35.410(a), the laboratory and ambient air monitoring quality assurance program activities of the subject agency were evaluated to determine agency performance in meeting air objectives in the FY-78 Program Plans. The evaluation was conducted by an EPA Surveillance and Analysis Division team, using the "Criteria and Procedures for the Evaluation of Ambient Air Monitoring Programs," Draft IV, June 1, 1978. Specific details of the August 9, 1978 evaluation are contained in the attached questionnaires. The laboratory's activities were evaluated by using the criteria questionnaire in lieu of an on-site visit. In general, the findings of the evaluation were as follows:

Laboratory:

- This excellent laboratory appears to have the capability to produce quality analytical data from samples received in the laboratory from the field. Samples are analyzed under exacting conditions with excellent operational quality control, using reference and/or equivalent methods.

- The subject agency's laboratory activities exceed the minimum criteria that an air pollution control agency laboratory must meet for Regional approval of the ambient air surveillance program.

Field:

- This agency continues to be a technological leader in the ambient air monitoring field. It produces high quality ambient air data.

- The subject agency's field-monitoring activities meet the criteria that an air pollution control agency's field monitoring program must meet for Regional approval of the ambient air surveillance programs.

ACTION

One of the attached copies is for your information. We request that, in the very near future, the additional copy be forwarded to the director of the subject agency.

BACKGROUND

- Criteria and Procedures for the Evaluation of Air Monitoring Programs. Laboratory and Field, Draft IV, June 1, 1978.

- FY-78 Program Plans

Enclosures

cc: Paul Traina
Bobby J. Carroll
Doyle Brittain

UNITED STATES ENVIRONMENTAL PROTECTION AGENCY
Region IV, Surveillance & Analysis Division, College Station Rd., Athens, GA 30605

DATE: JUL 20 19[illegible]

SUBJECT: Evaluation of the Air Laboratory and Monitoring Activities of the Division of Air Pollution Control, Kentucky Department for Natural Resources and Environmental Protection

FROM: Director
Surveillance and Analysis Division

TO: Joseph Franzmathes, Director
Office of Program Integration Operation

Summary

In accordance with 40 CFR 35.410(a), on April 17 and 18, 1979, the laboratory and ambient air monitoring quality assurance program activities of the subject agency were evaluated to determine agency performance in meeting air objectives in the FY-79 Program Plans. The on-site evaluation was conducted by an EPA Surveillance and Analysis division team, using the "Criteria and Procedures for the Evaluation of Ambient Air Monitoring Programs," Draft IV, June 1, 1978. Specific details of this evaluation are contained in the attached questionnaires. In general, the findings of the evaluation were as follows:

- This agency continues to be a leader in the field of Ambient Air Monitoring. This is demonstrated by excellent field and laboratory activities, genuine co-operation, and sincerely dedicated personnel.

- Of special note is that personnel of the subject agency conducted an on-site evaluation of the Jefferson County Air Pollution Control District and reported their findings to the SAD evaluation team.

Laboratory

- This excellent laboratory appears to have the capability to produce quality analytical data from samples received in the laboratory from the field. All samples are analyzed under exacting conditions with excellent operational quality control, using reference and/or equivalent methods.

- The subject agency's laboratory activities exceed the minimum criteria necessary for Regional approval of the ambient air surveillance program.

Field

- This agency continues to be a technological leader in the

ambient air monitoring field and continues to produce high quality ambient air data.

- The subject agency's field-monitoring activities exceed the minimum criteria necessary for Regional approval of the ambient air surveillance program.

Action

One of the attached copies is for your information. We request that, in the very near future, the additional copy be forwarded to the director of the subject agency.

Background

- Criteria and Procedures for the Evaluation of Air Monitoring Programs, Laboratory and Field, Draft IV, June 1, 1978.

- FY-79 Program Plans

Billy N. Adams/bar
James H. Finger

Enclosures

cc - Sanford Harvey
Bobby Carroll
Doyle Brittain
Tom Devine

APPENDIX L[43]

Process of Ozone Transport

On a "first" day in an urban area, NOx and HC are emitted into the atmosphere, mixed with any pollutants transported from upwind, and exposed to solar radiation. The resulting chains of reactions generate ozone over a period of several hours. Local transport of the precursors causes the peak ozone concentrations during the day to occur downwind of the urban area. Other mechanisms contributing to this downwind peaking include the time required for the reactions to proceed, the relative lack of NO scavenging (NO reacts with ozone) downwind, and the "dilution effect;" i.e., the apparently more efficient ozone production which occurs when the precursor mixture is diluted.

At night, atmospheric temperature inversion, which inhibits vertical air circulation, protects ozone aloft from destruction by NO scavenging,while ground level ozone tends to be destroyed (although high ground levels have been measured at night). The following day, inversion breakup allows downward mixing of the ozone and any unreacted precursors aloft with the new day's local precursor emissions and any other pollutants transported from upwind. Some investigators believe that the downward mixed ozone can contribute substantially to the new day's urban ozone levels; other believe that only ozone generated in the mixed layer is important--that ozone aloft will be scavenged by NO upon downward mixing into urban areas. Such may not be the case in rural areas where NO concentrations are low. Some also believe that the second day irradiation of unreacted precursors aloft may contribute substantially to the new day's ozone levels. In any event, the downwind ozone levels are not necessarily primarily determined by local or same-day emissions.

When regional high pressure systems (with accompanying features of high solar radiation, slow ventilation, and nocturnal inversion) move across areas of anthropogenic sources, elevated ozone blanket levels tend to occur on the backsides of the systems where ozone and/or its precursors aloft have been resident and accumulating in parcels of air 2-4 days. Under such conditions, ozone can be transported up to 1,000 km. Multiple-day irradiation of less reactive anthropogenic precursors and high minimum ozone levels left over from previous days,on which the photochemical generation can build,are among the mechanisms cited to account for high rural ozone blanket levels.