Attachment No. CCC 1

SCAQMD Rule 445 and associated studies

RULE 445. WOOD BURNING DEVICES

(a) Purpose

The purpose of this rule is to reduce the emission of particulate matter from wood burning devices.

(b) Applicability

The provisions of this rule shall apply to specified persons or businesses within the South Coast Air Basin portion of the South Coast Air Quality Management District:

(1) Any person that manufacturers, sells, or offers for sale, or installs a wood burning device;

(2) Any commercial firewood facility that sells, offers for sale, or supplies wood intended for burning in a wood burning device or portable outdoor wood burning device; and

(3) Any land owner or land occupier that operates a wood burning device or portable outdoor wood burning device.

(c) Definitions

(1) COMMERCIAL FIREWOOD FACILITY means any operation that has a business license that sells, or offers for sale, bulk firewood.

(2) COOKSTOVE means any wood-fired device used primarily for cooking food as defined in Title 40 of the Code of Federal Regulations Section 60.531, February 28, 1988, or subsequently revised.

(3) DEDICATED GASEOUS-FUELED FIREPLACE means a fireplace, including, but not limited to, a gas log fireplace, either constructed on-site, or factory built, fueled exclusively with a gaseous fuel such that the burner pan and associated equipment are affixed to the masonry or metal base of the fireplace.

(4) FIREPLACE means any permanently installed masonry or factory-built device used for aesthetic or space-heating purposes and designed to operate with an air-to-fuel ratio greater than or equal to 35-to-1.

(5) LOW INCOME HOUSEHOLD means any household that receives financial assistance through reduced electric or gas bills from an electric or natural gas utility based on household income levels.

(6) MANDATORY WOOD BURNING CURTAILMENT DAY means any calendar day so declared to the public by the Executive Officer when levels of particulate matter of 2.5 microns in size or less (PM2.5) is forecast to exceed 35 $\mu g/m^3$.

(7) MANUFACTURED FIRELOG means a commercial product expressly manufactured for use to simulate a wood burning fire in a wood burning device.

(8) MASONRY HEATER means any permanently installed device that meets the definition of a masonry heater in ASTM E 1602-03.

(9) NEW DEVELOPMENT means residential or commercial, single or multi-building unit, which begins construction on or after March 9, 2009. For the purposes of this definition, construction has begun when the building permit has been approved or when the foundation for the structure is started, whichever occurs first.

(10) PELLET-FUELED WOOD BURNING HEATER means any wood burning heater that is operated on any pellet-fuel, and is either U.S. EPA Phase II-certified or exempted under U.S. EPA requirements as defined in Title 40 Code of Federal Regulations, Part 60, Subpart AAA, February 28, 1988, or subsequent revisions.

(11) PERMANENTLY INSTALLED means any device built or installed in such a manner that the device is attached to the ground, floor, or wall, and is not readily moveable. A free standing stove that is attached to an exhaust system that is built into or through a wall is considered permanently installed.

(12) PORTABLE OUTDOOR WOOD BURNING DEVICE means any portable outdoor device burning any solid fuel for aesthetic or space heating purposes including, but not limited to, fireplaces, burn bowls, and chimineas located on property zoned for residential uses.

(13) SEASONED WOOD means wood of any species that has been sufficiently dried so as to contain 20 percent or less moisture content by weight as determined by ASTM Test Method D 4442-92 or a hand-held moisture meter operated in accordance with ASTM Test Method D 4444-92, Standard Test Methods for Use and Calibration of Hand-Held

Moisture Meters or an alternative method approved by the Executive Officer, the California Air Resources Board, and the U.S. Environmental Protection Agency.

(14) SOLE SOURCE OF HEAT means the only permanent source of heat that is capable of meeting the space heating needs of a household.

(15) SOUTH COAST AIR BASIN means the non-desert portions of Los Angeles, Riverside, and San Bernardino counties and all of Orange County as defined in California Code of Regulations, Title 17, Section 60104.

(16) TREATED WOOD means wood of any species that has been chemically impregnated, painted, coated or similarly modified to improve resistance to insects or weathering.

(17) U.S. EPA PHASE II-CERTIFIED WOOD BURNING HEATER means any device certified by the U.S. EPA to meet the performance and emission standards as defined in Title 40 Code of Federal Regulations, Part 60, Subpart AAA, February 28, 1988, or subsequent revisions.

(18) WOOD BURNING DEVICE means any fireplace, wood burning heater, or pellet fueled wood heater, or any similarly enclosed, permanently installed, indoor or outdoor device burning any solid fuel for aesthetic or space-heating purposes, which has a heat input of less than one million British thermal units per hour (Btu/hr).

(19) WOOD BURNING HEATER means an enclosed, wood burning device capable of space heating that meets all the criteria defined in Title 40 Code of Federal Regulations Section 60.531, February 28, 1988, or subsequent revisions including, but not limited to, wood stoves and wood burning fireplace inserts.

(20) WOOD BURNING SEASON means the consecutive entire months of November, December, January, and February.

(d) Requirements

(1) No person shall install a permanently installed wood burning device into any new development.

(2) Notwithstanding the requirements of paragraph (d)(1), effective September 8, 2008, no person shall sell, offer for sale, supply, or install, a new or used permanently installed indoor or outdoor wood burning device or gaseous-fueled device unless it is one of the following:

(A) A U.S. EPA Phase II-Certified wood burning heater; or

(B) A pellet-fueled wood burning heater; or

(C) A masonry heater; or

(D) A wood burning device or fireplace determined to meet the U.S. EPA particulate matter emission standard established by Title 40 Code of Federal Regulations, Part 60, Subpart AAA, February 28, 1988 or subsequent revisions; or

(E) A dedicated gaseous-fueled fireplace.

(3) No person shall burn any product not intended for use as fuel in a wood burning device including, but not limited to, garbage, treated wood, particle board, plastic products, rubber products, waste petroleum products, paints, coatings or solvents, or coal.

(4) A commercial firewood facility shall only sell seasoned wood from July 1 through the end of February the following year. Any commercial firewood facility may sell seasoned as well as non-seasoned wood during the remaining months.

(e) Wood Burning Curtailment Program (effective during the months of November, December, January, and February)

Effective beginning November 1, 2011, no person shall operate an indoor or outdoor wood burning device or portable outdoor wood burning device when a mandatory wood burning curtailment day is forecast for their specific region within the South Coast Air Basin.

(f) Exemptions

(1) The provisions of this rule shall not apply to cookstoves.

(2) The provisions of paragraph (d)(1) shall not apply to new developments where there is no existing infrastructure for natural gas service within 150 feet of the property line or those 3,000 or more feet above mean sea level.

(3) The provisions of paragraph (d)(2) shall not apply to an indoor or outdoor wood burning device that is permanently installed and included in the sale or transfer of any existing development.

(4) The provisions of (d)(2) shall not apply to properties that are registered as a historical site, or are contributing structures located in a Historic Preservation Overlay Zone, as determined by the applicable, federal, State, or local agency. Contributing structures are those buildings which

are examples of the predominate styles of the area, built during the time period when the bulk of the structures were built in the Historic Preservation Overlay Zone.

(5) The provisions of (d)(3) shall not apply to manufactured firelogs.

(6) The provisions of subdivision (e) shall not apply under the following circumstances:

(A) Residential or commercial properties where a wood burning device is the sole source of heat; or

(B) A low income household; or

(C) Residential or commercial properties where there is no existing infrastructure for natural gas service within 150 feet of the property line; or

(D) Residential or commercial properties located 3,000 or more feet above mean sea level; or

(E) Ceremonial fires exempted under Rule 444 - Open Burning.

(g) Administrative Requirements

The Executive Officer will provide public notice of a mandatory wood burning curtailment through one or more of the following methods:

(1) A recorded telephone message;

(2) Messages posted on the South Coast Air Quality Management District web site;

(3) Electronic mail messages to persons or entities that have requested electronic notice;

(4) Notifying broadcast and print media operating within the boundaries of the South Coast Air Basin; and

(5) Any additional method that the Executive Officer determines is appropriate.

(h) Penalties

Any person that violates the provisions of subdivision (e) is subject to the following:

(1) For first time violators during each wood burning season, attendance at a wood smoke awareness course that has been approved by the Executive Officer or payment of a penalty of $50;

(2) For second time violators during each wood burning season, payment of a penalty of $150 or submission of proof of installation of a dedicated gaseous-fueled fireplace within 90 days after receiving the notice of violation; and

(3) For third time violators during each wood burning season, payment of a penalty of $500 or implementation of an environmentally beneficial project as derived through the mutual settlement process.

South Coast AQMD Efforts to Reduce Wood Smoke Emissions

(April 2008)

Background

People that live and work in the South Coast Air Basin breathe unhealthful amounts of fine particulate matter, also referred to as PM2.5 which stands for "particulate matter less than 2.5 microns in diameter." These particles are so small that the body's natural defense mechanisms can't keep them from entering deep into the lungs where they can harm us by reducing lung function, making bronchitis and other lung conditions worse, and triggering asthma and heart attacks. The State and federal government set health-based air quality standards, which this area does not yet meet. Although not the largest source, wood smoke from fireplaces and wood stoves contributes to the poor air quality in our area.

Answers to frequently asked questions on PM2.5 health effects and AQMD Rule 445 – Wood Burning Devices, are found below. You may also want to review the detailed information that has been prepared to answer to frequently asked questions for local government building officials, home builders, architects, installers or other interested parties.

Health Effects from Wood Smoke

Wood smoke consists of very small particles, generally less than 2.5 microns in diameter (1/30th the diameter of a human hair) that can evade the body's natural defense mechanisms. A study by the California Air Resources Board (CARB) estimated that the elevated ambient 1999-2000 PM2.5 levels from all sources here in the South Coast Air Basin result in 5,400 premature deaths, 140,000 asthma/lower respiratory symptoms and 980,000 lost work days per year. For more information on the health effects from air pollution, refer to Appendix I of the 2007 Air Quality Management Plan (AQMP).

Wood smoke contains a mixture of gases and fine particles that can cause burning eyes, runny nose, and bronchitis. Even limited exposure to smoke can be harmful to human health, particularly to the health of children, the elderly, and those with chronic health conditions. Beside fine particulates, wood smoke also contains carbon monoxide and toxic compounds, such as polycyclic aromatic hydrocarbons

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also known as PAHs. You may also want to visit the following web sites if you are interested in learning more about heath effects from wood smoke:

http://www.epa.gov/woodstoves/healtheffects.html

http://www.chhi.org/woodsmoke/health_effects.shtml

http://www.pubmedcentral.nih.gov/articlerender.fcgi?artid=1026893

Frequently Asked Questions about AQMD Rule 445 – Wood Burning Devices

1. What action is the District taking to address wood smoke emissions?

The AQMD Governing Board adopted Rule 445 – Wood Burning Devices on March 7, 2008 and has also initiated an incentive program to encourage the public to switch to gaseous-fueled hearth products.

2. What does Rule 445 require?

The main elements of the rule are:

- standards for new construction and existing structures;
- no burning of items such as trash in a wood burning device;
- requirements for commercial bulk wood sellers relative to selling unseasoned wood during certain months; and
- a mandatory curtailment element that will be implemented during high pollution days beginning in 2011 during winter months, if any.

The following is a link to the Rule 445 Fact Sheet.

3. What does the incentive program offer?

The AQMD Governing Board has approved the release of a request for proposals to hire one or more contractors to implement a program that would offer the public financial incentives to switch to gaseous-fueled hearth products. Under the

program, the gaseous-fueled hearth product (i.e., log set, fireplace insert, or stove) would have to be professionally installed in order to receive the financial incentive. Details are still being explored, however, it is anticipated that the financial incentive will be in the $100 to $150 range per residence.

For more information you can view the Program Announcement:

4. What portions of Southern California are subject to Rule 445?

Rule 445 applies to the South Coast Air Basin portion of the South Coast Air Quality Management District. As shown in the map below, this area is generally defined as all of Orange County and the non-desert portion of Los Angeles, Riverside and San Bernardino Counties. Rule 445 is not applicable in the Coachella Valley. The standards for new construction and the mandatory wood burning curtailment program that will be implemented in 2011 are not applicable to properties 3,000 and more feet in elevation.

You can also view a more detailed map.

South Coast AQMD Efforts to Reduce Wood Smoke Emissions

(April 2008)

5. What types of hearth products (fireplaces, inserts or stoves) can be built under AQMD Rule 445?

It is important to note that Rule 445 establishes separate standards for new and existing developments. New developments are subject to tighter restrictions while existing developments would have a wider range of options for remodels or room additions. The standards for new developments go into effect on March 9, 2009 and the standards for existing developments become effective September 8, 2008. The following information outlines the standards for new and existing developments.

New Developments
(Effective March 9, 2009)

- ✓ Permanently installed indoor and outdoor wood burning devices not allowed in new developments.
- ✓ Open hearth fireplaces with gas logs or other design features that preclude wood burning are allowed.

Exemptions

- ✓ A wood burning device can be installed in new developments above 3,000 feet in elevation or where there is no natural gas infrastructure within 150 feet of the property line **if** it is a cleaner device as allowed for installation in existing developments under Rule 445.

Existing Developments
(Effective September 8, 2008)

- ✓ Permanently installed indoor and outdoor wood burning devices can only be installed in existing homes and businesses if it is one of these cleaner burning options:
  - U.S. EPA Phase II-certified fireplace insert/stove or equivalent device;
  - Pellet-fueled fireplace insert or stove;
  - Masonry heater (not an open hearth fireplace); or
  - dedicated gaseous-fueled fireplace

Exemptions

- ✓ Existing properties that are officially registered as a historic site are not subject to this restriction.

Local government building officials, home builders, architects, chimney installers or other interested parties may also want to review the detailed information prepared for them on Rule 445.

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6. How is the sale of firewood in Southern California covered by the Rule?

There are no restrictions on the sale of seasoned firewood (less than 20 percent moisture content) at any time during the year. AQMD Rule 445 does, however, specify that commercial firewood facilities may not sell green firewood (greater than 20 percent moisture content) from July through February. This restriction is only for commercial firewood facilities that have a business license and does not apply to private parties that trim or remove trees and give away or sell small amounts of firewood. Properly seasoned wood is darker, has cracks in the end grain, and sounds hollow when smacked against another piece of wood.

7. Is wood burning going to be banned during the entire winter in Southern California?

No. AQMD Rule 445 includes a mandatory wood burning curtailment program that could be implemented in 2011 during periods of poor air quality in specific areas. A mandatory wood burning curtailment would be issued for specific areas where PM2.5 levels are forecast to exceed the standard (35 $\mu g/m^3$). The mandatory curtailment program would only be effective during November through the end of February and would be for specific areas. During a mandatory curtailment day, the public will be required to refrain from both indoor and outdoor solid fuel burning in specific areas. Based on historical data, 10 to 25 mandatory curtailment days could be expected in specific areas, however, due to anticipated improvements in air quality, it is expected that there would be fewer curtailment days in the future.

South Coast AQMD Efforts to Reduce Wood Smoke Emissions

(April 2008)

8. How can I find out if there is a mandatory wood burning curtailment day in my neighborhood?

As mentioned, the earliest time that a mandatory wood burning curtailment day could be implemented in any portion of the South Coast Air Basin is November of 2011. If air quality conditions have not improved sufficiently and a mandatory curtailment program is required, the public will be notified through one or more of the following methods:

- AQMD web site,
- toll free phone number;
- list serve e-mail message, or
- other media outlets.

Additional information will be developed in the future.

9. How can I learn more about air quality conditions?

You can also check current air quality conditions in your neighborhood or by calling 1 (800) CUT-SMOG.

10. How can I reduce emissions from my fireplace to help improve air quality in my neighborhood?

Smoke from neighborhood fireplaces and wood stoves, a common source of both odor and reduced visibility, contributes to the air pollution problems people complain about most. When you include the health-related problems caused by inhaling smoke pollutants, health costs for individuals and the community can be significant. Cleaner alternatives to wood burning include gaseous-fueled or electric devices. If you do decide to burn firewood, follow these simple principles to limit the amount of smoke produced.

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(April 2008)

- **Never:**
  - burn garbage, plastic, colored paper, treated wood, or wet wood
- Always:
  - ✓ use seasoned, dry wood that has been split properly. Properly seasoned wood is darker, has cracks in the end grain, and sounds hollow when hit against another piece of wood.
  - ✓ Store wood outdoors, off the ground with the top covered.
  - ✓ Burn hot, bright fires.
  - ✓ Use smaller fires in mild weather.

Additional tips to reduce wood smoke emissions can be obtained from the U.S. EPA or CARB web sites at:

http://www.epa.gov/woodstoves/efficiently.html

http://www.arb.ca.gov/cap/handbooks/wood_burning_handbook.pdf

FINAL 2007 AQMP
APPENDIX I

HEALTH EFFECTS

JUNE 2007

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GOVERNING BOARD

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HEALTH EFFECTS

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INTRODUCTION

This document presents a summary of scientific findings on the health effects of ambient air pollutants. The California Health and Safety Code Section 40471(b) requires that the South Coast Air Quality Management District prepare a report on the health impacts of particulate matter in the South Coast Air Basin, in conjunction with the preparation of the Air Quality Management Plan revisions. This document, which was prepared to satisfy that requirement, also includes the effects of the other major pollutants.

HEALTH EFFECTS OF AIR POLLUTION

Ambient air pollution is a major public health concern. Excess deaths and increases in illnesses associated with high air pollution levels have been documented in several episodes as early as 1930 in Meuse Valley, Belgium; 1948 in Donora, Pennsylvania; and 1952 in London. Although levels of pollutants that occurred during these acute episodes are now unlikely in the United States, ambient air pollution continues to be linked to increases in respiratory illness (morbidity) and increases in death rates (mortality).

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

- Increased mortality
- Increased health care utilization (hospitalization, physician and emergency room visits)
- Increased respiratory illness (symptoms, infections, and asthma exacerbation)
- Decreased lung function (breathing capacity)
- Lung inflammation
- Potential immunological changes
- Increased airway reactivity to a known chemical exposure - a method used in laboratories to evaluate the tendency of airways to have an increased possibility of developing an asthmatic response
- A decreased tolerance for exercise.

The evidence linking these effects to air pollutants is derived from population-based observational and field studies (epidemiological) as well as controlled laboratory studies involving human subjects and animals. There have been an increasing number of studies focusing on the mechanisms (that is, on learning how specific organs, cell types,

and biochemicals are involved in the human body's response to air pollution) and specific pollutants responsible for individual effects. Yet the underlying biological pathways for these effects are not always clearly understood.

Although individuals inhale pollutants as a mixture under ambient conditions, the regulatory framework and the control measures developed are mostly pollutant-specific. This is appropriate, in that different pollutants usually differ in their sources, their times and places of occurrence, the kinds of health effects they may cause, and their overall levels of health risk. Different pollutants, from the same or different sources, may sometimes act together to harm health more than they would acting separately. Nevertheless, as a practical matter, health scientists, as well as regulatory officials, usually must deal with one pollutant at a time in determining health effects and in adopting air quality standards. To meet the air quality standards, comprehensive plans are developed such as the Air Quality Management Plan (AQMP) and the Air Toxics Control Plan (ATCP). These plans examine multiple pollutants, cumulative impacts, and transport issues related to attaining healthful air quality. A brief overview of the effects observed and attributed to various air pollutants is presented in this document.

This summary is drawn substantially from reviews presented previously (SCAQMD, 1996 and 2003), and from reviews on the effects of air pollution by the American Thoracic Society (ATS, 1996), the U.S. EPA reviews for ozone (U.S. EPA, 2006), Carbon Monoxide (U.S. EPA, 2000), and Particulate Matter (U.S. EPA, 2004), from a published review of the health effects of air pollution (Brunekreef and Holgate, 2002), and from reviews prepared by the California EPA Office of the Environmental Health Hazard Assessment for Particulate Matter (Cal EPA, 2002) and for Ozone (Cal EPA, 2005) . More detailed citations and discussions on air pollution health effects can be found in these references.[1]

OZONE

Ozone is a highly reactive compound, and is a strong oxidizing agent. When ozone comes into contact with the respiratory tract, it can react with tissues and cause damage in the airways. Since it is a gas, it can penetrate into the gas exchange region of the deep lung.

The EPA primary standard for ozone is 0.08 ppm averaged over eight hours. The California Air Resources Board (CARB) has established standards of 0.09 ppm averaged over one hour and at 0.070 ppm averaged over eight hours.

The major subgroups of the population considered to be at increased risk from ozone exposure are outdoor exercising individuals including children and people with preexisting respiratory disease(s) such as asthma. The data base identifying the former

[1] Most of the studies referred to in this appendix are cited in the above sources. Only more recent specific references will be cited in this summary.

group as being at increased risk to ozone exposure is much stronger and more quantitative than that for the latter group, probably because of a larger number of studies conducted with healthy individuals. The adverse effects reported with short-term ozone exposure are greater with increased activity because activity increases the breathing rate and the volume of air reaching the lungs, resulting in an increased amount of ozone reaching the lungs. Children may be a particularly vulnerable population to air pollution effects because they spend more time outdoors, are generally more active, and have a higher ventilation rate than adults.

A number of adverse health effects associated with ambient ozone levels have been identified from laboratory and epidemiological studies (EPA, 1996; ATS, 1996). These include increased respiratory symptoms, damage to cells of the respiratory tract, decreases in lung function, increased susceptibility to respiratory infection, and increased risk of hospitalization.

The Children's Health Study, conducted by researchers at the University of Southern California, followed a cohort of children that live in 12 communities in southern California with differing levels of air pollution for several years. A publication from this study found that school absences in fourth graders for respiratory illnesses were associated with ambient ozone levels. An increase of 20 ppb ozone was associated with an 83% increase in illness related absence rates (Gilliland, 2001).

The number of hospital admissions and emergency room visits for all respiratory causes (infections, respiratory failure, chronic bronchitis, etc.) including asthma show a consistent increase as ambient ozone levels increase in a community. These excess hospital admissions and emergency room visits are observed when hourly ozone concentrations are as low as 0.08 to 0.10 ppm.

Numerous recent studies have found positive associations between increases in ozone levels and excess risk of mortality. These associations persist even when other variables including season and levels of particulate matter are accounted for. This indicates that ozone mortality effects are independent of other pollutants (Bell, 2004).

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

A recent publication from the Children's Health Study focused on children and outdoor exercise. In communities with high ozone concentrations, the relative risk of developing asthma in children playing three or more sports was found to be over three times higher than in children playing no sports (McConnell, 2002). These findings indicate that new cases of asthma in children are associated with heavy exercise in communities with high levels of ozone. While it has long been known that air pollution can exacerbate symptoms in individuals with respiratory disease, this is among the first studies that indicate ozone exposure may be causally linked to asthma.

In addition, human and animal studies involving both short-term (few hours) and long-term (months to years) exposures indicate a wide range of effects induced or associated with ambient ozone exposure. These are summarized in Table 1.

Some lung function responses (volume and airway resistance changes) observed after a single exposure to ozone exhibit attenuation or a reduction in magnitude with repeated exposures. Although it has been argued that the observed shift in response is evidence of a probable adaptation phenomenon, it appears that while functional changes may exhibit adaptation, biochemical and cellular changes which may be associated with episodic and chronic exposure effects may not exhibit similar adaptation. That is, internal damage to the respiratory system may continue with repeated ozone exposures, even if externally observable effects (chest symptoms and reduced lung function) disappear.

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

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

The susceptibility to ozone observed under ambient conditions could be due to the combination of pollutants that coexist in the atmosphere or ozone may actually sensitize these subgroups to the effects of other pollutants.

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

A recent study of birth outcomes in southern California found an increased risk for birth defects in the aortic and pulmonary arteries associated with ozone exposure in the second month of pregnancy (Ritz et al., 2002). This is the first study linking ambient air pollutants to birth defects in humans. Confirmation by further studies is needed.

TABLE 1

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

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

From: SCAQMD, 1996

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

PARTICULATE MATTER

Airborne particulates are a complex group of pollutants that vary in source, size and composition, depending on location and time. The components include nitrates, sulfates, elemental carbon, organic carbon compounds, acid aerosols, trace metals, and material from the earth's crust. Substances of biological origin, such as pollen and spores, may also be present.

Until several years ago, the health effects of particulates were focused on those sized 10 μm (micrometers) aerodynamic diameter and smaller. These can be inhaled through the upper airways and deposited in the lower airways and gas exchange tissues in the lung. These particles are referred to as PM10. EPA initially promulgated ambient air quality standards for PM10 of 150 $\mu g/m^3$ averaged over a 24-hour period, and 50 $\mu g/m^3$ for an annual average. EPA has very recently rescinded the annual PM10 standard, but kept the 24-hour standard.

In recent years additional focus has been placed on particles having an aerodynamic diameter of 2.5 μm or less (PM2.5). A greater faction of particles in this size range can penetrate and deposit deep in the lungs. The EPA recently lowered the air quality standards for PM2.5 to 35 $\mu g/m^3$ for a 24-hour average and reaffirmed 15 $\mu g/m^3$ for an annual average standard. There was considerable controversy and debate surrounding the review of particulate matter health effects and the consideration of ambient air quality standards (Kaiser, 1997; Vedal, 1997) when the EPA promulgated the initial PM2.5 standards in 1997.

Since that time, numerous studies have been published and some of the key studies were closely scrutinized and analyses repeated. The result is that there are now substantial data confirming the adverse health effects of PM2.5 exposures.

There are also differences in the composition and sources of particles in the different size ranges that may have implications for health effects. The particles larger than 2.5 μm (often referred to as the coarse fraction) are mostly produced by mechanical processes. These include automobile tire wear, industrial processes such as cutting and grinding, and resuspension of particles from the ground or road surfaces by wind and human activities.

In contrast, particles smaller than 2.5 μm are mostly derived from combustion sources, such as automobiles, trucks, and other vehicle exhaust, as well as from stationary combustion sources. The particles are either directly emitted or are formed in the atmosphere from gases that are emitted. Components from material in the earth's crust, such as dust, are also present, with the amount varying in different locations.

Attention to another range of very small particles has been increasing over the last few years. These are generally referred to as "ultrafine" particles, with diameters of 0.1 μm

or less. These particles are mainly from fresh emissions of combustion sources, but are also formed in the atmosphere from photochemical reactions. Ultrafine particles have relatively short half lives (minutes to hours) and rapidly grow through condensation and coagulation process into larger particles within the PM2.5 size range. These particles are garnering interest since laboratory studies indicate that their toxicity may be higher on a mass basis than larger particles, and there is evidence that these small particles can translocate from the lung to the blood and to other organs of the body.

The health effects of ambient particulate matter have been recently reviewed (ATS, 1996; U.S. EPA, 2004, Brunekreef, 2002). In addition, the California Air Resources Board (CARB) and the Office of Environmental Health and Hazard Assessment (OEHHA) have reviewed the adequacy of the California Air Quality Standards for Particulate Matter (Cal EPA, 2002).

The major types of effects associated with particulate matter include:

- Increased mortality
- Exacerbation of respiratory disease and of cardiovascular disease as evidenced by increases in:
  - -Respiratory symptoms
  - -Hospital admissions and emergency room visits
  - -Physician office visits
  - -School absences
  - -Work loss days
- Effects on lung function
- Changes in lung morphology

The U.S. EPA has recently lowered the short-term ambient air quality standard for fine particles (PM2.5) and has rescinded the annual standard for PM10. The current federal and California standards are listed below:

Standard	Federal	California
PM10 24-Hour average	150 $\mu g/m^3$	50 $\mu g/m^3$
PM10 Annual Average	--	20 $\mu g/m^3$
PM 2.5 24-Hour Average	35 $\mu g/m^3$	--
PM 2.5 Annual Average	15 $\mu g/m^3$	12 $\mu g/m^3$

Short-Term Exposure Effects

Epidemiological studies have provided continued and consistent evidence for most of the effects listed above. An association between increased daily or several-day-average concentrations of PM10 and excess mortality and morbidity is consistently reported from studies involving communities across the U.S. as well as in Europe, Asia, and South America. A review and analysis of epidemiological literature for acute adverse effects was undertaken by Dockery and Pope to estimate these effects as percent increase in mortality associated with each incremental increase of PM10 by 10 $\mu g/m^3$. The estimates are presented in Table 2. It appears that individuals who are elderly or have preexistent lung or heart disease are more susceptible than others to the adverse effects of PM10.

TABLE 2

Combined Effect Estimates of Daily Mean
Particulate Pollution

	% Change in Health Indicator per each 10 $\mu g/m^3$ Increase in PM10
Increase in daily mortality	
Total deaths	1.0
Respiratory deaths	3.4
Cardiovascular deaths	1.4
Increase in hospital usage (all respiratory diagnoses)	
Admissions	1.4
Emergency department visits	0.9
Exacerbation of asthma	
Asthmatic attacks	3.0
Bronchodilator use	12.2
Emergency department visits*	3.4
Hospital admissions	1.9
Increase in respiratory symptom reports	
Lower respiratory	3.0
Upper respiratory	0.7
Cough	2.5
Decrease in lung function	
Forced expiratory volume	0.15
Peak expiratory flow	0.08

* One study only

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

Many recent studies have confirmed that excess mortality and morbidity are associated with particulate matter levels. Estimates of mortality effects from these studies range from 0.3 to 1.7% increase for a 10 $\mu g/m^3$ increase in PM10 levels. The National Morbidity, Mortality, and Air Pollution Study (NMMAPS), a recent study of the largest U.S. cities, determined a combined risk estimate of about a 0.5% increase in total mortality for a 10 $\mu g/m^3$ increase in PM10 (Samet, 2000a). This study also analyzed the effects of gaseous co-pollutants. The results indicated that the association of PM10 and mortality were not confounded by the presence of the gaseous pollutants. When the gaseous pollutants were included in the analyses, the significance of the PM10 estimates remained. The PM10 effects were reduced somewhat when O_3 was also considered and

tended to be variably decreased when NO_2, CO, and SO_2 were added to the analysis. These results argue that the effects are likely due to the particulate exposures; they cannot readily be explained by coexisting weather stresses or other pollutants.

The NMMAPS study (Samet 2000b) was one that used a flawed statistical software package. The investigators have reanalyzed their data using corrected settings for the software (Dominici, 2002a, Dominici 2002b). When the estimates for the 90 cities in the study were recalculated, the estimate changed from 0.41 percent increase in mortality for a 10 $\mu g/m^3$ increase in PM10 to a 0.27 percent increase. There remained a strong positive association between acute exposure to PM10 and mortality. Thus while the quantitative estimate is reduced, the major findings of the study did not change.

Studies of PM2.5 also find associations with elevated mortality. The estimates for PM2.5 generally are in the range of 2.0 to 8.5% increase in total deaths per 25 $\mu g/m^3$ increase in 24-hour PM2.5 levels. The estimates for cardiovascular related mortality range from 3.0 to 7.0% per 25 $\mu g/m^3$ 24-hour PM2.5, and for respiratory mortality estimates range from 2.0 to 7.0% per 25 $\mu g/m^3$ 24-hour PM2.5.

Several studies have attempted to assess the relative importance of particles smaller than 2.5 μm and those between 2.5 μm and 10 μm (PM10-2.5). While some studies report that PM2.5 levels are better predictors of mortality effects, others suggest that PM10-2.5 is also important. Most of the studies found higher mortality associated with PM2.5 levels than with PM10-2.5. For example, a study of six cities in the U.S. found that particulate matter less than 2.5 μm were associated with increased mortality, but that the larger particles were not. Other studies in Mexico City and Santiago, Chile reported that PM10-2.5 was as important as PM2.5. Overall effects estimates for PM10-2.5 fall in the range of 0.5 to 6.0 % excess mortality per 25 $\mu g/m^3$ 24-hour average.

The relative importance of both PM2.5 and PM10-2.5 may vary in different regions depending on the relative concentrations and components, which can also vary by season. More research is needed to better assess the relative effects of fine (PM2.5) and coarse (PM10-2.5) fractions of particulate matter on mortality.

A number of studies have evaluated the association between particulate matter exposure and indices of morbidity such as hospital admissions, emergency room visits or physician office visits for respiratory and cardiovascular diseases. The effects estimates are generally higher than the effects for mortality. The effects are associated with measures of PM10 and PM2.5. Effects are also associated with PM10-2.5. Thus, it appears that when a relatively small number of people experience severe effects, larger

numbers experience milder effects, which may relate either to the coarse or to the fine fraction of airborne particulate matter.

In the NMMAPS study, hospital admissions for those 65 years or older were assessed in 14 cities. Hospital admissions for these individuals showed an increase of 6% for cardiovascular diseases and a 10% increase for respiratory disease admissions, per 50 $\mu g/m^3$ increase in PM10. The excess risk for cardiovascular disease ranges from 3-10% per 50 $\mu g/m^3$ PM10 and from 4-10% per 25 $\mu g/m^3$ PM2.5 or PM10-2.5.

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

Some studies have reported that short-term particulate matter exposure is associated with changes in lung function (lung capacity and breathing volume); upper respiratory symptoms (hoarseness and sore throat); and lower respiratory symptoms (increased sputum, chest pain and wheeze). The severity of these effects is widely varied and is dependent on the population studied, such as adults or children with and without asthma. Sensitive individuals, such as those with asthma or pre-existing respiratory disease, may have increased or aggravated symptoms associated with short-term particulate matter exposures. Several studies have followed the number of medical visits associated with pollutant exposures. A range of increases from 3% to 42% for medical visits for respiratory illnesses was found corresponding to a 50 $\mu g/m^3$ change in PM10. A limited number of studies also looked at levels of PM2.5 or PM10-2.5. The findings suggest that both the fine and coarse fractions may have associations with some respiratory symptoms.

The biological mechanisms by which particulate matter can produce health effects are being investigated in laboratory studies. Inflammatory responses in the respiratory system in humans and animals exposed to concentrated ambient particles have been measured. These include effects such as increases in neutrophils in the lungs. Other changes reported include increased release of cytokines and interleukins, chemicals released as part of the inflammatory process. The effects of particulate matter may be mediated in part through the production of reactive oxygen species during the inflammatory process. Recent reviews discuss mechanistic studies in more detail (Brunekreef, 2002; Brook, 2004).

Long-Term Exposure Effects

While most studies have evaluated the acute effects, some studies specifically focused on evaluating the effects of chronic exposure to PM10 and PM2.5. Studies have analyzed the mortality of adults living in different U.S. cities. After adjusting for important risk factors, these studies found a consistent positive association of deaths and exposure to particulate matter. A similar association was observable in both total number of deaths and deaths due to cardiorespiratory causes. A shortening of lifespan was also reported in these studies.

Significant associations for PM2.5 for both total mortality and cardiorespiratory mortality were reported in a study using data from the American Cancer Society. A re-analysis of the data from this study confirmed the finding (Krewski, 2000). The Harvard Six Cities Study evaluated several size ranges of particulate matter and reported significant associations with PM15, PM2.5, sulfates, and non-sulfate particles, but not with coarse particles (PM15 – PM2.5). An extension of the Harvard Six Cities Cohort confirmed the association of mortality with PM2.5 levels (Laden, 2006). These studies provide evidence that the fine particles, as measured by PM2.5, may be more strongly associated with mortality effects from long-term particulate matter exposures than are coarse compounds.

A follow-up study of the American Cancer Society cohort confirmed and extended the findings in the initial study. The researchers estimated that, on average, a 10ug/m3 increase in fine particulates was associated with approximately a 4% increase in total mortality, a 6% increase in cardiopulmonary mortality, and an 8% increase risk of lung cancer mortality (Pope, 2002). The magnitude of effects is larger in the long-term studies than in the short-term investigations. An analysis of the American Cancer Society Cohort from the Los Angeles area used a more detailed estimate of long-term PM2.5 exposures and found that the risk of mortality was up to three times higher than estimated with the national cohort (Jerrett, 2005). These findings indicate that long-term exposures may be more important in terms of overall health effects.

Recent studies report evidence indicating that particulate matter exposure early in pregnancy may be associated with lowered birth weights (Bobak, 1999). Other studies from the U.S., the Czech Republic and Mexico City have reported that neonatal and early postnatal exposure to particulate matter may lead to increased infant mortality. A more recent study in Southern California found increased risks for infant deaths associated with exposures to particulates and other pollutants (Ritz, 2006). These results suggest that infants may be a subgroup affected by particulate matter exposures.

In addition, some long-term effect studies have reported an increased risk of mortality from lung cancer associated with particulate matter exposures. A study involving California Seventh Day Adventists (very few of whom smoke) has reported an association of lung cancer mortality with PM10 levels. It is not clear from these studies

whether the association relates to causation of disease, or whether individuals with cancer are more susceptible to other effects of particles leading to the observed mortality association. A recent study that followed a large number of individuals living in the largest U.S. cities found elevated lung cancer risk associated with long term average PM2.5 levels (Pope, 2002).

Several studies have assessed the effects of long-term particulate matter exposure on respiratory symptoms and lung function changes. Associations have been found with symptoms of chronic bronchitis and decreased lung function. A study of school children in 12 communities in Southern California showed significant association of particulate matter with bronchitis or phlegm in children with asthma. These effects were also associated with NO_2 and acid vapor levels.

A cohort of fourth graders from the Southern California communities was followed over a period of four years by the Children's Health Study. A lower rate of growth in lung function was found in children living in areas with higher levels of particulate pollution (Gauderman, 2000). Decreases in lung function growth were associated with PM10, PM2.5, PM10-2.5, acid vapor, and NO_2. There was no association with ozone levels. The investigators were not able to identify independent effects of the pollutants, but noted that motor vehicle emissions are a major source of the pollutants.

A follow-up study on a second cohort of children confirmed the findings that decreased lung function growth was associated with particulates, nitric oxides, and elemental carbon levels (Gauderman, 2002). Elemental carbon is often used as a measure for diesel particulate. Additionally, children who moved to areas with less air pollution were found to regain some of the lung function growth rate (Avol, 2001). By the time the fourth graders graduated from high school, a significant number showed lower lung function. The risk of lower lung function was about five times higher in children with the highest PM2.5 exposure when compared to the lowest exposure communities (Gauderman, 2004). These deficits are likely to persist since the children were at the end of their growth period.

Despite data gaps, the extensive body of epidemiological studies has both qualitative and quantitative consistency suggestive of causality. A considerable body of evidence from these studies suggests that ambient particulate matter, alone or in combination with other coexisting pollutants, is associated with significant increases in mortality and morbidity in a community.

In summary, the scientific literature indicates that an increased risk of mortality and morbidity is associated with particulate matter at ambient levels. The evidence for particulate matter effects is mostly derived from population studies with supportive evidence from clinical and animal studies. Although most of the effects are attributable to particulate matter, co-pollutant effects cannot be ruled out on the basis of existing studies. The difficulty of separating the effects may be due to the fact that particulate

levels co-vary with other combustion source pollutants. That is, the particle measurements serve as an index of overall exposure to combustion-related pollution, and some component(s) of combustion pollution other than particles might be at least partly responsible for the observed health effects.

ULTRAFINE PARTICLES

As noted above, numerous studies have found association of particulate matter levels with adverse effects, including mortality, hospital admissions, and respiratory disease symptoms. The vast majority of these studies used particle mass of PM10 or PM2.5 as the measure of exposure. Some researchers have postulated, however, that ultrafine particles may be responsible for some of the observed associations of particulate matter and health outcomes (Oberdorster, et al, 1995; Seaton, et al, 1995). Ultrafine particles are generally classified of 0.1 μm and small diameter.

Several potential mechanisms have been brought forward to suggest that the ultrafine portion may be important in determining the toxicity of ambient particulates, some of which are discussed below.

For a given mass concentration, ultrafine particles have much higher numbers and surface area compared to larger particles. Particles can act as carriers for other adsorbed agents, such as trace metals and organic compounds; and the larger surface area may transport more of such toxic agents than larger particles.

Smaller particles can also be inhaled deep into the lungs. As much as 50% of 0.02 μm diameter particles are estimated to be deposited in the alveolar region of the lung.

CARBON MONOXIDE

The high affinity of carbon monoxide (CO) to bond with oxygen-carrying proteins (hemoglobin and myoglobin) results in reduced oxygen supply in the bloodstream of exposed individuals. The reduced oxygen supply is responsible for the toxic effects of CO which are typically manifested in the oxygen-sensitive organ systems. The effects have been studied in controlled laboratory environments involving exposure of humans and animals to CO, as well as in population-based studies of ambient CO exposure effects. People with deficient blood supply to the heart (ischemic heart disease) are known to be susceptible to the effects of CO. Protection of this group is the basis of the existing National Ambient Air Quality Standards for CO at 35 ppm for one hour and 9 ppm averaged over eight hours. The health effects of ambient CO have been recently reviewed (U.S. EPA, 2000).

Inhaled CO has no known direct toxic effect on lungs but rather exerts its effects by interfering with oxygen transport through the formation of carboxyhemoglobin (COHb, a chemical complex of CO and hemoglobin). Exposure to CO is often evaluated in

terms of COHb levels in blood measured as percentage of total hemoglobin bound to CO. COHb levels in non-smokers range between 0.3 and 0.7% and 5 to 10% in smokers. COHb levels in excess of 1.5% in a significant proportion of urban nonsmoking populations can be considered as evidence of widespread exposure to environmental CO.

Under controlled laboratory conditions, healthy subjects exposed to CO sufficient to result in 5% COHb levels exhibited reduced duration of maximal exercise performance and consumption of oxygen. Studies involving subjects with coronary artery disease who engaged in exercise during CO exposures have shown that COHb levels as low as 2.4% can lead to earlier onset of electrocardiograph changes indicative of deficiency of oxygen supply to the heart. Other effects include an earlier onset of chest pain, an increase in the duration of chest pain, and a decrease in oxygen consumption.

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

Recent epidemiological studies conducted in Southern California have indicated an association with CO exposure during pregnancy to increases in pre-term births. (Ritz, 2000). However, the results were not consistent in different areas studied. The increase in the pre-term births was also associated with PM10 levels. Another study found increased risks for cardiac related birth defects with carbon monoxide exposure in the second month of pregnancy (Ritz, 2002). Further study is needed to confirm these observations.

NITROGEN DIOXIDE

The California EPA is currently reviewing the health effects of nitrogen dioxide (Cal EPA, 2006). Evidence for low-level nitrogen dioxide (NO_2) exposure effects is derived from laboratory studies of asthmatics and from epidemiological studies. Additional supportive evidence is derived from animal studies.

Epidemiological studies using the presence of an unvented gas stove as a surrogate for indoor NO_2 exposures suggest an increased incidence of respiratory infections or symptoms in children.

Recent studies related to outdoor exposure have found health effects associated with ambient NO_2 levels, including respiratory symptoms, respiratory illness, decreased lung function, increased emergency room visits for asthma, and cardiopulmonary mortality. However, since NO_2 exposure generally occurs in the presence of other pollutants, such as particulate matter, these studies are often unable to determine the specific role of NO_2 in causing effects.

The Children's Health Study in Southern California found associations of air pollution, including NO_2, PM10, and PM2.5, with respiratory symptoms in asthmatics (McConnell, 1999). Particles and NO_2 were correlated, and effects of individual

pollutants could not be discerned. A subsequent analysis indicated a stronger role for NO2 (McConnell, 2002).

Ambient levels of NO_2 were also associated with a decrease in lung function growth in a group of children followed for eight years. In addition to NO_2, the decreased growth was also associated with particulate matter and airborne acids. The study authors postulated that these may be a measure of a package of pollutants from traffic sources. (Gauderman, 2004).

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

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

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

SULFUR DIOXIDE

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

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

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

Animal studies have shown that despite SO_2 being a respiratory irritant, it does not cause substantial acute or chronic toxicity in animals exposed at ambient concentrations. However, relatively high exposures (10 ppm of SO_2 for 72 hours) in mice can lead to tissue damage, fluid accumulation and sloughing of respiratory lining. Sensitization to allergies is observable in guinea pigs repeatedly exposed to high levels (72 ppm) of SO_2. This effect needs further evaluation in clinical and population studies to identify any chronic exposure impact on both asthmatic incidence and attacks in a population.

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

SULFATES

Based on a level determined necessary to protect the most sensitive individuals, the California Air Resources Board in 1976 adopted a standard of 25 $\mu g/m^3$ (24-hour average) for sulfates.

In recent years, a vast majority of effects (mortality and morbidity) associated with fine particles (PM2.5) and sulfur dioxide have shown a similar association with ambient sulfate levels in some population studies. The efforts to fully separate the effects of sulfates from other coexisting pollutants have not been successful. This may be due to the fact that these pollutants covary under ambient conditions, having been emitted from common sources; and the effects observed may be due to the combination of pollutants, rather than a single pollutant.

A clinical study involving exposure of human subjects to sulfuric acid aerosol indicated that adolescent asthmatics may be a susceptible population subgroup with some changes in lung function observed with exposures below 100 $\mu g/m^3$. In general, however, laboratory exposures of human volunteers to sulfates at or near ambient levels have not found significant changes in lung function.

Results from animal studies involving exposures to sulfuric acid aerosol, ammonium bisulfate and ammonium sulfate indicate that acidic particles (former two) are more toxic than non-acidic particles (latter). In addition, the severity or magnitude of both mortality and morbidity effects is relatively higher in population studies of the eastern United States and Canada where sulfate concentrations are higher than for those

observed in the western United States. Mixed results have been reported from studies which attempted to ascertain the role of acidity in determining the observed toxicity.

TOXIC AIR CONTAMINANTS

Toxic air contaminants are pollutants for which there generally are no ambient air quality standards. Under California's Air Toxics Program, CARB staff and OEHHA assess the health effects of substances that may pose a risk of adverse health effects. These effects are usually an increased risk for cancer or adverse birth outcome. After review by the state Scientific Review Panel, the CARB holds a public hearing on whether to formally list substances that may pose a significant risk to public health as a Toxic Air Contaminant.

CARB and OEHHA also establish potency factors for air toxics that are carcinogenic. The potency factors can be used to estimate the additional cancer risk from ambient levels of toxics. This estimate represents the chance of contracting cancer in an individual over a lifetime exposure to a given level of an air toxic and is usually expressed in terms of additional cancer cases per million people exposed.

The SCAQMD conducted a study on the ambient concentrations and estimated the potential health risks from air toxics (SQAQMD, 2000). A one year monitoring program was undertaken at 12 sites throughout the SCAB. Over 30 substances were measured, and annual average levels were calculated. The results showed that the overall risk for excess cancer from a lifetime exposure to ambient levels of air toxics was about 1,400 in a million. The largest contributor to this risk was diesel exhaust, accounting for 71% of the air toxics risk. A breakdown of the major contributors to the air toxics risk is shown in the following graph.

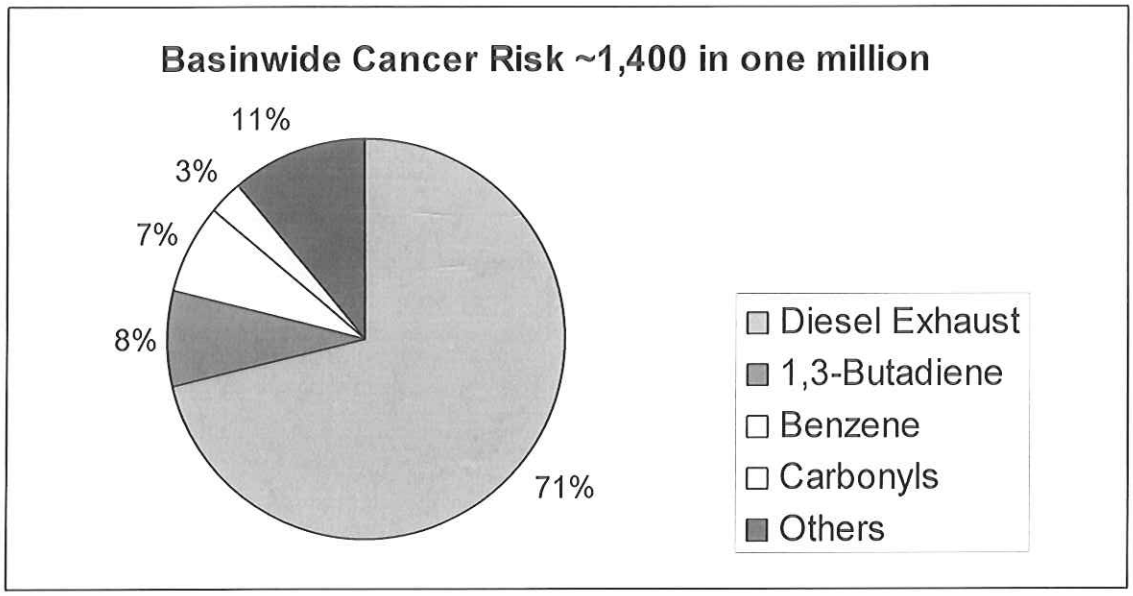

FIGURE 1

Major pollutants contributing to Air Toxics Cancer Risk in the South Coast Air Basin

For non-cancer health effects, OEHHA has developed acute and chronic Reference Exposure Levels (RELs). RELs are concentrations in the air below which adverse health effects are not likely to occur. Acute RELs refer to short-term exposures, generally of one-hour duration. Chronic RELs refer to long-term exposures of several years. The ratio of ambient concentration to the appropriate REL can be used to calculate a Hazard Index. A Hazard Index of less than one would not be expected to result in adverse effects.

The key air toxics contributing to risk from mobile and stationary sources are listed below.

TABLE 3

Key Air Toxic Air Contaminants in the SCAB

Mobile Sources	Stationary Sources
Acetaldehyde	Hexavalent Chromium
Benzene	Methylene Chloride
1,3 Butadiene	Nickel
Diesel Exhaust	Perchloroethylene
Formaldehyde	Trichloroethylene

CONCLUSION

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

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ATTACHMENT 1

COMMENTS RECEIVED ON DRAFT APPENDIX I FROM SCAQMD ADVISORY COUNCIL

The letter requesting comments and a copy of comments received follow.

Staff responses to comments are in Attachment 2.

COUNTY SANITATION DISTRICTS
OF LOS ANGELES COUNTY

1955 Workman Mill Road, Whittier, CA 90601-1400
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STEPHEN R. MAGUIN
Chief Engineer and General Manager

March 27, 2007

South Coast Air Quality Management District
21865 E. Copley Drive
Diamond bar, CA 91765-4182

Attention: Elaine Chang, DrPH

Comments on Appendix I
Draft 2007 Air Quality Management Plan

Dear Elaine:

Thank you for the opportunity to represent LACSD and the Home Rule Advisory Group (HRAG) in submitting comments on Appendix I of the 2007 Draft Air Quality Management Plan. Speaking on behalf of the HRAG, while the AQMP has varying degrees of significant impacts on all the participants around the table, we all recognize the very considerable effort that is involved in developing an AQMP and applaud your efforts. We have the following comments on Appendix I and the health aspects of the draft 2007 AQMP:

1. While we believe Dr. Ospital has done a very good job assembling the documentation in support of the health effects associated with criteria air pollution, there was little or no explanation within the appendix or the plan as to how the plan was going to better things. There is, off course, a tacit understanding that lower levels of pollution should reduce health effects. Perhaps a few paragraphs could be added to the document to this effect. Perhaps enhanced health effects might be used as an additional consideration in the ranking of control measures so that the most beneficial measures health-wise are implemented first.

2. We continue to be confused about the focus on health effects of toxic air contaminants. On Page I-20 of the Appendix, the basin-wide cancer risk is reported to be 1400 in a million, largely the impact of Diesel particulate matter and other mobile source emissions. Page 3.1-54 of the Draft AQMP Program EIR also says that exposure to environmental pollution only accounts for two percent of cancer cases. But then we also looked at Dr. Thomas Mack's 2004 work Cancers in the Urban Development[1], a detailed analysis of which was presented to the Mobile Source Committee by Dr. Ospital in 2004. In the last paragraph on Page 7 of the 645 page tome, in a section entitled *Environmental and Other Causes of Cancer* the author states, "...no local increase in

[1] Cancers in the Urban Environment *Patterns of Malignant Disease in Los Angeles County and its Neighborhoods*; Thomas Mack, Dept. of Preventive Medicine, Keck School of Medicine, Norris Comprehensive Cancer Center, University of Southern California; Elsevier Academic Press, 2004.

cancer due to pollution has yet been clearly identified in the United States. Even such highly publicized sites of pollution as the Love Canal, Three Mile Island and those popularized in the movies *Erin Brockovich* and *A Civil Action* did not produce clear evidence of a cancer excess, although each of these examples of irresponsible industrial contamination represented a clear potential danger to local residents and may have produced other medical problems."

In the very last sentence of that same book on Page 645, Dr. Mack also states, "As of this writing, no evidence of a malignancy caused by a strictly environmental carcinogen has yet been confirmed." We believe some clarification should be considered in the AQMP that acute and chronic effects of toxic air pollution should take priority, as far as regulations are concerned, over carcinogenic health effects.

3. It is also unclear how readings at air quality monitoring stations correlate with AQMP strategy. We are aware of SCAQMD efforts at the Rubidoux station, for instance, to improve the local road surfaces to reduce PM emissions, that hopefully will result in reduced monitor readings. We believe that a check should be done of all the District monitoring stations to confirm that they are not impacted by unusual site conditions and that they are reading truly representative air.

4. We believe that some analysis of indoor air quality and PM2.5 is appropriate at this time. A significant portion of human exposures to PM2.5 occurs indoors, where people spend ~85-90% of their time.[2]

We thank you for this opportunity to comment.

Yours very truly,

S. R. Maguin

Gregory M. Adams

Gregory M. Adams
Assistant Departmental Engineer
Air Quality Engineering
Technical Services Department

GMA:ch

cc: Jane Carney
Barry Wallerstein
Jean Ospital

[2] Journal of the Air and Waste Management Association, March 2007, *Indoor/Outdoor Relationships, Trends, and Carbonaceous Content of Fine Particulate Matter in Retirement Homes of the Los Angeles Basin*, p.366.

March 25, 2007

Dr. Elaine Chang
Deputy Executive Officers of Planning,
Rule Development and Area Service

Dear Dr. Chang,

It was nice talking with you and thank you for allowing me an extension on my comments for the AQMP - Health Effects of Air Pollutions- Appendix I document.

The overall documentation is excellent, and the following recommendations are not corrections but simply suggestions.

OZONE- I-2: Since ozone is such a strong oxidizing agent, perhaps a short explanation of how oxidizing agents affect biological tissues could be added.

PARTICULATE MATTER-I-11: The percentage change in health indicator for PM-10 is well documented in Table 2, page I-7-12. However, there is no percentage change for PM-2.5 health indicators. Does that imply that biological mechanisms, mortality and morbidity data for both particulate matter are the same, despite their variations in size and sources? If they are the same, that should be stated. If not, a second table for percentage change for PM 2.5 health indicators should be added.

ULTRAFINE PARTICLES-I-15: There are some reports indicating that the ultrafine particles might be embedded in cellular mitochondria. If that is correct, it would mean ultrafine particles are ubiquitous in every cell, therefore affecting cellular ATP production. That could explain why particulate matter might exacerbate diabetic conditions.

SULFUR DIOXIDE and SULFATES I-18-19: Perhaps a short definition of the differences between sulfur dioxide and sulfates could be included.

Respectfully Submitted,

Sam Huang, Ph.D
Member -SCAQMD Advisory Council
7458 Whitegate Ave.
Riverside, CA 92506

South Coast
Air Quality Management District
21865 Copley Drive, Diamond Bar, CA 91765-4178
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Office of the Executive Officer
Barry R. Wallerstein, D.Env.
909 396 2100, fax 909 396 3340

February 28, 2007

Mr. Greg Adams
Los Angeles County Sanitation District
1955 Workman Mill Road
Whittier, CA 90607

Re: SCAQMD Advisory Council

Dear Mr. Adams:

I would like to congratulate and welcome you as a member of the South Coast Air Quality Management District Advisory Council. I have attached a copy of the council's policies as well as a copy of the current roster.

Attached also for your review is Appendix I of Draft 2007 Air Quality Management Plan (AQMP)–Health Effects of Air Pollutants. This document presents a summary of scientific findings on the health effects of ambient air pollutants. The California Health and Safety Code Section 40471(b) requires that the South Coast Air Quality Management District Board prepare a report on the health impacts of particulate matter in the South Coast Air Basin in conjunction with the preparation of the AQMP. This document was prepared to fulfill that requirement. The Health and Safety Code also directs that the report be submitted to the Advisory Council for review and comment.

Please review the attached document and provide your comments by March 21, 2007.

Thank you again for agreeing to serve as a member of the Advisory Council. Should you have any questions on this matter, please do not hesitate to contact Elaine Chang, Deputy Executive Officer of Planning, Rule Development & Area Sources, at (909) 396-3186.

Sincerely,

Barry R. Wallerstein, D.Env
Executive Officer

BRW:lrw
Enclosures
cc: Home Rule Advisory Group
(w/o enclosures)

Emily D.P. Nelson, D.Env.
Health and Environmental Risk Consultant
P.O. Box 3703
Palm Desert, CA 92261-3703
760-333-1776

March 26, 2007

Dr. Barry Wallerstein
Executive Officer
South Coast Air Quality Management District
21865 Copley Drive
Diamond Bar, CA 91765-4178

Re: Appendix I Review
Draft 2007 Air Quality Management Plan

Dear Barry,

Thank you for the opportunity to serve as a member of the South Coast Air Quality Management District Advisory Council. I look forward to making a positive contribution.

I have reviewed Appendix I of the Draft 2007 AQMP Health Effects of Air Pollutants. Since the document's summary is substantially taken from prior reviews, it was an easy review for me. The relevant studies have been discussed or referred to in a concise yet complete way for these purposes. I did note that two studies referred to in the Ultrafine Particles discussion on page I-15 (Oberdorster, et al, 1995 and Seaton, et al, 1995) are not included in the references section at the end of the appendix. I have spoken with Dr. Jean Ospital about this today.

I am sorry for the delay in my response. I was in Washington, DC last week and did not realize this review had not been completed.

Sincerely,

Emily Nelson, D.Env.

cc: Supervisor S. Roy Wilson

ATTACHMENT 2

STAFF RESPONSES TO COMMENTS RECEIVED

Comment:

Citations missing in References section.

Response:

The missing citations are below.

> Oberdorster G, et al. 1995. "Association of Particulate Air Pollution and Acute Mortality: Involvement of Ultra-Fine Particles." Inhalation Toxicol 7:111-124.
>
> Seaton A, et al. 1995. "Particulate Air Pollution and Acute Health Effects." Lancet 345:176-178.

Comment:

Include an explanation of how oxidizing agents affect biological tissue.

Response:

Oxidants such as ozone can readily react with biochemicals in tissues to alter their chemical structure and affect their biochemical functioning.

Comment:

Are percent changes in health indicators for $PM_{2.5}$ the same as for PM_{10}? Include a table for percentage change for $PM_{2.5}$ indicators as was done for $PM_{2.5}$.

Response:

The table for PM_{10} was used to provide a brief summary of information related to daily changes in PM levels with various health indicators, and was taken from a previous review paper. The discussion for $PM_{2.5}$ associated outcomes includes a brief description of some of the more recent studies of health effects, which does include percentage changes associated with changes in ambient exposures. The discussion shows that the relative changes associated with $PM_{2.5}$ levels are not the same as those with PM_{10}. The discussion also points out that some studies indicate the effects from PM_{10} exposures may be attributed to the $PM_{2.5}$ component, whereas other studies indicate that the fraction of PM_{10} larger than 2.5 μm diameter also contribute. The relative contribution of the different fractions of PM_{10} to health outcomes is an ongoing area of research.

Comment:

Ultrafine particles may affect cellular ATP production, and this could explain why particulate matter might exacerbate diabetic conditions.

Response:

Studies have indeed shown that ultrafine particles can penetrate into cell mitochondria and result in cell death. The mechanism is thought to include oxidative injury to cellular components. While there are studies that indicate individuals with diabetes may be more sensitive to the effects of air pollutants, staff opinion is that there are not sufficient data available to determine whether particulate exposures exacerbate diabetic conditions.

Comment:

Include a definition of the differences between sulfur dioxide and sulfates.

Response:

Sulfur dioxide (SO_2) occurs as a gas in the atmosphere, whereas sulfates (referring to the ion SO_4^{-2}) are generally found as a component of particulate matter.

Comment:

Add a discussion on how the AQMP would reduce health effects.

Response:

Staff assessment of the benefits of implementing the AQMP and attaining the ambient air quality standards is included in the Socioeconomic Report.

Comment:

Some clarification should be considered in the AQMP that acute and chronic effects of toxic air pollutants should take priority, as far as regulations are concerned, over carcinogenic effects.

Response:

Staff position is that all adverse effects are important.

Comment:

A check should be done of all the District monitoring stations to confirm that they are not impacted by unusual site conditions.

Response:

This is beyond the scope of Appendix I. The air quality monitoring network stations are sited in conformance with federal and state guidelines to be at locations representative of regional air quality.

Comment:

An analysis of indoor air quality and $PM_{2.5}$ should be included.

Response:

There are few studies on the health effects on indoor $PM_{2.5}$ exposures. The study cited by the commenter did not provide information on health effects.

Environmental Health

Potential Adverse Health Effects of Wood Smoke

WILLIAM E. PIERSON, MD, *and* JANE Q. KOENIG, PhD, *Seattle, and* EMIL J. BARDANA, Jr, MD, *Portland*

The use of wood stoves has increased greatly in the past decade, causing concern in many communities about the health effects of wood smoke. Wood smoke is known to contain such compounds as carbon monoxide, nitrogen oxides, sulfur oxides, aldehydes, polycyclic aromatic hydrocarbons, and fine respirable particulate matter. All of these have been shown to cause deleterious physiologic responses in laboratory studies in humans. Some compounds found in wood smoke—benzo[a]pyrene and formaldehyde—are possible human carcinogens. Fine particulate matter has been associated with decreased pulmonary function in children and with increased chronic lung disease in Nepal, where exposure to very high amounts of wood smoke occurs in residences. Wood smoke fumes, taken from both outdoor and indoor samples, have shown mutagenic activity in short-term bioassay tests. Because of the potential health effects of wood smoke, exposure to this source of air pollution should be minimal.

(Pierson WE, Koenig JQ, Bardana EJ Jr: Potential adverse health effects of wood smoke. West J Med 1989 Sep; 151:339-342)

Homeowners have turned to the use of wood as a heating fuel because of the increasing cost of oil and natural gas. This trend has been especially striking in the northeastern and northwestern United States.[1] The Washington State Department of Ecology estimated in a study in 1984 that wood was burned in 60% of Washington households, with about 2.2 million cords of wood consumed per year. Several studies have shown the potential for wood-burning stoves and fireplaces to pollute indoor as well as outdoor air. Although wood-burning stoves and fireplaces are vented to the outside, many circumstances facilitate the access of combustion products into the indoors, including improper installation, such as insufficient stack height, cracks, leaks in or poor fitting of the stovepipe, negative air pressure indoors, downdrafts, and accidents, such as wood spilling from the fireplace.[1] Also, about 70% of the outdoor wood smoke reenters the house (T. V. Larson, PhD, University of Washington, Department of Civil Engineering, unpublished data). Combustion products of wood are highly irritating to the eyes, nose, and respiratory system. Duncan and co-workers have developed data on the types of pollutants associated with wood burning (Table 1).[2]

A wood-burning stove functions differently than a fireplace. In a fireplace, as much as 90% of a fire's heat is lost up the chimney along with exhaust gases. With a stove, the air supply is controlled and the rate of combustion is also controlled so that as much as 60% of the heat produced can be delivered indoors.[3]

Sexton and associates compared particle mass, size distribution, and chemical composition of indoor and outdoor air in a residential neighborhood.[4] They found that indoor concentrations of particles were often higher than outdoor, and although wood smoke contributed greatly to the mass, other sources also were important. They also found great variation among residences in the same neighborhood.

Traynor and colleagues measured indoor air pollution and found that both airtight and nonairtight stoves produced measurable particulate matter and polycyclic aromatic hydrocarbons within the home.[5] Nonairtight stoves emitted as much as 650 μg per m^3 for a 24-hour period. Polycyclic aromatic hydrocarbons, including benzo[*a*]pyrene, are also expelled into the indoor environment. Benzo[*a*]pyrene is a known carcinogen, and any exposure above local background levels should be avoided when possible. Other pollutants can cause both acute and toxic adverse health effects. Thus, it seems appropriate to highlight our understanding of the toxic exposures involved.

Effects of Individual Pollutants

Carbon Monoxide

Carbon monoxide is one of the most ubiquitous indoor pollutants. It is a major product of tobacco combustion, and concentrations from 2 to 110 ppm have been measured in dwellings, depending on the size of the space, the number and type of tobacco products smoked, and the adequacy of ventilation.[6] An indoor level of no higher than 5 ppm has been recommended by the American Society for Heating, Refrigeration, and Air Conditioning Engineers (ASHRAE). Incomplete combustion in fuel-rich flames due to wood burning also can produce substantial amounts of carbon monoxide, an odorless, colorless gas that has the potential to be an invisible and silent hazard. Carbon monoxide competes with oxygen on the hemoglobin molecule, forming carboxyhemoglobin. The current outdoor standard is 9 ppm for an eight-hour period, or 35 ppm for any given hour. Research on persons with coronary artery disease has shown that the amount of exercise that could be done before an attack of angina was notably shortened after carbon monoxide exposure.[7]

From the Department of Environmental Health, University of Washington School of Public Health and Community Medicine, Seattle (Drs Pierson and Koenig), and the Department of Medicine, Oregon Health Sciences University School of Medicine, Portland (Dr Bardana).

Reprint requests to Jane Q. Koenig, PhD, Research Associate Professor, Department of Environmental Health, SC-34, University of Washington, Seattle, WA 98195.

Nitrogen Oxides

Nitrogen is capable of forming several types of gaseous oxides. Nitric oxide and nitrogen dioxide are found in very hot wood combustion flames, and both are toxic.[8(p103)] Nitrous oxide is not formed in normal combustion. Nitric oxide and nitrogen dioxide result from the oxygen-rich combustion of wood, coal, natural gas, or oil in a variety of stoves. These gases are very reactive and can interconvert. Nitric oxide binds to hemoglobin to produce methemoglobin. Many of the adverse effects attributed to carbon monoxide in the past may be related to the combined effect of carboxyhemoglobin and methemoglobin.[1] An indoor level of no higher than 2.5 ppm has been recommended by ASHRAE. The National Ambient Air Quality Standard for nitrogen dioxide set by the Environmental Protection Agency is 0.05 ppm averaged over one year. Nitrogen oxides may produce hematologic aberrations, affect the activity of several enzyme systems, and may also cause vascular membrane injury and leakage leading to edema. Exposures to nitrogen dioxide have been associated with toxicologic effects including pulmonary edema, bronchoconstriction, and increased infection rates. An indoor level of no higher than 0.5 ppm has been recommended by ASHRAE. Some epidemiologic evidence indicates that an increased prevalence of respiratory tract infections in young children and adult men as well as lower pulmonary function performance are associated with a history of exposure to gas stove emissions.[9] Consistent lung effects in children due to nitrogen dioxide exposure have been difficult to characterize, however.[9]

Sulfur Oxides

Sulfur dioxide is a common air pollutant from wood-burning stoves that has known airway irritating effects. Kerosene heaters have been shown to emit sulfur dioxide levels that can exceed certain occupational health standards. An indoor level of not greater than 0.5 ppm has been recommended by ASHRAE. Koenig and co-workers[10-12] have shown that adolescent subjects with asthma or exercise-induced bronchospasm or both experience large changes in pulmonary function after exposure to either 1.0 or 0.5 ppm sulfur dioxide during moderate exercise. Symptoms related to lower airway dysfunction such as dyspnea and chest tightness are generally confined to those with asthma, but healthy subjects usually complain of an unusual taste or odor.

Aldehydes

Formaldehyde is ubiquitous in our environment, and the primary indoor source of this and other aldehydes is the combustion of tobacco products. Polyurea foam insulation, particle board, and other construction products also can release formaldehyde into the indoor environment. The indoor levels reported with wood-burning stoves range from 0.3 to 1.0 ppm. The Department of Housing and Urban Development has recommended indoor levels not higher than 0.4 ppm, but ASHRAE has recommended 0.1 ppm. Formaldehyde is associated with an annoying odor and at higher concentrations—generally more than 0.8 ppm—can produce a transient irritation of the eyes and mucous membranes of the upper respiratory tract.[13] It is so soluble and rapidly metabolized that it rarely reaches the lower respiratory tract to inflict damage, except when inhaled in cigarette smoke. Formaldehyde may, on rare occasions, induce bronchial asthma at relatively high exposure doses. It appears to be carcinogenic at exceptionally high cumulative doses in rodents, but there are no conclusive studies proving its carcinogenic effects in humans.

TABLE 1.—*Emissions From Residential Wood-Fired Stoves**

Pollutant	*Emission Range, lb/cord†*
Particulates	3-93
SO_x	0.5-1.5
NO_x	0.7-2.6
Hydrocarbons	1-146
Carbon monoxide	300-1,220
Polycyclic organic materials	0.6-1.22
Formaldehyde	0.3-1.0
Acetaldehyde	0.1-0.3
Phenols	0.3-8
Acetic acid	5-48

*Adapted from Duncan et al.[2]
†The factor used to convert 1 lb per ton was 1.65 lb per cord.

Polycyclic Aromatic Hydrocarbons

Incompletely burned hydrocarbons from wood stoves are frequently found in indoor air. Usually they are all the gaseous or vaporizable hydrocarbons, such as hydrocarbons with 1 to 16 carbon atoms. Toxic hydrocarbons are produced if plastic materials are incinerated in wood stoves. Resulting polycyclic aromatic hydrocarbons have been shown to be carcinogenic in animal studies. Coke oven workers have exposure to polycyclic aromatic hydrocarbons in levels similar to those measured in wood smoke fumes; these hydrocarbons can serve as a surrogate for wood smoke exposure. Studies have shown that coke oven workers with 15 years' or more exposure have a 16-fold excess risk of having lung cancer as compared with the general population.[14] One of the polycyclic aromatic hydrocarbon compounds, benzo[*a*]pyrene, is a proven carcinogen in animals.

Effects of Pollutant Mixtures

Most experimental studies, because of experimental design constraints, investigate the health effects of exposure to only one agent at a time. In real life, however, people are exposed to many pollutants. The effect of the described pollutants in combination with one another is reason for additional concern. Only a few studies have been conducted with wood smoke itself. One has investigated the incidence of respiratory illness among 31 preschool children living in homes heated with wood-burning stoves as compared with 31 children living in homes heated by other means.[15] Moderate to severe respiratory symptoms such as wheezing and cough at night were notably greater in the wood stove group compared with the control group. Possible confounding factors were investigated. Approximately the same proportion of children in each group had exposure to parental cigarette smoking in the home. There was no significant difference between groups in terms of the presence of urea formaldehyde insulation or the use of humidifiers. On the other hand, another study with older children—kindergarten through sixth grade—indicated that having a wood stove in the home did not increase significantly the frequency of acute respiratory episodes.[16] In this study formaldehyde exposure, estimated from construction or remodeling products, showed a small association with respiratory symptoms. Controlling for wood stove use did not diminish the formaldehyde effect.

Epidemiologic studies of long-term exposure to wood smoke have found an increased prevalence of respiratory illness in both children and adults. A study by Anderson[17] suggested an association between wood smoke exposure and chronic lung disease in adults in Papua New Guinea. Another study with children in the same region found no difference dependent on exposure except for an excess of wheeze in boys.[18] In a study in the hill region of Nepal the prevalence of chronic bronchitis among nonsmoking women increased substantially with the duration of time per day spent near the fireplace.[19] Houses there are poorly ventilated and have no chimneys. The absence of chronic bronchitis in men was suggested to be due to the lesser amount of time they spent indoors with the burning wood.

A unique case was reported of a 61-year-old woman who had shortness of breath. She was evaluated for interstitial lung disease of an unknown cause. Bronchoalveolar lavage revealed the presence of numerous particles and fibers that were identified as wood. The patient lived in a home heated with a solid-fuel radiant room heater that roasted wood to produce heat.[20]

A review of the health effects of indoor air pollution in general with some mention of wood smoke was published in 1987.[9] Also in 1987, Dockery and colleagues[21] concluded that wood stove use was associated with an increased relative risk for respiratory illness in children selected from grades 2 through 5 in six cities where wood stove use ranged from 46% to 5%. The odds ratio was 1.32 (95% confidence intervals 0.99 to 1.76).

Particulate Matter

Wood stoves have been shown to emit substantial amounts of fine particulate matter of less than 10 μm in size. Fine particulate matter ranging in size from 0.02 to 10 μm is of concern to public health because it has been shown to be readily inspired and deposited into lungs. The finest particles are deposited more deeply in lungs where some can remain indefinitely and cause morphologic and biochemical changes.

In a study in Steubenville, Ohio, decreases in lung function in children correlated with elevated concentrations of total suspended particulate matter.[22] In a similar study in the Netherlands, lung function measurements were followed in 179 children aged 7 to 11 years during a winter season in which total suspended particulate matter was being monitored and 3% to 5% reductions in lung volume measurement were found during air pollution episodes when high concentrations of particulate matter were present.[23] Taken together, these studies suggest that declines in lung function associated with episodic exposures to total suspended particulates occur rapidly and persist for as long as two to three weeks before recovery. Particles of less than 10 μm were measured during the winter of 1985-1986 in Olympia, Washington. For a period of five days, the concentration was greater than 150 μg per m^3. Wood smoke concentration was high and responsible for 80% to 90% of the fine particles. For comparison, the concentration at the same sampling site during the summer of 1986 was approximately 20 μg per m^3.

Airborne wood smoke fumes, collected both inside and outside homes using wood stoves, have been analyzed for their toxic properties. Kamens and associates[24] have shown that wood smoke fumes contain mutagens according to the Ames short-term mutagenicity assay. The volatile organic compounds, polycyclic aromatic hydrocarbons, and especially semivolatile compounds all showed mutagenic activity, with some having as much as 100 times the activity of some well-known carcinogens. Alfheim and co-workers showed that the polar fraction of organic extracts from emissions of wood combustion had direct mutagenic activity in a modified Ames *Salmonella* assay.[25] Using another test of mutagenicity, sister chromatid exchange, Hytonen and colleagues showed the capacity of emission from an airtight residential wood stove to induce sister chromatid exchange.[26] Burnet and Insley reported that emissions from both traditional and advanced technology wood stoves caused sister chromatid exchange in mammalian cells.[27] Even though the newer stoves produced less particulate matter and carbon monoxide, the emissions from these stoves did give a positive response to the sister chromatid exchange test. In another study, air samples were collected from occupied homes using wood as an energy source and mutagenic activity was found in the air of 8 of the 12 homes sampled.[28]

Other Combustion Sources of Indoor Pollution

Whenever unvented combustion takes place indoors or venting systems attached to stoves, boilers, or heaters are malfunctioning, a wide variety of combustion products can be emitted directly indoors. Besides tobacco combustion, the primary sources of combustion by-products in residential buildings are usually space heaters, gas stoves, and gas water heaters, as well as wood stoves. Exhaust from automobiles in homes with attached carports or garages and oil and kerosene lamps and candles can be additional sources of combustion by-products.

Summary

Concern over the quality of outdoor air has been an active issue in the United States for many years. A substantial portion of the Environmental Protection Agency's budget of $300 million is devoted to concern about the contamination of our outdoor environment, such as the selenium contamination of water in California, acid rain in the Great Lakes, or a toxic waste dump in New York.[29] Though these are important issues, most people spend 80% to 90% of their time indoors, each taking well over 10,000 breaths per day to provide the necessary oxygen for human metabolism. More time is spent indoors in the harsh winter months.

The increasing use of wood as a heating fuel has precipitated concern for its potential to further pollute the outdoors but, perhaps more important, to contaminate the home. Many irritating and potentially carcinogenic compounds have been identified in wood smoke, including carbon monoxide, nitrogen and sulfur oxides, fine particulate matter, and various aromatic hydrocarbons, including benzo[*a*]pyrene. In addition to wood smoke, various other combustion sources have been identified that could directly augment the problem. Several studies have identified dozens of toxic chemicals that all appear to originate in common household activities and practices in homes.

With respect to burning wood, it is advised as well that only dry, cured wood—less than 20% water content—be burned because combustion will be more complete and fewer products of incomplete combustion will be released into the air. All stoves should be operated in a manner to produce efficient burning with minimum smoke emissions. It is important to emphasize that even the newest airtight wood

stoves emit a considerable amount of fine particles to the outdoor air.[26] Plastic and other synthetic products should never be incinerated in wood stoves. We also strongly recommend curtailing all indoor wood burning during air pollution episodes or stagnations. Finally, we propose that public health authorities increase their research and monitoring efforts directed at this important issue.

REFERENCES

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